WORLD METEOROLOGICAL ORGANIZATION

TECHNICAL NOTE No. 191

ANIMAL HEALTH AND PRODUCTION AT EXTREMES OF WEATHER

(Reports of the CAgM Working Groups on Weather
and Animal Disease and Weather and Animal Health)

WMO - No. 685

Secretariat of the World Meteorological Organization - Geneva - Switzerland

The World Meteorological Organization

The World Meteorological Organization (WMO), of which 160 States and Territories are Members, is a specialized agency of the United Nations. It was created:

(a) *To facilitate world-wide co-operation in the establishment of networks of stations for the making of meteorological observations as well as hydrological and other geophysical observations related to meteorology, and to promote the establishment and maintenance of centres charged with the provision of meteorological and related services;*

(b) *To promote the establishment and maintenance of systems for the rapid exchange of meteorological and related information;*

(c) *To promote standardization of meteorological and related observations and to ensure the uniform publication of observations and statistics;*

(d) *To further the application of meteorology to aviation, shipping, water problems, agriculture and other human activities;*

(e) *To promote activities in operational hydrology and to further close co-operation between Meteorological and Hydrological Services; and*

(f) *To encourage research and training in meteorology and, as appropriate, in related fields and to assist in co-ordinating the international aspects of such research and training.*

(Convention of the World Meteorological Organization, Article 2)

The Organization consists of the following:

● The **World Meteorological Congress**, the supreme body of the Organization. It brings together the delegates of all Members once every four years to determine general policies for the fulfilment of the purposes of the Organization, to approve the WMO Long-term Plan, to authorize maximum expenditures for the following four-year financial period, to adopt Technical Regulations relating to international meteorological and operational hydrological practice, to elect the President and Vice-Presidents of the Organization and members of the Executive Council other than the presidents of the regional associations and to appoint the Secretary-General;

● The **Executive Council,** composed of 36 directors of national Meteorological or Hydrometeorological Services. It meets at least once a year to conduct the activities of the Organization, to implement the decisions taken by its Members in Congress and to study and make recommendations on any matter affecting international meteorology and related activities of the Organization;

● The six **regional associations** (Africa, Asia, South America, North and Central America, South-West Pacific and Europe), composed of Member governments. They co-ordinate meteorological and related activities within their respective Regions and examine from the regional point of view all questions referred to them;

● The eight **technical commissions,** consisting of experts designated by Members. They are responsible for studying any subject within the purposes of the Organization. Technical commissions have been established for basic systems, instruments and methods of observation, atmospheric sciences, aeronautical meteorology, agricultural meteorology, marine meteorology, hydrology, and climatology;

● The **Secretariat,** located at 41 Avenue Giuseppe-Motta, Geneva, Switzerland. It is composed of a Secretary-General and such technical and clerical staff as are required for the work of the Organization. It serves as the administrative, documentation and information centre of the Organization, makes technical studies as directed, supports all the bodies of the Organization, prepares, edits and arranges for the publication and distribution of the approved publications of the Organization, and carries out duties specified in the Convention and other Basic Documents and such other work as Congress, the Executive Council and the President decide. The Secretariat works in close collaboration with the United Nations and its specialized agencies.

WORLD METEOROLOGICAL ORGANIZATION

TECHNICAL NOTE No. 191

ANIMAL HEALTH AND PRODUCTION AT EXTREMES OF WEATHER

(Reports of the CAgM Working Groups on Weather
and Animal Disease and Weather and Animal Health)

WMO - No. 685

Secretariat of the World Meteorological Organization - Geneva - Switzerland
1989

NOTE

C O N T E N T S

CONTENTS

                                        *

                              *   *

FOREWORD

The success of the world campaign against hunger depends no less on man's ability to improve and increase the productivity of farm livestock than on his effectiveness in growing more and better varieties of crops. The importance of meteorological parameters in animal experiments, in animal diseases and in animal health has been well recognized.

The WMO Commission for Agricultural Meteorology has been involved since its third session in studies related to weather effects on animals. The work of the Commission has been published as WMO Technical Notes. The Commission, at its seventh session in 1979 (Sofia, Bulgaria), recognized that there was a need to concentrate on the operational aspects of forecasting the incidence and intensity of animal disease outbreaks as they related to weather. The Commission therefore established a working group to study various aspects of weather effects on animal health with a view to improving the control and prevention of weather-related animal problems. The present technical note deals exclusively with cold- and hot-weather stress on cattle, sheep, pigs and poultry.

It gives me great pleasure to express my thanks to Dr Hugh-Jones (Chairman), the members of his working group and the various other contributors for their active role in the preparation of this technical note. I am confident that this technical note will be welcomed by all those engaged in studies relating to animal production and animal health.

G.O.P. Obasi
Secretary-General

PREFACE

      The technical reports of the Working Groups on Weather and Animal Disease and Weather and Animal Health submitted to the eighth and ninth sessions of the Commission for Agricultural Meteorology were approved for publication. However, they were so voluminous that it was clear that the material would be long out-of-date by the time the Organization could publish them. Therefore, the reports of the members of the two working groups, as well as of the numerous individuals who had also generously contributed scripts, were abstracted and brought together in this document. The intention was to provide an over-view of the effects and impacts of heat and cold stressors on animals and their diseases.

      Through this document it is hoped to encourage meteorologists, animal scientists, and veterinarians to work in this area and, in particular, to work together. There can be no doubt that meteorologists have an important role to play in livestock production and disease control, and in helping to ameliorate the effects of severe climates.

      The text is retrospective and readers wishing to keep abreast of future developments are encouraged to read the report by the CAgM-VIII Rapporteur on Weather and Climate and Animal Performance (Dr J.R. Starr) and subsequent reports as they are issued by WMO.

      The objective of the working groups was to show how meteorologists can help the livestock industries by forecasting extreme climatic conditions, whether temperate or tropical, short-term fluctuations or in season, because of climatic effects on reproduction, production, or disease in livestock. This was to be achieved by demonstrating that the possibility exists to accurately forecast animal health effects through meteorological data; by explaining some of the underlying biological processes, both to improve the understanding of why certain observations are used and by indicating areas of future concern. It is an area of applied and basic sciences where there have been some resounding successes but much more is still to be learned.

Several authors have contributed to this assembly of papers dealing with Animal Health and Production at Extremes of Weather.

The papers deal with cold- and hot-weather stress on cattle, sheep, pigs and poultry. Other subjects covered are: cold and heat exposure and the immune function; animal parasites in cold and hot climates; effects of environmental requirements (including nutrition requirements) and the role of agrometeorologists in animal health and production. Some aspects of the meteorological forecasting of animal health and diseases are discussed.

Résumé

Les articles sur la santé et la production animales dans des conditions météorologiques extrêmes qui constituent le présent ouvrage sont dus à différents auteurs.

Ils traitent de l'agression subie par les bovins, les ovins, les porcins et les volailles par des conditions de temps très chaud ou très froid, de l'exposition au froid ou à la chaleur et de la fonction immunitaire, des parasites des animaux dans les climats chauds ou froids, des effets des besoins dus à l'environnement (y compris des besoins en fait de nutrition) ainsi que du rôle joué par les agrométéorologistes dans le domaine de la production et de la santé animales. Il y est question également de certains aspects de la prévision des maladies et de l'état sanitaire des animaux en fonction des conditions météorologiques.

РЕЗЮМЕ

В сборнике представлены работы нескольких авторов по вопросам здоровья и разведения домашних животных в экстремальных погодных условиях.

В некоторых статьях рассматривается воздействие холода и жары на крупный рогатый скот, овец, свиней и домашнюю птицу. В других поднимаются такие вопросы, как: функционирование иммунной системы в холод и жару; паразиты животных в холодном и жарком климате, влияние условий окружающей среды (включая условия питания) и роль агрометеорологов в обеспечении здоровья и разведения домашних животных. Рассматриваются также некоторые аспекты метеорологического прогнозирования здоровья и болезней домашних животных.

RESUMEN

Los trabajos recopilados en el presente documento, que trata de la salud y de la producción animales en condiciones meteorológicas extremas, son obra de varios autores.

En ellos se estudia la tensión causada por el frío o el calor en el ganado vacuno, ovino, porcino y en las aves de corral. También se analizan en estos trabajos los efectos de la exposición al frío y al calor y sus funciones inmunitarias, los parásitos de los animales en los climas fríos y cálidos, el impacto de las necesidades en función del medio ambiente (en particular las necesidades de nutrición) y el papel que incumbe a los agrometeorólogos en la salud animal y la producción animal. También se examinan algunos aspectos de la predicción de la salud animal y de las enfermedades de los animales gracias a la meteorología.

LIST OF CONTRIBUTORS

I.   Members of CAgM - VII Working Group on Weather and Animal Health and Weather and Animal Disease

M.E. Hugh-Jones (Chairman)	Department of Epidemiology and Community Health School of Veterinary Medicine, Louisiana State University Baton Rouge, LA 70803, USA
J.A. Clark	Department of Physiology and Environmental Science School of Agriculture Sutton Bonington Loughborough LE12 5RD United Kingdom
J.D. Coulter	Fiji Meteorological Service Private Bag Nadi International Airport Fiji
G.L. Hahn	Roman L. Hruska US Meat Animal Research Center Agricultural Research Service US Department of Agriculture PO Box 166 Clay Center, NE 68933 USA
W.O. Haufe	Research Station Research Branch, Agriculture Canada Lethbridge, ALTA Canada T1J 4B1
M. Mannathoko	Director, Veterinary Services Department of Veterinary Services and Tsetse Control Private Bag 0032 Gaborone Botswana
D.J.G. O'Brien	Parasitology Section Veterinary Research Laboratory Abbotstown Castleknock, Co Dublin Eire
P. Yvore	Station de Pathologie Aviare et de Parasitologie Institut National de la Recherche Agronomique Ministere de l'Agriculture Nouzilly - 37380 Monnaie France

II.   Other Contributors

D.K. Ames   Department of Animal Sciences
                                    Colorado State University
                                    Fort Collins, CO 80523 USA

J. Armour   Department of Veterinary Parasitology
                                    University of Glasgow Veterinary School
                                    Bearsden Road, Bearsden
                                    Glasgow G61 1QH Scotland
                                    United Kingdom

F. Blecha   Department of Anatomy and Physiology
                                    College of Veterinary Medicine
                                    Kansas State University
                                    Manhattan, KS 66502 USA

R.K. Christenson   Roman L. Hruska US Meat Animal Research
                                    Center
                                    Agricultural Research Service
                                    US Department of Agriculture
                                    PO Box 166
                                    Clay Center, NE 68933 USA

G.I. Christison   Department of Animal and Poultry Science
                                    University of Saskatchewan
                                    Saskatoon, S7N OWO Canada

J.W. Deaton   South Central Poultry Research Laboratory
                                    USDA/ARS
                                    Mississippi State, MS 39762 USA

D.J. Farrell   Department of Biochemistry and Nutrition
                                    University of New England
                                    Armidale NSW 2351
                                    Australia

V.A. Finch   Tropical Cattle Research Centre
                                    CSIRO Division Tropical Animal Sciences
                                    North Rockhampton, Queensland 4701
                                    Australia

S.M. Gaafar   Department of Veterinary Microbiology and
                                    Pathology and Public Health
                                    Purdue University
                                    West Lafayette, IN 47907 USA

G. Gettinby   Department of Mathematics
                                    University of Strathclyde
                                    Glasgow
                                    Scotland
                                    United Kingdom

R.D. Ingraham   Department of Physiology and Toxicology
                                    School of Veterinary Medicine
                                    Louisiana State University
                                    Baton Rouge, LA 70803 USA

N.St.G. Hyslop (deceased)   Animal Disease Research Institute
(Member of CAgM VI and   Health of Animals Branch
CAgM VII W.G.)   Agriculture Canada
                                 PO Box 11300
                                 Postal Station "H"
                                 Ottawa, Ont. K2H 8P9 Canada

H.D. Johnson   Department of Dairy Husbandry
(Member CAgM VI Working Group)   103 Eckles Hall
                                 University of Missouri
                                 Columbia, MO 65201 USA

K.W. Kelley   Department of Animal Sciences
                                 University of Illinois
                                 126 ASL
                                 2107 West Gregory Drive
                                 Urbana, IL 61801 USA

V. Lopez   Head, Veterinary Diagnostic Laboratory
                                 Veterinary Division
                                 Ministry of Agriculture
                                 PO Box 309, Hope Gardens
                                 Kingston 6 Jamaica

M.G. MacLeod   Agricultural Research Council's Poultry
                                 Research Centre
                                 Roslin, Midlothian EH 25 9PS
                                 Scotland
                                 United Kingdom

H.J. Mertsching   Department of Animal Sciences
                                 College of Agriculture
                                 Washington State University
                                 Pullman, WA 99164 USA

M.A. Mitchell   Agricultural Research Council's Poultry
                                 Research Centre
                                 Roslin, Midlothian EH 25 9PS
                                 Scotland
                                 United Kingdom

S.R. Morrison   Department of Agricultural Engineering
                                 University of California, Davis
                                 Davis, CA 95616 USA

M.D. Murray   CSIRO, McMasters Animal Health Laboratory
                                 Private Bag #1
                                 Glebe NSW
                                 Australia 2037

C.B. Ollerenshaw   Department of Parasitology
                                 Central Veterinary Laboratory, MAFF
                                 New Haw
                                 Weybridge, Surrey
                                 England
                                 United Kingdom

LIST OF CONTRIBUTORS

J.A. Regnier	Department of Veterinary Microbiology and Pathology College of Veterinary Medicine Washington State University Pullman, WA 99164 USA
S.M. Shane	Department of Epidemiology and Community Health School of Veterinary Medicine Louisiana State University Baton Rouge, LA 70803 USA
H.S. Siegel	Southeast Poultry Research Laboratory USDA/ARS 934 College Station Road Athens, GA 30605 USA
J. Slee	ARC Animal Breeding Research Organization Field Laboratory Roslin, Midlothian EH 25 9PS Scotland United Kingdom
C.V. Smith	Meteorological Office Met 0 8 London Road Bracknell, Berks. RS12 2S2 England United Kingdom
C.E. Terrill	National Program Staff USDA/ARS Beltsville, MD 20705 USA
C.J. Thwaites	Department of Animal Sciences University of New England Armidale, NSW 2351 Australia
H.G. Turner	Tropical Cattle Research Centre CSIRO Division of Tropical Animal Sciences North Rockhampton, Queensland 4701 Australia
A.E. Wrathall	Head, Diseases of Breeding Central Veterinary Laboratory MAFF New Haw, Weybridge, Surrey England United Kingdom

C H A P T E R   1

INTRODUCTION

1.1   ANIMAL HEALTH - A ROLE FOR AGRICULTURAL METEOROLOGISTS

      Aims: The emphasis in this study will be on the response of farm animals to extremes of the thermal environment in the presence of organisms that are likely to challenge and infect.

      The reader will find significant implications for methods of managing farm animals and their total environment. The effective control of stress can be expected to increase rates of animal performance and productivity; control of stress will lead to better animal health (in so far as these rates are a reflection of the ability of the immune system to contain the challenge of pathogens and parasites). A comment (Smith, 1988) which is not fully explored, is that in intensive systems for housed livestock, control of the thermal environment should be complemented by control of air hygiene.

      However the expectation must be that stressful weather will inevitably occur. Somehow agricultural meteorological services must reach farmers and others so that they understand the role played by the weather and climate so that the decisions and actions they take will be consistent with the constraints set by the conditions, with outcomes predictable by the physical sciences.

      Problem areas which should be noted by the agricultural meteorologist:

(a)   Education and training: To face up to the problems of animal health generally, the meteorologist must move beyond an understanding of the processes and variations of the atmosphere in the boundary layer, and the role of local ground cover and topography in these variations. He should learn something of current farm practice and the implications of ambient conditions for animals, for pests and diseases, for vegetation and soils, and for environments within farm buildings and stores, etc. Generally the meteorologist should educate himself in these matters, and as self-motivation and education are ongoing processes, training courses are at best only passive pointers to the way. Useful results and applications will not follow unless individual meteorologists set questions and provide answers for themselves.

(b)   The thermal environment:

      (i)   In his region the meteorologist should look at the thermoneutral ranges of environmental variables for important classes of livestock in the light of the weather and the seasonal variations that he knows can occur. Past weather data (both conventional and derived climate data) should be analysed and interpreted for the specific purpose of establishing risks and probabilities.

      (ii)   A detailed energy budget for individual animals and groups of animals can indicate imbalances between metabolic heat

production and heat losses to the environment under various realistic combinations of weather variables. The weather data must be at an appropriate resolution, i.e. hourly, or perhaps daily, values. For each class of stock, but particularly for young or newborn animals, the maximum possible (peak) rate of metabolic heat production is of considerable interest, together with the length of time it can be sustained; the likely duration of spells of weather that take the animal beyond its comfort zone needs to be known, whilst the accumulation of such periods over a season (when interpreted in terms of implied weight loss, etc.) will provide some measure of economic performance. Extended weather episodes that affect the availability or amount of food intake are clearly stressful. If they are also linked with thermal stress and if there are competing demands for body reserves (as in pregnancy), then the metabolic disorders induced may have effects which extend beyond the weather episodes themselves and which may not be fully recognized until the young are born.

(iii)   An understanding, preferably quantitative, of how environmental variables affect the heat budget of animals should suggest how their ambient environment might be manipulated by natural and man-made shelter against wind, sun, precipitation, by site selection to increase or decrease exposure, and by artificial aids that would provide additional heating or cooling directly.

(iv)   Where (intensive) animal housing offers improved animal and economic performance, then a plan to change the external macro-environment into an acceptable micro-environment would commonly also call for an energy budget approach, with the house and its animals as the unit, and ventilation (natural or fan-assisted) as the primary control variable. Minimum ventilation rates set simply from consideration of the thermal environment and desirable temperatures are, in cold climates, likely to provide environments of poor air quality, highly contaminated with the products of respiration, etc., (high CO_2 levels, high aerosol and pathogen content).

(c)   The disease environment:

(i)   Better feeding, improved hygiene, advances in veterinary medicine, all play a part in the control of infection in animals. But clearly the timing and the scale of the problem, together with the immune response of animals are weather-dependent phenomena in many instances. If the meteorologist is to be of assistance as to the control measures then he must visualize, quantitatively if possible, the life cycle of the organism or vector involved. He should identify opportunities for comment by himself, and for intervention by farmers and veterinarians (primarily through control of the animal host availability, the animal host exposure to disease, and the effectiveness of animal immune response) which will affect the success of the disease organism.

(ii)   Whilst many species of pathogens or parasites may have
        similarities in their constitution, their life cycles and
        their response to environmental factors, experience
        suggests that each organism has to be seen as representing
        a separate and distinct problem to the meteorologist. The
        same environmental variables (temperature and moisture in
        the air, in or on the soil or vegetation, air movement) are
        likely to have the same general significance for broad
        classes of micro- and macro-organisms, but the response of
        populations of different species to weather and environ-
        mental factors will differ in detail.

(iii)   Typically, in the case of virus spread by an airborne route
        it becomes necessary to explore the volume of virus output
        and the associated aerosol particle size spectrum;
        mechanisms and routes for dispersion from the primary host;
        virus viability in the external world and modes of virus
        uptake; relations between virus virulence, dosage and
        immune response in the secondary host, and incubation and
        progress of the emission intensity/time profile in the
        secondary host. Ideally one should aim to quantify many of
        these aspects of the total cycle.

(iv)   The aim is towards an integrated control procedure in which
        understanding of the implications of husbandry practice and
        of environmental factors (including weather and climate),
        together with veterinary medicine options, are applied in
        ways that are complementary. A role for the meteorologist
        is particularly emphasized in regions where preventive
        veterinary medicine and resources for primary animal health
        care are minimal.

(v)   In any epidemic, or at times of seasonal peaks of disease,
        the meteorologist should look for ways to give operational
        advice to those actively involved in control measures in
        the field, so that limited resources can be deployed most
        effectively. The meteorologist has to set about the
        interpretation of climate data in order to suggest
        husbandry practices which reduce the burden, the
        occurrence, or the spread of infection within and between
        successive generations of animals, and regions. A
        knowledge of disease epidemiology and population dynamics
        is clearly essential; this knowledge should be applied to
        give numerical assessments of disease risk through
        associations developed from past case studies, (e.g.
        regression relations employing gross climate data,
        simulation models employing weather data at finer
        resolution). The aim is to use the data flowing from
        weather monitoring networks to suggest a primary reason for
        change in disease risk.

Problem areas for the meteorological services:

(a)   Data gathering:

    (i)   Meteorological services provide and archive weather
            observations. Where the conventional set of observations

is seen as inadequate (in the light of known environmental requirements of specific disease vectors), agro-meteorological services should press for the introduction of additional measurements such as soil temperature, pasture canopy wetness, etc. If comparable biological field data on the incidence of animal stress and disease are not available from other agencies, then the agricultural meteorological services should obtain and archive the biological information that they need for operational advice or for research and development studies. However, liaison and co-operative work is clearly preferable.

(ii)   For detailed epidemiological case studies, where the objective is to unravel weather or environmental criteria of significance, the need is for dates, times, locations of disease outbreak, the numbers of animals involved and the sequence of events regarding the disease spread and progression. If information on this scale is not available, summaries of the recollections of individual and groups of farmers, etc. on the extent of particular diseases in particular seasons can provide a useful entry point for weather-based investigations. Experience would suggest that the resolution of the weather data employed in initial investigations should be the finest that records allow, i.e. hourly rather than daily, daily rather than monthly. Later analyses should attempt to replace these optimum data with those from standard equipment used in routine activities.

(b)   Advisory services:

(i)   A recasting of conventional weather forecasts to suggest the probability of direct weather hazards or spells of weather stressful to farm animals is an obvious requirement. This implies an adequate briefing of synoptic forecasters and the provision to them of simple rules and guidelines that will enable them to make the transfer from scales of weather variables to scales of stress. Information from weather observing networks (both the observations and automatically derived indices) should be available, in near real-time, to be accessed by the knowledgeable farmer himself if necessary.

(ii)   Conventional climate data of good quality, including information on station histories with site and instrument changes, should be available. These should find application in land-use studies and in disease risk assessment in broad terms, and in epidemiological case studies. They should also find application in the environmental engineering design of animal houses and storage facilities; at times the meteorologist may be required to carry out the engineering calculations himself to suggest a size or rating for the equipment of a particular project.

(iii)   Recent climate data (of the past days, weeks, months, decades) should find application in current advice on land-use rotations, on husbandry and veterinary practice. But there must be sufficient lead time between the significant weather and the consequences for parasite and/or animal host, so that the farmer and veterinarian can react in time to achieve some control. Appropriate interpretation of extended current weather forecasts may be needed to complement this work.

(iv)   Field work by agricultural meteorologists should establish relations between weather and environmental variables, monitored by standard equipment under standard exposure; and environmental conditions in parasite and vector habitats, in the soil, herbage, and in tree canopies etc. It should take in local studies to assess the effects of topographical features, on exposure and local variations in environmental variables of significance, as well as the monitoring of environments inside animal houses, and the siting and design of animal houses, feedlots, and so on. Sufficient field work needs to be undertaken to enable the experienced meteorologist to comment on the advantages and disadvantages of particular sites without necessarily taking measurements that would extend over weeks and months.

(c)   Liaison:

(i)   The nationwide channels of communication used by the weather observing networks and by forecasting services are unlikely to be duplicated by other services with biological field monitoring, reporting and advisory programmes. The scope for co-operation, rationalization and joint agricultural meteorological bulletins should be explored. An active link, with day-to-day contact with the agricultural extension and veterinary services, is essential and can provide the necessary contacts to those concerned at the national level.

(ii)   Personal contact within universities, agricultural research institutes, commercial farms, and agricultural businesses that at times enter into advisory work can be helpful in assessing priorities for agricultural meteorology and for assistance in its development. Positive encouragement and facilities for free-ranging contacts are needed in order to tackle problems that cross the boundaries of scientific disciplines and individual institutes.

(d)   Research and Development:

(i)   If answers are needed to questions that demand more than simple recall of conventional climate data, then research and development work is needed to develop the necessary expertise and to develop operational systems that give useful and timely advice.

(ii)   Inquiries put to the meteorologist should help his
         assessment of priorities, but at the same time he must
         trust his judgement of what meteorology might contribute in
         order to initiate projects of a multi-disciplinary nature.
         To build up useful working relations with "customers" it is
         necessary to specialize in animal biometeorology.

(iii)   On questions of thermal stress, fundamental studies are not
         likely to be needed by most agricultural meteorological
         services, since enough is known and reported in the
         literature to enable simple advice to be given. What is
         needed is assimilation and application of understanding;
         meteorological data and a willingness to undertake field
         work is all that is required. Increasing collaboration
         with animal scientists will enable more and more expert
         advice to be given.

(iv)   The move into advice on thermal and nutritional stress as a
         factor in immune response is a more demanding task. The
         search for relationships between weather, environment and
         animal health and disease, where the object is control, has
         to be seen as a step into the unknown. However, in the
         literature, there are already valid examples which treat
         both micro- and macro-parasites, their occurrence in space
         and in time and their numbers (in broad terms). The
         predictions made are based on procedures that have varying
         degrees of sophistication. There are analogues on which to
         proceed. The starting point might be collaboration with a
         farmer, an animal husbandry worker or veterinarian who
         admits to a problem that does not respond to his normal
         approaches, a problem which he thinks must in the end be
         due to weather and about which he is sufficiently worried
         or curious to collect and provide data from the field, from
         the farm or even from the laboratory.

1.2   METEOROLOGICAL GLOSSARY

Air mass: A large body of air that acquires through surface
                     contact uniform characteristics of temperature and
                     humidity. Primary air masses are labelled according to
                     their origin - tropical, polar, maritime, or continental.

Albedo: A measure of the reflecting power of a surface for solar
                     radiation. The albedo of a grass surface is about 0.25,
                     i.e. 25% of the incident solar radiation is returned to
                     space without change of wavelength. The albedo of bare
                     soil is commonly in the range 0.10 to 0.25.

Ambient air   The uncontrolled temperature characteristic of where an
temperature: animal is housed or grazes. (AT)

Boundary layer: In the 'surface boundary layer' of the atmosphere, i.e.
                     for depths up to around 100 metres, the air motions are
                     significantly influenced by the presence and nature of
                     the Earth's surface; within an overlying layer (the
                     planetary boundary layer) i.e., for depths up to around

600 m, the effects of the Earth's surface on air movements are still considerable.

Climate:

The collective state of day-to-day weather at a place or over an area expressed by the statistical properties of various weather elements. The average temperature and the total precipitation for a given time interval are often chosen as characteristics of climate. In the context of the present Note there is also interest in the probability of the occurrence of extreme values of weather variables and their duration, e.g. the occurrence of large diurnal ranges of temperature, hours above or below some threshold value.

Climagram:

A diagram comprising a plot of (monthly) values of two selected meteorological variables (commonly temperature and rainfall are taken as abscissa and ordinate). The plotted points are joined by a line which represents the annual variation of the relationship between the variables.

Conduction:

The transfer of energy within and through a conductor by means of internal particle or molecular activity and without any net external motion. As air is a poor conductor, it is heated by this means only within a few centimetres of the heat source. The distribution of heat away from that source is by convection and eddy-heat conduction.

Continental climate:

A climate characterized by a wide range in temperature between the warmest and coldest month (and from night to day), apt to have a dry, sunny regime, often with a pronounced rainy season.

Convection:

The transfer of heat (energy) within a fluid such as air through processes that involve the movement (and mixing) of substantial volumes of the air. In 'free' convection, buoyancy forces operate to effect the vertical transport and mixing of warmed air streams or 'bubbles'. In 'forced' convection, the heat transfer is effected by eddies in a turbulent airstream maintained by mechanical means (e.g., the wind or a fan jet) and directed over the animal.

Depression:

A centre of low air pressure around which winds circulate in a counter-clockwise direction in the northern hemisphere (clockwise in the southern); a cyclone of extra-tropical latitudes; a 'low'.

Front:

A boundary or transition zone between atmospheric air masses, usually labelled a cold or warm front, depending on the temperature of the actively advancing air.

High-pressure area or system:

A centre or region of high air pressure, inducing in the northern hemisphere a clockwise out-flowing circulation (counter-clockwise in the southern); an anticyclone.

Humidity:	A measure of the water vapour content of the air.
	● Absolute Humidity – grams of water (vapour) per cubic metre of air.
	● Relative Humidity – the ratio (written as a percentage) of the water vapour pressure in the air to the saturation vapour pressure at the prevailing temperature. If the water vapour content of a parcel of air is not changed but its temperature is increased, Relative Humidity (RH) will fall. Tables are available which show the relationships between simultaneous readings of Dry Bulb and Wet Bulb temperatures and Vapour Pressure, Relative Humidity and Dew Point.
	● Dew Point – that temperature to which a parcel of air must be cooled in order that water vapour pressure reaches saturation vapour pressure.
Infra-red:	Part of the spectrum of electromagnetic waves beyond the visible red but of shorter wave length than radio waves; infra-red radiation is often designated less precisely as long-wave or heat radiation.
Inversion:	In meteorology, refers to a vertical temperature distribution where warmer air is lying over cold air; specifically a ground inversion where air temperature is lowest near the ground and increases with elevation; a stable stratification of air in which vertical motion is inhibited.
Ion:	In meteorology, electrically charged air molecules or particles floating in air.
Isobar:	A line of constant (atmospheric) pressure.
Isohyet:	A line along which the amounts of rainfall are equal.
Isotherm:	Line along which the air temperature has the same value.
Low-pressure area or system:	A centre or region having low barometric pressure, usually applied to a large middle or high latitude depression (see also depression).
Maritime climate:	A climate characterized by a small range between the warmest and coldest month of the year; diurnal variations of temperature are also small; often cloudy and marked by an even distribution of precipitation throughout the year; humidities are usually high.
Occlusion:	A front which develops during the later stages of the life cycle of a depression initially associated with well marked cold and warm fronts; the term arises from the occluding (shutting-off) of the warm air from the Earth's surface.
Photochemical:	Narrowly pertaining to chemical actions induced by light, but more generally including changes caused by other wave lengths, such as ultraviolet; attributed to photons, which are quanta of energy.

Radiation:

The transmission of energy by electromagnetic waves. Any body emitting radiation loses energy; any body absorbing radiation receives energy. The wavelength of maximum energy emittance depends on the temperature of the radiator. For solar radiation peak emittance occurs at wavelengths of around 0.5 (shortwave radiation). For radiators at the temperature of the Earth's surface emittance peaks at wavelengths of around 10μ (long wave radiation – terrestrial radiation).

Saturation deficit:

The difference between the actual vapour pressure and the saturation vapour pressure at the existing temperature, expressed in hectopascals.

Spectrum:

The range or dispersal of the various wavelengths from a light or energy source.

Temperature-humidity index: THI

THI = 0.4 (T dry bulb + T wet bulb) + 15 (for °F).
THI = T dry bulb – 0.55 (1-RH) (for T dry bulb in °F and RH as a proportion).
THI = T dry bulb + 0.36 (T dew point) + 41.2 (for T dry bulb in °C).
THI = 0.72 (T dry bulb + T wet bulb) + 40.6 (for °C).

Temperature inversion:

A layer in which temperature increases with altitude. The principle characteristic is its static stability which ensures that very little turbulent exchange can occur within it. Strong wind shears often occur across inversion layers and abrupt changes in concentrations of atmospheric particulates and water vapour may be encountered on ascending through the layer.

Ultraviolet:

Part of the electromagnetic wave spectrum shorter than the visible violet light but longer than x-rays; a photochemically active radiation.

Vapour pressure:

The partial pressure of water vapour mixed with air, expressed in hectopascals.

Vaporization:

The physical process by which a liquid or solid is transformed into the gaseous state; in meteorology it is usually restricted to the change of water from liquid to gas. The latent heat of vaporization of water is approximately 597 calories/gm.

Weather forecast:

A statement of anticipated weather conditions for a specified area (or place) over a period of time.

Weather report:

A statement of values of meteorological variables observed at a specified place and time. It is a record of an observation, not a forecast.

Weather map:

A chart of a geographical area on which are plotted selected meteorological elements observed at a particular time at various sites over the area; a

'synoptic' chart or 'surface' chart. Analysis of
weather maps almost invariably involves the use of
isobars to relate the observed values of weather
elements to pressure patterns (which show considerable
coherence or persistence, albeit with some displacement,
from one time chart to another time chart).

1.3   PHYSIOLOGICAL AND PATHOLOGICAL GLOSSARY

Acclimatization: The gradual accommodation within hours, days or at most
a few weeks, of physiological processes to a new
climatic environment.

Adaptation: The process by which the animal adjusts physiologically
to maintain a relatively stable state (homeostasis) in
the face of disruptive external environmental challenges.

Adrenal: Pertaining to a small ductless gland located adjacent to
the upper part of the kidney, secreting a hormone
raising the blood pressure.

Allergy: An excessive sensitivity to substances breathed or
ingested or to other environmental conditions, causing
reactions such as hives, sneezing, or asthma.

Anoestrus: Absence of oestrus.

Biologic response
functions: Quantified relationships established between the
responses of biological organisms (B) (dependent
variables) and environmental factors (E) (independent,
nongenetic variables) on the basis of experimental data,
so that $B = f(E)$. An example for milk production
decline (MDec) as related to the temperature-humidity
index (THI) is: $MDec = -1.075 - 1.736 NL + 0.02474 (NL)
(THI)$ where NL is the normal level of milk production,
kg/cow-day, at THI 70.

Calving-conception
interval: The period in days from calving to conception. For
dairy cows the optimum is less than 90 days.

Calving interval: The period in days between subsequent calvings. It can
be used both for individual cows and for a herd or
herds. The optimum is less than 365 days.

Calving rate: Percentage of heifers or cows calving in a twelve-month
period. The optimum is 95% - 100%.

Circadian: A rhythm during the course of a calendar day, usually
applied to physiological phenomena.

Cold stress: Physiological state induced by ambient temperatures
below the lower critical temperature of the
thermoneutral zone (see Figure 1.1).

Conception rate: Percentage of animals conceiving after insemination.
Conception is based upon the production of a calf, alive

or dead, or on the basis of a rectal palpation of the uterus and ovaries, 35 to 60 days after insemination. (Readers should be aware of this and check which definition was used by an author. The optimum depends on circumstances but is usually in the range of 55% to 65%.)

Critical temperature: Upper critical temperature (UCT): The upper limit of the thermoneutral zone. Above this temperature, evaporation becomes the dominant method of heat loss in most species. Lower critical temperature (LCT): The lower limit of the thermoneutral zone. Below this temperature, metabolism must increase to offset losses to the environment (see Figure 1.1).
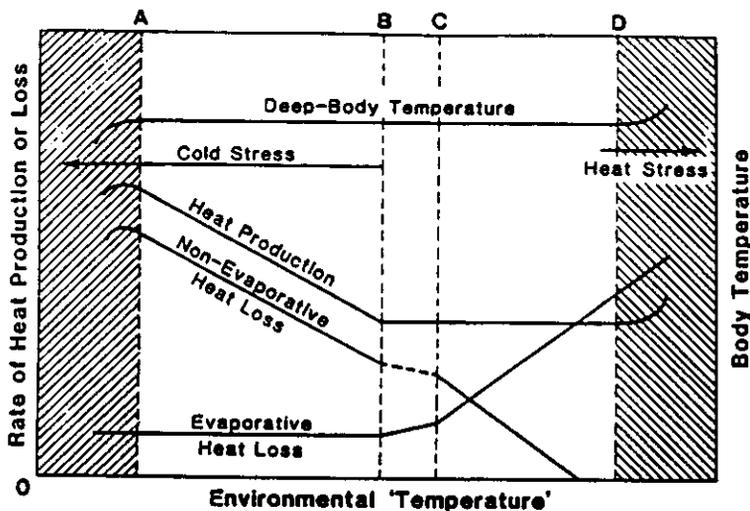

Figure 1.1 – Schematic representation of components of energy balance of a homeotherm (after Mount, 1973); A, temperature of summit metabolism; B, lower critical temperature; C, temperature associated with marked increase of evaporative loss; BC, zone of least thermoregulatory effort; D, upper critical temperature

Deep body temperature: The temperature of the body core, often represented by the rectal temperature (RT).

Digestibility: Proportion of food digested, usually based on dry matter of food eaten and faeces voided.

Endocrine: Pertaining to body glands producing internal secretions which, carried by blood or lymph, control or regulate various organs or functions.

Environmental demand: A requirement placed on an animal as a result of the environment; regarding the thermal environment: cold ambient temperatures, especially when combined with wind, draughts or precipitation, require the animal to increase heat production to maintain homeothermy.

Environmental load: A burden imposed on an animal as a result of the environment; as regards the thermal environment: solar radiation imposes an additional load on the animal subjected to high temperatures.

Gangrene, gangrenous: Decay of body tissue usually because of obstruction or loss of blood supply to an injured area of the body.

Heat stress: Physiological stress induced by ambient temperatures above the upper critical temperature of the thermoneutral zone (see Figure 1.1).

Homeothermy: The ability of warm-blooded animals to maintain a constant body temperature through basal metabolism in the face of fluctuating ambient temperatures.

Homeothermal: The property of warm-blooded beings to keep approximately constant body temperature.

Hormone: A substance formed by and circulated from one of the glands of internal secretion, generally causing powerful stimuli or regulation of body functions.

Incidence: The frequency with which new events occur, usually disease. The incidence rate is the number of newly reported events, or cases, occurring over a stated period divided by the average population during that period.

Incubation period: The interval between infection with a disease agent and the appearance of symptoms.

Lethal body   The deep body temperature at which all metabolic defence
temperature: mechanisms fail and death occurs.

Maintenance   Basal metabolism; Resting metabolism; the minimal energy
metabolism: expended for the maintenance of respiration, circulation, peristalsis, muscle tone, body temperature, glandular activity, and other vegetative functions.

Metabolism: A process characteristic of living beings in which food substances are transformed into tissue with release of energy and waste.

Necrotic: Pertaining to dead tissue in the body.

Oestrus: The behaviour of the female when 'on heat' and accepting insemination by the male; usually associated with ovulation. It is the physiological and behavioural response to the release of oestrogens in the non-pregnant female.

Optimal performance: The maximum growth, efficiency, reproduction or other performance measure. Optimal economic performance or optimal welfare performance may or may not be coincident with optimal productive performance. For example, optimal performance for growth rate is near 18°C, while that for energetic efficiency is near 21°C.

Pathogen: A micro-organism or virus causing disease.

Pathology: The branch of medicine dealing with disease, especially the morphological and functional changes caused by disease.

Peak metabolism: The maximum production of energy that can be produced biochemically.

Physiology: The branch of biology dealing with the functions of, and processes in, living organisms, usually referring to normal conditions.

Poikilothermal: The exhibition of body temperature that varies with the environmental temperature (cold-blooded).

Prevalence: The number of cases known. The Point Prevalence Rate is the number of cases known at a stated time divided by the population at that time. The Period Prevalence Rate is the number of cases occurring during a stated period divided by the average population during that period, usually at the mid-point.

Spermatogenesis: The process of formation of spermatozoa.

Stressor: An environmental factor which imposes stress on an animal as evidenced by altered physiological, behavioural, immunological or other measurable parameters, and which may or may not alter overall performance.

Summit metabolism: The maximal rate of heat production that is achieved in response to cold and can be sustained for some time without hypothermia.

Systolic blood pressure: Arterial pressure of the blood at the time of heart contraction when blood is forced from the heart chambers into the circulatory system.

Thermoneutrality: The range of ambient temperatures in which normal metabolism provides sufficient by-product heat to maintain an essentially constant body temperature. The limits of the thermoneutral zone (TNZ) are dependent on acclimatization, age, sex, breed and species, body conformation, feed and even the time of day (see Figures 1.1 and 1.2).

Toxicity: The state or level of poison or drug action.

Vasoconstriction: Constriction of a blood vessel or blood vessels.

1.4   CRITICAL TEMPERATURES - A DISCUSSION

It should not be thought that the critical values of environmental parameters can be readily defined for a given animal species. This would imply a "typical" animal while, as we know, there can be significant differences between individuals. There are major breed differences, affecting pelage, hair colour(s), body conformation, metabolism, as well as genetic differences within breeds. Age, productive and physiologic states, activity, nutrition, and other stressors, such as parasites or disease, all affect animal tolerance. Animals pass through cycles of acclimatization with season, so that a given thermal environment in spring may have different effects than the same kind of day in summer. Similarly, the variation between day and

night, and over a series of days, is related to the relative significance of acute and chronic thermal stress. For example, under summer conditions and the risk of heat stress, the opportunity for night-time cooling is crucial. Reaching a critical body temperature for a limited period may be relevant for, say, the induction of abortion or the surfacing of latent infection, but in general the chronic level of stress, either of heat or cold, rather than its peaks, are likely to be more important to overall productivity.

Figure 1.2 indicates the critical ambient temperatures and zones for optimal performance and nominal losses in farm livestock. The values shown represent the majority of each species population. These values should be taken as approximations only and are subject to circumstances. For example, wetted skin and pelage, or air velocities above 0.3 m/sec, shift all critical temperatures upward, just as an elevated humidity or exposure to solar radiation shifts all temperatures downward.

The impact of environmental factors and the reacting animal are integrated in the body temperature, which in turn interacts with the physiological and behavioural responses. Rectal temperature has advantages over climatic factors because it is a measure of the efficiency with which the animal is able to handle its excess heat load, or demand, and its adjustment to stressful weather.

The energy exchange between an animal and its environment is complex, involving radiation, convection, and conduction. While air temperature is frequently used for simplicity to measure the physical environment it does not describe the true thermal environment. For example, cold, uninsulated walls increase heat loss by radiation, and draughts increase convective heat loss. If accurate predictions of animal response to temperature stress are to be made from climate factors, allowance must be made for acclimatization to the weather during the previous two or three weeks. Humidity, thermal radiation, air velocity, light, precipitation, barometric pressure, dust, and gases will modify the influence of temperature. However, in spite of the possible modifying factors, as weather is generally consistent, some useful and fairly accurate predictions can be made.

Hahn, _et al_., (1983) have described an approach to the development of rational environmental criteria, which is relevant both to the theoretical selection as well as to the application and use of criteria in the accompanying papers. The development of criteria requires: (a) quantification of the environment; (b) performance measures and biological response functions; (c) consideration of threshold limits; (d) effects of compensation; and (e) statistical evaluation of environmental factors.

(a)   Environment quantification

Because the environment is so complex, quantifying its overall relationship with animal responses is difficult. But by using either single factors or small groups of factors to represent the environment, quantified relationships become possible though sometimes difficult to reproduce because of the impacts of the ignored factors. Ideally, integrative sensors or indices would combine the relative heating or cooling potentials of convection, radiation and conduction with the cooling potential of evaporation. Measuring sensors have been constructed and various thermal indices are available. However, three major problems exist in the use of integrative sensors and indices: (i) None
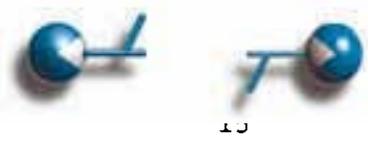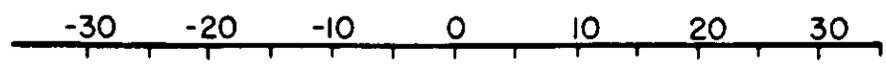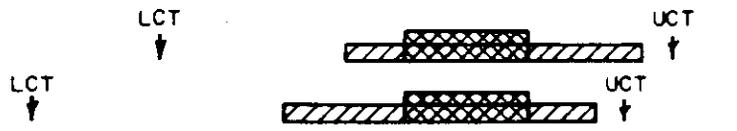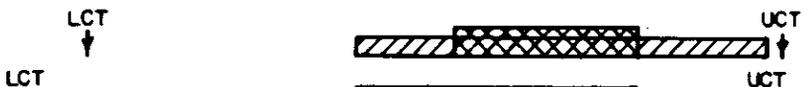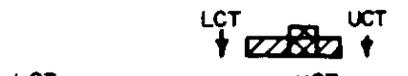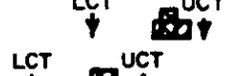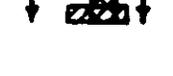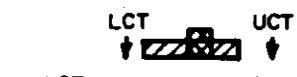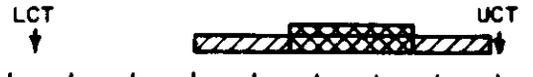
TEMPERATURE, °C

Figure 1.2 – Critical ambient temperatures and temperature zones for optimal performance (centrally shaded areas) and nominal performance losses (extended areas) in livestock; L, lower critical temperature; U, upper critical temperature

represent a single widely accepted and therefore routinely measured parameter. As a result, probability measures for assessing thermal impacts for animals in a given situation are few. (ii) Sensor locations often represent the mesoclimate, or even the macroclimate, while it is the animal's microclimate, that is of most concern. Variations between the microclimate and the mesoscale conditions can be considerable. (iii) Sensors and indices do not reflect the complex, physiological, behavioural and adaptive capabilities of the animals represented.

(b)   Performance measures and biological response functions

The specific response of an individual animal is influenced by many external and internal factors and is complicated by their interactions, especially when considering time-dependent factors such as age, gestation, or adaptability. Because of wide individual variations, statistical descriptions representative of the population must be used. Historic measures of growth, milk, eggs, wool, reproduction, feed conversion and mortality serve as integrative parameters and also for economic assessment. In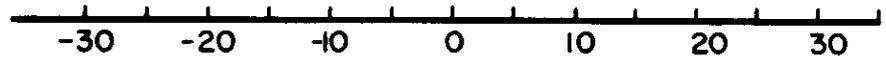 recent years additional parameters have been used, such as: morbidity, behaviour and well-being, endocrine and other physiological measures, immunity and disease resistance, and lastly, energy balances and quality evaluations of the final product. Animal performance should be tied to pre-targeted production levels of the livestock enterprise. As management improves productivity, the management targets and potential optima will coincide.

Quantified biological response functions are necessary when modelling animal performance. Early models used crude growth rates and feed conversions at various temperatures. Later, more refined models reflected the operational realities of interacting effects of temperature and humidity in modern livestock production units. Recent studies incorporate knowledge into "best-guess" response functions for temperature-nutritional interactions to suggest appropriate feeding in adverse weather.

(c)   Threshold limits

Thresholds are an important concept in developing rational criteria. They represent discontinuities in otherwise continuous biological functions. Within limits, the overall performance of animals resulting from various environmental interactions can be fairly constant. In Figure 1.2 temperature thresholds have been used to delineate the optimal and nominal performance zones. Other external environmental factors, such as humidity, radiation, and wind can alter the threshold limits as can intrinsic factors such as diet, genetic characteristics, health and activity. It must be emphasized that the limits need statistical evaluation for a population. The limits are biologically dynamic; for example, cold-conditioned range beef cattle in Canada can become heat stressed when ambient temperatures rise quickly to near 0°C, normally considered to be well below a heat-stressing temperature. Therefore, the upper and lower critical temperatures must be considered to be approximations.

Animal health is well suited to the general management concept of the treatment of losses and vulnerability to increased risk of losses. Nominal performance losses, in performance, morbidity, or mortality, are those considered to be negligible in terms of impact on management decisions; for example, a fall in growth rate of less than 50 g/day or a piglet mortality rate of under 5% of piglets born. These performance losses should be explicitly stated in terms such as mortality or disease incidence, or subclinical disease, and reduced productive performance. When these losses reach a preselected threshold, management by exception can institute the appropriate tactics in order to return the losses to acceptable levels.

During nominal losses, the biological response functions are of no other value than to establish loss thresholds. Beyond the loss thresholds, response functions are of major importance in assessing the increased risk of performance penalties and alternative strategies. For example, there is a slight vulnerability with modest production and mild environmental stress. But a high-performance animal, even in a moderate environment, can be at risk. Combining high performance with an adverse environment increases the risk. Inherent genetic characteristics which are disadvantageous for coping with the environment, coupled with high performance demands, mean that any environment other than the optimal increases animal vulnerability and managerial risk to unacceptable levels.

(d)   Compensatory effects

Within threshold limits, animals have considerable ability to rebound when stressors are removed. Compensatory capacity should be considered in the design and management of experiments, housing, and environments. The early models and resulting energy concepts tended to be based on short-term measures of stressor effects and indicated precise optimal temperatures for growth and feed conversion. In the longer term the animals' adaptive mechanisms blur the sharp edges of the short-term optimal and threshold values. As a result the response relationships for total efficiency tend toward continuous rather than discontinuous functions. Typically the response functions have relatively flat peaks so that losses are nominal over a wider range of temperatures either side of the optimum. While the healthy animal can withstand these wider ranges, they are detrimental to the diseased or neonatal animal.

(e)   Statistical evaluation

Climatological records will provide data on the frequencies of singular factors, as well as the likelihood of the occurrence of interacting factors, such as extreme temperature and humidity. Design temperatures are seldom based on the most extreme value experienced at a site, but are used to allow an acceptable level of risk being included. Appropriate livestock housing can be designed to accept a risk of approximately 10% of seasonal extremes.

*

*   *

C H A P T E R   2

COLD WEATHER STRESS

2.1   CATTLE – COLD STRESS

2.1.1   General observations

In general, cattle are tolerant of cold, although rate and efficiency of production may be reduced during exposure to cold when compared with more optimal conditions. There are, however, wide genetic differences among breeds of cattle with respect to productivity during cold, and in particular, little is known of the effect of cold on heat-adapted cattle such as the Brahman. Some unique aspects of cattle that are important to cold hardiness are: (a) relative maturity at birth which aids the survival of the newborn, (b) the ability to increase external insulation (haircoat) so reducing the rate of of heat loss, (c) the ability to increase the rate of heat production during exposure to cold, (d) the relatively large body size which helps to minimize the rate of heat loss per unit of mass, and (e) being ruminants they can consume large quantities of feed which is both a source of heat by fermentation and a valuable mechanism for withstanding periods of fasting when ice and snow may limit the intake of feedstuffs.

Thermal zones for cattle (including LCT) are dynamic; they change when any factor increases either the rate of heat loss or heat production. Increased insulation, for example, is an added barrier to heat flow and influences the rate at which sensible heat is exchanged with the environment. Insulation for cattle includes tissues insulation (fat, skin), external insulation (haircoat) and the insulative value of the air surrounding the animal. These insulative barriers are additive and are a major factor in established LCT and rate of heat loss below LCT. As an animal's insulation increases, the TNZ and LCT are lowered.

The predicted LCT for cattle on high feeding diets are considerably lower than for poultry or swine not on maintenance diets, or for cattle on maintenance diets. Examples are given in Table 2.1. These values should be considered only as indicators of cold susceptibility as, in practice, the actual LCT may vary considerably depending upon specific housing and pen conditions, age, breed type, lactational state, nutrition, time after feeding, history of thermal acclimation, haircoat, and behaviour. The extremely low values for the feed lot animal and dairy cow result from the large amounts of heat produced as an inevitable consequence of digestion and metabolism at high levels of production, from the small surface area to the mass ratio of these relatively large animals, and from their large amount of insulative tissue.

Dry-bulb temperature, precipitation, wind and solar radiation are the main physical variables affecting the cold hardiness of cattle. While the most important climatic factor is air temperature (McDowell and Johnston, 1971), it is essential to evaluate the impact of a given thermal environment on specific cattle in terms of the remaining climatic factors. Wind affects both the convective and evaporative losses of heat from cattle. For animals exposed to cold, increased heat loss is primarily a function of decreased external insulation (both haircoat and air insulation). The wind chill table

TABLE 2.1

Estimated lower critical temperature of cattle

Description	Critical Temp.(C)	Basis of Estimate	Source
8 mm hair, fasting	18	Calculated	Blaxter, 1967
8 mm hair, maintenance	7	Calculated	Blaxter, 1967
8 mm hair, full feed	-1	Calculated	Blaxter, 1967
Newborn calves	9	Calculated	Webster, 1974
One-month-old calves	0	Calculated	Webster, 1974
Fat stock, 0.8 kg gain/day	-36	Calculated	Webster, 1974
Fat stock, 1.5 kg gain/day	-36	Calculated	Webster, 1974
Beef cow, maintenance	-21	Calculated	Webster, 1974
Dairy cow, dry and pregnant	-14	Calculated	Webster, 1974
Dairy cow, 2 gal/day	-24	Calculated	Webster, 1974
Dairy cow, 8 gal/day	-40	Calculated	Webster, 1974
Beef cow,			
Summer coat, maintenance	15	Performance	Ames, 1980
Autumn coat, maintenance	7	Performance	Ames, 1980
Winter coat, maintenance	0	Performance	Ames, 1980
Heavy winter coat, maintenance	-7	Performance	Ames, 1980

TABLE 2.2

Wind chill table for cattle with haircoat

Wind Speed (mph)	Dry Bulb Temperature (°C)							
	-15	-10	-5	0	5	10	15	20
Calm	-15	-10	-5	0	5	10	15	20
5	-17	-13	-8	-3	2	7	12	17
10	-21	-16	-11	-6	-1	4	9	14
15	-24	-19	-14	-9	-4	1	6	11
20	-26	-21	-17	-12	-7	-2	3	8
25	-30	-25	-20	-15	-10	-5	0	5
30	-35	-30	-25	-20	-15	-10	-5	0
35	-43	-38	-33	-28	-23	-18	-13	-8
40	-53	-48	-43	-38	-33	-28	-23	-18

Source: Ames and Insley, 1975

developed specifically for cattle (Table 2.2) shows the combinedeffect of ambient temperature and air speed on equivalent temperature in terms of dry-bult temperature. Under cold outdoor conditions there is a high correlation between ambient temperature and wind chill or dewpoint (Milligan and Christison, 1974). Precipitation which wets the haircoat of cattle causes a lowering of insulatory value. In the case of heavy rain, water accumulates in the animals' haircoat, thereby displacing still air. This reduction in external insulation increases rate of heat loss and therefore lowers effective temperature. Solar radiation may change calculated lower critical temperature as much as 30°C when one compares animals exposed to direct solar radiation to those not so exposed.

2.1.2   Reproduction and lactation

There are no well-controlled studies of the effects of cold on reproduction and virtually none with respect to lactation which meet present-day criteria of statistical evaluation. Many past trials were based on comparisons of barns where the confinement systems, feeding programmes, or genetic potential of two herds differed. Other trials were based on few animals or results from a single season with no control animals in the design. These reservations must be considered when interpreting this section.

Seasonal effects on milk output have been documented. The standard values for the record of performance for milk production by dairy cattle (Agriculture Canada, 1974) indicate that cows which give birth in January have an 8 to 10% greater output per lactation than those which give birth in July. McDowell, et al. (1976) have shown that the major difference in output between cows calving in different seasons occurs during the first 60 days of lactation, and that climate is more important than feed intake during the first 30 days.

Milk production declines over the course of lactation and there is a general increase in the percentages of fat and non-fat solids (Bath, et al., 1978). Evidence concerning the influence of cold milk fat percentage is inconsistent. At low ambient temperatures, an increase in milk fat and non-fat solids percentages has been reported (Ragsdale, et al., 1950; Cobble and Herman, 1951; Johnson, 1965) but others have found no changes in milk fat percentage or non-fat solids (MacDonald and Bell, 1958c; Williams and Bell, 1964). Production level, feed intake, and feed type play important roles in affecting the composition of milk (Bath, et al., 1978) and such factors in turn are directly affected by cold. Changes in composition with changing temperature are probably of secondary origin.

To avoid the confusion of concurrent changes in yield and composition, results may be given as fat-corrected milk where the yield is reported in terms of an equivalent amount of milk containing 4% milk fat. In discussing milk yield, the distinction between yield and fat-corrected yield should be kept in mind.

Records from animals that have not had an opportunity to adapt to cold suggest that ambient temperature in the range of 0 to 10°C has a transient depressing effect on milk output (Rako and Dumanovsky, 1956). In Dutch studies (Oosterlee, 1959), effects of cold were present only for the first few days of exposure. Similarly, when cows in a climatic laboratory were exposed to an abrupt decrease in temperature from 10 to -16°C, the effect on milk production was much more pronounced than when temperature was

lowered from 10 to −13°C over an eight-week period (Ragsdale, et al., 1949). Noeffect of relative humidity in the range of 65 to 85% was found on milk production (Ragsdale, et al., 1953) or on the rate of vaporization from the skin (Kibler and Brody, 1953) at ambient temperatures of 4 or −9°C.

Under farm conditions, cows are continuously exposed to the changing seasons and thus have an opportunity to adapt as winter progresses. Summaries of the results of several trials are presented in Table 2.3 and are discussed in more detail below.

In reviewing the older literature from USA concerning lactation and cold, Dice (1940) drew on information from Maryland, Pennsylvania, and Wisconsin which indicated that winter temperatures had little effect on fat-corrected milk yield. Dice's results collected over four winters in North Dakota showed that dairy cows on an adequate ration, with good shelter and bedding, produced the same amount of fat-corrected milk in an open shed as in a conventional stanchion barn (Table 2.3). A nine-year study in Wisconsin (USA) (Witzel and Hiezer, 1946; Heizer, et al., 1953) compared a conventional stanchion barn with a well-bedded, uninsulated loose housing shed. An insulated loose housing shed was also included for two years of the trial. For the winter months, fat-corrected milk production was not affected by barn type or ambient temperature (Table 2.3) (Heizer, et al., 1953).

The most useful data on cold weather effects on dairy cattle come from Saskatchewan (Canada) (MacDonald and Bell, 1958a,b,c; Williams and Bell, 1964). Cows were stanchioned in a well-bedded free stall barn over the winter months. Daily ambient conditions were related by regression equations to daily water intake, feed intake, mild yield, and milk composition. The milk yield data initially were adjusted for the anticipated decline resulting from advancing lactation. No effect of temperature on daily yield of fat-corrected milk (Table 2.3) was found in either of the years studied (MacDonald and Bell, 1958c; Williams and Bell, 1964). In one year there was a statistically significant effect of ambient temperature on non-corrected milk yield. This was equivalent to a decline in output from 21.6 to 20.7 kg/day at mean daily ambient temperatures of 10 and −18°C, respectively (slope = 0.032 Kg/°C; r^2 = 0.05). The authors (MacDonald and Bell, 1958c) concluded that the response of milk yield to cold could be represented best by two regression lines which indicated that mean daily ambient temperature had no effect on non-corrected milk yield between 10 and −4°C but that below −4°C a decrease in ambient temperature caused a more pronounced fall in daily yield (slope = 0.052 kg/°C; r^2 = 0.04). The coefficients of determination (r^2) of these slopes emphasize that variations in daily average temperature could explain a maximum of 5% of the variation in milk yield. After reviewing the literature for warm and cold environmental temperatures, McDowell, et al. (1976) found that under field conditions the variance in milk yield associated with climate ranged from 3 to 10%.

A number of experiments carried out in the Missouri Climatic Laboratory with lactating daily cattle at low temperatures have been summarized by Yeck and Stewart (1959) who concluded that milk production by Holstein cows remained normal when temperatures were reduced from 10 to −12°C over an eight-week period. For Jersey cows, milk production began to decline at environmental temperatures below 4°C and was depressed by 7% at −7°C and by 20% at −12°C. Changes in fat-corrected milk production over the course of the experiments were extremely erratic (Ragsdale, et al., 1950; 1953) and the data reported in Table 2.3 are for non-corrected milk yield. Exposing Holstein

TABLE 2.3

Summary of relative effects of cold environments on the productivity of Holstein dairy cows

Lactation	Mean outdoor January temperature (°C)	Housing	Housing temp. (°C)	Fat corrected milk Production	Digestible energy intake	Gross efficiency of milk production	Author
North Dakota	-13	Stanchion barn.	11[a]	100	100	100	Dice, 1940
		Uninsulated shed, Loose housing	-2[a]	100	109[b]	104[b]	
Wisconsin	-7	Stanchion barn.	12[c]	100	100	100	Heizer et al., 1953.
		Uninsulated shed. Loose housing.	2[c]	101	104[b]	100[b]	
		Insulated shed, Loose housing.	10[c]	99	100	–	
Saskatchewan	-18	Uninsulates shed, stanchions.	7[d]	100	100	100	MacDonald and Bell, 1958b, c.
		Uninsulated shed, stanchions.	-18[d]	100	113	90	
Missouri	-1	Climatic lab, stanchions.	10	100[e]	100	100	Johnson, 1965
		Climatic lab, stanchions.	-13	93[e]	100	86	

Source: Bryson and Hare (1974)

a   Overall means
b   Animals gained weight thus affecting the efficiency of milk production
c   Weighted 9-year average (2 years for insulated shed)
d   Extremes of the temperature range
e   Data are not for fat-corrected milk

cows to temperatures which fluctuated daily from -12 to 4°C (Brody, et al., 1955) resulted in the same milk yield as an exposure to a diurnal fluctuation of 4 to 21°C. Yeck and Stewart (1959) noted that in the Missouri experiments which they summarized, the data were obtained from few animals and there was great variation among individuals, particularly for the Jersey cows. They recommended that the experiments should be repeated before the data could be used with confidence. This warning is repeated because the summary by Yeck and Stewart is used as the basis of such standard works as the ASHRAE (1984) Handbook of Fundamentals.

It appears clear from this review of the literature that transient or longer term low environmental temperatures have little or no direct effect on the milk production of Holstein cows allowed access to additional feed at the cold temperature extremes in latitudes where cows are normally found. If the increase in milk fat percentage (whether from reduced milk yield, increased hay intake, or other indirect causes) at lower temperature, is taken into account, then virtually no effect can be demonstrated on fat-corrected milk production. An overall estimate of the effect of cold on milk production is that for every Celsius degree that ambient temperature falls below 5°C, the daily output of milk may be reduced by 0.025 kg. In an uninsulated barn in Saskatchewan such an effect would reduce the total winter milk production by approximately 30 kg.

TABLE 2.4

Predicted performance of a 400 kg finishing steer exposed to different magnitudes of cold[1] (Ames, 1980)

Degrees of cold (C)	Dry matter intake (kg)	NEm (Mcal)	NEg (Mcal)	Gain (kg)	F/G
0	10.3	6.9	6.3	1.25	8.2
5	10.6	8.0	5.9	1.19	8.9
10	10.8	9.1	5.5	1.11	9.7
15	11.0	10.2	5.0	1.03	10.7
20	11.1	11.3	4.9	.94	11.8
25	11.1	12.4	4.2	.81	13.7

Source: Ames, 1980

[1]   This table was prepared using the feed values of $NEm = 1.7$ Mcal kg^{-1} and $NEg = 1.1$ Mcal kg^{-1} where feed net energy (NE) is net quantity of feed energy useful to an animal; NEm = portion of the net energy value of feed used to maintain essential body functions; NEg = portion of the net energy value of feed allocated to growth.

2.1.3   Neonatal survival and growth

In the cold-exposed newborn calf, non-shivering thermogenesis by metabolism of brown adipose tissue allows increased metabolic heat production

(Alexander, et al., 1975). This ability is thought to be lost in the first week of postnatal life. However, calves adapt rather quickly to cold temperatures and by one month of age, a high degree of survivability is exhibited at an average daily temperature of 0°C provided they have a dry, draught-free shelter. Unheated, bedded calf hutches that permit free access to small outside runs have been demonstrated to provide an excellent, healthful environment for two-month old calves in cold climates such as Minnesota (Anderson and Bates, 1982). Mild cold stress has only a major effect on rate of growth because increased intake compensates for increased maintenance requirement. However, during extremely cold weather, physical limits of intake can be reached while maintenance needs continue to increase. The result is lowered rate of performance. The effect of cold on efficiency of performance is noticeable with the beginning of cold stress. More feed is consumed without increased rate of performance, so that efficiency is reduced. Table 2.4 illustrates the typical rate and efficiency response of cattle to cold in terms of gain and efficiency. Increased insulation or environmental modification (i.e., windbreaks, sheds, etc.) reduce or eliminate the effects of cold on rate and efficiency of performance.

2.1.4   Acclimatization, chronic and acute exposures and reactions

Cattle respond to cold temperature by seeking protection from wind, reducing surface area by changing body posture, huddling, and other attempts to reduce rate of heat loss. At first these may be subtle changes that become more pronounced as cold stress becomes more severe. Physiological responses begin with vasoconstriction, particularly in the extremities (Whittow, 1962). This reduces rate of sensible heat loss by lowering skin temperature and increasing tissue insulation. When these responses fail to maintain energy balance, the animal has by definition reached its lower critical temperature. During prolonged exposure to severe cold, vasoconstriction may be so complete that blood flow does not reach surface tissues. In this situation, cold induced vasodilation is logically valuable in preventing necrosis of tissue during cold exposure.

To ensure constant body temperature, cattle must increase heat production when exposed to cold stress. Cattle have the ability to do this in several ways. Shivering is observed in cattle and can be an important method of increasing heat production, particularly in cases of acute cold exposure. An additional avenue of heat production that is effective for cattle and other ruminants compared with monogastrics is heat produced by digestive processes. The fact that cattle may consume relatively large quantities of food (both concentrates and roughages) make this source of energey uniquely important to cattle.

Feed intake of ad libitum fed cattle is responsive to cold temperatures. Below the LCT, feed intake increases to meet the need for increased heat production necessary for maintenance of homeothermy. At temperatures which are cool, but still above the LCT, increased feed intake may represent an adjustment of feed intake to compensate for the declining digestibility of the feed (Young and Degen, 1981). Christopherson (1976), who conducted extensive studies of digestibility, had a 0.31%, 0.21% and 0.08% decline/°C cold stress for sheep, calves, and steers, respectively. Kennedy and Milligan (1978) suggest that effects of cold in digestibility is greater with higher levels of food intake.

During severe cold, cattle may reduce level of intake because of difficulty in obtaining food due to snow cover or ice or because products such as silage or other high moisture feedstuffs may be frozen (NRC, 1981). An additional energy need for cattle consuming frozen feeds (or cold water) is the heat required to warm food or water to body temperature. Blaxter (1967) calculated that, when consuming high dry-matter diets, heat of warming amounted to less than 4% of daily heat loss, but for high moisture feedstuffs it could be as high as 14%. Young, et al. (1979) predicted that for cows, consuming only snow as a source of water, heat of warming may represent 15 to 20% of energy intake.

In the dairy cow, short-term effects of cold on milk synthesis may be mediated by hormones or by other factors. It has been suggested (Robertshaw, 1981) that prolactin could be one hormone involved but it has not been studied in lactating animals at low temperatures. Under cold conditions the blood supply to the udder may be reduced (Holmes, 1971) but a short-term, mild cold stress which decreased milk output did not affect mammary blood flow (Thompson and Thomson, 1977). Under more severe cold conditions mammary blood flow was reduced on the first day of cold exposure but was not significantly depressed on the second day. These short-term responses, and the small long-term effect of cold on milk yield suggest that the mammary system, in conjunction with the body's other systems, is capable of adapting to cold conditions. In farming practice, the effective environment to which the udder is exposed (e.g., warm, dry bedding vs. wet, hard, cold concrete) may be of more importance than ambient temperature.

The effect of cold exposure on cell-mediated immune defences is relatively unknown. Cell-mediated immunity was enhanced in calves exposed to severe cold (-15°C) for 12 hours, with suppression occurring after two weeks (Kelley, et al., 1982). The change in cell-mediated immune responses caused by cold exposure also depends on the type of immunogen. Mitogen responses were reduced by about one-third in calves after two weeks of both constant and alternating cold exposure. Even though mitogen responses were slightly reduced after one week of cold exposure, tuberculin reactions were significantly enhanced. The results suggest that cold exposure is likely to have different effects on different infectious organisms, a result that depends on the type of lymphoid cells affected by cold exposure. It is also likely that timing of the stressor, in relation to exposure to pathogenic organisms, affects development of clinical disease. The huddling of animals during cold weather can also enhance the transmission of infectious agents.

Cold acclimation of cattle is well documented and is essential to the well-being of cattle exposed to both acute and chronic cold exposure. Acclimation is associated with hormonal and metabolic functional changes that develop as a consequence of prolonged exposure. Therefore it is more associated with seasonal changes in the thermal environment than with daily or short-team weather fluctuations. Insulative acclimation is primarily a function of increased hair growth and is valuable in lowering lower critical temperature and reducing the rate of heat loss per degree of cold. Although photoperiodicity is involved in hair growth and shedding, reports indicate increased hair depth and coat density in cattle exposed to cold. Metabolic acclimation is also apparent for cattle exposed to cold. Repeated cold exposure increased the resting metabolic rate in calves (Christopherson, et al., 1979) and in mature animals (Young, 1975). Young's data suggest an increase in resting metabolic rate of approximately 1% for each degree celsius of cold. In effect, increased metabolic heat production shifts the lower

critical temperature to a lower value with the result that behavioural and physiological responses occur at a lower effective ambient temperature. Metabolic acclimation does not necessarily infer lowered energy needs because it does not lower the environmental energy demand.

Mean monthly temperatures would be an appropriate basis for evaluating the impact of metabolic acclimation. On the other hand, acute response to extreme heat or cold is an immediate response more clearly related to daily or even hourly weather fluctuations.

2.2   SHEEP - COLD STRESS

2.2.1   General observations

Northern latitude sheep breeds are generally well adapted to their cold climates and many survive productively under nutritional conditions which would be impossible for cattle. This is probably best characterized by the hill breed ewe on her own hill or mountainside, successfully raising a lamb. Like all livestock they are however affected by extreme cold, by sudden changes in temperature, and by the synergistic effects of cold, rain, and wind.

Certain diseases, pregnancy toxaemia, hypomagnesaemia, and swayback, are associated with cold weather. Pasteurellosis can occur in epizootic proportions when sheep are driven in cold weather; it is associated with a sudden reduction of the normal resistance mechanisms of the body, such as the macrophage efficiency, the ciliary clearing, the mucous blanket in the bronchioles, and sometimes is coincident with viral infections.

Pregnancy toxaemia usually occurs in ewes carrying twin lambs in flocks at pasture all year round and on limited grazing during the winter labm-bearing months. The disease is caused by a shortage of readily available dietary energy when demands are high. The amount of serum fatty acids is an index of the rate of mobilization of body fat and indicates the extent to which the energy demand exceeds that available from the current nutrient intake; these demands can be severe in very cold weather when grazing is absent or covered by snow. Acclimatized sheep survive by mobilizing fewer free fatty acids than non-acclimatized sheep. Serious losses can be produced by two sets of circumstances: (1) when ewes suffer a prolonged and progressive undernourishment during preganancy; (2) when well-nourished ewes face a sudden and almost complete lack of food, such as during a severe snow storm. Twin-bearing ewes normally have a mild anorexia in late pregnancy, possibly due to abdominal compression as well as the depressing effects of the increasing volumes of oestrogens produced at this time. It is possible to forecast the incidence using winter rainfall and temperature data (Smith, 1970).

Swayback, a congenital condition of lambs, is associated with a progressive copper deficiency in the lamb and its ewe. It is concomitant with a wide variety of soils, some with higher than average molybdenum content. The annual incidence is very variable and the condition is more common after a mild winter than one with many days of snow cover. During a hard winter sheep will normally receive supplementary feed, containing more utilizable copper than is usually available from natural grazing. Also, severe winters limit the involuntary ingestion of soil and herbage molybdenum. The British Ministry of Agriculture has been successfully issuing swayback forecasts for many years based on Smith (1970).

Hypomagnesaemia takes two forms: "spring" and "autumn and winter" grass tetany; the former caused by a mineral imbalance in the herbage and the latter from under-feeding. Ewes with twin lambs are especially vulnerable as are older ewes, that have significally less serum magnesium than young ewes (1.62 mg/100ml vs 2.26 mg/100ml). The period of highest risk is from just before lambing to six weeks after, with the highest incidence just after lambing when lactation is at its maximum. Spring grass tetany occurs when, after a period of cold weather, there is a sudden rise in temperature triggering the growth of young grass; the greater the temperature change, the more marked is the effect of the temperature rise. This spring grass can have 15% or less dry matter, which is itself a component of under-feeding and will accentuate other hypomagnesaemic effects. It results in an excessive production of rumenal acid which reduces the absorption of magnesium. The temperature rise is associated with an increase of potassium in the grazing. This magnesium antagonist will upset the K:Mg ratio and set off a clinical attack of the disease, usually about five warm days after the temperature rise. In autumn and winter grass tetany, the seasonal diet of poor quality and quantity with little or no magnesium slowly depletes the animal of its mobilizable reserves of magnesium; occasionally accompanied by hypocalcaemia at subzero temperatures. It is exacerbated by any digestive upset with anorexia. The colder seasonal temperatures increase thyroid activity and the need for magensium; thyroxine itself has an antagonistic effect on magnesium. Normal levels can only be maintained through proper nutrition and mineral supplementation. The major factors in this form of hypomagnesaemia are acute and chronic lack of feed and sudden and prolonged cold. The following weather can be tetanogenic: sudden temperature drops to 0°C, showers with cold winds, and snowfalls preventing grazing.

Winds can chill adult sheep by opening the fleece and destroying the insulating microclimate, also by driving cold water into the fleece. There are differences in ability to withstand climatic stress based on fleece type. A flock will respond to winter winds by actively seeking shelter and all sheep look for shelter when the temperature drops below freezing. Some protection can be provided by geomorphic shelter near the ground and by windbreaks. Strong winds will drive sheep before them to seek impermeable windbreaks.

Management plays a large part in the minimization or maximization of cold-related problems. For example, housing sheep with the intention of minimizing the effect of cold can result in many other problems unless the shepherd is competent. Housed sheep can suffer from pneumonia due to faulty building ventilation, acidosis due to boredom and overeating, rumenitis from low roughage intake, and a range of other georgogenic conditions. Because of the increased stocking density and shared air space, there is an increased risk of a number of infectious diseases, includng jaagsiekte. However, many of the disease problems of housed sheep in winter are more potential than real and can be prevented by good management (Hugh-Jones, 1976).

The removal by shearing or fleece-shedding of up to 6 calories of insulation from a sheep in winter instantly subjects it to thermal strain. There are three major forms of insulation in a sheep: sub-cutaneous fat assists the reduction of heat flow, on some skin areas essentially the legs, ears and face there is vasoconstriction (but there is no effective vasoconstriction in the wool-covered areas) therefore when the wool has been removed the heat drains from the trunk into the environment. The third form of insulation is the fleece, which when dry, is effective but loses its insulation value when wet. The thermoneutral point of a sheep without wool is

25° to 27°C and the balance point drops with the amount of fleece. In full
fleece a sheep can be in thermal balance at -30°C in still air, but in wind
and rain this can rise to +6°C.

When sheep are shorn in winter, the consequences on the metabolism
and water economy are a function of previous nutritional state. Fluid intake
is proportional to dry matter intake; chilled newly-shorn animals eat less
than they did immediately before shearing. The more food eaten the more water
is turned over and the more water retained in the body spaces. As fat
reserves are used up in poorly fed sheep they are in part replaced by
increased water in extracellular spaces. Undernourished sheep increase the
volume of fluid in cell and gut space as well as in extracellular volume, an
adjustment than can conceal real losses of tissue weight. Shearing during
cold weather is a way of inducing acute starvation since metabolic rates may
rise to three or four times above normal. Shorn sheep on poor, overgrazed
pasture, can be burning over 18,000 KJ per day compared to a normal 6,000 KJ.
With this high energy turnover and the inadequate food intake, a starvation
type of water distribution occurs. In practical terms the effects are not
immediately evident as the undernourished sheep appear normal and do not seem
to have lost weight. However, the extravascular volume of the animal will
have increased and as it has compensated for the lack of dry matter in its
feed by taking in water, twice as much fluid as normal will be found in its
rumen. (Macfarlane, 1976).

Deaths after shearing are usually negligible, but significant
losses from 2% to 8%, or even 36%, have occurred after wind and rain on the
newly shorn sheep. The situation is made more precarious if the sheep are
underweight or if there has been a loss of condition prior to shearing. Half
the deaths will occur in the week immediately following shearing and will be
ascribed to "pneumonia" but the actual cause may be either hypothermia and/or
an endocrine crisis precipitated by the cold exposure. The sheep's critical
temperature is affected by the fleece length, nutritional status, and body
size. Close-clipped sheep on a high level of nutrition can have a critical
temperature of 24°C while on a sub-maintenance diet it is 38°C. On a
maintenance diet and a fleece length of 0.1 cm the critical temperature is
32°C, 2.5 cm and 13°C, and 12 cm and 0°C (Armstrong, et al., 1969). The
effect of chilling on some sheep is to decrease their feed intake, which will
affect the summit metabolism. Losses can be prevented either by leaving about
3 cm of fleece on the sheep, or by providing sheared sheep with plastic coats
(which are only economically worthwhile if the expected losses are over
3%-6%), or by patrolling the flock and placing any "downer" sheep, head and
all, overnight in an empty feed-sack to recover body temperature. Rapid
acclimatization during the following weeks enables the animal to maintain its
body temperature and regain its normal feed intake.

Sheep appear to be more susceptible to excess heat than cold
during spermatogenesis, ovulation, and conception. In the field a significant
drop in conception rates has been noted following severe drops in air
temperature (27°C to 16°C) followed by rain, which affect both libido and
fertility (Davis, 1973; Webster, 1939). For the ewe on the hill with her
normal undernourishment, single lambs can be 10% underweight and twins 25%
under their expected birthweight. The same effect can follow prolonged
underfeeding, such as can be expected if the ewe has to provide enough for her
chilled self as well as growing foetus. If the ewe is properly fed after
lambing there should be no meaningful reduction in her lactation but small
lambs would be unable to fully exploit her potential, and the cold may have

affected her udder development. A malnourished foetus, if it survives, would
be born with a small body-weight and therefore a relatively large body surface
area, a reduced birth-coat density with low thermal insulation, and poor
vigour. The lack of vigour can cause the lamb to fail to trigger the
appropriate maternal responses. All of which increases the risk of chilling
and fatal hypothermia in the newborn lamb. This is discussed in more detail
in the next section.

2.2.2   Neonatal survival

2.2.2.1   Lamb mortality

          In many parts of the world, the newborn lamb is confronted with a
hostile environment from the moment of birth. Normal management systems in
the major sheep-rearing countries vary from indoor lambing in mainland Europe
and the USSR to outdoor lambing in Australia, New Zealand, Britain, and parts
of USA. In the latter countries, neonatal losses can be severe and
weather-dependent. The annual mortality of young lambs in Britain has been
conservatively estimated at 1-4 million (Wiener, et al., 1973) and in severe
winters can be much higher (Blaxter, 1964; Burroughs, 1978). Pre-weaning
mortality can range from 6%-20% and to 45% in special circumstances (Samson,
982); Houston and Maddox, 1974; Purser and Young, 1964; Johnston, 1977;
Wilson, 1954). In New Zealand, mortality rates by breed vary from 17% for
Perendale lambs to 40% in Merinos, with seasonal effects causing the overall
mortality to vary from 14% to 34% in different years (Dalton, et al., 1980)
against a normal background of 18% (Hight and Jury, 1969). In Australia there
are many instances of high mortality: 35%-53% in Merinos in south eastern
Australia in various years (Dennis and Nairn, 1970) and 35% in Polwarth lambs
near Melbourne (McDonald, 1966). In North America similar death rates are
known and reported (Sidwell, et al., 1962).

          Where the causes of death have been analysed the two most
important factors were dystokia and starvation/exposure (Wiener, et al., 1982)
with the latter accounting for 50%-60% of all deaths not ascribed to
stillbirths and dystokia. Under-nutrition and exposure are synergistic and
their relative contributions vary with circumstances. On Kangaroo Island in
Australia (Obst and Day, 1968) the neonatal mortality rate has varied from
15%-90% in Merinos and from 16%-54% in Corriedales according to changes in
rainfall, windspeed, and temperature. In dry weather with windspeeds below 2
m/sec the postnatal mortality was 5%-10% but up to 70% with rain and winds
exceeding 5 m/sec (Obst and Ellis, 1977). In Britain short-term variations in
neonatal mortality of 2%-10% in long-coated Welsh Mountain lambs and 7%-42% in
similar but short-coated lambs were noted with severe weather (Purser, 1967).
In parts of Argentina perinatal lamb losses of 41% were a direct result of
severe weather, which indirectly contributed to a further 19% loss (Olaechea,
et al., 1981).

2.2.2.2   Physiological and environmental factors

          The newborn lamb is abruptly transferred from a controlled uterine
environment of about 39°C to a hostile external world, where the effective
ambient temperature may be close to 0°C. The lower critical temperature of
newborn lambs is high for the first 24 hours (see Table 2.5) so that lambs in
almost all climates are subject to some cold stress at birth.

TABLE 2.5

Critical temperatures of newborn lambs

Age	Birthweight	
	4 g	6 Kg
1 - 6 hours	37°C	32°C
14 - 20 hours	33°C	26°C
30 - 34 hours	31°C	22°C

Source: Mercer, 1974

       Using a climate chamber, Alexander (1974a) has defined the environmental temperatures and windspeeds capable of producing hypothermia in newborn Merino lambs with different birthweights and coat types (see Table 2.6). These results amply demonstrate the effectiveness of wind and wetness in destroying insulation and increasing the lamb's vulnerability, as well as the remarkable resistance of dry heavy lambs.

TABLE 2.6

Environmental temperatures (°C) at which the peak metabolism was
just capable of maintaining normal body temperature in newborn lambs

Coat Type	Birthweight and Conditions					
	2 Kg			5 Kg		
	Still Air	Wind	Wind Wet Lamb	Still Air	Wind	Wind Wet Lamb
Long	−56	−22	+13	−105	−54	−19
Short	−32	− 4	+23	− 69	−25	+ 4

Source: Alexander, 1974a

Slee (1978) has also measured the effects of birthcoat type in relation to body cooling in climate chambers. The effects of birthweight and coat were clear, but his results (Table 2.7) also show the importance of genetic variations.

TABLE 2.7

Resistance to body cooling expressed as the number of minutes
exposed to -20°C and a 1.8 m/sec wind needed to produce
a 1°C decline in rectal temperature.

	Breed Types				
	Welsh clipped	Welsh short coated	Welsh long coated	Merino	Blackface
Number of Lambs	5	10	10	10	10
Mean Birthweight (Kg)	2.8	2.8	2.8	3.8	3.9
Mean Resistance	19 min	57 min	245 min	22 min	267 min
Mean Resistance adjusted for birthweight	22 min	66 min	281 min	22 min	263 min

In the field, birthweight is associated with survival (Purser and Young, 1964). Smaller than average lambs may have poor birthcoats and do have a higher ratio of surface area to mass (Alexander, 1974b), which is a disadvantage as heat production is directly related to mass and heat loss to surface area. However, Eales and Small (1980) suggested that the metabolic rate could be higher in small lambs. Excessively large lambs may suffer from the effects of dystokia and those surviving may have central nervous system lesions, which might affect thermoregulation (Haughey, 1980). Lambs with average birthweights possess adequate energy reserves and a satisfactory mass/surface area ratio. The U-shaped curve describing birthweight and mortality is slightly skewed so that the optimum birthweight for survival is slightly above the average weight (Alexander, 1974b; Hight and Jury, 1969). Twins and triplets are therefore generally less viable than single lambs.

The prenatal nutrition of the ewe and the adequacy of energy reserves in the neonate are also important determinants of survival. The newborn's energy reserves may vary from 100-200 Kcals/Kg depending on the ewe's nutrition during pregnancy (Alexander, 1964). These reserves last for 6-18 hours even during severe cold exposure requiring heat production of four to five times more than the basal level. However, these reserves are finite and the lamb must be eventually suckle to survive. A vicious circle can be

set up by the mutually reinforcing effects of cold and starvation. Lambs which do not die quickly from acute hypothermia can become hypoglycaemic and die slowly as their energy stores are depleted (Eales, et al., 1982a). Sublethal hypothermia can induce immobility (Slee, 1979) compounded by a reduced suckling drive (Alexander and Williams, 1966).

For details on the physiological responses of the lamb to cold and the importance of the birthcoat, readers are referred to Alexander (1962, 1964, 1974a), Alexander and Bell (1975 (a) and (b)), Alexander and Peterson (1961), Houston and Maddox (1974), and McCutcheon (1982). They have investigated the effects of birthweight, birthcoat type, and nutritional status, along with heat production, thyroid and adrenal activity, cardic efficiency, acclimation, and thermogenesis of brown fat. Cold exposure combined with under-nutrition is the most common cause of death for lambs born outside and surviving birth.

2.2.2.3   Methods of reducing lamb mortality

The problem of survival for the newborn lamb depends upon ensuing that heat production equals heat loss, and that the necessary level of heat production is sustainable from the available energy reserves until establishment of the maternal bond secures the necessary continous supply of milk. Effective action can be taken in four general management areas to reduce the lethal impact of cold exposure.

(1)   Genetic improvement. Is cumulative, usually permanent, and does not require capital or recurrent expenditure as does the improvement of shelter or nutrition. It can be obtained through crossbreeding, the choice of cold-resistant breeds, or within-breed selection. Selection tests for cold resistance have been developed using cold-chambers (Slee, 1978), or a wind tunnel and artificial rain (Slee, et al., 1979), or a water bath (Slee, et al., 1980). The latter shows particular promise as it is quick and easy to perform and has a 90% repeatability. There are significant breed differences in resistance to cold (Samson and Slee, 1981), which rank similarly against perinatal mortality and susceptibility to hypothermia for non-feral breeds in the field (Sykes, et al., 1976). Within-breed selection for cold resistance in newborn Scottish Blackface lambs shows a heritability estimate of about 30%. It may be that "cold resistance" can be regarded as the summation of all factors affecting thermoregulation. The outlook for genetic improvement in this area is therefore encouraging.

(2)   Behaviour. Mismothering, desertion, or poor maternal behaviour may accelerate death by disrupting the maternal bond and delaying or preventing suckling. Breeds differ in postnatal behaviour (Kilgour and de Langen, 1980). Slee (1981) noted that feral and mountain lambs suckled earlier and developed a maternal bond sooner than the observed lowland breeds. Desirable behavioural characteristics appear to be generally associated with the ability to resist hypothermia.

(3)   Shelter and Weather Forecasting. It has been frequently demonstrated that shelter at lambing time will reduce lamb mortality in adverse climates. However, shelter requires investment, not always possible

in low-cost enterprises, and may be impracticable in extensive systems. Recent research, largely in Australia, has centred on the evaluation of cheap windbreaks or shelter belts. A non-palatable phalaris grass hybrid has been successfully used as a windbreak. This reduced neonatal lamb mortality in single lambs from 12% to 9% and significantly reduced twin-lamb mortality from 33% to 19% (Egan, et al., 1976). Over a five-year period single lamb survival increased by 10% and by 32% for multiple lambs (Alexander, et al., 1980). The effectiveness can be further enhanced by shearing the pregnant ewes 4-6 days before lambing and encouraging them to seek shelter. (Note: the editor was shown similar results in 1960 in southern Patagonia where 20 m wide grazing strips had been cut out of the common heather-like scrub at 90° to the prevailing wind.) Meteorological forecasts of weather likely to be detrimental for newborn lambs should theoretically have high benefit:cost ratios. Using published data, Starr (1981) defined the energy demand exerted by various combinations of wind, rain, and temperature on the newborn lamb. Actual weather conditions were then related to the lamb mortality in a lowland flock. During one year weather forecasts were apparently used with some success as the basis for decisions on the housing of young lambs.

(4)   Resuscitation. When all else has failed and/or shepherds have ignored advice, some lambs will become hypothermic and will be at risk. The problem is first to identify hypothermic lambs and then to apply appropriate resuscitative measures (see Table 2.8). A lamb that appears to be immobile should be checked for a low temperature. Hypothermic lambs should be divided into two categories, those with excessive heat loss and those with subnormal heat production (Eales, et al., 1982a).

TABLE 2.8

Hypothermia in newborn lambs

Rectal Temp (°C)	Prognosis	Action
39-40	Normal	none
38-39	Very slight hypothermia	check again in 30-60 minutes
36-38	Hypothermic; some danger as suckling and mobility is impaired	Feed as necessary resuscitation necessary
30-36	Serious hypothermia	resuscitation necessary
25-30	Should survive if	resuscitation used
20-25	Almost lethal even if	resuscitation used
< 20	Survival unlikely even if	resuscitation used

Source: Slee, 1976 and Eales, 1982

Lambs under five hours old are likely to have suffered excessive heat loss and can be revived by shelter and rewarming. Those over five hours old may have suffered excessive heat loss and are likely to be also suffering from depressed heat production and hypoglycaemia. For these an

intraperitoneal injection of 20% glucose solution is effective and a feed of 100-200 ml of colostrum by stomach tube may be helpful in some cases (Eales, et al., 1982b). Resuscitation is an action of last resort as it is labour intensive, needs the sheep to be easily accessible, and runs counter to the usual Darwinian approach of naturally culling undesirable genotypes in the interests of future lower management costs.

2.3   PIGS - COLD STRESS

2.3.1   General observations

Pigs are relatively sensitive to cold when compared to cattle and sheep. As a result, a high proportion of pigs in temperate and cold climates are housed to moderate the impact of cool or cold conditions on survival, health, productivity and feed utilization.

Even in housed situations, thermal conditions can be adverse. For example, Mount (1975) developed an effective temperature approach for evaluating less-than-optimal conditions. Figure 2.1 illustrates his estimations of the impact of different situations encountered in production facilities. The first vertical bar, designated optimal, indicates that the best thermal environment for a given group of pigs is 21°C. In this optimal situation, air and wall temperatures are equal, with no draughts, a relative humidity of 50% and dry, solid, insulated floors. Departures from this optimal situation are also presented. For instance, in a slight draught of 1.5 m.sec^{-1}, air temperature must be increased to 31°C . Air temperature must be raised 2 Celsius degrees to account for poor insulation and 5 Celsius degrees if concrete slats have been used. Wet floors increase the requirement by 8 Celsius degrees. However, if straw bedding has been used, optimal air temperature could be reduced from 21°C to 17°C and still maintain the same thermal comfort for pigs. An added difficulty is that the air temperature that is required for optimal thermal comfort declines as the pig grows and consumes more feed.

What happens if air temperature is not raised to account for deficits in the thermal environment? For example, assume that young pigs are housed in a modern, well constructed nursery that is well insulated. Floors are concrete slats, but because of a poorly designed ventilation system, a 0.5 m.sec^{-1} draught is present. Concrete slats effectively reduce the pigs' thermal comfort by 5°C, and the draught causes an additional reduction of 7°C. Therefore, even though the thermometer may indicate an air temperature of 21°C, the effective air temperature that the pigs experience is 21°C -12°C, or 9°C. This suboptimal air temperature will increase feed costs, and may increase disease risks as well.

In the final analysis, swine managers should watch their pigs, not their thermometers. The pigs give the producer an estimate of effective air temperature. If pigs are piled up, huddled together, shivering or lying with all four legs beneath them, the thermal environment is inadequate and should be improved. The trade-off between cold nurseries, high mortality, reduced feed efficiency and the cost of paying the additional fuel bill to keep the nursery warm is widely appreciated. Unfortunately, direct experimental information to support these calculations is lacking.
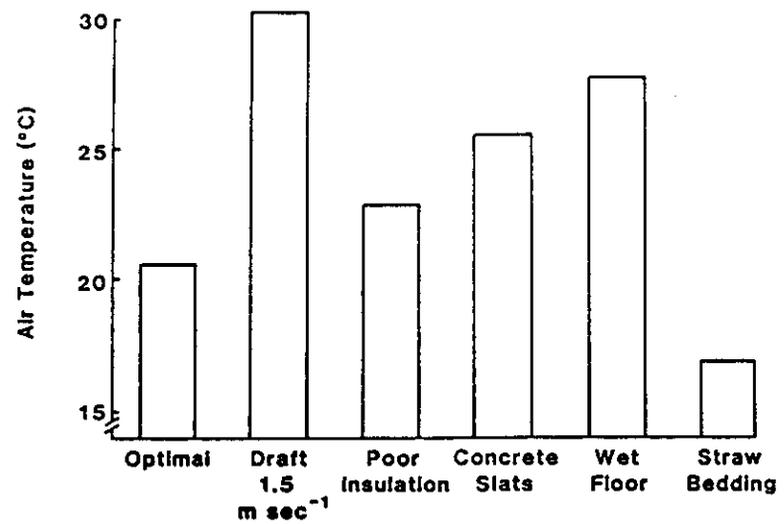
Figure 2.1 – The concept of effective environmental temperature (Mount, 1975)

2.3.2   Reproduction and lactation

For efficient reproduction pigs should be maintained under carefully controlled climatic conditions for, in comparison with other domestic mammals, their hair coat is minimal and their thermoregulatory system relatively inefficient. In practice, however, adult pigs can successfully cope with a wide range of conditions outdoors and, in most instances, their reproductive performance in cold is satisfactory. Situations do arise when reproductive failure is attributable to cold but usually these are associated with climatic extremes accentuated further by badly constructed, poorly insulated and ventilated buildings, or in outdoor systems, when inadequate shelter is provided. Alternatively, the susceptibility of the animals to reproductive problems may be increased by poor nutrition (especially low feed energy levels), parasitism, or by keeping them in individual sow stalls or crates. Further conditions that can influence the impact of climatic factors include provision of bedding, the presence of other pigs and the presence of water or general dampness.

Considerable progress has been made towards the definition, in precise physiological terms, of the optimum climatic conditions for newborn and growing pigs (see section 2.3.1). This information has been incorporated into practical recommendations for building design and management procedures for these types of pig (Ministry of Agriculture, Fisheries and Food, 1972; Sainsbury, 1976; Baxter, 1978; Baxter and Robertson, 1980). The optimum climatic conditions for adult breeding pigs have been less thoroughly studied than those for young pigs. There is still a need for precise and specific recommendations based on physiological principles. It is generally agreed that exposure to cold has little direct harmful effect on reproduction. Studies by Swierstra and Rahnefield (1972) on the reproductive performance of adult sows and boars kept out of doors in subzero winter conditions in Canada showed that over a period of eight years cold had no adverse effect on ovulation rate, conception rate, or prenatal survival in sows. Boar performance also appeared to be unaffected by cold; in fact, records seemed to indicate that fertility of boars was inversely related to environmental temperature (i.e., better in winter than in summer). Fahmy (1975) also reported that extremely cold environmental conditions had little adverse effect on reproductive performance in adult pigs. Adequate provision of feed and bedding must undoubtedly have enabled the pigs to acclimatize themselves

to the severe conditions, and to satisfy their increased metabolic requirements in these studies. Hacker, et al., (1973), however, showed that exposure of young prepuberal boars to excessively cold conditions, without measures to mitigate the effects of cold, led to testicular hypoplasia and delayed puberty.

In contrast to other stages of reproduction, low temperatures are particularly liable to have adverse effects at farrowing because of the immaturity of the thermoregulatory system of the newborn piglets (see sections 2.3.3 and 2.3.4). Randall and Penny (1970) observed a high "stillbirth" rate (approx. 10%) during winter months on farms in the UK where environmental control in the farrowing rooms was minimal. On other farms where the environment of the sows was well controlled at farrowing the stillbirths amounted to only 4% of the piglets born. The authors make the point that the 6% difference between the two types of housing probably reflects an increase in very early neonatal mortality rather than a true increase in stillbirths.

2.3.3   Neonatal survival and growth

Cold-exposed pigs lose heat from their bodies by radiation, convection, conduction and evaporation. In most situations, radiation and convection account for over 75% of the loss of heat from pigs. The major exception to this general rule occurs when floors are damp and cold, which causes pigs to become wet resulting in increased heat dissipation by conductive heat loss to the floor and evaporation from wet skin surfaces.

A significant proportion, perhaps as many as twenty per cent of all live-born baby pigs, may not survive until weaning. Half of these deaths occur during the first three days of life. Cold wather can triple or quadruple death of neonatal pigs without heated hover or "creep" facilities. Cold exposure also reduces growth rate and feed efficiency and increases susceptibility of young pigs to bacterial and viral diseases. Piglets that die before weaning are lighter at birth, are born later in the litter, have had longer birth intervals than their surviving littermates and have lower concentrations of colostal antibodies in their blood at 12 hours of age (Hendrix, et al., 1978). Therefore experiments have been conducted to learn whether cold exposure contributes to disease and death losses by reducing serum levels of colostral immunoglobulins (Blecha and Kelley, 1981). Piglets were removed from the dam at birth and subjected to a single or intermittent cold exposure (10°C) for two to three hours. Cold-exposed piglets had lower blood concentrations of colostrum-derived immunoglobulins than piglets held at thermoneutrality (36°C). Further tests (Kelley, et al., 1982) indicated that when newborn pigs received a constant amount of colostrum, serum levels of colostral immunoglobulins were similar among thermoneutral and cold-exposed piglets. These data demonstrated that absorption of colostral macromolecules was not reduced by cold exposure. They inferred that under natural farrowing conditions, cold exposure reduced serum levels of colostral immunoglobulins because colostrum consumption was reduced. Other observations support this conclusion (Parker, et al., 1980), and independent research by another laboratory has offered direct evidence to support this explanation: cold exposure reduced colostrum consumption by 27% (LeDividich and Noblet, 1981).

For young, newly weaned pigs, suboptimal air temperature does not normally cause death unless cold exposure is severe. However, the typically occurring transient reduction in feed intake during the first few days after weaning may deplete energy reserves necessary to combat cold exposure. During

this particularly susceptible period, the newly-weaned pig may require an increased effective environmental temperature. During the growth period from 25 to 60 kg, the animals needed about 25 g d^{-1} of feed to compensate for each degree Celsius below thermoneutrality. During the fattening period from 60 to 100 kg, this requirement was 39 g d^{-1} (Verstegen, et al., 1982). Once the pigs are eating to meet the energy needs for maintenance and growth, the need for increased effective temperature decreases, although the extra feed energy results in a reduced feed efficiency (NRC, 1981).
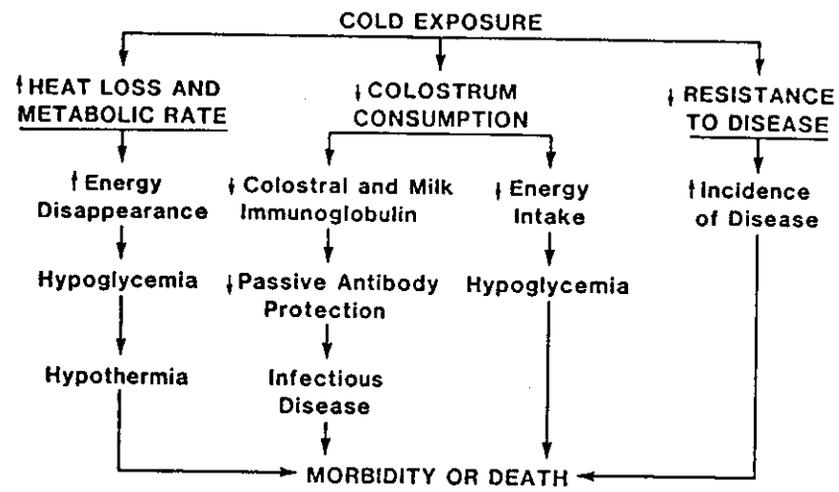

Figure 2.2 - Cold exposure and death in pigs

2.3.4   Acclimatization, chronic and acute exposure and reactions

        Several physiological mechanisms are activated to compensate for the heat loss in cold conditions. For newborn piglets, a natural reponse is for the piglets to seek warmth by lying near the sow or in a heated hover. Another response is to minimize body surface area. Huddling with littermates is an effective method to reduce surface area and one that participates in energy exchange with the environment. These types of thermoregulatory behaviours are beneficial in reducing heat lost to the environment, but they may have detrimental side-effects. If thermoregulatory behaviour becomes more critical than teat-seeking and other nursing behaviours, colostrum consumption will be reduced which will impair the acquisition of resistence to disease. Reduced consumption of colostrum in newborn pigs as a result of cold exposure also results in lowered energy intake and serum levels of colostrum-derived antibodies (Blecha and Kelley, et al., 1982; LeDividich and Noblet, 1981). Sub-optimal air temperature also increases the piglets' metabolic rate. The newborn pig has limited stores of glycogen and body fat and a defective capacity for gluconeogenesis (Mersmann, 1974). The neonate is then in double jeopardy because heat loss and metabolic rate are raised and food intake is reduced. Resistance to infectious disease is lowered and fewer colostral antibodies are available in the blood and intestine to provide protection (Kelley, 1983). This is summarized in Figure 2.2.

Cold exposure also directly affects the pigs' active immune system by causing changes in the regulatory systems that control IgM antibodies synthesis for degradation (Blecha and Kelley, 1981b). If biological function of the IgM molecules is compromised, disease could prevail when in the presence of high titres of antigen-specific antibody. High concentrations of antibodies could also exacerbate certain pathological lesions. Experiments with cold-exposed young pigs show an age-dependent pulmonary bacterial clearance (Curtis, et al., 1976); however, cold exposure has little apparent effect on bacterial clearance in older pigs.

2.4   POULTRY - COLD STRESS

Prolonged low ambient temperature is of economic significance to commercial poultry. This is partly due to the elevated lower critical temperature of chickens and turkeys and the susceptibility of neonate and juvenile birds to low ambient temperature. The lower critical temperature in the context of cold stress may be regarded as the ambient temperature below which metabolic rate increases in inverse proportion to the ambient temperature. The value of the lower critical temperature ranges from 30°C in the 10-day-old turkey poult (Nichelmann, et al., 1976) to 16°C in the adult laying hen (Arieli, et al., 1979). All Anseriformes are the most tolerant of commercial poultry to cold stress, by virtue of their plumage and subcutaneous fat (Hagan and Heath, 1976). From the standpoint of practical poultry production, lower critical temperatures under 35°C result in an increase of indigenous heat production in 50-day-old broilers (Farrell and Swain, 1977) and feed intake increases in a linear relationship as temperature declines from 30°C to 5°C (Sykes, 1977).

Adult hens are tolerant to air temperatures as low as -50°C for up to 1 hour (Giaja and Gelineo, 1933) and a lethal deep body temperature of 22°C has been established for chickens (Moreng and Shaffner, 1951).

Poultry are dependent on the integrity of the outer feather covering for protection against low ambient temperature (Wathes and Clark, 1981) and defective feathering at temperatures below 30°C results in excessive heat loss and elevated feed intake (Tauson and Svenson, 1980). Under commercial conditions, huddling occurs in neonates subjected to low temperature; for example during transport chicks may die due to suffocation (Mission, 1976).

The demand for feed energy increases by about 2% for every 1°C decrease in ambient temperature below lower critical temperature. Heat is generated from voluntary muscle activity and derived from the heat increment of feed. Involuntary shivering is a significant method of augmentting thermogenesis in poultry. Adult bantam chickens subjected to an ambient temperature of 15°C shivered continously and showed a 40% increase in metabolic rate (Aulie, 1976). Non-shivering thermogenesis occurs in poultry and is associated with increased protein catabolism and gluconeogenesis (Davison, 1973) and a decrease in liver glycogen (Palokangas, et al., 1973).

Peripheral thermoceptors occur in the tongue and pharynx of the chicken (Kitchel, et al., 1959) and may be associated with voluntary rejection of extremely cold or warm drinking water during conditions of environmental stress. Thermoceptors may also be present in the skin of birds; Rautenberg (1971) showed that cooling of the skin resulted in shivering with no change to the deep body temperature. The dominant cold signal input to the central

thermoregulatory system in poultry begins in the thermoceptors situated in the walls of veins and viscerae, and in muscle spindles. Central integration of cold-signal inputs and effector activity occurs in the hypothalmus (Freeman, 1971). Pinealectomy has been shown to cause thermoregulatory dysfunction in chickens (Cogburn, et al., 1976) and it is therefore assumed that pineal input to the hypothylamic integrating amines are involved in the response to cold stress (Hensel, 1981).

The endocrine system is intimately involved in the effector response to cold stress. Thyroid activity is inversely related to ambient temperature and it has been demonstrated that plasma T3 and T4 levels are higher during acute cold stress in quail (Bobek, et al., 1980). Plasma adrenalin and noradrenalin are higher following chronic exposure of domestic chickens to low temperatures (Lin and Sturkie, 1968). Plasma levels of prolactin are increased by chronic cold stress but the physiological significance is unknown (Ensor, 1975). Glucocon is involved in response to cold stress (Palokangas, et al., 1973) and has been demonstrated to raise the metabolic rate of chickens (Keller, 1980).

Commercial poultry are precocial and are capable of independent thermal homeostasis at the time of hatching. Turkey poults are the most susceptible to the effect of cold stress (Poczopko, 1981) and ducklings are the most resistent to the effect of low ambient temperature due to their thermal insulation and high summit metabolic rate (Untergasser and Hayward, 1972). Cold stress of relatively short duration during the immediate post-hatch period may have a deleterious effect on subsequent growth rate (Davison and Lickiss, 1979).

Cold stress has an adverse affect on the immune system. Regnier and Kelley (1981) report on the effect of cold stress on the in vivo and in vitro immune response of chickens. This finding may relate to earlier studies which showed that low brooding temperatures increased mortality in chicks infected with Salmonella pullorum (Ram and Hutt, 1955), S. typhimurium (Soerjadi, et al., 1979), and pasteurellosis (Simensen, et al., 1980). The seasonal prevalence of septicaemia associated with E. coli infection is well recognized in most broiler-producing areas of the world where it is standard procedure to increase the ambient temperature of affected flocks concurrently with the administration of antibiotics. Studies on the epizootiology of Newcastle disease have demonstrated that outbreaks are more prevalent in winter, and are exacerbated during periods of unseasonal cold weather (Brandly, et al., 1946). Low ambient temperatures are known to prolong the viability of highly pathogenic strains of avian influenza virus under field conditions.

2.5   COLD EXPOSURE AND IMMUNE FUNCTION

2.5.1   General observations

Animal scientists, veterinarians and food animal producers often associate cold, damp, draughty or fluctuating air temperature conditions with outbreaks of clinical disease. In these situations, improvement of the thermal environment often leads to an improvement in the performance of animals, less dependence on supplemental antibiotics and a reduction in the incidence and severity of certain infectious diseases. Although these observations are only empirical, they suggest an important relationship

between cold exposure and clinical disease. Current knowledge about the cause of this relationship is meagre. Several physical, behavioural, physiological and immunological phenomena are believed to be involved. This section will discuss effects of cold exposure on the immune system of domestic animals. These changes in immune function provide important insights into the relationship between stress and disease.

There are several possible reasons for the association between weather and infectious disease (Ferguson and Branagan, 1972). This relationship could be caused by: (a) direct insults (adverse weather conditions can cause frost-bite, sunburn or heat stroke); (b) pathogen host resistance (air temperature can affect survival of some airborne bacteria); (c) thermoregulatory behaviour (cold weather causes cattle to remain inside or huddle, which contributes to the transmission of infectious agents); (d) quantity and quality of food (cold, rainy, windy conditions may also discourage cattle from eating at outside feeders. Deep snowfall restricts the amount of foodstuff available to domestic animals, particularly for ruminant species); and (e) changes in host resistance (cold weather can have a direct effect upon the resistance of domestic animals to infectious disease).

2.5.2   Disease

When animals are exposed to certain pathogenic organisms, the incidence and severity of disease is often greater when the animals are maintained at sub-optimal environmental temperatures. Some of these diseases result in reduced performance of domestic animals. For example, in pigs, cold air temperature increases the incidence of diarrhoea caused by enterotoxigenic Escherichia coli (Armstrong and Cline, 1977). Cold exposure also increases the severity of lesions caused by the transmissible gastroenteritis virus (Shimizu, et al., 1978) and can reactivate latent influenza virus (Shope, 1955). In calves, alternating temperatures may (Jennings and Gloer, 1952; Hamdy, et al., 1963) or may not (Jericho and Langford, 1978) increase the severity of respiratory disease. Cold exposure also increases the susceptibility of chicks to salmonella (Soerjadi, et al., 1979) and increases the incidence of a chemically-induced skin cancer in mice (Weiss, et al., 1981). Many other examples of cold exposure effects on resistance to disease have been cited (Kelley, 1980).

2.5.3   Non-specific resistance

Non-specific (natural) resistance refers to that line of nonspecific host defence that operates before and independently of, exposure to a particular immunogen. For example, these lines of defences include local pH, sloughing of epithelial cells, ciliary action and genetic resistance. These natural barriers can compromise survival and growth of pathogenic microbes. Unfortunately, in many experiments that use common infectious agents it is not reported whether the animal had developed effective immunity to the challenging agent. In those cases, it is impossible to determine if the cold exposure is affecting only a non-specific mechanism, or whether specific immune defences are also involved.

Complement, a group of serum proteins that aid in the antibacterial action of antibody, is present in the serum of all normal animals, regardless of any previous exposure to antigen. It is possible that complement concentrations are affected by cold exposure. This question has

been investigated with newborn calves exposed to moderate cold (Woodard, et al., 1980) when complement activity was not impaired.

A very important constituent of host resistance to infectious disease is phagocytosis. Phagocytic cells are located throughout the body, either within tissues (histiocytes) or wandering in the blood stream (monocytes). Phagocytic cells include macrophages and polymorphonuclear cells. An evaluation of macrophage activity can be determined in vitro by migration inhibition from antigen-loaded capillary tubes. Macrophage inhibition was depressed in cold-exposed mice that were infected with Pasteurella hemolytic, even in the absence of antigen (Tizard and Ellicott, 1974). This disease in macrophage migration could impair the animal's capability to deal effectively with invading micro-organisms.

Phagocytic activity of reticuloendothlial cells can also be determined by the rate of removal of colloidal carbon particles from the blood. Clearance of carbon particles in rabbits exposed to only cool conditions (22°C) was lower than in rabbits held at 30°C (Donald, 1972). Similar seasonal trends were observed. If rabbits were infected with pathogenic organisms before exposure to cold, there was an increase in clearance of carbon. This finding demonstrates that infections can increase activity of the reticuloendothelial system.

Experiments with cold-exposed young pigs showed an age-dependent pulmonary bacterial clearance (Curtis, et al., 1976). Pulmonary clearance of a nonpathogenic strain of Escherichia coli was depressed in cold-exposed 2-day-old pigs. Green and Kass (1965) also reported that cold exposure reduced bacterial clearance of Staphylococcus albus and Proteus mirabilis from the lungs of mice. This reduction was attributed to impairment of alveolar macrophage activity.

Phagocytic cells are also important in limiting infections of the upper respiratory tract. Reduction in phagocytic activity in cold-stressed animals may partially account for the higher incidence of respiratory disease in domestic animals in the winter months. However, Rodbard (1981) suggested that fluctuations in air temperature may also alter mucin production, ciliary activity and other natural host defences that affect virulence and infectivity of micro-organisms. Furthermore, since macrophages participate in a wide variety of antibody and cell-medicated immune events, cold exposure may indirectly affect other components of the immune system.

2.5.4   Passive antibody immunity

Cold exposure reduces consumption of colostrum in newborn pigs (Blecha, et al., 1982; Kelley et al., 1982; LeDividich and Noblet, 1981) and lambs (Slee, 1981). This, in turn, reduces energy intake and lowers serum levels of colostrum-derived antibodies. The neonate is then in double jeopardy because heat loss and metabolic rate have been raised and food intake reduced. Resistance to infectious disease is lowered and fewer colostral antibodies are available in the blood and intestine to provide protection. This topic has been more fully discussed in paragraphs 2.3.3 and 2.3.4.

2.5.5   Active antibody immunity

Cold exposure reduces the half-life of serum immunoglobulins and increases turnover rate of IgC and IgM (Sabiston and Rose, 1976; Trapani and Campbell, 1959). Generally, cold exposure increases the capability of pigs

(Blecha and Kelley, 1981; Shimizu, et al., 1978), rabbits (Ste. Rose and Sabiston, 1971; Sabiston and Ste. Rose, 1976; Kiorpes and Yuill, 1975), chickens (Subba Rao and Glick, 1977) and mice (Sabiston, et al., 1978) to synthesize antibodies. However, if air temperature is substantially below the lower critical temperature of an animal species, a reduction in antibody synthesis will occur (Sabiston, et al., 1978; Eiseman, et al., 1964). It is also important to note that breed differences exist that relate to the effect of cold exposure on antibody synthesis; i.e., antibody responses may be affected by cold in some breeds, but not in others (Sabiston, et al., 1978; Regnier, et al., 1980). This finding supports the concept of genetic-disease-environment interactions.

It has been suggested that cold exposure somehow results in higher levels of viremia when animals are infected with live virus (Kiorpes and Yuill, 1975; Shimizu, et al., 1978). High concentration of virus in the circulation may therefore provide a greater amount of antigen to stimulate antibody synthesis, which could account for the greater antibody synthesis in cold-exposed animals. However, new research has shown that it is more likely that the immune system is affected directly (Blecha and Kelley, 1981). In experiments with young pigs, a non-replication antigen (sheep erythrocytes) was used to vaccinate pigs (Table 2.9). Sheep erythrocytes do not replicate in pigs, so confounding effects of virus multiplication were eliminated in this experiment. However, the short, four-day cold exposure still caused a 360 per cent increase in haemaglutination titres to sheep erythrocytes. Serum immunoglobulin was increased by 60 per cent. As expected, elevated antibody titres were mostly of the IgM class (2 mercaptro-ethanol sensitive). Cold exposure also increased serum levels of IgM in cold-exposed calves (Kelley, et al., 1981; Kelley, et al., 1982).

These findings indicate that cold exposure causes important changes in regulatory systems that control IgM antibody synthesis or degradation in pigs and calves. If the biological function of these molecules is compromised, disease could prevail in the presence of high titres of antigen-specific antibody. High concentrations of antibodies could also exacerbate certain pathological lesions.

TABLE 2.9

Antibody immune events in cold-exposed pigs

Immune Event	Time After 4-Day Cold Exposure[a]	
	3 days	10 days
Gamma globulin	21%	61%
Total antibody titre	146%	359%
IgM antibody titre	130%	273%

Source: Blecha and Kelley, 1981

[a]   Expressed as a percentage of values of control immunized pigs maintained at thermoneutrality.

2.5.6   Cell-mediated immunity

Cell-mediated immune defence mechanisms are important to domestic animals' resistance to infectious diseases. Unfortunately, the effect of cold exposure on cell-mediated immune defences is relatively unknown. It is important to measure cell-mediated immune events in vivo in the presence of the physiologic changes that are known to occur in cold-stressed animals. Therefore, efforts were concentrated on measuring skin-test responses of immunized animals that were cold stressed. In laboratory animals, immunity to the immunogens listed in Table 2.10 can be transferred by immune cells, but not by immune serum. Therefore, these results gave an indication of how cold exposure affected cell-mediated immunity in calves, chickens and mice.

Expression of contact sensitivity and tuberculin reactions depended on how long calves were exposed to cold. Enhancement was measured if calves were exposed to severe cold (-15°C) for 12 hours, but suppression occurred after two weeks. For mitogens, both constant and alternating cold exposure caused a consistent reduction in skin swelling, an effect which became more severe as duration of cold exposure was lengthened. It appears that plasma from cold-exposed calves reduced the capability of normal peripheral blood mononuclear cells to respond to concanavalin A in vitro (Kelley, et al., 1982b). This effect may partially explain the reduction in contact sensitivity and tuberculin reactions after one and two weeks of cold exposure.

The change in cell-mediated immune responses caused by cold exposure also depended on the type of immunogen. For instance, expression of mitogen responses does not depend on prior exposure to the immunogen. However, expression of contact sensitivity and tuberculin reactions required previous immunization with the appropriate immunogen. Mitogen responses were reduced by about one-third in calves after two weeks of both constant and alternating cold exposure. Even though mitogen responses were slightly reduced after one week of cold exposure, tuberculin reactions were significantly enhanced. Similar conclusions were reached with other species. Cold exposure caused a 27 per cent reduction in mitogen-driven skin tests in chickens, but contact sensitivity reactions increased even though cellular reactions to sheep erythrocytes were suppressed. These results suggest that cold exposure is likely to have different effects on different infectious organisms, a result which depends on the type of lymphoid cells affected by cold exposure.

It is also likely that timing of the stressor in relation to exposure to pathogenic organisms affects development of clinical disease. Some evidence exists to support this postulate. When mice were vaccinated with sheep erythrocytes and maintained in the cold for three days, a modest but significant suppression was recorded when mice were challenged at a thermoneutral temperature. However, if mice were exposed to cold during the induction phase and also challenged with sheep erythrocytes during cold exposure, a highly significant immuno-enhancement occurred. These results suggest that certain sets of regulatory cells are sensitive to environmental stressors at discrete times during their ontogeny. New results with pigs and mice also indicate that physiological concentrations of cortisol may differentially inhibit the function of different types of lymphoid cells (Kelley, et al., 1982c; Blecha, et al., 1982b).

TABLE 2.10

Cold exposure and cell-medicated immune responses measured in vivo

Species	In vivo skin tests[a]			
	Mitogen	Contact Sensitivity	Tuberculin Reactions	Sheep Erythrocytes
-15C, 1/2 day[b]	—	+31	+60	—
-5C, 7 days[c]	-7	-19	+28	—
-5C, 14 days[c]	-29	-17	-30	—
Alternating cold, 5 days[d]	-16	NO	-14	—
Alternating cold, 12 days[d]	-33	-13	-16	—
Chickens				
1C, 5 days[e]	-27	-56	—	—
Mice				
5C, 3 days, induction[f]	—	+56	—	-19
5C, 10 days, induction and expression	—	+57	—	+109

[a]   Expressed as a percentage of control immunized animals maintained at thermoneutrality.   "+" indicates an enhanced % response, "-" a suppressed % response.

[b]   (Kelley, et al., 1981a). Tuberculin averaged from 12 to 48 h; contact sensitivity averaged from 4 to 24 h.

[c]   (Kelley, et al., 1982a). Tuberculin averaged from 12 to 48 h; contact sensitivity averaged from 4 to 24 h; mitogen averaged from 4 to 48h.

[d]   (Kelley, et al., 1981b). Alternating cold = 5C for 12 h and -10C for 12 h. Tuberculin averaged from 12 to 48 h; contact sensitivity averaged from 6 to 24 h; mitogen averaged from 6 to 48 h.

[e]   (Regnier and Kelley, 1981).

[f]   (Blecha, et al., 1982a).

*

*   *

2.6   ANIMAL PARASITES IN COLD CLIMATES

2.6.1   Internal parasites and cold weather stress

Readers are encouraged to refer to the WMO Technical Note No. 159, "Weather and Parasitic Animal Diseases" which is a valuable reference text. The following will highlight some recent developments and general principles.

2.6.1.1   Cattle parasites - Introduction

In areas of the world with cold climates, there are three major internal parasites adversely affecting the productivity of cattle. These are the stomach worm, Ostertagia ostertagi, the lungworm, Dictyocaulus viviparus, and the liver fluke, Fasciola hepatica. In both roundworms, the sexes are separate and life cycles direct with a free-living phase involving development through three larval stages (L_1, L_2, and L_3), the L_3 being the infective stage. In the parasite cycle further development takes place through stages L_4 and L_5 to the mature adult. In contrast, the flatworm, F. hepatica, is a hermaphrodite that has an indirect cycle with most of the development taking place asexually outside the final host, in snails of the genus Lymnaea. From an epidemiological viewpoint, it should be realized that under favourable conditions one roundworm egg or larva would give rise to one infective larva and, if ingested, one adult worm. However, one fluke egg could eventually yield several hundred infective stages, or metacercaria, through asexual multiplication in the intermediate host.

In cattle, previous exposure to an internal parasite generally has more effect on the outcome than does the age of the animal. Therefore clinical infections with these parasites can occur in adult cattle with no previous exposure, whereas re-infection is reduced to a varying degree. Cattle exposed to lungworm infection rapidly acquire an immunity and are highly refractory to re-infection with very few of the ingested larvae developing to the adult stage. Resistance to ostertagiasis is slowly acquired and less absolute. Young cattle require a prolonged exposure before a significant immunity is built up (Armour, et al., 1979). Though it is quicker in older stock (Michel, et al., 1979), it is not uncommon to find several thousand O. ostertagi in "immune cows." The situation regarding liver flukes is similar to that of stomach worms with a slower and less absolute protection developing. While a reduction in the number of established flukes following re-infection has been reported (Doyle, 1972), there is doubt as to whether this is due to acquired immunity or to the unfavourable milieu of cirrhosis created by the previous infection; sheep do not develop this cirrhosis nor protection from subsequent infections. There is evidence that with re-infection flukes remain stored in the adult parenchyma and may subsequently migrate to the bile ducts of the developing foetus (Over, 1982).

The host/parasite relationship is disturbed around parturition giving rise to a periparturient increase in numbers of parasite eggs in the faeces (Jacobs, 1966). This occurs in calving cows with O. ostertagi infections, though the increase is less than for sheep or pigs. This increase has yet to be reported for lungworm infections. Clinical salmonellosis can be triggered off at calving by fluke-infected cows.

2.6.1.2   Seasonal considerations

In Western Europe the number of free-living stages of many helminths increases markedly during the later months of the spring-autumn grazing season, i.e., August to October, and then declines to low levels by the following spring and virtually zero by the summer (Michel, 1969). The pattern appears to be relatively constant but varies in magnitude from year to year. This applies equally to O. ostertagi and to the metacercaria of F. hepatica. The seasonal availability of lungworm larvae varies due to differences in dissemination and longevity. The increase of infective stages of O. ostertagi originates from eggs or L_1 larvae deposited by infected cattle in spring and early summer which with increasing rapidity become infective L_3 larvae by July and August; thereafter development slows with little or none after September. The same lag in development occurs with the larval stages of F. hepatica within the Lymnaea snails. Therefore, one or at most two, generations of internal parasites occur annually in cold climates and the important period of contamination is the spring or early summer. The infective larval stages are capable of surviving in reasonable numbers from the autumn to the following midsummer and thereby to infect next year's calf crop.

The migration of the infective larvae from the faecal mass to the surrounding herbage depends on the larvae being able to travel on a thin film of water. O. ostertagi L_3 larvae are completely dependent on this but the more sluggish L_3 larvae of D. viviparous are not. The lungworm larvae climb the sporangium of the fungus Pilobolus, which abounds in bovine faeces, and can be carried up to 13m on ripe spores in windy conditions. So while dry weather limits the translation and availability of infective Ostertagia larvae, it has little effect on lungworm larvae.

Very dry conditions during the summer and early autumn delays the migration of O. ostertagi L_3 larvae onto the pasture until the return of wet conditions in the late autumn. If these larvae are subjected to 4°C temperatures their later development will be affected so that instead of becoming adults, they only develop to the early fourth stage in the host and will remain at this stage for approximately four months. Then they break out of the glands in the host's stomach, almost "en masse", causing a severe outbreak of what is known as Type II ostertagiasis (Armour and Ogbourne, 1981). Cold temperature sensitivity is an inherited trait in certain strains of O. ostertagi and not all strains posses it; some strains possess a warm temperature sensitivity (Smeal, et al., 1980; Smeal and Donald, 1982). Therefore dry conditions can enhance the prevalence of arrested larval development and the risk of Type II ostertagiasis.

All lymnaead snails need water to breed and develop. Dry conditions will stop all snail activity and the multiplication of F. hepatica is terminated in aeastivating snails and at temperatures below 10°C. Both situations significantly reduce the potential level of exposure to the parasite. As long as the temperature is above 0°C wet weather will favour snail population and parasite multiplication, as well as increasing the areas suitable for snail colonization. Because of other factors, such as predators and disease, snail population growth is constrained, but infective metacercaria will be spread in large numbers over a bigger area thus increasing the probability of ruminant infection.

2.6.1.3   Climatic models of disease

An empirical analysis of 1953-1964 field data led Ollerenshaw and Smith (1969) to note that for each year the early summer helminth disease levels could be expressed as a linear function of the observed disease level and the mean monthly rainfall during August to October of the previous year. Late summer levels were a function of the observed earlier disease level and the soil moisture deficit at the end of July. In contrast, Gettinby, et al., (1979) modelled the dynamics of the Ostertagia population in response to weather conditions. Sufficient information exists from laboratory and field studies to quantify the relationships of daily temperature and rainfall levels with larval development rates (Gibson, 1980), survivorship of infective larvae (Pandey, 1972), and fecundity of adult worms (Anderson, et al., 1966). The predictive levels were found to be in good agreement with recorded weekly pasture counts. The model has now been successfully modified to deal with different parasite strains (Gettingby and Paton, 1981), that have been reviewed by Salih (1981).

A successful model for dictyocauliasis has yet to be constructed. Undoubtably the extreme complexities peculiar to the life-cycle of D. viviparus make it a formidable task.

Temperature controls the rate of development of egg and snail stages of the life-cycle of F. hepatica, as well as the survivorship of the parasite at the free-living stage. Rainfall is essential for the translation of the parasite from egg to snail and from snail to herbage. Both climatic factors regulate the growth and availability of the snail population. Because of the lengthy development period in the egg and snail stages in cool climates, the disease is absent in Iceland, the Faroes, and parts of Norway. In the United Kingdom and Ireland fascioliasis is endemic and generally follows the pattern of a single rise each year, the timing depending on the geographic location. In the wetter parts of Western Ireland and Scotland the moisture conditions are not limiting but the low temperatures slow development. Conversely, in southern England higher temperatures accelerate development and moisture conditions limit survivorship and translation.

The earlier successful predictor was Ollerenshaw and Rowlands' (1959) M_t climatic index. This was modified by Ross (1970) for the Stormant Wet-day index. A long-term average index (Ollerenshaw and Smith, 1969) can be operated successfully in the warmer climatic conditions of the Netherlands (Ollerenshaw, 1974).

Nice and Wilson (1974) were able to model the growth of the parasite in the snail as a function of temperature. Smith and Wilson (1980) have tackled the microclimate conditions of snail habitat, interrelating air temperature, soil surface temperature, and moisture. They have gone on to develop two interesting and valuable models of fascioliasis: an analytical model of fluke trasmission and the interactions resulting in equilibrium and a simulation model of the parasite-host system. Along with an admirable review of the disease, the models are described in Wilson, et al., (1982) and the mathematical components in Smith, 1982 and 1984). A series of models based on the development of the liverfluke egg and snail stages as a function of temperature, mortality, and the limiting effects of moisture were constructed to predict pasture contamination with metacercaria (Hope-Cawdery, et al., 1978). Snail predation can be modelled as a function of temperature (Hope-Cawdery, 1981). Various operational methods of forecasting parasitic disease incidences have been reviewed by Starr (under publication).

2.6.1.4   Management considerations

In cold climates many cattle and sometimes flocks of sheep are housed during the winter. The timing of that housing affects the probability of Type II ostertagiasis in the spring. Similarly if the spring turnout is delayed until after the majority of overwintered L₃ larvae have succumbed to the spring temperature rise, the incidence of ostertagiasis and parasite bronchitis (Jacobs and Fox, 1985) will be reduced. With outwintered stock, the overwintered L₃ remain the main source of pasture infection. For D. viviparus the survival of even a few larvae either in the animal or on the pasture will constitute a potential hazard since the numbers needed to produce disease are only a fraction of those necessary to precipitate ostertagiasis. The prevalence of fascioliasis can be reduced by early autumn housing of stock, which would be denied access to metacercariae during the autumn and winter. As fluke infections last for five months in untreated cattle, a delayed turnout can result in fewer flukes remaining and thus fewer eggs infecting the spring generation of snails. All these housing benefits, relating to parasites, would be undone if the manure and slurry from housed infected stock were to be spread in late winter on the fields that would be grazed during the coming summer (Nansen, et al., 1978)

2.6.1.5   Pig parasites

Internal parasite infections have a significant effect on the health and productivity of pigs. The short life cycle and fast growth rates of pigs encourages quick economic returns. Because of their gregarious nature and management crowding, they easily succumb to parasitisms. Modern management systems have increased efficiency as pig rearing has moved from the pasture and dry-lot to partial and total confinement systems which have led to a decline in the overall intensity of parasitisms, but not always in the prevalence of infection (Gaafer and Jones, 1965; Stewart and Hale, 1975). However Ames (1976) in 1,461 herds in Missouri, USA, found 8% of pastured herds parasite free, 19% in dry-lots, 25% on concrete, and over 40% on slotted-floors were uninfected. The stomach worm, Hyostrongylus rubidus, is naturally eliminated from pigs on slotted floors (Morriss, 1979). Some previously unimportant parasites, such as Coccidia and Strongyloides, have become important pathogens, particularly in neonates (Stewart and Hale, 1975) affected by Escherichia coli infections (Wade and Gaafar, 1982).

Baby pigs, young weaners and fatteners are usually more susceptible to parasitisms. While parasites are seldom a cause of obvious disease, they are a significant component of reduced weight gain and feed efficiency. Parasites with longer incubation periods and prepatent periods manifest themselves in replacement and breeding stock, for example the swine kidney worm (Stephanurus dentatus) and the nodular worm (Oesophagostomum spp).

Immunity plays an important part in parasite infections. Piglets can passively acquire immunity through maternal colostrum, but its absorption is reduced in cold stressed newborn piglets (Blecha and Kelley, 1981a). This stress enhances antibody production in weaned piglets (Blecha and Kelley, 1981b).

The development of pig parasite eggs and larvae in relation to climate and environmental conditions is largely undescribed. Cool temperatures will slow down the embryonation of eggs or even comletely arrest

development.   This development within the egg shell is an aerobic process requiring an exogenous supply of oxygen which is altered by changes in the lipid layer brought about by temperature variations.   Prolonged and significant changes from the optimum will cause irreversible alterations and cell death from lack of oxygen.   The requirements are not so critical for fully larvated eggs.   Helminths with an intermediate host have the additional influence of climate on the intermediate host as well as interactions between the intermediate host and the infesting larval parasite.

Some of the common parasitisms of pigs are reviewed in relation to cold stresss:

Balantidiosis.   Balantidium coli is cosmopolitan in distribution and found in cold regions as far north as Scandinavia and northern Russia. Svenson (1955) found different strains varied in their resistance to environmental conditions.

Coccidiosis.   Isospora suis is the most pathogenic coccidian affecting piglets (Hoefling and Todd, 1981).   An intensive production system with continous farrrowing will result in a massive build-up of oocysts which can withstand freezing for 26 days and remain infective (Avery, 1942).   Viable infective oocysts have been recovered after 15 months in soil ranging in temperature from 40°C to -45°C, which explains the prevalence of this infection in cold climates.

Toxoplasmosis.   Cats are the definitive hosts for Toxoplasma gondii and about 200 species of birds and mammals are intermediate hosts.   The disease affects young and adult pigs.   The oocysts remain infective as long as they are maintained at 80% relative humidity (Fayer, 1980).   Under natural conditions, viable oocysts were maintained for 2-12 months in soil in Costa Rica, but up to 18 months in Kansas, when soil temperatures had dropped to -20°C (Frankel, et al., 1975).

Ascariasis.   This is the most common helminth infection of pigs in the world.   It is acquired through the ingestion of infective Ascaris suum eggs.   The optimum temperature range for development of the larvae within the eggs is 30°C to 33°C.   However some development may occur at 15°C.   An 18-month study in England by Stevenson (1979) indicated that there was no development in the eggs between the winter months of November and May in pig houses, a period when the outside average temperature was 15°C.   Development in late spring and summer, May to July, was rapid when the maximum outside temperature had reached 33.6°C.   At a regional slaughterhouse it was noted that this period coincided with a high incidence of condemnation rates of pig livers with focal interstitial hepatitis, or "milk spots", due to the migration of ascarid larvae.   Seasonal retardation of the development of eggs in cool weather is only a temporary condition and the eggs resume development soon after the temperature becomes optimum.

In addition, it was noted by Potskhveriya (1979) in eastern Georgia in the USSR that the earthworm, Eisenia foetida, in pig pastures had an infection rate of 45.3% with A. suum larvae.   The larvae can overwinter in the earthworm, which burrows down into the loose soil below the frost line. These earthworms return to the surface in May the next year.

Trichuriasis. The eggs of the whipworm, Trichuris suis, are equally resistant to the environment. Hill (1957) demonstrated that in Maryland, USA pigs were infected from pastures that had been left fallow for six years. Usually larvation of T. suis eggs is completed in 18 days at 37.5°C; 22 days at 33°C (Alicata, 1935); and 102 days at 20°C (Beer and Rutter, 1972).

Burden and Hammet (1979) artifically seeded pasture plots with undeveloped T. suis eggs in late winter and early spring. The eggs did not complete larvation until the following summer, 62 to 90 weeks later. The plots remained infective for at least two years. The eggs were most abundant in the 10-20 cm layer where the soil was frozen in winter. This ability for prolonged infectivity must be considered when pig slurry is used to fertilize pastures.

Hyostrongylosis. The stomach worm, Hyostrongylus rubidus, is a parasite of pigs at pasture. With confinement systems this parasite is of minor importance. The infective larvae cannot survive freezing (Connan, 1971).

Oesophagostomiasis. The nodular worms, Oesophagostomum spp., are confined to pigs at pasture or in partial confinement. Larvae exposed to -19°C for ten days were still viable but not after 31 days (Alicata, 1935). In southern England, Rose and Small (1980a) noted that infected larvae developed only during the summer and autumn in contaminated pastures, but did not overwinter (Rose and Small, 1980b); the same has been noted in Canada (Smith, 1979). Infection persists through adult worms within the host.

Metastrongylosis. Lungworm infections from the genus Metastrongylus are acquired from infected earth-worms; several genera and species of earthworms have been shown to be vectors. The lungworm eggs are excreted and disperse with the host's faeces on the pastures where they are ingested by the earthworms. The eggs are relatively resistant if covered by soil and survive up to two years when the soil is continually moist (Rose, 1959). Larval development to the infective stage takes 16 days at 22°C to 23°C but 219 days at 10°C to 11°C. These larvae survived for two weeks at -8°C to -5°C, 62 weeks at 5°C to 6°C, and 16 weeks at 10°C to 11°C. Infective larvae were rapidly killed by desiccation.

Trichinosis. The introduction of Trichinella spiralis into a herd is usually by feeding infected meat scraps to pigs as well as through the pigs' eating of wild animals, such as rats and raccoons. While T. sprialis is not resistant to cold, T. nativa of bears and wolves has adapted to cold weather and can survive for 300 days at -15°C, as well as during repeated thawing and freezing (Smith, 1981).

Cysticercosis. The pig is the intermediate host of three important taeniid tapeworms of amn and carnivores, Echnicoccus granulosus, Taenia hydatigena, and T. solium. The latter parasite develops in the pig in the cysticercus form, while the others can develop in other mammals. Most taeniid eggs survive in moist, shady areas for weeks, but are rapidly killed by sunlight (Soulsby, 1965). In New Zealand in areas of modest rainfall, (76 mm) when the eggs were not washed deep into the soil and the cold dry weather lasted for four months, T. hydatigena and E. granulosus eggs remained viable despite repeated freezing and thawing. However the succeeding summer sunlight disinfected these same pastures (Sweatman and Williams, 1963).

2.6.2   Ectoparasites of livestock in cold climates

2.6.2.1   Introduction

        Parasitism in farm animals accounts for a serious reduction of
efficiency of livestock management and consequently causes significant loss of
food production to the livestock industry. The prevalence and severity of
many parasitisms in livestock are strongly influenced by weather and climate.
The nature of these influences on some of the commonly recognized parasites of
animals has been described in a technical note on weather and parasitic animal
diseases (Gibson, 1978).

        Despite scientific recognition of the biological implications of
weather and climate in the etiology of parasitism, the applied aspects of the
biometeorology of parasites have remained relatively undeveloped. More
definitive assessments of the development of biometeorological methods are
needed to support the environmental management and control of parasites in
livestock production systems. In this chapter, selected types of parasitism
are identified with applications of meteorological services that could
significantly improve efficiency of the practical management and control of
external parasites. Benefits from the use of meteorological services in
parasite control have special significance for livestock managment in extreme
climates. This section assesses applications of meteorological information
and services in cold environments.

2.6.2.2   Applied biometeorology in etiology of parasites

        Weather and climate are limiting factors in parasitism to the
extent that they interact with the life cylce of the parasite. Tis
interaction may involve the impact of the atmospheric and extra-terrestrial
environment directly on the parasite or indirectly through the host on the
host-parasite process. Many livestock parasites are highly specialized
organisms that have adapted to specific associations with their homeothermic
hosts. Under these conditions, the direct impact of weather and climate on
the parasitic phase of life cycles may be considerably reduced or found to
vary considerably between different parasites sharing the same physical
environment. Consequently, general interpretations of meteorological
variables for the interphasing of parasites with weather and climate in the
host environment are of limited use. They are highly impractical for purposes
of optimizing the management of livestock production systems.

        The specialized nature of host-parasite associations in livestock
production systems should not discourage practical use of meteorological
information to improve livestock management. Although there is limited
opportunity to apply general meteorological forecasts to specific pest
management, the highly specialized host-parasite association is often
vulnerable to the meteorological environment during certain stages of its life
cycle. Use of meteorological services under these conditions can be turned to
practical advantage for more efficient management and control of parasites in
animal production. At the same time, an animal's natural resistance to
parasitism is vulnerable to the detrimental influence of extreme weather
during certain seasons. Therefore, the use of meteorological services can be
effective in making the most of the preventive aspects of animal protection
procedures to obtain economic benefit from management of livestock production.

Life cycles of external parasites display phenomenal variation in the degree and, more importantly, in the nature of their dependence on the external environment (Table 2.11). Dependence of each parasite on its host and the external environment is the result of combinations of biological adaptations. For this reason, each parasite is usually a unique case study defining host-parasite-environment interactions that are fundamental to its practical economic management. Consequently, unlike other areas of agrometeorology, general meteorological services and forecasts tend to have limited application. Applications of meteorological data and services to the management of parasites are conceived frequently in terms of selective requirements for specific problems with few exceptions for the general case.

Selective requirements for the application of meteorological services and data are determined by one or more bioclimatic conditions of parasitism:

(a)   Climate influences the overall distribution and geographic range of parasitic species and, for practical purposes, weather determines local and temporary changes in their epidemiology.
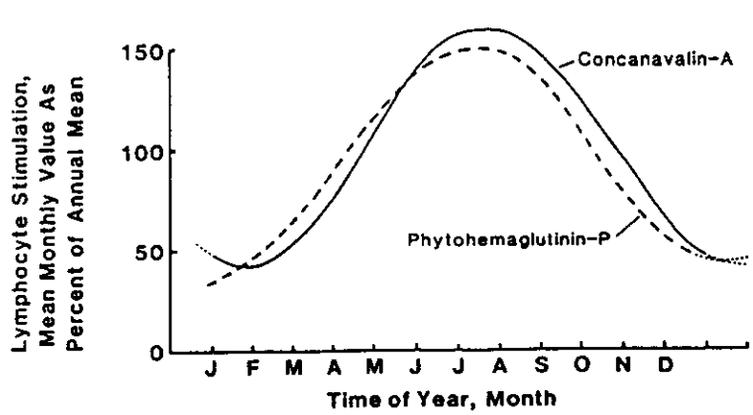
Figure 2.3 - Seasonal variation in the stimulation of lymphocyte mitogenesis as a measure of cell-mediated immunity in clinically normal dogs in an outdoor environment as a speculative relation of the underlying resistance in all homeotherms to weather and climate. (Normalized percentage of annual means fitted as an harmonic function, from Shifrine, et al., 1981)

(b)   Seasonal development of specific parasite problems or of episodes within infestation cycles are highly influenced, if not determined, by weather variations. Accurate anticipation of these events from meteorologicl projections of weather and climate can significantly reduce the magnitude of risk of infestations and also increase the effectiveness of pest management practices.

(c)   Behavioural patterns of adaptation (or tolerance) develop with age in some host animals modifying distribution of physiological adapted parasites in well established host-parasite systems. These processes may be elicited in part by certain weather factors, such as high temperature or drought, to mediate the effects of meteorological conditions on the host-parasite system through the stress syndrome and animal behaviour.

(d)   Physiological mechanisms in animals for resisting parasites, such as a the thickening of cutaneous tissues in response to excessive blood-feeding, or tissue injury by certain ectoparasites, modifies susceptibility of animals to reinfestation. These conditions, e.g., those produced by severe infections of lice or mange mites, may lead to extensive denuding of the skin. Parasitized animals affected in this way are exposed to increased stress from cold or wet weather for the maintainence of thermoregulation.

(e)   The sensitivity of the underlying immunological system of homeothermic vertebrate animals is influenced by weather and climate. The seasonal course of infestations of parasites observed on various animals confirm results of experiments on dogs (Shifrine, et al., 1980) in which the cell-mediated immune response, as measured by rate of division in lymphocytes, displays a typical pattern of seasonal variation in response to meteorological environment in a temperate climate (Fig. 5). Weather resumés of seasonal and annual variations are useful meteorological aids to the timing of therapeutic treatment of some common ectoparasites to exploit maximum reinforcement of seasonal self-cure through natural immunological processes.
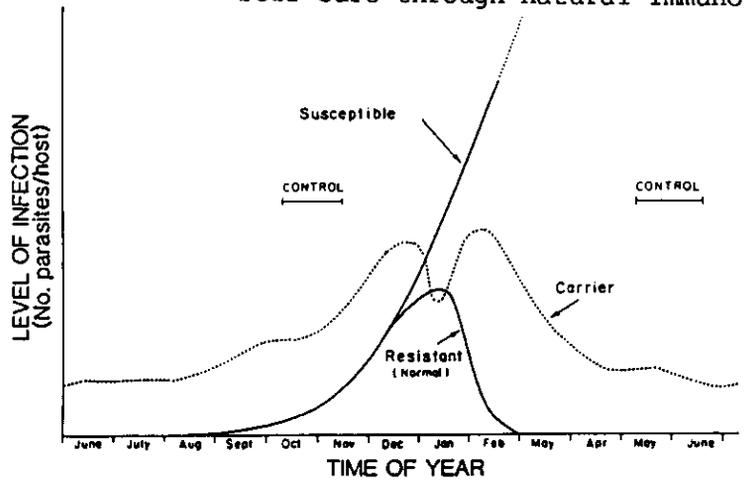

Figure 2.4 – Comparative annual course of infection by the sucking louse, Haematopinus eurysternus, in resistant, carrier, and susceptible cattle in response to continous exposure to climate and weather

2.6.2.3   Applications_for_meteorological_services_in_management_of ectoparasites

Insufficient modern resources and technology have been dedicated to the development of functional models of host-parasite systems in a quantitative biometeorological context. Until this is done, concepts for the practical development and application of meteorological services for animal management tend to be relegated to trial cases based on relatively ad hoc and sparsely recorded biometeorological knowledge. Nevertheless, advantages from meteorological considerations of parasite control or management may be obtained with practice from selected host-parasite associations of long-standing economic importance.

The major difficulty for meteorological services in animal production is that the biometeorological modelling essential for practical operations tends to be a special case for each host-parasite association. Unlike usual meteorological services that are based on the concept of general forecast systems, the requirements for animal management are perceived more as modules developed to accommodate certain types of host-parasite systems. Parasite systems with different implications for meteorological conditions

range from highly adapted relations of "obligatory" forms at one extreme to relatively unadapted but highly variable interactions of "free-living" species at the other. Meteorological applications are generally determined by exposure of the parasite system to the influence of weather and climate within this range of host-parasite relations (Table 2.11).

2.6.2.4   Sedentary obligate ectoparasites

Sedentary obligate ectoparasites, such as the blood-sucking lice on farm animals, represent the extreme host-parasite adaptation. Interactions between populations of parasites and hosts combine to maintain a host-parasite regulatory system which normally limits each species of parasite to a relatively low infection rate. It is estimated from available studies in the western regions of Canada and the United States, for example, that the infection rate for the blood-sucking species of lice generally remains below 8 per cent of the cattle population. Departures from this normal infection rate are usually related to external stresses imposed on the system by management and the meteorological environment.

Sedentary obligatory ectoparasitism is a prime example of the simultaneous interaction of meteorological conditions with the immunological system of the host and the infectivity of the parasite. Susceptible range and feedlot cattle in Western Canada succumbed rapidly to increasing densities of lice (Fig. 2.4) when exposure to the coldest day of winter coincided with an apparent low in immunological resistance during December and January. Resistant animals, representing the vast majority in herds, develop low attenuated peak infestations at the same time of year but also display rapid self-cure during the following 6-8 weeks. "Carrier" animals, that maintain year-round infestations and are the source of annual reinfection in herds, characteristically display a distinct bimodal peak in numbers of parasites. The first mode corresponds with severe low winter temperatures in December and early January. In contrast with resistant animals, the second mode in "carrier" animals is a manifestation of the re-establishment of host-parasite regulation by a weak immunological system following the destabilizing influence of extreme cold in mid-winter.

Meteorological services have useful adaptations in the management of obligate sedentary ectoparasites to "fine-tune" schedules and procedures in control practices. With optimum timing for interaction of weather with varying seasonal potentials for infections and self-cure (Fig. 2.5), preventive control can be achieved with single effective treatment before extreme cold limits operations in late autumn. Under similar host-parasite relations, treatment of susceptibles not responding normally to self-cure can be precisely timed to minimize or eliminate the reservoir of subsequent infection of animals in closed herds.

Meteorological services can be of significant advantage to the management of livestock primarily by defining seasonality in climate and its local variations, a major factor for the effective control of adapted sedentary parasites on obligate hosts. These applications involve calculations of departures from normals in weather variables in order to adjust annual and seasonal time scales for life cycles and the related optimum control schedules (Fig. 2.5). On-line weather data, however, must be available for direct analysis by users as an integral and operational part of the decision making necessary for the optimum management of livestock production.

TABLE 2.11

Variation in external parasitism and its response to weather and climate

Type of parasitism	Meteorological exposure	Sensitivity to meteorological change	Immunological influence	Example	Stage of impact in infestation cycles
Sedentary obligate	host-sheltered micro-climate	low	high	lice, sheep keds, mange mites	infection & reproduction
Free-living obli- gate parasite	all stages exposed	high	low	horn flies, fleas	infestation, development & reproduction
Free-living adult/ obligate larval parasite	non-feeding adult vulnerable/ immature sheltered	very high/ very low	high	cattle grubs	reproduction & infection
Facultative parasite	adult exposed/ immatures sheltered	high/low	low	flesh flies & other myiasis flies	reproduction & infection
Free-living blood feeders	all stages exposed	very high	low or none	dipterous blood-sucking flies, ticks, fleas	infestation, development & reproduction
Disease trans- mission (vectors)	all stages exposed	very high	none	dipterous blood feeders, ticks, flies	infection, development & reproduction

2.6.2.5   Free-living obligate ectoparasites

A free-living obligate parasite, such as the horn fly Haematobia irritans, is physiologically dependent on a specific host during at least one of its life stages, but it retains habits of free dispersal, development, and reproduction away from the host and is essentially an inhabitant of the pasture. The adult remains closely associated with its host because of its daily requirement of multiple blood meals. The frequent blood-feeding of flies is a significant factor of the productivity of grazing animals (Haufa, 1978).

The impact of summer infestations depends on the fly's capacity to develop as a larva in cattle faeces dropped on permanent pasture and to emerge, disperse, and reproduce the highly mobile, adult stage of the life cycle. Although fully adapted as an obligate parasite, the meteorological conditions limit all the phases of its life cycle and the time and rate at which the number and size of generations contribute to its seasonal abundance. As a result of its host-parasite adaptation, the life cycle of the parasite is also under indirect influence of meteorological conditions through its bovine host. A chemical factor in the bovine blood meal, triggered in the host by a decreasing intensity of ultravoilet radiation, induces the blood-feeding adult flies to produce diapausing progeny increasingly during August and September that eventually terminates the cattle infestation. Overwintering diapause will be broken by rising temperature in spring and the post-diapause generation of a new summer infestion will resume development.

Locally available on-line meteorological data are advantageous in pest control practices if used to document the seasonal variation from normal temperature in the immediate post-diapause period. Unseasonable warm periods in winter and early spring followed by low temperatures can reduce post-diapause survival. Severity and rate of establishment of an annual infestation in early summer can be forecast from weather data in early spring as a measure of probability for the survival of the diapausing generation.

Local on-line meteorological data can also be used to advantage in forecasting the relative abundance of successive summer generations of flies for the optimum scheduling of the economic control of infestations. The meteorological relationship is of the form:

$$H_{Ft} = f (T_t, R_t, M_t, I_t)$$

in which: H_F = log number of parasite per animal
T = average temperature (°C)
R = amount of rainfall (mm)
M = mixing ratio for atmospheric humidity
I = application of irrigation (0 or 1)
t = time in the projection period.

*

*   *

TABLE 2.12

Predictive indices for the equation of weather variables
with average number of horn flies (log) per animal per week
at Lethbridge, Alberta, over 6 years [e](1971-76)

Estimated coefficients for:

Date	Intercept	Temp (°C)	Rain (mm)	Mixr	Irr'	R^2
1971	1.106	.1083	.0125	.1802	-.1867	53.79
1972	2.238**	.0045	-.0300**	.2929*	-.3340	66.59
1973	-0.399	.1734**	-.0093	.2518	.1686	60.85
1974	0.319	.0297	.0449	.3969	.4202	31.58
1975	0.687	.2942**	-.6105	-.1659	-.2746	73.84
1976	2.180**	.0937	.0026	.1223*	.4407	63.80

** Means significant at the .05 level
* Means significant at the .10 level
' Yes = 1 and no = 0
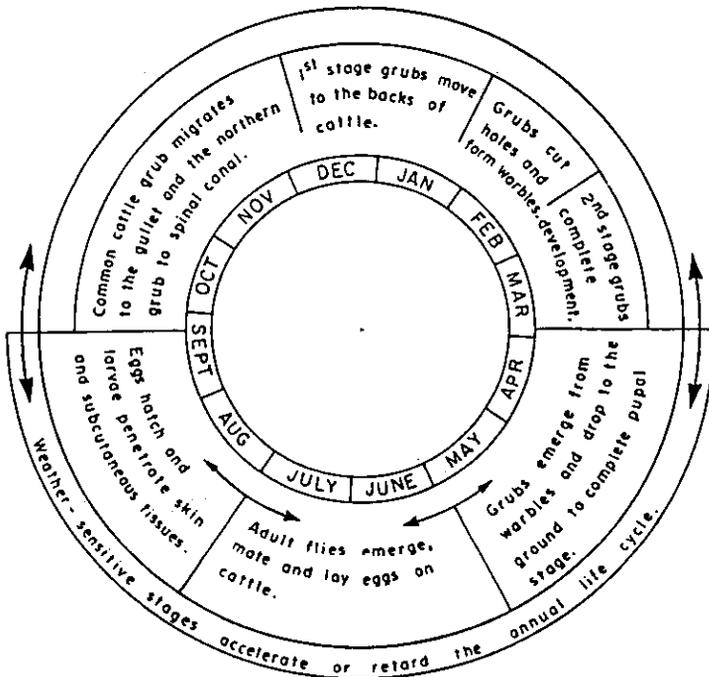
Figure 2.5 - The annual cycle for infections of blood-feeding species of lice on cattle exposed to the weather and climate year-round in the north temperate zone
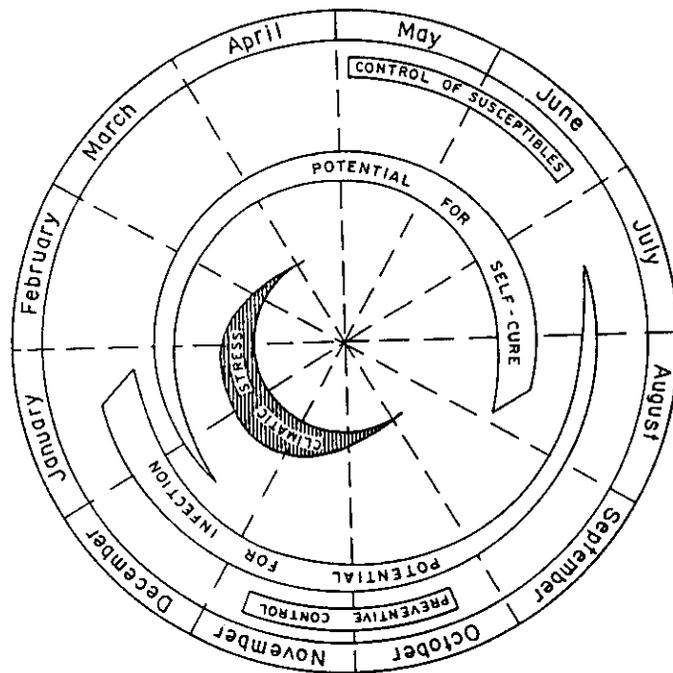
Figure 2.6 – The annual cycle for infections of the grubs _Hypoderma lineatum_ and _H. bovis_ in cattle in southern Alberta showing the dependence of weather-sensitive "free-living" stages on climatic variations in the timing of seasonal events

Prediction equations, such as those calculated from on-line weather data for southern Alberta (Table 2.12), may be used on a short-interval basis to project the need that parasite controls should meet economic thresholds when growing beef animals in large production units. Such weather-related equations may also be incorporated in beef-forage-grain simulation models in order to select the optimum economic control practice of parasites in various kinds of farm operations and different agricultural environments (Gordon, et al., 1984).

2.6.2.6   Free-living adult obligate larval parasite

        Some parasites, such as the cattle grubs _Hypoderma_ spp., retain a combination of the fully adapted obligate and the free-living phase of the host-parasite association of different stages of the life cycle. The growing immature stages are endoparasitic and, in a homeothermic host, functionally isolated from the direct efforts of the atmospheric environment. The influence of meteorological conditions on immature grub development is indirect due to the general suppression of the host's immune response in mid-winter. The living span and survival of the parasite's free-living pupal and adult stages are determined primarily by weather conditions. Since adult flies do not feed, the successful use of its limited stored energy for reproduction is directly related to favourable meteorological conditions after emergence.

        Meteorological services have significant practical applications to the timing of the annual infection as a result of acceleration or retardation

of the activities of weather-sensitive free-living stages (Fig. 2.6). These applications are most useful in the form of variations from the norm in temperature and moisture during critical periods, i.e. after the grubs drop from the backs of cattle, when probabilities of success as to the emergence of adults, the effective dispersal and mating for reproductive efficiency, and host reinfestation, can be predicted. Systematic use of local on-line weather data is particularly advantageous for the critical scheduling of therapeutic treatment with systemic parasiticides that can minimize or eliminate the critical side-effects associated with the killing of migratory grubs in sensitive tissues of the gullet or spinal cord, a hazard of indiscriminate treatment. Meteorological services are more essential in the recent development of integrated pest management of cattle grubs. Biological phases of integrated control procedures, such as release of sterile male flies to disrupt the reproduction of flies surviving chemotherapeutic treatments, depend on critical predictions from biometeorological models to maximize probabilities for eradication of infestations.

2.6.2.7   Facultative ectoparasites

Facultative parasites such as the dipterous flesh- and blowflies that cause primary and secondary myiasis through their larval infections are free-living ectoparasites open to direct influence from the meteorological environment. Parasitisms, such as those in mink on fur farms or of infants, are usually confined to infrequent or sporadic local outbreaks. Although subject to weather conditions, outbreaks of parasitisms are the result of uncommon deviations from the normal oviposition or viviposition behaviour in flies that occasionally are deprived of their usual breeding sites for larval development. Meteorological information has relatively little application in forecasting infrequent infestations determined primarily by conditions of environmental sanitation.

2.6.2.8   Free-living blood feeders

Numerous species of dipterous flies (mosquitoes, black flies, tabanids, ceratopogonids, snipe flies, etc.) and various species of ticks and fleas attack livestock and man as blood-sucking ectoparasites. This wide variety of parasites is completely free living and functionally unadapted to specific host-parasite relations. However, largely because of their lack of host specificity and unadapted host-parasite associations, they have the potential to make a devasting impact on livestock exposed to intermittent mass invasions, even from relatively remote breeding grounds. Development, behaviour, dispersal, and the blood-feeding activities of these parasites are highly dependent on weather and climate. Of all known types of parasitism, control of these species has the highest potential for economic application of specialized on-line meteorological data services. The terrestrial distribution, habits, activity, and dispersal of these species are mediated in poikilothermic relationships with their climatological and meteorological environments.

The potential of practical applications of meteorological services in more general forecast systems for the dipterous pests of animals and man have been described in the larger context of biometeorology (Haufe, 1965; 1976). Comprehensive and comparative studies of mosquitoes, black flies and tabanids indicate a common basis for the regulation of a large number of species by their hygrothermal environment. The design of a hygrothermogram for mosquitoes (Fig. 2.7) is an example of a biometeorological model that can

be used by meteorological services to define atmospheric environments favourable or unfavourable to spatial activity and displacement of pests. Practical applications are limited only by the availability of the established biological limits for designated species of economic importance categorized in a basic psychronergetic regression for poikilothermic efficiency in dipterous insects. Biological applications of the hygrothermogram (Q) in Fig. 2.7 for various species or groupings of species, are readily established either from field correlations or laboratory measurements of pest activity under recorded atmospheric conditions of temperature, humidity, and radiation.
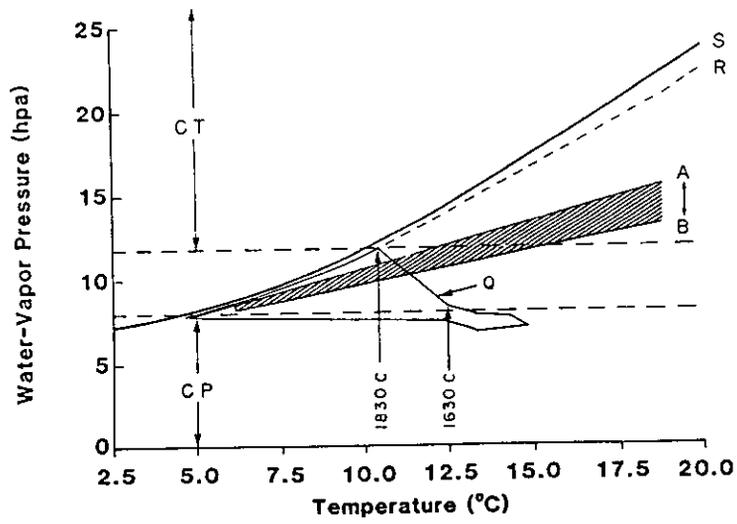
Figure 2.7 — The hygrothermogram (Q) as a meteorological method of projecting potential activity of blood-sucking flies from conventional synoptic on-line weather for practical applications; 24-hour trajectory (Q) for hygrothermal environment in relation to resting (S-R) and active (A-B) conditions for the Aedes punctor-communis mosquito complex and the continental tropical and polar air masses in sub-arctic Canada

     Hygrothermal models of the activities or behaviour patterns of blood-feeding dipterous insects have significant applications as to their extension as disease vectors and the prediction of epidemics of arbovirus diseases. Models for these specialized applications require a meteorological definition and classification of atmospheric environments conducive to unusual contact of arthropod vectors with both the animal reservoir and the accidental host. The means of employing meteorological services to predict the environmental conditions necessary for epidemics of arbovirus disease such as the encephalitides, are possible through studies of vector behaviour. Development of forecast systems are hampered, however, by a lack of on-line weather data and their use in order to test probabilities of biometeorological relations during the course of epidemic.

2.6.2.8   Conclusion

    A biometeorological understanding of host-parasite relations is an essential part of the background knowledge needed to manage livestock efficiently and to protect livestock production from ectoparasites. The control of infestations has for too long depended on the use of chemical parasiticides instead of moving to improve operational practices for the effective management of ectoparasites through the use and application of meteorological services and data. As future emphasis on integrated pest management will combine biological, cultural, and other methods of control, reduced use of the environmentally objectionable synthetic chemicals will depend on increased on-line availability of the relevant meteorological information and services.

C H A P T E R   3

HOT WEATHER STRESS

3.1   CATTLE – HEAT STRESS

3.1.1   General observations

        Domestic cattle fall into two main groups: European Bos taurus breeds which evolved in temperate or cold regions, and Bos indicus or zebu breeds which evolved in tropical regions of southern Asia and Africa. The relative heat tolerance of zebu cattle is an important factor favouring their use in warmer environments, although these breeds are by no means immune to the effects of heat on their physiological and productive functions. Bos taurus breeds, however, carry genes for higher production potential, so that the crossing of the two types to combine productive potential with tropical adaptation is a widespread practice. Improvement in productivity is to be sought through moderation of the effects of heat either by management or by breeding for heat tolerance.

        Stability of body temperature is a prerequisite for high productivity in cattle (Frisch, 1981; Turner, 1982). Because of the limited ability of cattle to thermoregulate in hot environments, the impact of various weather conditions on the resultant net energy exchanges between the animal and its environment tend to displace body temperature. The forms of energy exchange by which the animal dissipates its metabolic heat production are shown in Figure 3.1 (Hahn, 1983). A representative perspective of the relative magnitude of energy exchange fluxes is given in Table 3.1. Heat stored in the animal's body must be dissipated during the cooler evening hours or the point of exhaustion would soon be reached.

        Animal factors which mediate the impact of environmental factors on body temperature are schematically summarized in Figure 3.2. The physiologic measures of most interest for cattle in hot environments relate to heat dissipation by evaporation, since the potential for convective, conductive and radiative dissipation is greatly reduced (Figure 3.3). Although cattle are obviously able to markedly increase evaporative heat loss at high temperatures, and will reduce feed intake in an attempt to reduce heat production and maintain homoethermy, body temperature increases when heat production exceeds heat dissipation. Since body temperature impinges on the functions of the endocrine, cardiovascular and digestive systems, and as the fluid balances, the animal must maintain it within relatively narrow limits; therefore, the measurement of body temperature (e.g., rectal or tympanic) is critical.

        When high ambient temperature and radiation inhibit the outflow of sensible heat, cattle become more dependent on evaporative cooling (Figure 3.3). The inhibitor effect of humidity on the evaporation of water increases in importance. Since the saturation deficit of air in contact with and at the temperature of the evaporating surface (respiratory passages and external body surfaces) determines the capacity for moisture removal, the absolute rather than the relative humidity is the more relevant measure. Air movement helps to maintain absorptive capacity of the air in contact with the external body surface, but it is beneficial only up to the level needed to displace air with reduced saturation deficit.
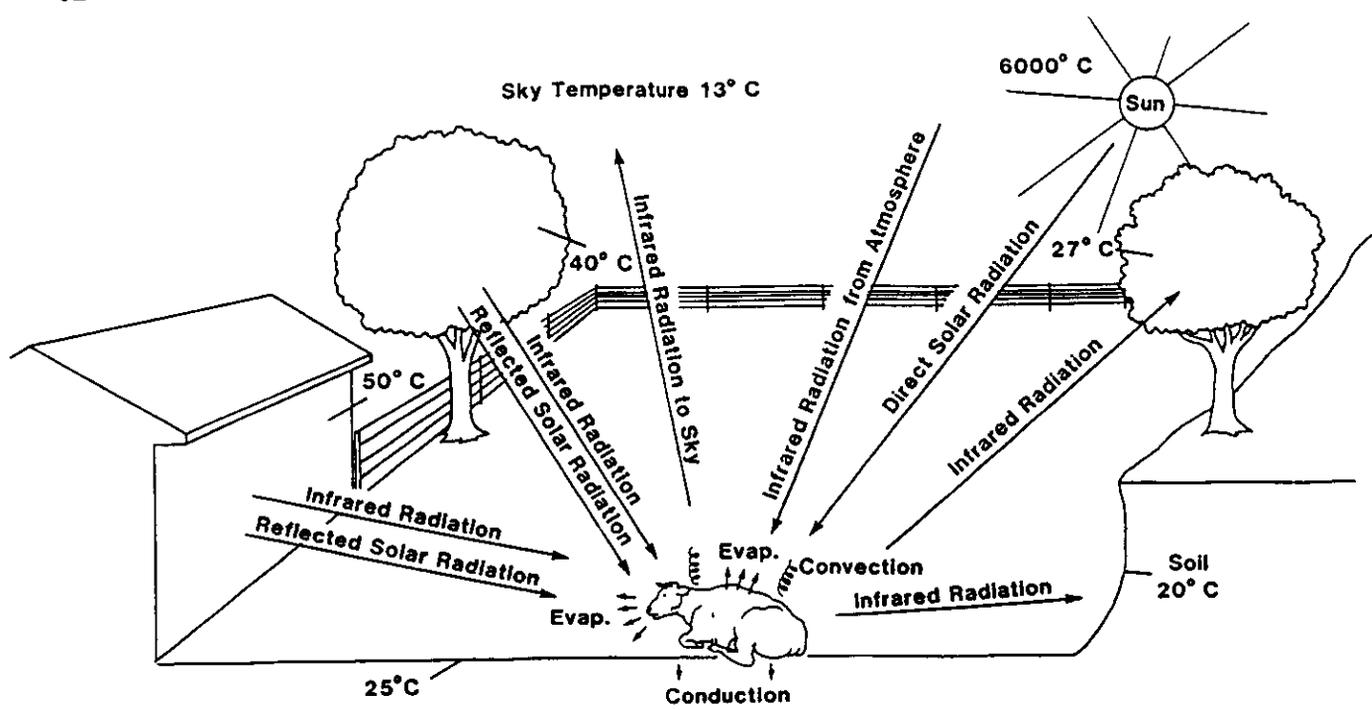
Figure 3.1 - Energy exchanges between a farm animal (39°C core body tempera-
ture) and its surrounding environment; with the air termperature at 30°C and
no wind movement, the skin temperature might be 30°C and the hair on the back
at 45°C (Hahn, 1983)

TABLE 3.1

Typical mean rates of energy exchange (W/m^2) of a steer in
a hot outdoor environment, between 0900 and 1500 hr

	W/m^2
Gains	
Metabolic heat	76
Radiation absorbed	676
Losses	
Radiation emitted	436
Convection	100
Respiratory cooling	39
Sweating (surface vaporization) . . .	169
Balance	
Heat storage . . . . (resulting in about 1°C body temperature rise for the period of observation)	8

After Finch, 1976

Environmental factors	Food	Ambient temperatures, air movement.	Insolation reflection, re-radiation.	Humidity, air movement.
Form of energy exchange	Metabolic heat Production	Convection, Conduction	Radiant exchange	Evaporative cooling
Animal Factors	Maintenance metabolism Heat increment of productive function and high food intake	Insulation: External- coat type Peripheral- tissue compo- sition & blood flow. Mass transfer: heat in excreta Direct cooling: to a heat sink, e.g. water	Coat colour Coat insulation Selection of environment (shading)	Respiratory evaporation Surface evaporation (sweating) Hydrodynamic properties of coat
Integrated effect		BODY TEMPERATURE		

Figure 3.2 – Energy exchanges and animal factors which are involved in the integrated influences of environmental factors on the body temperature of animals

Metabolic heat production is the inescapable by-product of the oxidation of food for the energy transactions necessary both to maintain basic vital functions and to synthesize products such as growth, the foetus, or milk production. Energy metabolism therefore has a dual significance in terms of heat load and the efficiency of feed utilization which should be considered separately in relation to animals at a maintenance level or at a productive level of feeding. Maintenance metabolism is about 14% higher in European than in zebu cattle (Frisch and Vercoe, 1976). Within a breed, there is genetic variation in maintenance heat production, with a coefficient of variation of about 6.5%. An animal with an inherently low maintenance metabolism would be doubly advantageous if, in a warm climate and at a low level of feeding, it suffered less heat load and also needed less food. It would be cheaper to maintain and would survive better under conditions of food shortage. On the other hand, the efficiency of a growing or lactating animal with access to abundant food is dependent on a high food intake and metabolism.
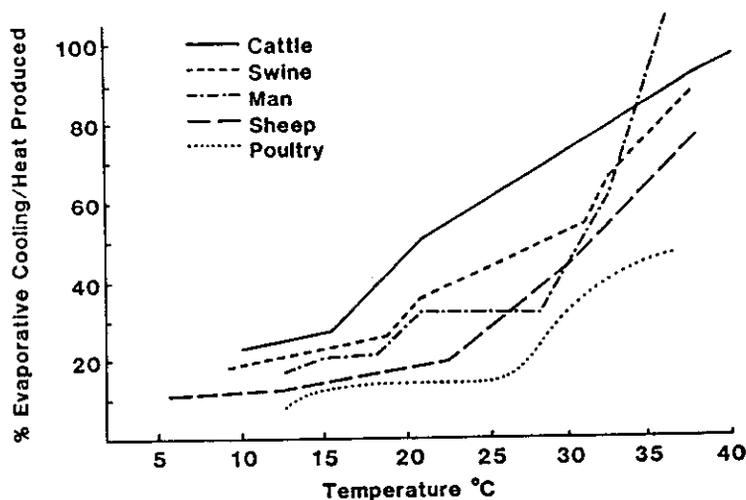
Figure 3.3 - Ratio of evaporative heat loss (skin and respiratory) to total heat production for cattle and other species (Johnson, 1965)

Phenotypic and genotypic differences among cattle affect the impact of a given stressful environment. Results of studies by da Silva (1973) and Turner (1982) indicate that in a stressful climate animals within a breed that differ by one standard deviation in inherited rectal temperature also differ by about 0.8 standard deviation in inherited growth rate or fertility. Genetic differences appear to be predominantly in temperature regulation, not in tolerance of raised body temperature (Vercoe and Frisch, 1970; Turner, 1982). This means that the animal factors shown in Figure 3.2 are rich in primary genetic diversity, whereas productive functions are largely secondary and automatic consequences of variation in body temperatures.

Erroneous conclusions or confusion concerning efforts to quantify the effects of heat stress on cattle responses have resulted from (a) failure to consider differences in the heat-stressing qualities of hot/dry as opposed to hot/humid climates, (b) failure to consider the limits of using an average daily heat stress index when there is a wide diurnal flux, (c) attempts to make projections concerning the effects of heat stress by extension of lines developed from known data, and (d) failure to consider the degree of acclimatization of animals to a given level of heat stress and the subsequent compensatory performance when predicting responses.

Selection of the most efficient weather parameter for estimating heat stress depends in part on the local climate and the available equipment for measurement. Maximum and minimum daily ambient temperature may serve well

for predictions, especially if air moisture is relatively constant as in a maritime location. The Temperature—Humidity Index (THI)* has been used (Thom, 1959) for the estimation of the level of heat stress. THI offers a method of combining two of the more important and easily measured weather factors into a single figure that can be easily used in calculations Also, it makes it possible to compare temperature and humidity data and animal response at different locations. With a THI of 75, 50% of the human population are predictably uncomfortable; interestingly, THI 75 also appears to be the average upper limit of relatively complete acclimatization for the European breeds of dairy cows.

Significant depressions in milk production and reproduction occur at an average daily THI of above 76. However, some depresssion may also occur between THI 68 and 76 in animals milking at high levels or acclimatized to a lower THI. Preferably, average daily THI should be the average of THI calculated at one or two—hour intervals but, where complete information is not available, an average THI calculated from maximum and minimum AT with respective THI will give an estimate of the average THI of the day that may serve well for predictive purposes.

Under pastoral conditions, rainfall has indirect effects that can increase the severity of heat stress. Since pasture abundance affects the distance cattle must travel between shade, water and food, it also affects the amount of heat-generating exercise they must take to gather their nutritional requirements and also the time spent exposed to solar radiation. Seasonal variation in surface water may affect the distance between food and water. Grass cover also affects the albedo and temperature of the ground to augment the radiation load on the animal. The nutritional value of pastures can be reduced by heat stress, which might increase the need for supplementation with concentrates despite the availability of grass in fodder. Mineral deficiencies can also be a serious problem; for instance, phosphorous deficiency is very common in Africa. Since phosphorous is essential for bone and muscle synthesis and reproduction, the productivity of animals feeding on pastures in areas without phosphorous supplementation is poor.

In the 1976 drought in England, reduced growth of grass subsequent to the rains that ended the drought in September, resulted in problems of undernutrition as farmers overestimated the feeding value of the pastures; high incidences of hypomagnesemia, ketosis and other metabolic disorders were noted (Gibson, et al., 1982).

Poisoning problems in cattle associated with drought conditions included nitrate/nitrite poisoning and salt poisoning. Nitrite accumulation may have followed the high level of nitrification by bacteria in the soil during the hot summer and also the absence of leaching during the drought. In one incident, undried hay and straw samples contained 2.2% and 3.4% nitrate, respectively (Jones and Jones, 1977). Heavy application of nitrogenous fertilizers before the drought may have led to rapid uptake of unchanged nitrite when the rains came in September. In other situations reported by Gibson, et al. (1982), six dry cows died within three hours of being turned out to pasture, but milking cows were unaffected. Nitrite poisoning was also suspected as the cause of 17 abortions in a herd of 110 milking cows. Salt poisoning was blamed for a few deaths in cattle that drank from riverside

---

* See 1.2. Meteorological glossary.

marshes commonly containing 0.5% NaCl, and occasionally as much as 1.8%. Other cattle showed diarrhoea and ataxia but recovered with treatment. However, overall, the adverse effects of the drought on cattle were less than had been feared.

3.1.2   Reproduction and lactation

Summer heat in temperate zones and the prolonged seasons of environmental stress associated with the tropics and sub-tropics cause reproductive problems in temperate-evolved cattle, especially dairy cattle Reproductive function is altered mainly by poor conception rates (CR) and reducted detection of oestrus. In addition, it may cause dwarf calves, abortions or a shortened gestation period.
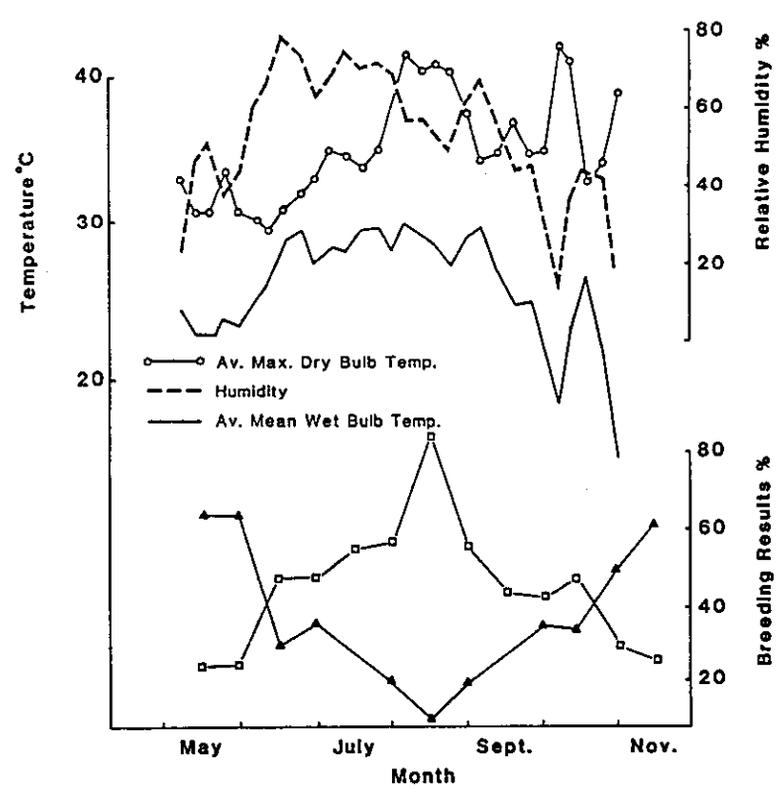
Figure 3.4 – Humidity and temperature effects on fertility of dairy cattle in Arizona (after Stott and Williams, 1962); per cent breeding results of cows having oestral cycles over 26 days (open squares), cows pregnant at 35 days post-breeding (closed triangles)

Heat production may be doubled in a high-producing dairy cow, causing the lactating cow to be more prone to heat stress than a non-lactating cow or bull. Therefore, bulls and heifers have less of an increase in body temperature at a given level of heat-stressing climate than the lactating cow. Consequently, their reproductive efficiency is not impaired to the same extent. In milder climates heifers may have near normal CR during the hot season. The classical data of Stott and Williams (1962) shown in Figure 3.4 clearly demonstrate the adverse effects of hot conditions on the fertility of dairy cattle. The Temperature Humidity Index remained at 80 or above during the period.

Data from Ingraham (1974) show changes in the diurnal rectal temperature flux with the increasing average daily THI in the naturally-varying environment (Figure 3.5). Average maximum and minimum temperatures with associated humidities and resulting THI for this period were
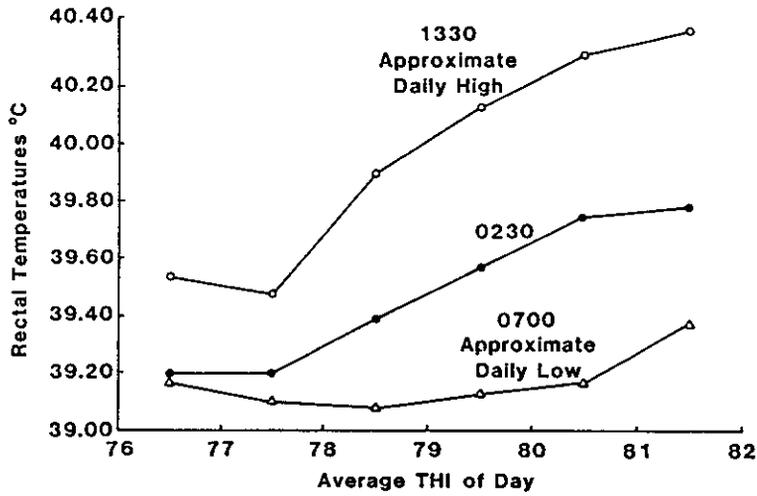
Figure 3.5 – Diurnal rectal temperature flux as a function of daily average THI (Ingraham, et al., 1976)
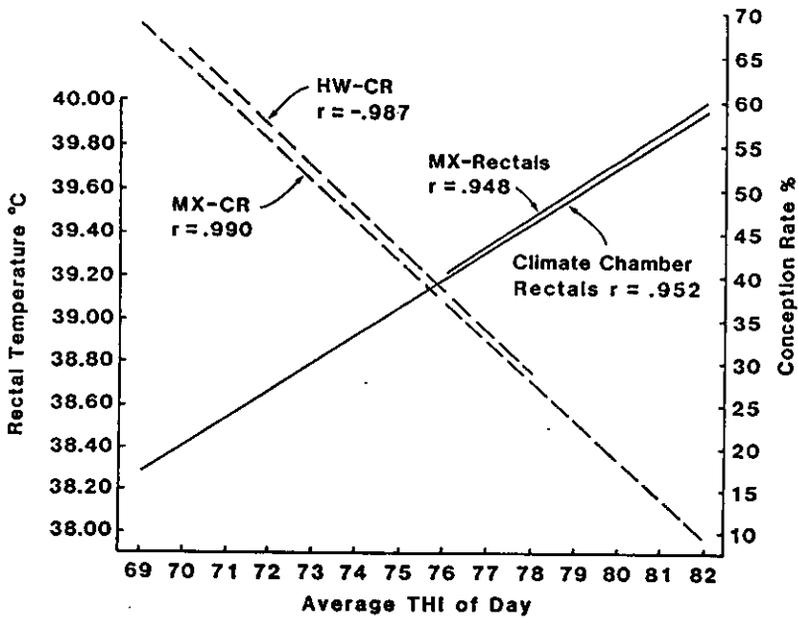

Figure 3.6 – Influence of THI on rectal temperature ("rectals") and conception rates (CR) of Holstein cows; data are from corralled cows during the hot season in Mexico (MX) and Hawaii (HW), and from constant temperature chambers (Ingraham, 1974)
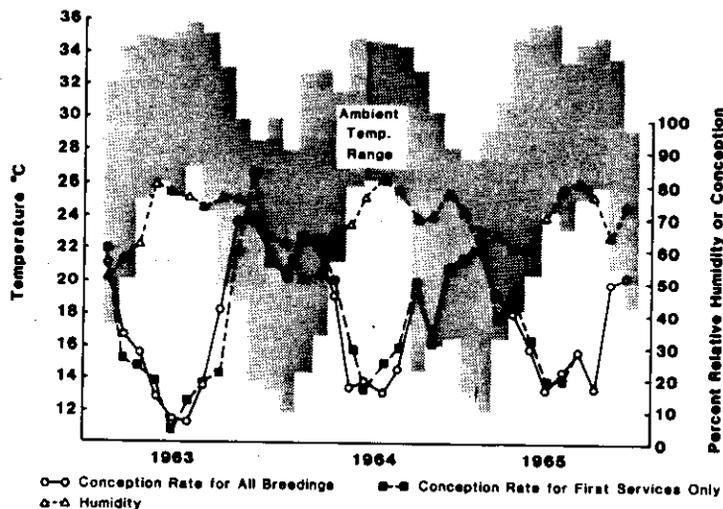

Figure 3.7 – Monthly conception rates for Holsteins in Mexico over a three year period, with monthly climatic data superimposed (Ingraham, 1973)

34°C, 54% and 84 and 25.8°C, 82% and 76. Cows were not able to attain normal
body temperatures at night, a factor that appears to be critical to the
problem of heat stress. The manner in which the diurnal body temperature
fluctuation may affect reproductive efficiency has not been established.
Several researchers have found a CR decline associated with rectal
temperatures above 39.7°C (Zakeri, et al., 1981; Gwazdaukas, et al., 1973;
Long, et al., 1969); however, since the rectal temperature varies during the
day, interpretation of such data reported without regard to time of day is
difficult to interpret. Data from Ingraham (1974) and Ingraham, et al. (1976)
were used to establish the relationship presented in Figure 3.6 between the
THI and the average rectal temperature for Holstein cows based on
measurementsat 0230, 0700, 1330, and 1900, and to further establish that CR is
linearly related to the THI on the second day prior to breeding by the
regression equation (Hahn, 1981):

$$CR = 388.3 - 4.62 \ THI.$$

Although THI level has a significant effect on CR for all the 11 days before
breeding, with the exception of the day immediately before, the effect is most
significant on the second day before breeding (Ingraham, et al., 1976).
Experience has indicated that this relationship is valid for summer seasons in
which the average THI of 1/3 or more of the days is above THI – 76; for
seasons with long periods below 76, the CR vs THI response was nearly level
(Ingraham, et al, 1976). For tropical breeds in heat-stressing climates, the
level of reproductive efficiency is likely to be attributable to the ability
of those cows to maintain a lower body temperature at higher THI values
(Zakeri, et al., 1981).

        Climate chamber studies have indicated that heat stress can impair
fertility in the bull. Casady, et al., (1953) concluded the impaired
spermatogenesis might result from 5-week exposure to 30°C. Skinner and Louw
(1966) reported that as little as 12 hrs at 40°C and 45% RH proved critical to
optimum spermatogenesis. Rhynes and Ewing (1973) exposed Hereford bulls to
35.5° and 50% RH for 7 weeks. Plasma testosterone fell to 43% of control
values during the first two weeks of the experiment, but rose to near control
levels in the subsequent weeks. While an extreme situation, spermatogenesis
was seriously impaired by heat treatment as evaluated by semen characteristics
and histological evaluation at the termination of the experiment. The net
effect of heat stress in field conditions is difficult to estimate. Stott
(1961), using semen from bulls in a desert climate on local cows and on cows
located in cooler areas concluded that the female was responsible for the
depressed fertility that occurred during the warm season. From a similar
evaluation in South Carolina, Kelly and Hurst (1963) concluded that males
and females were contributing about equally to the low summer conception rates
(about 10% each). Bulls were used for the study in Mexico depicted in
Figure 3.7. Bulls were taken to pens daily for mating with cows in oestrus.
The recovery of fertility as the weather cooled in October appeared to be
almost immediate. There was no indication of an extended period of reduced
fertility, as might be expected from chamber experiments, resulting from
damage to sperm at different stages of development. The extreme of average
daily THI in this experiment was 82. Conception rates using artificial
insemination with semen from a bull in California during the next summer were
not different from bulls used during the same period. In summary, it is
difficult to estimate the extent that the bull influences fertility during the
hot season but most evidence points to stress in the cow as the primary source
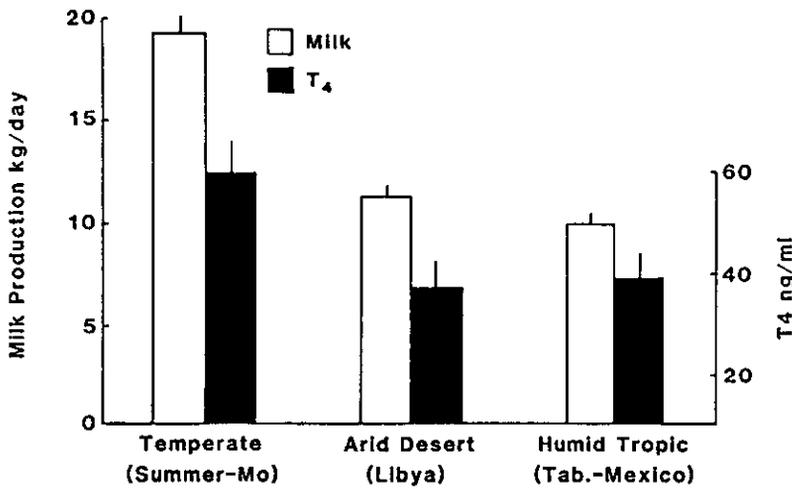of the problem. However, semen quality is depressed in bull studs in the

Figure 3.8 – A comparison of temperate zone milk production levels, (Missouri, USA), to the production in the Libyan coastal desert and the Mexican low-tropic region at Tabasco; all the animals were pure-bred Holsteins and Friesians (Johnson, unpublished data)
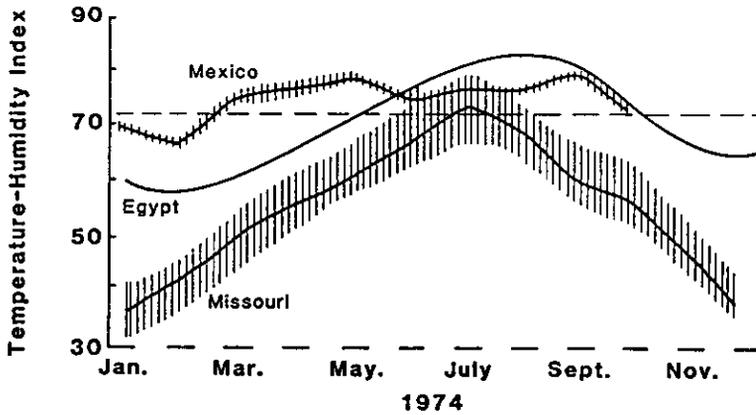
Figure 3.9 – A comparison of the Temperature Humidity Index (THI) at various seasons for a temperate zone (Missouri, USA), hot arid zone (Egypt), and hot humid zone (Tabasco, Mexico). An optimal zone for higher lactating cows is in the range of 40–72 THI (Johnson, unpublished data)
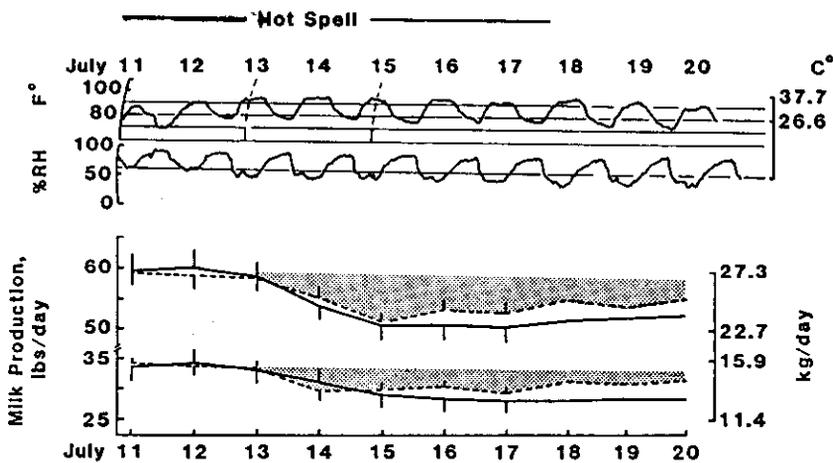
Figure 3.10 – Weather events and their influence on the level of milk production in the University of Missouri Holstein herd, 1977 (Johnson, UMC Climatic Laboratory, unpublished data)
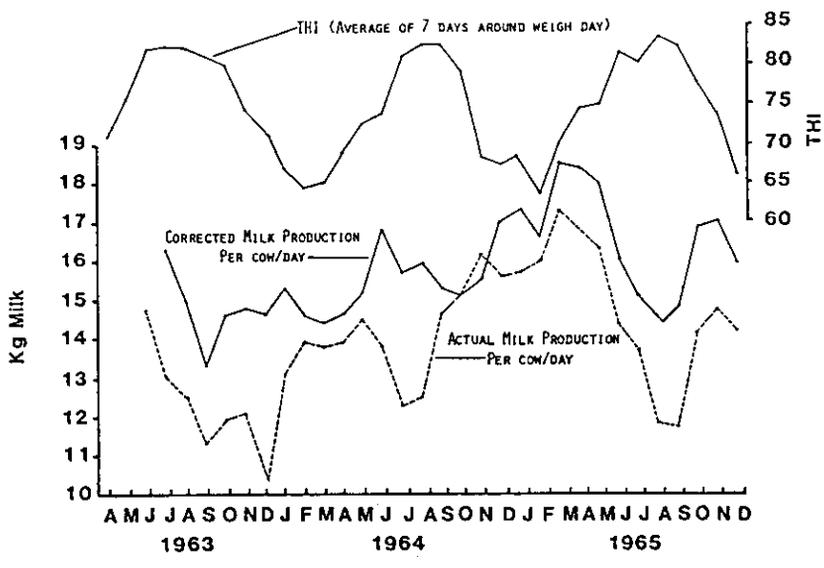
Figure 3.11 - Average THI versus milk production for Mexico. The dashed line gives the actual milk production averaged for each month from daily production records. Corrected milk production was computed from monthly milk production records of individual cows. It is the level of production that would have been obtained if the milking herd was composed of equal numbers of cows in each of the first ten months of lactation

southern USA during the hot months and some artificial insemination units do not collect semen for freezing during this season.

Generally, heat stress depresses the milk producing capabilities of cattle and reduces the percentage of milk fat. Figure 3.8 presents milk production and associated plasma T_4 values in purebred cattle acclimatized to temperate, hot-arid and hot-humid climates (Johnson, unpublished data). Associated climatic environments are shown in Figure 3.9. English Milk Marketing Board data indicated that milk sales from farms during the drought of July, August and September, 1976 decreased by 2.7, 6.7, and 6.3% from comparable values of 1975 (Gibson, et al., 1982). While heat was considered to be the primary factor in these studies, other environmental factors such as feed quality, management and disease may have also contributed to the overall effect.

Figure 3.10 illustrates the effect of a shorter period (approximately one week) of hot conditions in a temperate climatic zone. Daily maximum temperatures were about 35°C and minimum temperatures remained above 23.9°C, coupled with relative humidities ranging between 40 and 85%, considerable thermal stress was induced in the animals. Recorded feed intake dropped markedly with a resultant decreased average daily production of as much as 3.2 kg/cow in high-producing animals. Some recovery in milk production was noted as the temperatures moderated, however, the animals in this study did not return to their previous production levels (Johnson, unpublished data). Another report (Ansell, 1981) indicated a possibility of recovery of milk production over a five-month post-stress period sufficient to offset milk declines during the five months of heat stress. Cows in the Ansell study were subjected to the naturally varying conditions in the United Arab Emirates (Digdaga), where maximum daily temperatures were regularly in the range of 33°C to 43°C during the 5-month "summer"; subsequent maximum temperatures were usually between 25°C and 30°C.

Seasonal infertility due to heat stress depresses milk production in an indirect manner by lengthening the period between calves. Conception rates were low during the hot season (mid-June to mid-October). Consequently, relatively few cows calved between March and August of the succeeding year. Cows were milked beyond the normal 10 months in order to get all available

TABLE 3.2

Breed genotypes and various environmental interactions

1. Relative heat tolerance of breeds in sub-tropical Queensland

Breed	Increase Gain %
Queensland Hereford vs. New South Wales Herefords	50
Africander vs. Hereford-Shorthorn CX	25-30
Brahman CX vs. Hereford-Shorthorn CX	15

2. Nutrition

Breed	Native Pasture Weaning wt. kg.	Improved Pasture Weaning wt. kg.
Shorthorn	100	163
Brahman	150	152

3. Parasites Gastro-intestinal worms

	Untreated		Treated
	Eggs/GM Faeces	KG Gain	KG Gain
Hereford-Shorthorn	180	44.3	72
Brahman - CX	222	66-5	68-8

4. External Parasites

	Undipped		Dipped
	Mean No. Tick Per Side	KG Gain	KG Gain
Hereford-Shorthorn	17	44.3	84.3
Brahman - CX	3	99.5	90

Source: Turner, 1972

production during the period when cows were not calving. It was a period with a relatively small number of cows in early lactation and a disproportionately large number of cows at the low end of their lactation curve. Average milk production data is presented in two ways in Figure 3.11, actual and corrected. Actual milk production was calculated from daily records. Corrected milk production was calculated from monthly weights taken on each cow and production records beyond 10 months of lactation were eliminated from the calculations. For each monthly corrected milk average, cows were grouped by month in lactation (1 to 10), an average production calculated for each group, the averages for each of the ten months summed and the average production of the 10 groups calculated. This gave an estimate of expected milk production for the herd if there had been an equal number of cows in each month of lactation. It corrected for an unbalanced calving schedule without altering the effects of heat stress on production. It also demonstrated that over 50% of the hot season depression of milk production in this herd was due to the failure to maintain a consistent calving system. In the subtropics and tropics, a long period between calves causing cows to be milked beyond the period of efficient production is probably the major cause of low average herd production in herds in which nutrition is not a limiting factor.

3.1.3   Neonatal survival and growth

While increased stillbirth rates and mortality in the prenatal period increase during the hottest months in the tropics and semi-tropics, the data appear to be unreported in the formal literature. Newborn calves left in the sun by inexperienced dams or cows with poor mothering abilities in July and August can result in a lethal hyperthermia. Similarily, experience in Louisiana, USA, has amply shown that stock must not be worked after 10:30 a.m. in hot weather; in even hotter climates, this time would be as early as 8:30.a.m.

Limiting factors of the environment for specific breeds of cattle with variations in heat tolerance are summarized in Table 3.2 (Turner, 1972). The influence of nutrition and internal and external parasites were measured under field conditions on temperate-evolved shorthorns and tropic-evolved Brahma cattle. In the more adverse environments, the Brahma or crosses were relatively superior in performance (genotype by environment interactions). Inability to obtain normal body temperatures limited the growth of the shorthorn as compared to its control and as compared to the Brahma, as shown in Figure 3.12 (Ragsdale, et al., 1957).

Performance penalties resulting from short to intermediate exposure (a few days to 3-4 weeks) to moderate heat stress may also be overcome by compensatory performance during a subsequent non-stress period, as reported by Hahn, et al., (1974) and Baccari, et al., (1980). Such recovery is shown in Figure 3.13 for beef cattle. These results relate well to the findings of others (Strachan and Marnson, 1963, McDowell, 1977, Yazman, 1980) who found no significant correlation between ambient temperature and cattle growth rate during field tests over periods of 12 months or more. Balling (1982) also found that hot weather rarely appeared as a significant factor affecting overall feedlot beef cattle performance (growth and death losses) in Nebraska, USA. Yazman (1980), as cited by NRC (1981), noted that the average daily gain of dairy heifers declined by 50% during seven-day periods of above-average temperatures in Puerto Rico, but gains following the high-temperature periods were 50-70% greater. Although short-term changes in
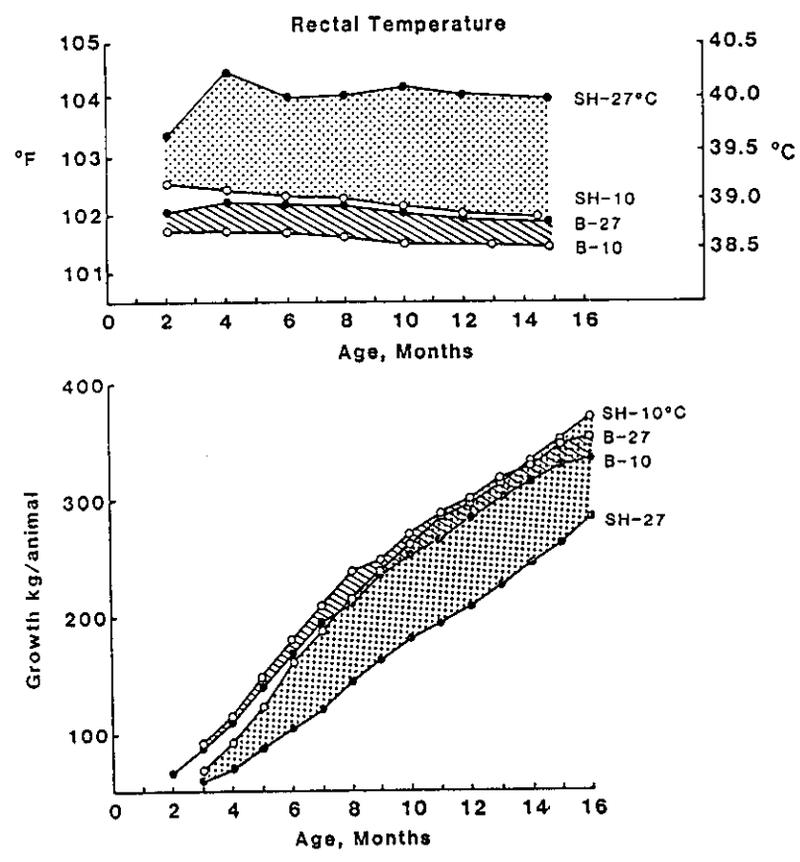
Figure 3.12 - Comparison of rectal temperatures and growth rates of Brahman (B) and shorthorn (SH) heifers raised under constant laboratory conditions of 10°C and 27°C (Ragsdale et al., 1957)
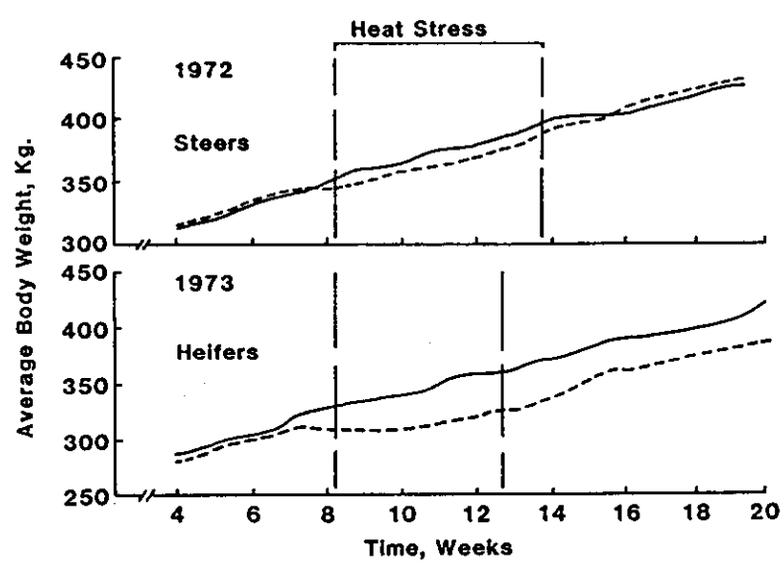

Figure 3.13 - Average growth curves for heat-stressed (treatment, dotted lines) and unstressed (control, solid lines) Herefords, the curves represent three steers in each group in 1972 and two heifers in each group in 1973 (Hahn et al., 1974)

feed efficiency occur during heat stress, cattle and other species have been noted to convert feed more efficiently subsequent to the exposure when compared to unstressed animals (Hahn, 1982). The net result has little effect on long-term efficiency. Body composition is also basically unaltered at the end of the total growth period when suppression is followed by recovery (Hahn, et al., 1974). However, there are limitations to the compensatory capabilities, as, for instance exceeding the stress threshold beyond which recovery would not occur within an acceptable time frame (Figure 3.13). Flexibility in managing the production system is also required to permit compensatory growth to occur before sale or slaughter of the animals, as discussed by Hahn (1982).

While index values such as the Temperature-Humidity Index better characterize the collective impact of an animal's thermal environment during hot weather, and have been successfully applied to assess declines in milk production of dairy cows, a successful application of indices to assess adverse environmental effects on growing cattle has not been demonstrated. This is in large part a result of the animal's ability to counter thermal stress by physiological and behavioural reactions, that may result in a minimal impact on the ultimate weight gain, feed efficiency, and carcass composition of that animal.

3.1.4   Acclimatization, chronic and acute exposures and reactions

High ambient temperature acts as a stressor to adversely affect short-term growth and efficiency of cattle. High levels of solar and terrestrial radiation associated with hot weather and high humidities exacerbate the adverse effect, while increasing wind speeds moderate the effects to a limited extent. Primary means of attempting to maintain homeostasis at temperatures immediately above the upper critical temperature are increased water intake and reduced feed intake. The latter is illustrated in Figure 3.14. However, high ambient temperatures also affect digestibility, both directly and indirectly (e.g., water intake and rate of passage), as shown in Figure 3.15. Reduced feed intake and digestibility both act to adversely affect growth and efficiency.

During heat stress, cattle decrease their feed intake to help reduce the heat load on the metabolic processes. Numerous physiological changes also occur. Environmental stimuli cause evaluation or depression of various factors in the neuroendocrine system. These in turn affect balances of water, heat, hormones, minerals and energy. Alterations in endocrine activities (for example, aldosterone, growth hormone, cortisol, progesterone, thyroxine, leutinizing hormone, insulin and prolcatin) induce changes in growth rates, levels of milk production and fertility. If the stressor is not critically adverse, the animal tends to acclimate to the adverse environment, and perhaps can even, as mentioned earlier, compensate for reduced performance when the adverse environment is no longer present. After acclimation, the animal may resume near-normal activity levels but remain at an abnormal performance level. Duration and intensity of environmental stressors determine whether an animal acclimatizes and perhaps eventually compensates, or eventually succumbs, to the stressors as a result of exhaustion. The genotype is an important variable affecting an animal's ability to adapt or compensate for environmental stress.

The effects of extreme temperatures are highly dependent on the genetic background of the animal, as well as the stage of growth (e.g.,

neonatal and market-weight animals are more vulnerable than cattle at intermediate growth stages), the level of prior conditioning (acclimatization) to the elevated temperature, the roughage/concentrate ratios being fed, and other compounding factors such as insects or other pests, handling, and transporting. The effects of long-term elevated temperature on the growth of various breeds of cattle are indicated in Table 3.3. The tropically-evolved Brahman breed is obviously better able to contend with hot weather than the other breeds tested. Chronic exposure to hot weather can, through acclimatization, shift the upper critical temperature upward to a limited extent so that adverse effects on performance are reduced or eliminated.

Figure 3.6 in an earlier section, shows the linear relationship between average rectal temperature (rectals at 0230, 0700, 1330 and 1900 averaged) and the average daily THI above 69.0, based on data from Holstein cows in Mexico (Ingraham, et al., 1974). Graphed with it is the least squares line for a rectal temperature versus a constant THI relationship from climate chamber data (Kibler, 1964). The two lines are not significantly different, supporting the hypothesis that within this THI range the average heat stressing effect of THI during the diurnal fluctuation is equal to that resulting from a constant THI equal to the average of the cycle. Other evidence from Ingraham, et al., (1974) emphasizes three points: (a) relatively small changes in average THI elicited acute RT responses; (b) these chronically heat-stressed cows were being subjected to intermittent periods of acute heat stress; and (c) the cows showed some degree of acclimation by reduced RT response to specific THI after 2 weeks or longer exposure to hot conditions. This effect of day-to-day changes in thermal stress on animal physiology is an aspect of climate stress for which data are not available.

Another aspect of rectal temperature response to THI concerns the fact that cows vary in their ability to maintain normal rectal temperatures at THI above 76. Figure 3.16 gives the cumulative distribution curves for rectal temperature response of a group of 49 cows (average of 0230 and 1130 rectal temperatures) at average THI of 76.6, 78.5 and 80.3. The percentage of the group with an average rectal temperature of 39.5°C, a figure sometimes cited as the threshold for depressing reproductive efficiency, increases from 30% at THI of 76.6 to 85% at THI of 80.3. The linearity of these graphs (r = 0.98, P<.001) for this group of Holstein cows in the first three months of lactation stimulates speculation that conception rates are a function of the percentage of the cow population with body temperature above some critical figure at some critical time or times. The linearity also emphasizes the relatively even distribution of cows across the range of rectal temperatures. In other words, while the rectal temperatures of cows in a thermoneutral climate can be expected to clump around a mean rectal temperature of 38.6°C, the bell-shaped curve of the normal distribution flattens in heat stress as each cow acclimates with a different level of heat storage. It also appears to indicate a wide range of adaptability to heat stress within the Holstein breed and consequently a potential for selection of heat tolerant animals. The cows in this study were not native to the area, but had been imported from California, USA, as pregnant heifers.

Some words of caution must be given concerning the use of the average ambient temperature or THI of the day as an estimator of heat stress of cattle. It may lead to some erroneous conclusions if the diurnal spread between maximum and minimum ambient temperature is greater than 12°C or the minimum temperature is below 18°C. As the daily minimum declines below 21°C the ability of the average Bos taurus cow to lose excess stored body heat increases considerably, especially at lower humidities. Conversely, as
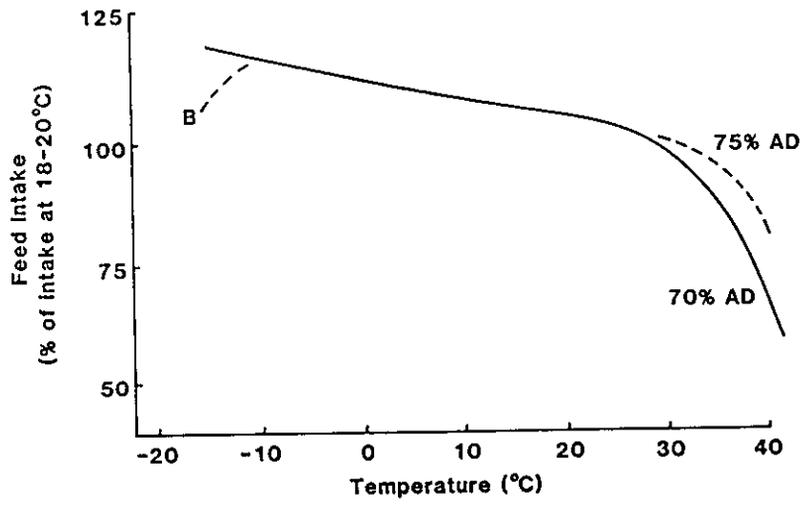
Figure 3.14 – Estimated changes in dry matter intake by feedlot cattle on a ration with 70% apparent digestibility or at temperatures above 27°C with 75% apparent digestibility (AD); "B" indicates behavioural changes (NRC, 1981)
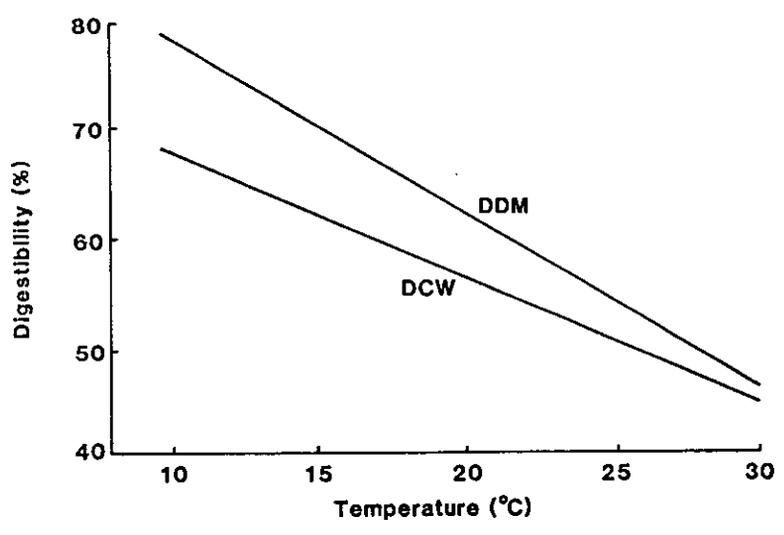

Figure 3.15 – Changes in the digestibility percentage by ruminants of the plant digestibility of dry matter (DDM) and the digestibility of cell wall (DCW) with changes in ambient temperature from 10°C to 28°C (NRC, 1981)
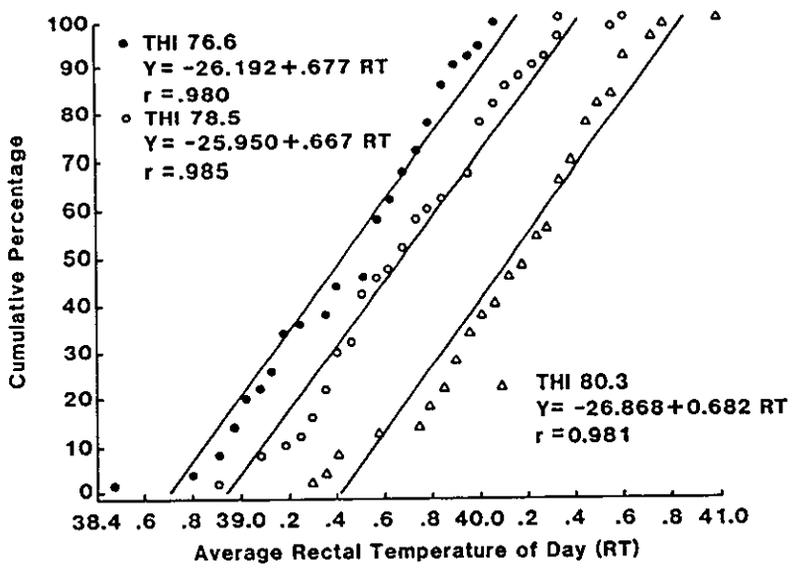

Figure 3.16 – Cumulative distribution curves for rectal temperatures (average of 0230 and 1330) of 49 cows at average daily THI of 76.6, 78.5, and 80.3 (after Ingraham, 1968)

TABLE 3.3

Ambient temperature effects on feed digestibility in cattle

Description of animals	Component	Temperature range, °C Low	High	Change in digestibility coefficient per 1°C	Reference source
Crossbred calves	Dry matter	17	38	+0.16	Colditz & Kellaway, 1972
Brahman calves	Dry matter	17	38	+0.13	Colditz & Kellaway, 1972
Friesian calves	Dry matter	17	38	+0.17	Colditz & Kellaway, 1972
Crossbred calves	Energy	17	38	+0.03	Colditz & Kellaway, 1972
Brahman calves	Energy	17	38	0	Colditz & Kellaway, 1972
Friesian calves	Energy	17	38	−0.04	Colditz & Kellaway, 1972
Steers	ADF	17	32	+0.38	Warren et al., 1974
Steers	Nitrogen	17	33	+0.17	Warren et al., 1974

Source: NRC, 1981

TABLE 3.4

Weight losses in terms of per cent shrinkage for beef cattle[1] fasted for 48 hours under controlled conditions in the Missouri Climatic Laboratory

Environmental conditions		% Shrink (cumulative) Hrs.[2]:	7	24	31	48	Hrs. to recover initial weight[3]
Temp.	Rel. Humidity						
21°C	50-60%		1.8	5.3	6.2	8.5	29 hrs.
28°C	50-60%		2.3	6.4	7.9	10.6	6.5 hrs.
35°C[4]	50-60%		-2.3	-0.4	0.5	1.6	3 hrs.

Source: Hahn, et al., 1978

Notes:

[1]   Average body-weight of animals: 200 kg. Animals were all crossbred steers with normal spring hair coat

[2]   Elapsed time after feed and water removed

[3]   Time needed to recover body-weight at start of shrinkage observations, after feed and water returned at 48 hrs; obtained by linear interpolation from observed weights

[4]   Feed withheld, but water available ad lib at 35°C to assure survival

minimum ambient temperature increases above 21°C, cattle are less able to cool down at night and consequently do not attain a normal body temperature at any time during each 24-hour period. When this occurs the effects of heat stress on production and preproduction begin to become evident. Probably the simplest indicator from which to predict a drop in milk production is an increase in minimum ambient temperature to above 21°C, especially if the humidity is high. In a characteristic desert climate with a wide diurnal range, cattle will store considerable heat during the day but lose it at night. This kind of climate, even with a maximum ambient temperature above 40°C may not show a significant effect of heat stress on milk production or reproduction, especially if shade is available for the animals during the day and if they can radiate to the open sky at night. To repeat, the ability to lose body heat and attain a near normal body temperature for a time during each 24-hour period appears to be important with respect to the effect of heat stress on both productive and reproductive function. If body heat is not lost at night the heat production of the next day will continue to build on it and cause it to go higher. Ingraham, et al. (1979) reported from a study in Hawaii that the rectal temperature with which a cow started the day was more highly correlated with afternoon rectal temperature than with the maximum THI of the day.

A sudden onslaught of extremely stressing conditions can be devastating to lactating cows, even when the animals are acclimatized to hot conditions. An example is the effect of a tropical storm in August, 1977 on the first Holstein dairy herds in the Chino Valley of California, USA (Oliver, et al., 1979). The succession of adverse conditions resulting from several hot days (daily maximum air temperature exceeding 37.8°C) followed by rains and subsequent high humidities as the moisture evaporated resulted in more than 700 cow deaths.

Bovine meat production must include considerations of handling and transport for animals in hot weather to minimize product losses and quality deterioration. The Temperature-Humidity Index (THI) has been used for several years in the United States of America as a guide for the use of precautionary measures (LCI, 1970). Recommendations for action are based on forecast THI values for the day, categorized as follows:

THI < 70   — Normal; no heat stress precautions needed.

THI = 71-80 — Alert; be prepared to take extra precautions and don't leave a vehicle loaded with animals standing in the sun.

THI = 79-83 — Danger; additional precautions should be taken to avoid overexertion of animals during handling and overcrowding during transport. Use of sprinklers and fans in loading areas is advisable.

THI > 84   — Emergency; minimize animal handling and transport. If absolutely necessary, take precautions listed for danger and complete operations in early morning hours. Wet down transport vehicle and bedding before loading and at any stops made during shipment; position vehicle so that breeze will help cool the animals, and park in any shade available.

Provision of water is particularly important in hot weather to minimize shrinkage of slaughter animals or to minimize the recovery time to regain pre-handling weight of feeder cattle. Weight losses at various temperatures as a function of time, along with recovery times to regain initial weight, are shown in Table 3.4 (Hahn, et al., 1978). These results are in reasonable agreement with weight losses measured at the end of a 36-hour truck shipment by Harston (1959)

The resistance of domestic animals to infectious diseases is adversely affected by extremes of temperature. Modern, intensively managed production facilities for livestock in many cases removed the impact of cold extremes by enclosed housing and adequately designed ventilation systems. In such facilities, high ambient temperatures tend to create more problems. One problem is the altering of host resistance (see also Section 3.5). Exposure to hot ambient temperatures reduces absorption of colostral immunoglobulins in newborn dairy calves (Stott, et al., 1976). Calves that have obtained passively-acquired antibody immunity via absorption of colostral immunoglobulin and are then exposed to hot environments have lower levels of serum IgG_1 (Kelley, et al., 1982b), although older calves raised in tropical climates may have higher serum IgG_1 as a result of active antibody synthesis (Curtain, 1971). Recent experiments with three-week-old Holstein bull calves indicate that heat exposure also suppresses cellular immune reactions (Kelley, et al., 1982a). Heat exposure may cause alterations in plasma factors that regulate lymphocyte blastogenesis in calves, but apparently does not directly alter lymphocyte blastogenic capability (Kelley, et al., 1982b). However, under longer natural summer conditions heat stress may directly enhance the mitogenic response of peripheral blood mononuclear cells (Soper, et al., 1978).

3.2   SHEEP AND GOATS - HEAT STRESS

3.2.1   General observations

Sheep and goats are kept over a wider range of climatic conditions than most other farm animals. Goats are more common in warm humid climates, while more sheep tend to be raised under warm dry conditions. Both are also well adapted to and are widely kept in temperate climates. Thus, they must also adapt to cold stress when they will suffer from heat stress more than those already acclimatized to hot climates. While many breeds of sheep and goats are adapted to high ambient temperatures, they still suffer from heat stress, and productivity is generally lower and mortality higher than for sheep and goats in the temperate zone. Reduction in voluntary feed intake and in reproductive efficiency are the main effects of heat stress. Effects of heat stress have been reviewed by Macfarlane (1964, 1976a, 1976b, 1982), Terrill (1968), Johnson (1976), Kamal (1976), Jindal (1980, 1984), and Singh and Rai (1981).

3.2.2   Heat tolerance

Sheep and goats have remarkable tolerance to heat: their body temperature is generally maintained at about 39°C, with a normal range from about 37.5° to 40.5°C. Body temperature generally increases under heat stress and may be associated with increased respiratory rates, heart rates, and water intake and loss. Goats, like sheep, reduce activity, seek shade and water, reduce feed intake, and generally lie down, while panting increases markedly under heat stress (Bianca and Kunz, 1978).

As temperatures increase, the body is able to adjust to accommodate a moderate rise in body temperature. Evaporation from the respiratory tract is the most important means of heat loss. Respiration rates in sheep may vary from rates of 20 to 50 up to over 300 per minute under heat stress. High humidity and high ambient temperature cause an increase in the respiratory rate that is related both to the rise in humidity and to the rise in deep body temperature (Bligh, 1963). Normal biphasic panting in response to severe heat stress in sheep is largely independent of arterial CO_2 tension (Maskrey, et al., 1981). Rectal temperatures increase as environmental temperatures increase, irrespective of CO_2 concentrations of environmental air, but decrease as the CO_2 increases (Tsuda, et al., 1973). Responses of respiration rate or panting may result from both central stimuli such as the temperature of the blood supply to the hypothalmus, or from peripheral stimuli to the skin. Such peripheral receptors are particularly sensitive, e.g. the inguinal region of the ram scrotum (Waites, 1962) or the udder of the goat.

Sheep sweating has some cooling value under heat stress especially in the shorn animal. Bligh (1961) has shown that the apocrine sweat glands of Welsh mountain sheep from time to time discharge their contents synchronously over a large area of the fleece-covered skin, resulting in a rise in skin temperature that frequently exceeds both ambient and deep body temperature. This discharge serves little or no thermoregulatory function and may be characteristic of a more primitive gland.

The wool coat protects the sheep against the heat of solar radiation. Parer (1963) observed merinos with varying wool lengths at a constant air temperature of 36°C and exposed to infra-red radiation of energy equivalent to that received from the sun. Respiratory rate and skin and rectal temperatures were inversely related to wool length. With wool length below 1 cm heat was conducted through the back wool very rapidly but increasing wool length beyond 4 cm gave little added protection

Body temperature of shorn sheep increases more rapidly wih high ambient temperature but also cools faster in shade, wind, or with lower environmental temperatures, than for unshorn sheep. Shorn sheep have lower respiration rates in shade and a higher critical temperature than unshorn sheep. Sheep are more heat tolerant in summer than in winter, and shearing may increase heat tolerance in summer. Apparently sheep improve in their ability to tolerate high temperature as their exposure to hot conditions is extended. Robinson (1969) found unshorn sheep were more heat tolerant than shorn sheep, and nutritional depletion reduced heat tolerance by a similar, if not greater, margin than shearing. Breed differences in heat tolerance were found by Singh and Rai, 1981, and Singh, et al., 1983, in higher body temperatures, pulse, and respiration rates in imported as compared to native breeds under heat stress.

The most noticeable effect of acute heat stress in sheep is an increase in the rate and volume of respiration. Rapid respiration may begin with a rectal temperature of 39.5°C and open mouth panting at 41°C (Lee, 1950). Death following muscular weakness and inco-ordination, tremors, and convulsions may occur at body temperatures above 42°C. The limit of heat tolerance in the goat is about 40°C when the heat regulatory system will fail (Appleman and Delouche, 1958). Wetting, wind, shade, and reduction in air temperature and humidity may bring relief.

Robertshaw (1982) has reviewed the effect of colour in the goat. Coat colour influences the amount of solar heat absorbed (Finch, et al., 1980) more being absorbed by black than by white animals. Goats tend to be white in the tropics but in the sub-tropical desert areas they are either predominantly or entirely black (Shkolnik, et al., 1972). This apparent paradox has been extensively investigated regarding its effect on heat tolerance (Finch, et al., 1980, and Dmi'el, et al., 1980). While black hair absorbs more solar radiation than white hair the penetration to the skin is a function not only of the colour but the structure of the coat (Hutchison and Brown, 1969). Thus, heat absorbed at the surface of black hair may be lost by convection given adequate wind speed (Walsberg, et al., 1978), while white hair reflects the heat inwards to the skin (Hutchison and Brown, 1969). Finch, et al. (1980) showed that black goats absorbed more heat than white goats. The extra heat was not stored but was lost through evaporation. The apparent paradox has been explained by the benefit to the animal in winter of the sparing radiation effect on its meagre energy reserve.

3.2.3   Temperature regulation

Evaporative temperature regulation in sheep has been reviewed by Johnson (1976). Increased respiration rates and thus heat loss from water evaporation, are the chief mechanisms for controlling body temperature under hot conditions, though water is also lost through the skin and the digestive tract. Panting and sweating lead to loss of body heat so long as the heat of vaporization of water is drawn from the body and not from the environment. Under heat stress, effective evaporation occurs mainly from respiration while loss from the skin may be small and unimportant. Loss through faeces was related to the level of food intake and not to air temperature (Degen and Young, 1981).

The sheep, in hot weather, can reduce its brain temperature by panting whilst the temperature of the remainder of the body is more slowly reduced (Rawson, 1976). This selective panting prevents cerebral overheating and may be a partial basis for the sheep's high heat tolerance. The cooling of cerebral arterial blood by means of the carotid rete located in the cavernous sinus has been described by Daniel, et al. (1953) and Baker and Hayward (1968a and 1968b). Experimental cooling of the hypothalmus will reduce panting even when the whole animal is under heat stress. However, there appears to be a hierarchy of heat regulators of which the hypothalmus is the most senior. In spite of a lowered brain temperature, sheep continue to pant until the body temperature is reduced. Other temperature regulators can be found in the skin and spinal cord. The skin receptors monitor the external environment and provide an immediate warning of external heat stress. The spinal neurones are stimulated by the heat transferred by the blood and pumped to the spinal cord. Intro-abdominal thermoreceptors using afferent pathways in the splanchnic nerves can increase respiratory heat loss in a hot environment and can be demonstrated experimentally when intro-abdominal temperatures are changed (Rawson, 1976).

While sheep incur evaporative heat loss in all environments, effective evaporation is principally from the respiratory tract and not from the skin. However, removal of the fleece at hot summer temperatures is traumatic. Macfarlane (1976a) observed that the surface of some merino fleeces reached 85°C, or higher, while the wool protected the skin from the long wave solar radiation. Rectal temperatures of sheep in fleece in the sun were 39.9°C and respiratory rates averaged 137 per minute. After shearing,

they no longer grazed during the day but actively sought even minimal shade with panting rates up to 200 per minute. The average rate was 178 per minute compared to 125 per minute of control unsheared sheep in the same pen. The surface of the shorn coat (6-10mm long) was 46°C, the skin 42°C and the rectal temperatures averaged 40.5°C. During the day, the sheep became increasingly sunburned and doubled their water intake. Shearing more than doubled energy and water turnover and increased the extracellular volume. The blood circulation compensated for the extra heat and water flow by increasing plasma and interstitial volumes to prevent dehydration and shock. Sunburn is a serious hazard of summer shearing as ultra-violet radiation penetrates to the skin, which can become necrotic, plasma ooze then adds to the general loss of fluid and death sometimes occurs.

Blood flow in non-respiratory muscles decreased during mild heat stress and increased during advanced stages of severe heat stress (Hales, 1973). This was probably part of an overall redistribution of cardiac output to combat heat stress. Panting during initial stages of heat stress is primarily diaphragmatic, but there is a significant thoracic component during advanced stages of severe heat stress. While the metabolic rate of respiratory muscles increases to perform the work of panting, the effect on total body metabolic rate is offset by a decrease in the metabolic rate of other tissues.

3.2.4   Water metabolism

Water loss is vital to temperature regulation under heat stress: accordingly water intake and conservation are essential to well-being and survival under hot weather stress. Total body water in sheep may vary from 50% in a cool temperate region to 68% in the arid tropics. The distribution of body water will vary with the temperature and the season as well as with the kind of feed and the physiological conditions of the sheep. Water of the alimentary tract makes up about 5 to 14% of body-weight, of the plasma 5 to 9%, and that of extra-cellular fluids from 21 to 43% (Macfarlane, 1964). Some water is produced in the body from oxidation in the tissues. Water deprivation (watering only once in 96 hours) did not influence lamb growth but did did result in a decline in milk yield and body-weight (More and Sahni, 1980).

Generally water needs rise as the ambient temperatures rise. Water intake may increase as much as 50% when ambient temperatures rise from 15°C to 20°C. The water temperature has little influence on water consumption but intake is closely related to feed intake. As water content of sheep responds to pasture composition (Macfarlane, 1976b) lush pasture will increase the animal's water content. A hot climate will also increase the water content mainly in extra-cellular components. When water turnover is increased by evaporative cooling or by the intake of salt, the total body water will increase. Water turnover is high in wet seasons and lower in dry and more water will be used by temperate zone sheep than by tropical sheep. When drinking water is restricted, Marwari sheep conserve their body water by reducing their rate of respiration despite rising ambient temperatures (Taneja, 1965).

Water loss through urine can vary from 1.5% of body-weight per day in summer to 4.7% in winter for merino sheep in the dry tropics (Macfarlane, et al., 1958). Daily water loss through the faeces is about 1% to 3% of body-weight. Evaporation would largely account for the remainder of a total loss of about 10% of body-weight per day in non-lactating sheep. Loss from

the skin is estimated to be about one-eighth of that of respiration. When deprived of water, sheep lose from 5% to 7% of body-weight per day for the first 3 days, depending on ambient temperature. This is reduced to 2% to 3% for subsequent days. Gut and cell water provide about half of the loss. The best adapted animals lose only 3% to 4% per day initially with relatively high urine and faecal water output. Survival is more likely when a slow rate of water excretion is combined with low potassium and high sodium secretion. Water content of the faeces falls to 45%. Low water excretion is probably the most important single factor of adaptation to desert conditions. When fluid is lost, circulation is maintained at low volumes (Macfarlane, 1964).

Urine output increases during hot-humid and hot-dry exposure but can be 70% greater during the hot-humid than during hot-dry exposure. Urine osmolality decreases by 38% during the hot-humid and 22% under the hot-dry exposure. Urine output and probably plasma volume increases during these exposures because evaporative heat loss is reduced due to lack of adequate air convection and mobility (Guerrini, et al., 1980).

During drought sheep eat little and, as their fat reserves are used up they lose body-weight. Their total water turnover may be less than three litres per day. Undernourished sheep grazing on dry grasses drink little more liquid than that needed for urine and respiratory losses. In mountains and the tropics little sodium alderstone is secreted so as to retain renal sodium and maintain extra-cellular volume. In deserts there are many plants that accumulate sodium and some that store potassium. When grazing on these plants, sheep will drink more water in proportion to the salt load, which is excreted. If water is unavailable, either the sheep will not eat or the nervous system will be damaged by the sodium load. Sheep have been poisoned by 2% sodium chloride in drinking water but can live satisfactorily on 1.3% to 1.5% concentrations. Breeds differ as to their tolerance of salt and individual sheep can vary greatly. Goats are more salt tolerant than sheep (Macfarlane, 1982).

Robertshaw (1982) has outlined the specific environmental adaptations of the goat to a hot desert environment. These include (a) the ability to desiccate the faeces, causing faecal water loss to fall by about 60%; (b) the ability to concentrate the urine and thereby reduce urine volume by as much as 50%; (c) the ability to reduce evaporative water loss including a reduction in metabolic rate; and (d) the use of the rumen as water reservoir.

3.2.5   Energy metabolism

Energy metabolism during heat stress has been reviewed by Knox (1976). Activity and feed intake add to heat production, and, therefore, tend to be reduced under high environmental temperatures. Sheep and goats tend to rest in the shade during the heat of the day. Their metabolic rate tends to fall under heat stress and heat storage may occur. The rise in body temperature with heat storage may be fatal if additional methods of reducing body temperature by an increased rate of respiration and sweating cannot hold body temperature within the normal range.

Sheep will voluntarily reduce their feed intake and thus its calorigenic effects during hot weather. Food energy may be retained or eliminated from the body. Energy may be lost in faeces but faecal energy losses tend to decrease at high environmental temperatures probably as a result of the reduced feed intake. Urinary energy losses are inconsistent.

Some energy may be retained as body substance or stored, the latter resulting in a rise in body temperature.

Bhattacharya and Hussain (1974) found that heat stress decreased feed intake, increased water consumption and urine volume, lowered the digestibility of dry matter, crude protein, ether extract, and energy but did not affect the digestibility of crude fibre and nitrogen-free extract of rations. The metabolizable energy of rations was also depressed and the maximum effect was in the 75% roughage ration.

Heat production by sheep in the thermoneutral zone is dependent on the quality and quantity of their diet. As the environmental temperature rises above the thermoneutral zone, heat production continues to decrease but at a lower rate, and then increases. This increase in heat production occurs once the animal is unable physiologically to dissipate the heat. Death from heat exhaustion will occur if not relieved. Accordingly the lower calorigenic capacity of the diet, the better sheep are able to handle heat production. Although individual sheep handle the problem by reducing intake, the actual mechanism is not well understood. The overall result is that at high environmental temperatures, dietary energy is generally not sufficient to allow for energy retention (Knox, 1976). Average daily gain and protein efficiency ratios decrease under heat stress (Ames and Brink, 1977). Altering protein intake to match the thermal environment seems indicated.

A high plane of nutrition tends to increase heat production in sheep, so that in hot atmospheres those on a high plane of nutrition will show a greater increase in respiration rate. High protein diets, however, have little effect on reactions to high temperatures. While high planes of nutrition may reduce heat tolerance, low planes of nutrition result in impairment of heat-regulating mechanisms so that sheep on a medium plane of nutrition can most effectively adjust to high temperatures. Starvation lowers resistance to both cold and heat because of lowered heat production and interference with the normal physiological mechanisms for adjusting to temperature change. Sheep exhibit their adaptability to a marked increase in temperature by the natural decrease in appetite.

The effect of temperature on the thyroid function appears to be initiated at the hypothalmic level. The voluntary restriction of feed intake under heat stress tends to reduce plasma thyroxine levels, but temperature per se provides an additional depression (Valtorta, et al., 1982).

3.2.6   Reproduction

Reproduction in either sex is adversely affected by hot weather (Alliston, 1976). Adaptation of breeds to heat stress from hundreds of years of natural selection is essential in tropical or sub-tropical climates. In the male, the testes should be several degrees cooler than normal body temperature for normal function and high fertility. The greatest sensitivity in females is in the early stage of gestation, but dwarfing may result from continuous high temperatures throughout pregnancy.

Decreased spermatogenesis in the ram during the summer months (McKenzie and Berliner, 1937) has also been observed under controlled temperature facilities and studied in detail (Dutt and Hamm, 1957; Moule and Waites, 1963). Seasonal changes in semen quality in the tropics were reviewed by Sahni and Tiwari (1981) and were reported for both sheep and goats by

Shukla and Bhattacharya (1953), Singh and Roy (1963), Ramamurty (1963), Mishra and Sengupta (1965), and Sinha, et al. (1979), but good quality semen was obtained throughout the year. Dutt (1954) found that low reproductive performance of ewes bred early in the breeding season (August and September) was principally due to failure of fertilization. Temperatures above 26°C appear to be the threshold for rams, but there is marked individual variation among rams in the tropics. Several days of very hot weather can cause complete seminal degeneration for many weeks, while shorter exposures to less extreme heat would cause only moderate damage, such as more abnormal sperm and a lower conception rate. A lag of about two weeks may occur between overheating and the first appearance of affected sperm, and as many as six weeks of freedom from excess heat may be required for full recovery.

Dutt (1960) summarized results in which ewes were subjected to 32°C at different stages around the mating season. A significant increase of unfertilized and abnormal eggs occurred when treatment was initiated prior to mating. The embryo loss did not differ significantly from control females when treatment was initiated prior to mating. The embryo loss did not differ significantly from control females when exposure to the high temperature was delayed until eight days post-breeding. Ewes exposed to high temperatures immediately after ovulation showed a significant increase in embryo mortality. Eggs following ovulation, but before entry into the uterus, were generally sensitive to high ambient temperatures.

The number of ewes showing oestrus was significantly reduced by about one-third following heating. However, most ewes ovulated. Exposure to high radiant heat load, either natural or artificial, at the time of insemination, contributed to reproductive wastage from ewes that mated, apparently conceived, but failed to lamb (Sawyer, 1979).

In the later stages of gestation in sheep, there will be a significant depression of birth weight and lamb survival at high temperatures. High temperature stress during late gestation can cause foetal stunting and increased mortality at birth (Yeates, 1956, 1958; Shelton, 1964; Goode, 1964, Alexander and Williams, 1966, 1971). Ewes under heat stress for 12 hours were essentially intermediate in birth weight and survival between unstressed ewes and those stressed for 24 hours (Shelton and Huston, 1968). If there had been a daily respite from the high temperatures, the effects on female reproduction would be migitated and reproduction not significantly decreased. Acclimatization also contributed to diminished effects (Alliston, 1976).

Birth weight and the weight of the placenta are considerably reduced by heat stress during middle and late pregnancy, and the reductions are more than can be accounted for by the partial loss of appetite arising from heat stress. Alexander and Williams (1971) showed that there was a close inverse relationship between birth weight and high rectal temperature of overheated ewes, but there was no foetal dwarfing in ewes with elevated rectal temperatures due to daily heating for nine hours at 44°C, indicating that higher body temperature was not the main cause of dwarfing. They also found that dwarfed lambs were not proportional miniatures, for the head components, body lengths, kidneys, and adrenal glands were disproportionately large in heated lambs, while the liver, thyroid and thymus glands, and the biceps femoris muscle were disproportionately small, and the ratio of secondary to primary wool follicles was very much reduced. There were cavities in the white matter of the cerebral hemisphers of heat-dwarfed lambs. Stunting of

the placenta also occurred from heat stress, but under some circumstances a heat-stunted placenta was capable of considerable growth in the absence of heating during the final third of pregnancy. Then the placenta normally shrinks and that shrinkage may be greatly accelerated by heat.

Cartwright and Thwaites (1976a) found that simple maternal under-nutrition was not a cause of foetal stunting in heat-stressed ewes, though the proportions of affected lambs closely resembled those of nutritional dwarfs. The adverse effects of high temperature appeared to arise from an extreme form of foetal under-nutrition. Cartwright and Thwaites (1976b) also found that the total number of secondary wool follicles tended to decline in unheated ewes with a decline in the level of nutrition and in the heated group was significantly reduced by a further 30%. Brown, et al. (1977) heated ewes during the last third of gestation at 30°C (day) and 28°C (night) for 25 and 53 days. Small lambs from heated ewes were proportional dwarfs and occurred independently of the level of nutrition of the ewe.

Environmental heat stress in tropical sheep suppressed lamb birth weight, but the animals that did not experience hyperthermia under natural environmental conditions gave birth to significantly heavier lambs than their less adapted counterparts. Heat stress during the last month of pregnancy, without nutritional intervention, significantly retarded foetal growth and maturation of wool follicles. Severe nutritional restrictions during the last three months of pregnancy also caused a significant reduction in lamb birth weight, but this difference was not so marked as that from heat stress (Hopkins, et al., 1980).

Shelton (1964) explained that foetal stunting or dwarfing would appear to be a nutritional deficiency of the developing foetus due to a reduced blood supply to the uterus. The latter would be expected to occur due to increased peripheral circulation as a means of dissipating the increased heat load. Suppression of placental development (Alexander and Williams, 1971) probably also resulted from reduced uterine blood flow. Roman-Ponce, et al. (1978) reported reduced mid-uterine artery blood flow in response to heat stress in non-pregnant ovariectomized ewes. Brown and Harrison (1981) showed that the central sympathetic transmission was not a major component in the control of uterine blood flow during acute heat stress.

3.2.7   Breed adaptation and disease

Many breeds of sheep and goats have become adapted to hot weather stress, especially those that evolved in the tropics. Those which evolved in the temperate zone, such as European breeds, often could not survive in the tropics because they could not adequately control their body temperature under the hot conditions. Crosses with native breeds might be misleading, as hybrid vigour tends to cover up the lack of adaptability (Terrill, 1982). Importation of exotic breeds should be based on survival and high productivity of the exotic breed in the new environment rather than on the performance of crossbreds. Devendra and Burns (1979) found that heat tolerance was critical for introduced breeds from cooler environments, since a failure to cope with the increased heat load led to a rapid decline in their performance.

Native breeds of India are generally well adapted to hot conditions (Acharya, 1982) and do better than imported breeds (Singh and Rai, 1981). Singh and Roy (1963a) reported that Corriedales had higher pulse and

respiration rates than Bikaneri sheep during hot dry and hot wet seasons. Soviet Merinos had higher body temperature, pulse, and respiration rates than Marwari and Malpura sheep (Taneja, 1966). On exposure to the sun, the Rambouillet showed a marked increase in respiration rate, the Malpura a slight increase and crossbreds were intermediate (Singh and Acharya, 1977).

The small black Bedouin goat, watered only once in two to four days under hot conditions, still gave daily yields of over 2 kg milk from animals weighing 15 to 25 kg, and growth rates may exceed those of other breeds (Shkolnik, et al., 1980). Thompson, et al. (1981) found that in Friesland sheep after heat stress milk secretion was reduced, the concentration of total N increased and the prevalence of short-chain fatty acids in milk fat also increased.

Breeds developed in Europe are sensitive to changes in day length so that the breeding season tends to start as the days shorten and carries on through the cooler weather into the winter. Tropical breeds are less reactive to photoperiod and, therefore, have a more extended or year-round breeding season. To survive, these breeds have had to adapt to higher temperatures and over a longer period. The Merino breeds, including the Rambouillet, evolved in Southern Europe and seem to have a wider range of adaptability than either the Northern European or tropical breeds. They also have a longer breeding season than many of the other European breeds with the exception of the Dorset. The migratory situation for the Merino may have contributed to their adaptability to a wide range of particularly hot, dry conditions such as in much of Australia and Patagonia. Hairy breeds such as the Barbados Blackbelly are well adapted to the extreme hot, humid areas of the tropics.

Effects of heat on diseases in sheep have been reviewed by Hugh-Jones (1976) although diseased animals are better referred to as suffering from ill health. Certain diseases are claimed to be associated with hot weather or seasons, but with scant evidence. Parasitism is generally more of a problem under hot humid conditions. Generally it is difficult to separate the problems of poor husbandry and management, inadequate nutrition and parasitism, all of which are common in the tropics, in order to identify specific causes of ill health.

3.2.8   Management

Proper management is most important at lambing time when lambs must be protected from heat stress. Lambs are very susceptible to excess heat (Smith, 1961; Morgan, et al., 1972) and are most susceptible to hyperthermia at birth, but tolerance improves with age. With time, there is a progressive drop in rectal temperatures. Merino lambs in shaded pens with an air temperature of 32°C can have rectal temperatures of 40.6°C. Further rises in temperature follow short periods of exercise or exposure to direct sunlight. With air temperatures over 37°C, half or more of the newborn lambs in unshaded pens will die, while none or few will die in nearby shaded pens. In Northern Australia where there is frequently little natural shade or water, and ewes need to walk long distances to find grazing and water lambs will collapse with heat prostration after following their mothers for some 200 metres in the sun. However the provision of adequate water can markedly reduce lamb mortality. Milk yield of the heat-stressed ewe may not decline in the first two weeks of lactation but generally does later (Stephenson, et al., 1980).

Generally rams and ewes should be shorn before breeding and ewes sometimes before lambing, leaving at least 4 cm of wool on sheep exposed to strong solar radiation. In humid climates wool may be shorn closely, as radiation is seldom so important. To maximize local cooling, woolled scrota should be shorn. Rams should never be allowed to become overheated in the breeding season and thus should always be moved carefully. All sheep should be provided with ready shade by the strategic planting or preserving of trees or by the provision of artificial shade (Yeates, 1976).

3.2.9   Sheep and goat comparisons

McDowell and Woodward (1982) have compared sheep and goats in relation to their adaptation to hot climates. Their body size and surface area are nearly the same but goats have fewer hair follicles and sweat glands per unit of body surface, lower subaceous gland secretion rate, and less body fat covering than sheep. Goats' skin is firmer, more elastic, and less spongy than sheep's. With a more open hair coat, thinner skin, and low fat covering, the goat will heat and cool more rapidly than sheep. Goats appear to have lower metabolic heat production, higher tolerance to dehydration, less susceptibility to respiratory alkolosis from high respiration rates, and fewer metabolic disorders than sheep. Goats may have advantages over sheep where water availability is low.

3.3   PIGS - HEAT STRESS

3.3.1   General observations

The large surface-to-volume ratio of new-born pigs, with a resultant high rate of heat loss, leads to fewer problems as a direct result of hot conditions. Indirect problems can be encountered, such as reduced milk flow in the sow. For larger pigs, heavier than about 25 kg, heat stress can be a problem at conditions of 30°C and 50% relative humidity or above (Close and Mount, 1978). Factors such as acclimatization are therefore extremely important as heat stress can occur suddenly, seasonally or continuously. Thermal radiation, air movement and humidity can alter the way animals react to high temperature. Moreover, environmental temperature is not normally constant; the responses of pigs to fluctuating temperatures are important considerations (Bond, et al., 1967).

Since humidity has a strong effect on pigs at higher air temperatues there have been several attempts to combine the two factors in a single index comparable to the temperature-humidity index for assessing human comfort. Beckett (1965) developed an "effective temperature" for pigs based on the assumption that the amount of air moved by the lungs was an indication of discomfort. Roller and Goldman (1969) determined the weight to be given the wet-bulb temperature in a wet bulb/dry bulb index by determining correlation coefficients over a range of wet-bulb temperatures when related to various physiological responses to acute heat stress. They determined the Wet-bulb/Dry bulb Index = $0.25T_{wb} + 0.75T_{db}$ to be the best formula. This compares with $0.35T_{wb} + 0.6T_{db}$ as suggested for the best index for young pigs by Ingram (1965). Morrison, et al. (1968) developed a semi-theoretical relationship for predicting the rate of weight gain of swine for any combination of temperature above optimum and humidity. This index expresses the temperature-humidity relationship in terms of actual weight gain and appears validated by the tests described above (Morrison, et al., 1969). The results are given in Figure 3.17 where the gain reduction factor is the
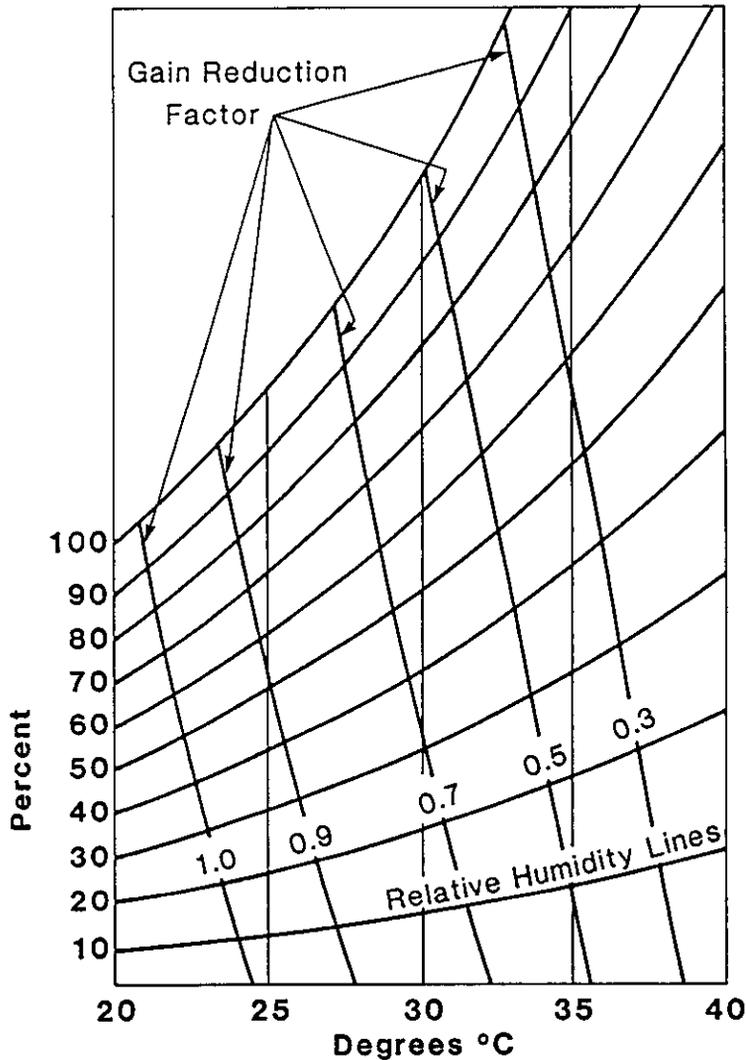
Figure 3.17 – Graphical representation of rate-of-gain of swine as a function of temperature and humidity. The Gain Reduction Factor refers to the fraction of gain which would occur under optimum conditions

fraction of the gain which would occur under optimum conditions. The weight given to the wet-bulb temperature in this relationship is close to 0.25, so the fraction detemined by Roller and Goldman from short-term physiological measurements is identical with that determined by long-term production tests. However, as will be pointed out later, the decline in gain with increasing temperature described by the Morrison relationship is greater than that observed in other experiments.

3.3.2   Reproduction and lactation

Summer infertility is a common problem in pigs, particularly in areas that experience marked seasonal fluctuations in temperature. The problem appears to be related to four classes of stock: the recently-mated sow, the sow during the final days of gestation, the postpartum, postweaned but unbred sow, and the boar. An example of seasonal variations in reproductive performance is shown in Figure 3.18, based on data representing 1,453 matings over a four-year period. Farrowing rates ranged from a low of 58% for sows bred in August to a high of 86% for those bred in March. Similar effects of season on farrowing rates have been found in France (Corteel, et al., 1964; Herak, et al., 1974). The decrease in farrowing rates could be related to the average daily maximal temperature. However the lowered farrowing rate in sows could be an influence by either the boar, the sow or the combined effects of high temperature on the boar and sow. These influences will be considered on the basis of available research information.

The adverse effects of high temperature appear to be most severe in gilts (Wells, 1977), and less critical prior to breeding than during early gestation when a reduction of embryonic survival occurs in sows subjected to a temperature of 37°C (Tompkins, et al., 1967). Teague, et al. (1968) and d'Arce, et al. (1970) have shown that gilts exposed to high temperatures during the oestrus cycle before breeding had reduced oestrous activity and, after breeding, a reduced pregnancy rate. Several studies have shown that embryo survival is adversely affected if gilts and sows are exposed to hot environmental temperatures immediately after breeding (Warnick, et al., 1965; Tompkins, et al., 1967; Edwards, et al., 1968; Omtvedt, et al., 1971). Omtvedt, et al. (1971) found a tendency for lower conception rates and a highly significant reduction in the number of viable embryos in gilts subjected
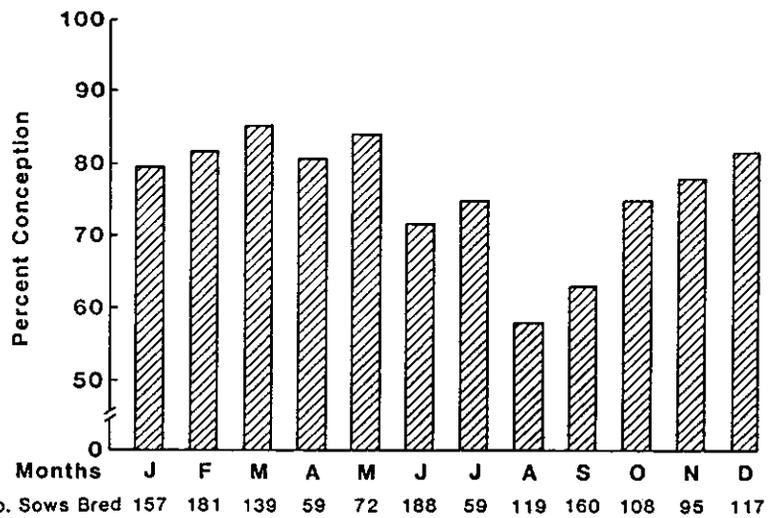
Figure 3.18 – Seasonal variation in percentage conception in the Iowa State swine nutrition herd, 1954 to 1957 (adapted from Teague, 1970; based on data provided by V. C. Speer)

to a cyclic elevated temperature, either 0 to 8 days or 8 to 16 days after breeding. Only 8 of 14 gilts in the 0- to 8-day exposure group and 11 of 14 gilts in the 8- to 16-day exposure groups and 28 of 28 in the control groups were pregnant at 30 days. Of the gilts that were pregnant, the number of viable embryos was lower in the gilts exposed to cyclic increased temperatures from 8 to 16 days after breeding than in gilts exposed from 0 to 8 days after breeding. Apparently conception and subsequent embryonic survival were adversely affected by increased temperatures during early implantation (days 12 to 16).

While gilts are less sensitive to heat stress from 20 days after breeding until midpregnancy, heat stress during late gestation can have strong adverse effects. Gilts exposed to heat stress during late gestation (102 to 110 days after breeding) farrowed fewer live pigs (6 vs 10.4 pigs), more stillborn pigs (5.2 vs 0.4 pigs) and litters with lower birth weights (8.6 vs 13.6 kg) than did the control gilts (Omtvedt, et al., 1971). Exposure of gilts to elevated environmental temperatures and the resulting increase in rectal temperature at this stage of gestation may have had a more direct foetal effect than at other stages of gestation. This suggests that while heat stress is detrimental to piglet production at all stages of gestation, near term foeti may be extremely sensitive to heating in utero and that late gestation heating may be of greater concern to the swine manager.

Research data on the physiological mechanism by which hot environmental temperature and high humidity reduce fertility in the sow are

limited (Christenson, 1980). Exposure of the gilt or sow to high temperatures could reduce fertility by two ways: the direct effect of increased body temperature, i.e. a higher uterine temperature, directly affecting the gametes or the product of fertilization (the most convincing data to support this direct theory have been reported in species other than the pig (Dutt, et al., 1959; Howarth, et al., 1965)); the second, the indirect influence of hot environmental temperature and high humidity on gametes and embryos by altered endocrine and uterine function.

Hot environmental temperatures combined with high humidity can alter pituitary, ovarian and adrenal hormone concentrations (Gwazdauskas, et al., 1972); Madan and Johnson, 1972; Mills, et al., 1972; Wagner, et al., 1972; Alvarez and Johnson, 1973). Changes in plasma progesterone concentration or progesterone to estrogen ratios may be involved in reduced fertility in the sow. The reproductive tract environment at oestrus and during the oestrus cycle or early pregnancy is influenced by these two ovarian hormones. An increase in the secretion of progestrone or a possible change in the progesterone to estrogen ratio as a result of temperature and humidity stress could alter the transport of sperm (Hawk and Conley, 1971), increase the speed of transport of fertilized ova through the reproductive tract (Chang, 1966) and alter secretions of the porcine uterus (Beier, et al., 1971; Knight, et al., 1974; Bazer, 1975). In addition, progesterone and estrogen appear to be the principal hormones associated with the regulation of blood flow to the reproductive tract of the sow (Ford and Christenson, 1979).

Seasonal infertility in postpartum postweaned sows has been described in detail (Herak and Sviben, 1974; Herak, et al., 1974; Sviben, et al., 1974; Hurtgen, 1976; Hurtgen and Leman, 1978; Love, 1978; Paterson, et al., 1978; Stork, 1979. Hurtgen(1976) reported that postweaning oestrus was commonly observed within seven days in sows weaned from November to May; however, 20 to 50% of the sows failed to show oestrus seven days after weaning during July to October. In this study, failure to show oestrus after weaning was more common in first-litter sows than in older sows. Coupled with the failure of sows to show oestrus seven days after weaning, a greater percentage of sows had not shown oestrus 30 days after weaning during August, September and October than during the cooler months and were classified as anoestrus. Examination of reproductive tracts from such sows revealed small atonic uteri and small ovaries that lacked luteral tissue and usually contained 20 to 50 follicles less than 4 mm in diameter per ovary (Hurtgen, 1976). Such observations have been recorded during late summer and autumn in weaned sows by others (Maurer, et al., 1984, unpublished data). Conception rates to a first service 30 days after weaning were markedly lower during the months of July, August, September, and October (Hurtgen, 1976) than during the rest of the year. Similarly, weekly farrowing rates for the years 1971 to 1976 were significantly lower than an acceptable 85% farrowing rate as a result of matings during the summer (late January to mid-April) in studies conducted in Australia (Love, 1978). In the studies of Hurtgen (1976), Paterson, et al. (1978) and Love (1978), the return to oestrus after unsuccessful breeding was delayed (25 to 35 days) during summer and early autumn. Paterson, et al. (1978) reported that when the mean maximum temperature during the week of mating exceeded 32°C, the number of sows that had had long and irregular intervals before returning to oestrus increased. Results of these studies suggest that hot temperatures prepartum or postpartum result in delayed postweaning oestrus, extended periods of anoestrus, lowered conception rates, delayed return to oestrus after mating and reduced farrowing rates.

In the postpartum postweaned sow, delayed oestrus and extended periods of anoestrus characterized by small inactive ovaries suggest an endocrine imbalance resulting in a hypothalmic-hypophyseal insufficiency. But the endocrine aspects of the problem are just beginning to be explored (Stevenson, et al., 1981). Low energy diets fed to first-litter sows during lactation (Reese, et al., 1980) and exposure to hot environmental temperature postweaning (Christenson, et al., 1984), unpublished data) have resulted in a delayed return to oestrus in first-litter sows.

When boars are exposed to heat stress there is a decrease in sperm quality: at 30 days after natural mating only 59% of gilts bred to heat-stressed boars were pregnant compared to 82% of gilts mated to control boars (Wettemann, et al., 1979). In previous work Wettemann, et al. (1976) had observed that boars exposed to 34°C for eight hours and 31°C for 16 hours each day for 90 days had, after two weeks, reduced sperm motility and semen quality compared with controls at 23°C. Only 29% of gilts bred with semen from heat-stressed boars conceived compared to 41% of gilts bred to control boars. At 30 days gestation embryonic survival was 71% in gilts bred with semen from heat-stressed boars. The problems of reduced semen quality in boars and of increased embryonic survival rates are only part of the overall deleterious effect of high temperature on reproduction; reduced libido is another (Wells, 1977).

Rectal temperature responses of the boars to temperature regimens of 21° and 33.3°C are shown in Figure 3.19. The average rectal temperature of all boars prior to the 72-hour exposure period was 38.1°C. During the 72-hour exposure period, the rectal temperature of the heat-stressed boars increased rapidly and remained significantly higher than the rectal temperature of control boars. Following the 72-hour treatment period, rectal temperature for the heat-stressed boars returned to near pre-exposure levels and was similar to rectal temperatures in control boars. Semen quality was similar and normal for all boars prior to the 72-hour exposure period. The first evidence of a detrimental effect of elevated temperature on semen quality occurred approximately two to three weeks after exposure. At this time there was a significant decrease in the total number and per cent of motile spermatozoa (Figure 3.20), and an increase in the percentage of abnormal spermatozoa. Semen quality continued to decline until four to five weeks after exposure. Based on semen evaluation, there was then a gradual return to pre-exposure values by eight to nine weeks after exposure. In control boars, semen quality measurements remained relatively constant throughout the entire experiment.

The effect of elevated temperature on semen fertility, as measured by pregnancy rate in artificially inseminated gilts, is presented in Figure 3.21 (Christenson, et al., 1972). Fertility data paralleled semen quality measurements during the pre- and post-exposure period for control and heat-stressed boars. Pregnancy rate was considered normal for gilts inseminated with semen collected from both groups of boars during the pre-exposure and first two weeks post-exposure. However, beginning two weeks post exposure, the percentage of pregnant gilts was markedly reduced after insemination with semen from the heat-stressed boars. During the 5th and 6th week post-exposure, the pregnancy rate was at the lowest point (56%) for gilts artificially inseminated with semen from heat-stressed boars. Eight weeks after treatment such an adverse effect on semen fertility had largely disappeared and the pregnancy rate (77.8%) had returned to near pre-exposure levels. No period of decreased reproductive performance, as measured by the pregnancy rate of artificially inseminated gilts, was observed for control boars. The pregnancy rate was significantly different for gilts inseminated
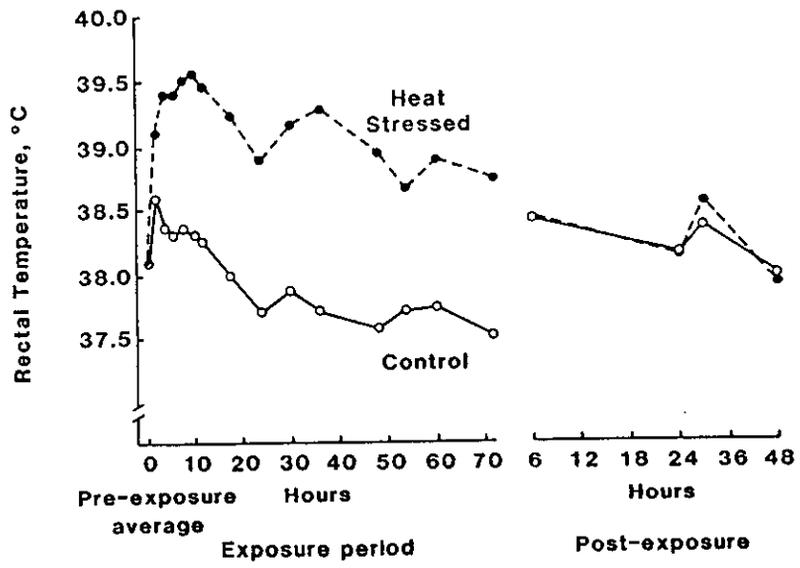
Figure 3.19 – Rectal temperatures of control and heat-stressed boars during exposure and post-exposure periods
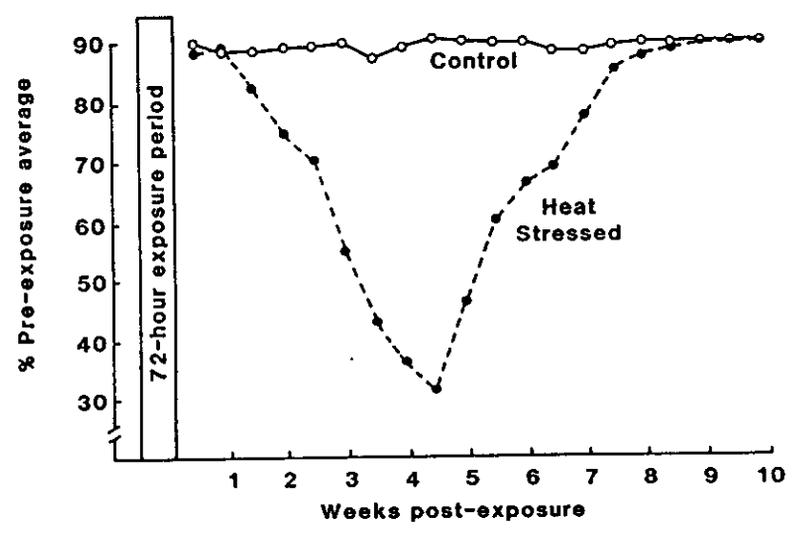

Figure 3.20 – Per cent of spermatozoa motility for control and heat-stressed boars
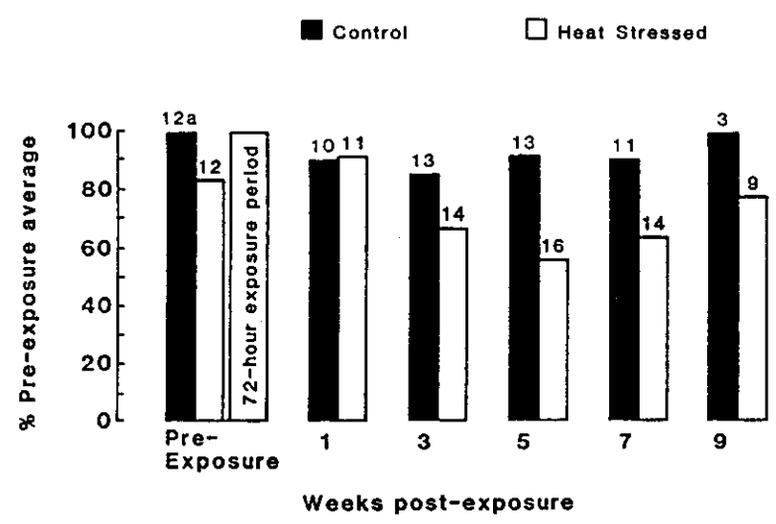

Figure 3.21 – Per cent pregnancy rates of gilts inseminated with semen from control and heat-stressed boars at various intervals after stressing; superscript the number of gilts inseminated

with semen from control and heat-stressed boars when evaluated for the entire post-exposure period (90% to 67%, respectively).

It is therefore apparent that short-term temperature stress on the boar does have a detrimental effect on measures of semen quality as well as on the fertility of semen. Such adverse effects were not immediate but were very evident three to four weeks after exposure, with a gradual return to near normal pre-exposure values nine weeks after heat stress. Thus, in practice, the ill effects of hot weather on boar semen quality and fertility can be expected to be delayed as much as two to eight weeks after exposure.

3.3.3   Neonatal survival and growth

Mount (1959) found that newborn piglets had a critical temperature of 34-35°C; heat stress was therefore rarely a problem before homeothermy was fully established by 10-14 days of age. Slightly older pigs preferred lower temperatures (Mount, 1963). Exposed to a temperature range of 23-27°C in a thermocline box, newborn pigs were most often found in the 32-34°C segment of the box and 70% of the pigs tested preferred an ambient temperature of greater than 32°C. Piglets two to seven days old were found to prefer an environment with an average temperature of 29.3°C. This high "critical" temperature is necessary to newborn piglets but may represent a moderate to severe heat stress in older pigs. Additionally, the high thermal requirements of the newborn pig may impose a significant heat load on the sow. Heat stress in the sow would decrease feed intake and thus could reduce milk yield and subsequent piglet performance.

The air temperature at which the maximum growth rate of growing-finishing pigs occurs has been determined by several investigators: Heitman, et al. (1958); Wagenbach (1961); Sorenson and Moustgaard (1961); Holme and Cooey (1967); Mangold, et al. (1967); and Comberg, et al. (1967). As might be expected, the values vary with animal weight, type of housing and other thermal factors, but they are generally in the range of 15 to 20°C for growing-finishing pigs. The effect of constant temperature above optimum on growth of full-fed swine for periods of a week was determined by Heitman, et al. (1958). Their results were verified for periods of 12 to 14 weeks by Morrison, et al. (1969). The test conditions for the latter work included pigs on slotted floors with .07 m^2/head/floor area, relatively low air velocity (0.05 m/sec) and no opportunity for use of water for cooling. For pigs grown under commercial conditions to 90 kg live weight the depression in gain was 10 g per day per °C rise in mean environmental temperature over the temperature range 15 to 32°C (Vajrabukka, et al., 1981). Close and Mount (1978) observed a similar reduction of 11 g/day in pigs weighing about 35 kg and housed in calorimeters. Morrison, et al., (1969) used their results to develop the gain reduction relationship shown in Figure 3.17. That relationship was combined with hourly temperature and humidity measurements from a number of stations in the United States to predict production losses from June to September inclusive (Morrison, et al., 1970). Two examples of the resulting probablity curves are shown in Figure 3.17. The cumulative probablity is an estimate of the amount of time the gain reduction factor would be a given value or greater.

Mangold, et al., (1967) found considerably less decline in the rate of gain for comparable temperatures of pigs in production facilities. Pigs in their facility being able to keep the floor wet with liquids from waterers and body wastes may partly account for this result. The ability of pigs to compensate for supressed growth by an increased growth rate subsequent

to heat stress may also be a factor (Hahn, 1982). Nelson, et al. (1972) also found less decline in rate of gain for pigs exposed to naturally varying conditions than the Heitman-Morrison results. They attribute this difference possibly to the fact that their pigs were on a concrete slab which was cooler than the air temperature and to the alternation of periods of hot and cool days under natural conditions. Smith and Tonks (1966) compared pigs in "hot-house" environment (28°C, 92% R.H.) with conventionally housed pigs (21°C, 70% R.H.) and found a 27% higher growth rate for the latter group. In the light of the foregoing discussion the values in Figure 3.22 are probably the maximum reduction in gain that might occur with no alleviation of heat stress.

Diurnal cycles result in growth responses comparable to constant temperatures when the mean of the cycle equals the constant temperature, providing that the cycle range is not too great or the mean temperature too high. Pigs will withstand temperatures as high as 45°C for periods of a few hours if there are sufficiently cool temperatures the remainder of the time. The effect of diurnally cycling temperatures on growth has been reported by Bond, et al. (1963) and Morrison, et al. (1975). In the former study it was found that pigs weighing from 32 to 114 kg had higher weight gains at a constant 21°C air temperature than pigs under diurnal cycles of 4.5 to 38°C, 10 to 32°C or 15.5 to 26.5°C. The mean of all the diurnal cycles was the same
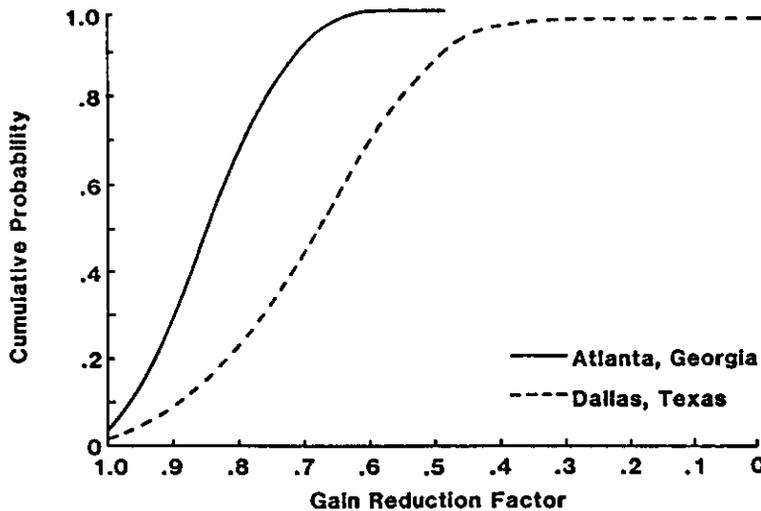

Figure 3.22 – The cumulative probability of gain reduction (the probability that the Gain Reduction Factor will be of a specified value or larger) for two US locations

as the constant temperature. In the second study, pigs weighing from 37 to 88 kg showed no difference in weight gains whether they were kept at a constant optimum temperature or at diurnal cycles within a range of 10 to 20°C with the mean at the optimum temperature. However, comparing constant temperature at 6°C above optimum and cycles with means also 6°C above optimum, the rate of gain declined significantly within a 20°C range though not within a 10°C range. The Bond study involved pigs with one-week test periods whereas the Morrison study used 36-day periods.

High humidity with increasing temperature has an increasingly detrimental effect on growth. As temperature rises the amount of heat capable of being lost by sensible means decreases so the proportion lost by evaporation increases. Bond, et al. (1952) found the percentage of total heat lost by evaporation increased from 28 to 90% as the temperature rose from 21 to 38°C. Similarly, Close and Mount (1978) found the increase in evaporative

net loss to be from 25 to 78% as air temperature rose from 10 to 30°C. However, in both these studies there was evaporation from faeces and urine on the floor. Restrepo, et al. (1977) measured somewhat lower values when pigs were on dry floors. Thus, it would be expected that air vapour pressure or humidity would affect the ability of the animal to lose heat and in turn affect its feed intake and growth. This was found to be true in studies by Morrison, et al. (1969). They found that the rate of growth was increasingly affected by a change in humidity as the air termperature increased. However, Addis, et al. (1967) found no difference in pigs kept at 85 or 30% relative humidity at an air temperature of 29°C. This result may be due to difference in breeds (Landrace or Duroc) or to the fact that the pigs gained very slowly (0.39 kg/day). A weight of about 0.25 for wet-bulb and 0.75 for dry-bulb temperature seems a good temperature humidity index to characterize swine growth.

As long as air temperature is cooler than animal surface temperature any increase in air velocity should increase the convective heat loss resulting in a decrease of heat stress, other factors remaining constant. Bond, et al. (1965) when comparing air velocities of 0.18 and 1.5 m/sec found no weight gain increase at the higher velocity except at air temperatures of 35 and 38°C. Morrison, et al., (1976) found increased gains with an air velocity of 0.5 m/sec compared with 0.05 m/sec for temperatures either 5.6 or 11.1°C above the optimum. However, an air velocity of 1.0 m/sec produced intermediate results leading them to believe that very high air velocities may be unpleasant to the pigs causing them to react by eating less.

Shielding animals from thermal radiation, especially solar radiation, helps to alleviate heat stress. Hale, et al. (1966) showed that pigs on pastures with movable shades, which reduced the black globe temperature about 16.5% as compared with unsheltered temperatures in the sun, had satisfactory weight gains.

References so far have been to growth or rate of gain. Producers are also interested in efficiency or conversion of feed to weight gain. As heat stress increases the resulting decline in feed intake might be thought to result in lower efficiency since the maintenance energy remains about the same. Holmes (1974) found a decrease in energy and nitrogen retained by pigs at 34°C compared with those at 25°C. Fuller (1965) observed a marked improvement in feed conversion at 30°C compared with lower temperatures; however, two major studies of heat stress (Mangold, et al., 1967 and Morrison, et al., 1969) found little decline in conversion feed to gain per se until air temperatures reached about 30°C. Also, humidity showed little additional effect on feed conversion. It is customary to distinguish between the energy cost of maintaining pigs (i.e., at zero energy retention) and that of depositing or synthesizing tissue. These two costs account for the major part of dietary energy and consequently changes to them would have an important influence on the gross efficiency of the utilization of dietary energy for body-weight gain. It is apparent from Table 3.5 that more energy is required to maintain pigs at 33-35°C than at 25°C. While the increment is related to the body-weight of the pig (Holmes, 1973), the situation is not simple because heat production is related to both ambient temperature and the level of feeding. It may be inferred from these relationships that the same situation exists for the requirement of maintenance energy.

The efficiency of utilization (k) of metabolizable energy for live-weight gain tends to be less in a hot, compared with a warm, climate (Table 3.5). Close (1978) suggested that this decrease in (k) would be expected because of the need to dissipate the additional heat associated with

TABLE 3.5

Maintenance energy requirements of pigs and net efficiency of utilization of metabolizable energy (ME) for gain of pigs growing at temperatures between 20° and 34°C

Live weight	Environmental temperature (°C)	Maintenance energy requirement $(kcal/kg^{w^{0.75}}/d)$	Energy retention (% ME)		Reference
37 to 45	25	85	64	)	
	33–35	92	66	)	
				)	Holmes (1973)
59 to 64	25	104	68	)	
	33–35	98	61	)	
31–78	25	115	83	)	Holmes (1974)
	34	125	75	)	
20 to 50	20	115	71	)	
	25	105	67	)	Close (1978)
	30	112	63	)	

TABLE 3.6

Nitrogen (N) retained by pigs growing at different environmental temperatures

Live weight (kg)	Temperature (°C)	Nitrogen retention (% dietary N)	Level of feeding		Reference
4 to 40	20	50	Ad libitum	)	Fuller (1965)
	25	51		)	
	30	46		)	
20 to 65	25	36	High	)	Holmes (1974)
	25	35	Low	)	
	34	27	High	)	
	34	36	Low	)	
25 to 65	25	37	High	)	Holmes (1973)
	25	36	Low	)	
	34	24	High	)	
	34	29	Low	)	
25 to 50	20	46	Four	)	Close, et al.,(1978)
	25	46	levels	)	
	30	39		)	

tissue synthesis. Moreover, Close (1978) calculated that at any given environmental temperature in the range 12.5-30°C, (k) declined with increasing metabolizable energy intake.

Data for nitrogen retention are summarized in Table 3.6 from which it is clear that a decline in retention occurs as environmental temperature increases. This decline is probably related in part to a reduction in feed intake in the warmth and to an increase in urinary nitrogen excretion (Holmes, 1973; 1974). While the apparent digestibility of dietary N has been reported to increase over the temperature range 10-30°C (Fuller, 1965), this is not a consistent observation (Holmes, 1974).

When pigs are exposed to high temperature for long periods of time, anatomical changes may occur. It is not clear whether these changes are due to high temperatures per se, or to reduced feed intake as a consequence of exposure to high temperature. Nevertheless these changes probably help pigs to increase their heat loss by sensible means. Fuller (1965) and Straub, et al. (1976) observed that pigs raised in a warm environment had less hair, larger ears and longer limbs than control pigs raised in a cool environment. Straub, et al. (1976) also observed that organs such as the heart, liver, kidney and spleen were heavier in boars kept at 35°C than in those at 15°C. There is also a marked reduction in thyroid size with increasing ambient temperature (Sugahara, et al., 1970; Stahly and Cromwell, 1979), and this may be associated with the concomitant reduction in metabolic rate.

The situation regarding carcass characteristics is less clear. There appears to be a decline in eye muscle area (Tonks, et al., 1972), an increase in backfat thickness (Houghton, et al., 1964; Holmes, 1971) and an increase in proportion of saturated fats (Fuller, et al., 1974) in pigs kept in a hot environment, although, as discussed by Holmes (1971) the latter finding is not consistent with the results of other similar experiments (Stahly and Cromwell, 1979). Because of biological variation among pigs' carcass characteristics it is extremely difficult to obtain convincing data on changes in carcass composition that can be attributed directly to the effects of temperature. In a large field survey, Vajrabukka, et al. (1981) found that there was a significant increase in carcass backfat depth with increasing ambient temperature among pigs grown under commercial conditions. Calorimetric studies are equivocal in that while Holmes' experiments (1973, 1974) indicated that at an ambient temperature of 33 to 35°C the ratio of the fat energy to protein energy increased, the results of Close, et al. (1978) did not confirm this observation. It is not known whether any increase in backfat depth is due to a redistribution of total body fat under hot conditions, although the data of Fuller (1965) and Stahly and Cromwell (1979) suggest that this is unlikely. In summary, it does appear that high temperature results in a slightly inferior carcass (Straub, et al., 1976).

3.3.4   Acclimatization, chronic and acute exposure and reactions

Postural changes are an important component of behavioural thermoregulation in pigs, which lie separately and spread-eagled when hot (Ingram and Mount, 1965). These changes regulate the skin area exposed for heat transfer, but markedly increase the space requirements for pigs in hot conditions; the possibility of overcrowding becomes very real. In appreciating the significance of postural changes it must be remembered that growing pigs in intensive production facilities lie down for about 22 hours out of 24, with approximately 20% of their body surface in contact with the floor (Kelly, et al., 1948).

Free access to water is important to pigs in hot conditions. Mount, et al. (1971) observed daily water use of 8.4 litres at 33°C as opposed to 4.6 litres, at 20°C air temperature for 50 kg pigs. The water/feed ratio for pigs at 7–20°C was between 2:1 and 2.7:1, while at 30°C and 33°C the ratios increased to 4:1 and 5:1, respectively. For boars weighing 70 to 110 kg water use of up to 15 1/day was observed at 25°C compared with about 10 1/day at 15°C (Straub, et al., 1976). Water consumption is not only related to ambient temperature and feed intake but also to the temperature of the drinking water. Vajrabukka, et al., (1981) found that pigs grown from 50 to 90 kg at a temperature of 35°C for 12 hours and 25°C for 12 hours, or at a constant 22°C, drank different amounts depending on whether the temperature of the water supplied was 12°or 30°C (Fig. 3.23). Pigs at 22°C drank only 3 litres per day when the water temperature was 11°C, or 4 litres when it was at 30°C. Pigs kept in the 35/25°C environment drank daily over 11 litres of water which had been cooled to 11°C compared to 7 litres when it was at 30°C (Fig.3.23). Those pigs in the hot environment that drank the cool water gained 60 g/day more than those whose drinking water was at 30°C. Pigs may thus use cooled drinking water to help maintain thermal equilibrium. It is apparent that in all treatments pigs drank mainly during the day. In the hot treatments this was when ambient temperature was 35°C.
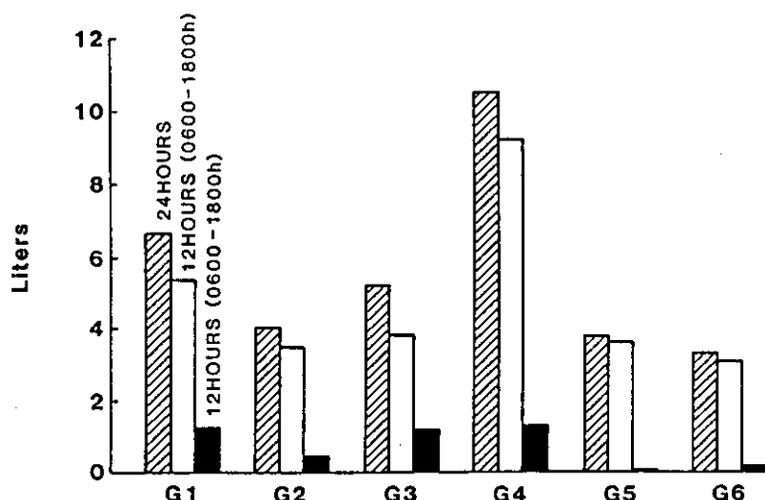

Figure 3.23 – Average daily water consumption of pigs in a hot room at 35°C for 12 hours and at 25°C for 12 hours, and continuously in a cool room at 22°C; G1, hot room, warm water, no spray, pair fed to G5; G2, hot room, warm water, sprinkled 120 secs/30 min.; G3, hot room, warm water, sprinkled 30 secs/30 min.; G4, hot room, cool water, no spray; G5, cool room, warm water, no spray, pair fed to G1; G6, cool room, cool water, no spray (Vajrabukka, et al., 1971)

Behavioural thermoregulatory responses in heat-exposed pigs also included control of heat production by regulation of feed intake. Additonal heat stress is imposed on pigs by the post-prandial rise in heat production associated with eating. Under these circumstances pigs reduced their feed intake and tended to consume their daily feed allowance over an extended period (Holmes, 1971) when housed in a warm environment. Pigs fed at a high level are known to exhibit greater increases in body temperature under hot conditions than those fed at a lower level (Robinson and Lee, 1947) and feed intake declined as heat stress became more acute. Morrison and Mount (1971) followed the response of 42 kg pigs over 28 days after the air temperature changed from 22 to 33°C. They found feed intake stabilized by the second day. Body-weight declined the first 24 hours, but by the end of the first week the rate of gain was about the same as that at the end of four weeks. However, about 12 days were required for water intake, rectal temperatures and respiration rate to reach steady values.

Physiologically, the pig attempts to maintain a constant deep body temperature of about 38°C. Neonatal piglets exposed to elevated environmental temperatures (36-38°C) were shown to have an elevated evaporative heat loss (Mount, 1962). Concomitant with the rise in evaporative heat loss was an increase in respiratory rate and an increase in body temperature from 30-40°C. Most of the increase in evaporative heat loss was attributed to the increased respiratory rate. However, because body temperature increased when convection and conduction were negligible, it was suggested that evaporative heat loss alone was not an effective thermoregulatory response (Mount, 1963). As the pig ages, dissipation of body heat by non-evaporative or sensible means becomes increasingly difficult. There is an increase in skin thickness and subcutaneous fat deposition resulting in a lower transfer of body heat to the environment (Blaxter, 1967). As the pig increases in live weight, the ratio of surface area to body-weight declines. But this evaporative heat loss alone is not an effective thermoregulatory response (Mount, 1963). As the pig ages, dissipation of body heat by non-evaporative or sensible means becomes increasingly difficult. There is an increase in skin thickness and subcutaneous fat deposition resulting in a lower transfer of body heat to the environment (Blaxter, 1967). As the ratio of surface area to body-weight has declined the effective surface area for dissipating body heat has been reduced.
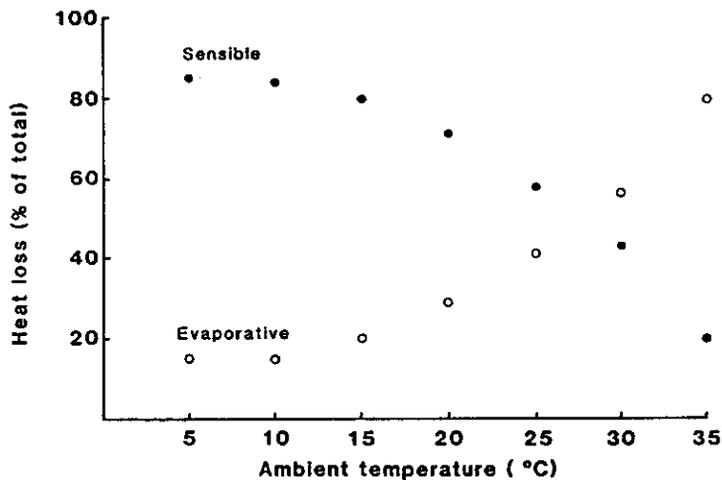

Figure 3.24 – The partition of total heat losses from a pig pen into sensible and evaporatiave components (Holmes and Close, 1977)

To lose heat at very high temperatures the pig relies almost entirely on evaporation of moisture from the surface of its respiratory tract (Fig. 3.24). The latent heat of vaporization is about 0.6 kcal/g; the exact value depending on the temperature of the respiratory surface and the temperature and humidity of the atmosphere. At high ambient temperatures, when heat loss by sensible means is very low, the pig can still dissipate heat by respiratory means even when the air is almost saturated with moisture. Provided inspired air, though saturated with moisture, is at a lower temperature than expired air, additional heat can be lost by this route. The mass of water required to saturate air also increases with increasing temperature. As a consequence of these effects evaporation represents 90% of the total heat loss of the pig at 38°C (Bond, et al., 1959). Holmes and Close (1977) state that at 30°C an increase of 18% in relative humidity is equivalent to a 1°C increase in air temperature as far as the effects on heat loss are concerned.

Unlike many other panting animals, the pig does not markedly change its pattern of breathing at high temperatures but can increase its panting rate to only a limited extent when compared to sheep and cattle (Bond, et al., 1967). Furthermore the pig does not have active sweat glands and loss of moisture from the skin is limited to about 30 $g/m^2/h$ (Ingram, 1965). Indeed, when ambient humidity is high, there may be a net gain of moisture through the skin (Ingram, 1974). On the other hand, the sparse bristle coat of the pig facilitates evaporative cooling by allowing water from a wallow or a sprinkler to make direct contact with the skin surface; this in turn allows loss of water to occur at nearly 800 $g/m^2/h$. It is not surprising, therefore, to observe pigs kept at a high temperature urinating and defaecating indiscriminately over the pen floor and rolling in urine or any other available water (Mount 1968; Close, et al., 1971) and in faeces (Tonks, et al., 1972). Effective though this may be, it has decided practical disadvantages to the producer in terms of dirty animals, dirty pens, lost feed and increased disease risk.

Although growth would not have meaning for short periods of acute heat stress, the ability of swine to survive is important. Robinson and Lee (1947) found that a 60-kg pig could not tolerate more than seven hours at 35°C, 65% relative humidity. At 38°C the tolerance time was reduced and at 41°C and above the pig was not able to tolerate atmosphere of any humidity for as long as seven hours. They also found no evidence of acclimatization to repeated exposures to hot atmospheres. However, Roller and Goldman (1969) kept pigs weighing from 80 to 114 kg at 43°C air temperature, 18°C dewpoint temperature and 41°C air temperature, 31°C dewpoint for 200 minutes. Bond, et al. (1967) reported pigs weighing about 50 kg surviving and gaining weight under diurnal cycling temperatures which reached a maximum of 49°C and a minimum of 21°C.

3.4   POULTRY - HEAT STRESS

Birds have evolved a thermoregulatory system designed to maintain deep body temperature within narrow limits with the minimal expenditure of energy. The sum of the heat gained from metabolism, muscle activity, heat increment of feed and heat loading from the environment must be balanced by heat losses via radiation, conduction, convection, and evaporation. The thermoneutral zone is defined as the range of ambient temperature wtihin which metabolism provides sufficient by-product heat to maintain essentially constant body temperature (Calder and King, 1974). The thermoneutral zone is bounded by lower and upper critical temperatures and is between 32° and 38°C in daylight and between 28°and 38°C at night. At ambient temperatures below the lower critical temperature, metabolism has to increase to offset heat losses to the environment but at values above the upper critical temperature, evaporation becomes the dominant route for heat loss. Although respiratory evaporation is accelerated by panting, additional heat is produced by increased muscular activity. As deep body temperature rises chemical reactions within the tissues are accelerated and there is a further increase in heat production.

The physiological mechanisms used by the bird to dissipate body heat under stress and methods of heat transfer are discussed by Whittow, 1976, van Kampen, 1976, Siegel, 1976, and Harrison, 1976. Continuously high environmental temperatures depress growth rate (Dale and Fuller, 1979, 1980) and an inverse linear relationship exists between growth rate and ambient temperature above 24°C (Ota and McNally, 1965). Heat production is increased

in birds exposed to high ambient temperature arising from the rise in energy demand associated with elevated respiratory and general metabolic activity (Keshavarz and Fuller, 1980).

Intense short duration exposure to high ambient temperatures produces extreme physiological stress often terminating in death. Endocrine response includes elevation in plasma cortico-steroid levels (Edens and Siegel, 1975) resulting in an increased rate of glycogenolysis (Snedecor, et al., 1963) accompanied by an increase in the excretion of non-protein nitrogen and uric acid (Brown, et al., 1958). Under conditions of prolonged heat stress, indigenous fat deposition is stimulated, and blood lipid and depot fat are increased (Heald, et al., 1965, Siegel and van Kampen, 1981). These reactions are not only attributable to cortico-steroid action but are ascribed to an elevation in secretion of catecholamines (El-Halawani, et al., 1973) which have been demonstrated to be potent glycogenolytic and hyperglycemic agents (Assenmacher, 1973).

High ambient temperature results in heat loss through evaporation of moisture from the respiratory mucosa, and increased ventilation is required to dissipate moisture (De Shazer, et al., 1974). This factor is of significance in relation to the fuel-conserving system of partial house brooding which has been adopted in many countries. Producers must balance the need to maintain house temperature with the requirement for adequate removal of moisture, carbon dioxide, and ammonia. Broilers maintained at an ambient temperature of 32°C demonstrated a decreased yield of edible meat in comparison to birds reared at 13°C (Huggins and Lewis, 1980). Swain and Farrell (1975) suggest that the relative increase in fat and corresponding decrease in water in stressed broilers is attributed to metabolic changes associated with acclimatization.

The growth rate of commercial poultry is suppreseed at temperatures above 30°C. An adverse effect on the weight gain was observed in turkey poults, brooded at 35°C declining by 3°C per week over a 5-week brooding period, compared to an initial brooding temperature of 30°C. Replacement pullets reared from six weeks of age at 30°C weighed approximately 130 grams less at 20 weeks than the control reared at ambient temperature. The effect of rearing at high temperature persisted up to 46 weeks of age, with fewer, smaller eggs being produced. These studies were confirmed by Vo, et al., (1978) who demonstrated that pullets reared at 35°C showed retardation in sexual maturity and weighed 25% less than controls maintained at 20°C. Depressed body-weight at 12 weeks adversely influenced subsequent body-weight at sexual maturity (McNaughton et al., 1977) and this in turn detracted from the attainment of optimum productivity for the specific strain (Krueger, et al., 1976).

Heat stress exerts a profound effect on the efficiency of commercial egg production, as high ambient temperatures result in a decline in egg production (Arima, et al., 1976), diminished egg size (Mueller, 1961), and a reduction of shell thickness (Warren and Schnepel, 1940). These effects are generally more pronounced in mature hens than pullets in the initial phase of production (eight months). Acute heat stress results in a decline in egg production and a marked increase in water intake. Deprivation of water will result in mortality associated with heat prostration (Jones and Huston, 1967), a factor which is of significance in large, intensive cage-laying operations. At an ambient temperature of 42°C, maintenance of voluntary water consumption will prolong flock survival (Fox, 1951).

Installation of insulation in poultry houses reduces the ambient dry bulb temperature during peak conditions with beneficial effects on growth rate and survivability of flocks subjected to temperatures exceeding 35°C (Reece, et al., 1976). Increasing the velocity of air flow over a bird has an ameliorative effect on high ambient temperature. The inter-relationship between temperature and air flow has been investigated by Siegel and Drury, 1968a, who demonstrated that hens were able to maintain body temperatures 1° to 2°C below controls maintained at 40°C under conditions of low air velocity. Increased air movement facilitates heat exchange between the body surface and the surrounding air (Whittow, 1976) and can alleviate the adverse effect of high environmental temperature (Wilson, et al., 1957). When the ambient temperature exceeds body temperature, increasing air velocity is detrimental to homeothermia as heat is transferred from warmer air to the body (Siegel and Drury, 1968b). The relationship between heat transfer and air velocity was investigated by Bouchillon, et al. (1970), and is of commercial significance when birds are transported in open coops, during periods of high ambient temperature. Mechanical ventilation can be used to alleviate the effects of extreme ambient temperature as shown by Reece, et al. (1972), in broilers in Mississippi, USA, and Oluyemi and Adebanjo (1979) in Nigeria. The operating characteristics of mechanical fan systems have been extensively reviewed by Carr and Carter, 1985, who indicated a ventilation requirement of 4.2 m^3/hour/kg live mass.

Under practical commercial conditions evaporative cooling is used to reduce the ambient temperature of houses in arid areas of the United States, the Middle East, and parts of African and Asian countries. The practical application of evaporative cooling has been reviewed by Reece, et al., (1970), who showed that an ambient temperature of 36°C at 44% relative humidity could be reduced to 27°C at 80% relative humidity with a resulting beneficial effect on growth rate and egg production. Evaporative pads, low pressure sprays and ultra-high presssure nozzle systems are used under different conditions. The selection of a system is dependent upon economic factors, the availability of water, the occurrence of sand storms, and the capacity of management to maintain the equipment (Shane, 1984).

Laying hens have a greater capacity to respond to high ambient temperature if they have free access to cool water. Drinking systems which allow the immersion of the wattles or the comb will enhance survival and productivity when compared to nipple systems that dispense small quantities of drinking water directly into the oral cavity.

Maintaining growth rate and egg production under conditions of high temperature requires the adjustment of nutrient intake (Scott, 1976). Daily food intake of a given ration will decrease in response to the lowered energy requirement (de Andrade, et al., 1977). They showed that increasing all nutrients in a ration by 25%, with the exception of dietary energy which was increased 10%, resulted in improved egg production and egg mass. Previous observations suggested that under conditions of high ambient temperatures, the energy content of rations should be reduced to compensate for decreased food intake. It has been demonstrated that when the ambient temperature exceeds 32°C, it is advisable to maintain the energy content of rations and supply a higher proportion of calories in the form of fat which should comprise at least 5% of the ration. All other nutrients, especially amino acids, should be balanced in relation to food intake.

High ambient temperatures, encountered in tropical countries, can contribute to an increased prevalence of specific diseases in poultry under commercial and subsistence farming systems. The relationship between disease and climate has been reviewed by Simensen (1985) who distinguished between specific environmental effects on the survival of pathogens, stress physiology of susceptible birds, and immuno-suppression associated with climatic extremes. The prevalence of Eimeria tenella infection in the south-eastern region of the United States is attributed in part to the warm temperatures and moist conditions which prevail (Jeffers, 1974). Lee (1978) indicated that elevated ambient temperatures increased the prevalence of parasitic diseases especially in hot and humid areas. Prolonged heat stress (five days at 36°C) decreased cell-mediated immune response (Regnier, et al., 1980) although short periods of exposure to high temperatures increased antibody titre to E. coli (Heller, et al., 1979). Laboratory studies suggest that ambient temperature does not play a direct role in enhancing the pathogenicity of viral and bacterial agents under commercial conditions of housing and management. A combination of high humidity and ambient temperature is an important factor in promoting parasitic conditions in poultry maintained on litter or in subsistence farming systems.

In reviewing the effect of high temperature on poultry production in hot climates, the following ameliorative measures can be applied:

- installation of adequate quantities of insulation to reduce solar heat load;
- the provision of mechanical ventilation to enhance physiological adaptation to high temperature;
- the installation of evaporative cooling systems to reduce the ambient temperature of air introduced into controlled environment houses;
- provision of adequate supplies of cool water;
- reversal of normal diurnal feeding and lighting programmes in controlled environment houses;
- implementing specific management procedures including routine and preventive maintenance of equipment, immunization to control prevalent infections, and appropriate programmes to control parasites.

Operation of commercial poultry flocks in countries where high temperatures prevail depends on an integrated approach to housing design and management.

3.5   HEAT EXPOSURE AND IMMUNE FUNCTION

3.5.1   Introduction

Meteorological factors are often implicated in health and disease of domestic animals. Economic losses due to death or a decline in productivity are increased when animals are relocated from one type of climate to another, e.g. to move from a temperate climate to the high temperatures of the tropics. However, animals need not necessarily be relocated in the tropics to encounter heat exposure. Modern, intensively-managed confinement facilities have increased the productive potential of many livestock operations, but failure and the poor design of ventilation systems, or overcrowding of animals, can result in acute, drastic elevations of the ambient temperature. Despite our awareness of thermally-induced changes in

the resistance of domestic animals to infectious diseases, little is known about physiological mechanisms that modulate host resistance.

TABLE 3.7

<u>Biometeorological effects on host resistance</u>   (Webster, 1981)

A.	Direct:	local resistance to infection at body surfaces, local clearance of infected organisms, systemic resistance to infection
B.	Indirect:	environmental damage at body surfaces, alterations to nutrition and behaviour, alterations to management

Exposure to hot environments alters host resistance in one of two ways (Table 3.7). Temperature and humidity may directly alter nonspecific local resistance of the skin and mucus membranes of the respiratory tract and thus facilitate entry of pathogenic microorganisms. Alternatively, thermal extremes may alter the general resistance of animals by directly affecting physiological and metabolic control systems that modulate specific immune events of the host. Heat exposure may also indirectly affect host resistance by causing changes in nutrition, behaviour or management of domestic animals. The folowing paragraphs review heat-induced alterations in antibody- and cell-mediated immune responses. Additionally, mechanisms responsibile for immune alterations in heat-exposed animals are discussed. Other sections will discuss effects of heat exposure on disease susceptibility.

3.5.2   <u>Antibody-mediated immunity</u>

Exposure to hot ambient temperatures has been shown to reduce absorption of colostral immunoglobulins in newborn dairy calves (Stot, <u>et al.</u>, 1976). This experiment was conducted in the south-western part of the United States and used conventional hot-weather housing conditions consisting of shade, cooled shade and hutches. Calves exposed to the warmer hutch housing had a higher mortality rate and lower serum concentrations of IgG_1, the major bovine immunoglobulin in colostrum, during the first 10 days after birth than did calves in the two cooler environments. The lowered serum antibody profile was related to an elevated plasma cortisol concentration. However, the correlation between immunoglobulin absorption and corticosteroid concentration may be coincidental since exogenous administration of corticoids fail to elicit premature gut closure (Stott, 1980)

Calves that had attained passively-acquired antibody immunity, via absorption of colostral immunoglobulin, and were then exposed to hot environments had lower levels of serum IgG_1. Two- to three-week-old calves, exposed to 35°C for 14 days had 25% less IgG_1 as compared to thermoneutral controls (Kelley, <u>et al.</u>, 1982a). However, studies with older calves raised in tropical climates suggested that serum IgG_1 is higher in calves raised in these environments than under temperate conditions (Curtain, 1971). The disparity between these two experiments probably relates to (a) the age of calves; the IgG_1 profile in younger calves reflects passively-acquired immunity whereas active antibody synthesis is relfected in older animals, (b) breeds; breed differences exist in immunoglobulin concentration (Norman,

et al., 1982) and heat tolerance (Joshi, et al., 1979), and (c) management conditions used in each study. Environmental chambers were used in the experiment by Kelley (Kelley, et al., 1982b), but natural conditions prevailed in the study by Curtain (1971).

An acute, intermittent heat exposure of certain strains of New Hampshire and Athens Randombred chickens reduced developing and circulating antibody to sheep erythrocytes. (Thaxton and Siegel, 1970, 1972, 1973 Subbarao and Click, 1970; Thaxton, et al., 1968). This reduction was dependent on time of the immunization relative to the initiation of heat exposure. However, more recent research has shown that heat-induced immuno-suppression in sheep erythrocyte antibody titres does not occur in outbred New Hampshire, Athens Randombred, White Rock or Leghorn breeds (Regnier, et al., 1980). Furthermore, a more severe heat exposure applied within four days of immunization in Rhode Island Red x Leghorn chickens resulted in higher sheep erythrocyte and Escherichia coli antibody titres (Heller, et al., 1979). This enhanced antibody response was associated with significantly elevated concentratons of plasma corticosterone.

3.5.3   Cell-mediated immunity

An acute, 60-min exposure to hot temperatures reduced contact sensitivity response to dinitrochlorobenzene in mice (Pitkin, 1965). However, longer exposure to hot environments yielded different results. Mice maintained for five days in a 35°C environment had enhanced delayed-type hypersensitivity responses to sheep erythrocytes, but contact sensitivity reactions to dinitrofluorobenzene were not different from thermoneutral controls (Blecha, et al., 1982). When mice were exposed to hot temperatures only during the inductive phase of these cell-mediated responses and then challenged at thermoneutral air temperatures, both dinitrofluorobenzene contact sensitivity responses and the delayed-type hypersensitivity responses to sheep erythrocytes were enhanced. These studies suggested that hot environments cause alterations in afferent, efferent and maturational processes of immune cells involved in cell-mediated immunity.

Cellular immune reactions in chickens exposed to hot environments are also suppressed (Regnier and Kelley, 1981). Dinitrofluorobenzene contact sensitivity responses, phytohemagglutinin skin tests and whole-blood, mitogen-stimulated blastogenic responses were lower in heat-exposed birds than in control birds. These suppresssions in cell-mediated immunity were generally of greater magnitude in the New Hampshire rather than in the Hubbard breeds.

Recent data from experiments with three-week old Holstein bull calves showed that heat exposure suppressed cellular immune reactions (Kelley, et al., 1982a). Mycobacterium tuberculosis-sensitized calves exposed to 35°C for two weeks had reduced delayed-type hypersensitivity reactions to a purified protein derivative of M. tuberculosis, suppressed contact sensitivity responses to dinitrofluorobenzene and diminished phytohemagglutinin-induced skin tests. Fluctuating hot environments, i.e., 37°C for 12 hours followed by 12 hours at 32°C, also suppressed expression of in vivo cellular immune responses in calves (Kelley, et al., 1981).

The capability of bovine lymphocytes to undergo blastogenesis in response to mitogen stimulation was not affected when calves were exposed to a constant 35°C environment (Kelley, et al., 1982b). However, when plasma from

heat-exposed calves was incorporated into culture medium with lymphocytes from a nonstressed animal, plasma from heat-exposed calves consistently enhanced the blastogenic response to plant lectins. These findings suggested that heat exposure causes alterations in plasma factors that regulate lymphocyte blastogenesis, but does not directly alter lymphocyte blastogenic capability. However, under longer, natural summer conditions, heat stress may directly enhance the mitogenic response of peripheral blood mononuclear cells (Soper, et al., 1978).

3.5.4   Possible mechanisms for heat-induced changes in the immune system

A single, definitive explanation of how exposure to hot environments alters host immunity is not possible. However, several postulations can be offered.

Most stressors, including heat exposure, cause characteristic changes in neuro-endocrine components, as well as eliciting typical changes in thymus and peripheral blood leukocytes (Riley, 1981; Stephens, 1980). Elevated glucocorticoids in acutely stressed animals (Heller, et al., 1979; Tache, et al., 1978) and immunoregulatory actions of glucocorticoids (Crabtree, et al., 1980; Gillis, et al., 1979) would suggest that these hormones may be involved in stress-induced alterations in host immune function. However, both a suppressed and facilitated immune response induced by heat exposure are difficult to explain by an increase in serum glucocorticoids only. It has been shown in cows (Christison and Johnson, 1972) and pigs (Marple, et al., 1972) that corticosteroids are depressed in plasma during chronic heat exposure. It may be that heat-induced enhancement in antibody- or cellular-immune responses are reflections of lower plasma glucocorticoid concentrations. Alternatively, other hormones may be responsible for heat-induced immune alterations. For example, prolactin is higher in heat-exposed heifers (Tucker and Wettemann, 1976; Wettemann and Tucker, 1974) and prolactin has been shown to modulate immune responses (Karmali, et al., 1974; Maiti and Chakroborty, 1979).

Timing of heat exposure in relation to immune responses may be an important determinant in expression of antibody- and cell-mediated immunity. Mice infected with Plasmodium berghei showed an increased resistance to the parasite if subjected to stress after inoculation (Friedman, et al., 1969). However, if animals were inoculated after a period of stress, the enhancing affect of stress on the host's resistance to the parasite was eliminated. Timing of a heat exposure episode relative to a phase of the immune response has also been shown to affect expression of contact sensitivity responses to dinitrofluorobenzene (Blecha, et al., 1982).

Duration and intensity of exposure are also important. Completely different responses in the immune system can be obtained by altering these two parameters. These variables partially explain why heat exposure has been reported to increase and decrease many types of immune events. Further research is needed before immunoregulatory mechanisms responsible for heat-induced immune alterations are understood.

3.5.5   Summary

Exposure to hot environments causes changes in antibody- and cell-mediated immune responses (Table 3.8). Both immuno-enhancement and immuno-suppression have been shown to occur in heat-exposed animals, and heat exposure has been shown to both enhance and suppress susceptibility to a

TABLE 3.8

Immune responses to heat exposure

Item	Response	
	Increase	Decrease
Absorption of colostral immunoglobulin		+
Serum IgG_1 concentration		+
Antibody synthesis	+	+
Tuberculin sensitivity		+
Contact sensitivity	+ (induction)	+
Delayed-type Hypersensitivity (SRBC)	+	
Lymphocyte blastogenesis	+ (calves)	+ (chickens)

After Kelley, 1980

variety of bacterial and viral diseases. Length of heat exposure and initiation of heat exposure in relation to vaccination are important parameters that may affect the outcome of the immune response. How high air temperatures exert regulatory influences on the host's immune system is not known. However, available data suggest that heat exposure causes alterations in homeostatic control mechanisms that influence the elicitation of immune events.

3.6   ANIMAL PARASITES IN HOT CLIMATES

3.6.1   Internal parasites and hot weather stress

        Heat stress in animals can occur due to natural extremes of heat in tropical and arid countries or come about artificially due to faulty housing, faulty ventilation or overcrowding. The effects depend not only on the degree of heat, relative humidity and associated factors, but also on the genetic and physiological status of stock. The fact that short-term exposure to temperature extremes has a stimulatory effect on the resistance of animals to internal parasites has little bearing on the animal's overall resistance, for long-term exposure lowers the immune status and so makes the animal more susceptible to parasites.

        Animals make a great effort to physiologically adapt to increased heat which causes expenditure of energy and a resultant increased water intake. In hot conditions appetite is depressed, and hot, dry conditions make herbage less digestible and of less nutritional value. Replacement energy is accordingly insufficient to balance the expended energy and the increasingly stressed animal is less resistant to larval challenge.

Hot, dry conditions affect the survival of free-living stages of helminth parasites. Faecal pats become dessicated and eggs and larvae may be killed. Intermediate insect or arthropod vectors suffer likewise. Otherwise the faecal mass may act as a reservoir of larvae to be freed by the rains at the end of the dry season. Dragging pastures to scatter faecal piles during hot, dry summers enhances the destruction of larvae, but the same practice in optimal times of larval development (i.e., wet seasons) can dangerously increase the number of available infective larvae during the following month. Thus parasitic conditions may, theoretically, not be a severe problem in hot, dry seasons though it seems that some parasites adapt to take advantage of the dry seasons.

At low stocking densities, cattle do not graze forage to ground level and consequently pick up fewer larvae than in conditions of high stocking densities when forage may be eaten very low. But the surplus forage may be of less nutritional quality and negatively influence the impact of parasitism. Under high stocking densities, though greater numbers of larvae may be acquired, the nutritional quality can remain high and offset the effects of parasitism. The nutritional benefits of heavily grazing tropical forages to maintain growth and digestibility is frequently not understood by management. However, in hot, dry seasons grazing becomes scarce and of minimal quality, and overgrazing common. Then with high numbers of larvae per volume of grazing a build-up of larvae can be established very quickly from infected animals. Pasture management and regular stock movement are accordingly as important under tropical climates as they are in temperate climates.

Parasite populations can rebound rapidly during the intervening wet sasons. Metabolism and movements of larvae and their insect vectors are greatly facilitated by hot, wet conditions. Under a massive build-up of infective larvae, host animals can rapidly succumb.

Native animals in hot weather conditions are physiologically more capable of coping with the stress, and a biological balance between host and parasite can develop. More important there is a lower expectation of productivity and losses elsewhere unacceptable may be unavoidable. Species and breeds introduced to improve production elevels are frequently physiologically unsuitable, unprepared for the nutritional stress, immunologically naive, and may not survive.

This section will explore some but not all of these aspects of parasitoses in hot climates. While dry seasons in the tropics are inimicable to the survival of free-living stages of all helminth and trematode parasites, not all dry seasons are hot. Some, such as in the Transvaal, are cool or even cold. Thus, the use in tropics of temperature climate season titles, such as "winter" and "summer", tend to confuse rather than enlighten. It will also be noted that it is long overdue for parasitologists to use microclimate data, especially of the dung pat, and not be satisfied with macroclimate observations, sometimes made even less appropriate by being averaged for the week or month.

3.6.1.1.   Fascioliasis

3.6.1.1.1   Fasciola hepatica

While there are many papers on seasonal surveys and abattoir collections, there is little literature on the quantified inter-relationships

of climate, snail populations, and Fasciola hepatica in the tropics and semi-tropics. Considering the importance of this disease, critical studies are urgently needed to accomplish efficient control through an understanding of its basic underlying dynamics. The following discussion is largely based on the work of Malone and his colleagues (1982a, 1984b).

3.6.1.1.2   Assessment of climatic variables

As soil moisture data is usually unavailable, the water budget method of Thornthwaite and Mather (1957) is useful as it calculates both surface (top 2.5 cm zone) and deeper (1.5-15 cm zone) moisture storage, as well as surplus water. The soil moisture values for the top zone indicate the conditions close to the snails' micro-environment at the soil surface; the deeper zone values cover the more severe, long-term moisture deficits in snail habitat as well as the area into which snails will retreat during dry periods and where young snails shelter. With allowances for the different soil types, the data needed are the daily maximum and minimum air temperatures, and precipitation. If the actual habitat (microclimate) temperatures are not available, which is usually the case, they can be usefully approximated with the air and 10 cm soil temperatures, though these will vary about the true micro-environment conditions (Smith and Wilson, 1980).

The onset, severity and length of the summer drought causes the snail populations to drop in the late spring and early summer. The lethal effect of drought is immediate, not cumulative. The Louisiana data suggest that deficits of 2.5-5 cm or greater represent the major stress. While high ambient temperatures are not usually a restraint so long as there is adequate soil moisture, it is unlikely that snails will survive when the microclimate temperature goes over 41°C, unless they can burrow deep and aestivate (Olsen, 1944). While Lindsay (1979) reported that the snail, Fossaria (Lymnaea) bullimoides, had two annual reproductive periods with two peak snail densities, one in the spring and the other in the early autumn, the true situation is certainly more complicated. The soil water recharge in the autumn, which can vary widely in onset from year to year, is marked by the emergence of aestivating snails. If it is late and/or the cold weather is early, any autumn snail activity will be limited. In the mild, wet winters (average temperature, 12.5°C) and springs in Louisiana, population trends, which increase the availability of snails for infection, coincide with the increased development and survival rates of free-living and snail-borne parasite stages. The variable periods of critical sub-10°C weather result in great year-to-year variations in snail and F. hepatica development and presumably of snail predation.

3.6.1.1.3   Snail population dynamics

A general "crash-boom" pattern of snail population dynamics occurs. Some medium sized snails survive the summer drought and emerge with the autumn soil moisture recharge. With favourable weather conditions there is a limited snail reproduction in the autumn. Snails in wet habitats grow slowly during the autumn and winter, and are active at temperatures over 10°C. Major reproductive efforts start in mid- or late winter in association with habitat water and warm temperatures over 10°C. The new generation repopulates habitats as older, larger snails die. Development can be rapid, being as short as 20 days from egg to egg and the maximum lifespan is about six months. This continues into the spring. When major soil moisture deficits occur in the late spring and early summer, the populations drop with

massive mortality of snails of all sizes in drying habitats. However, during the hot weather snail-infested sites can be maintained by soil moisture, such as by constantly overflowing water troughs, and at pond and river edges.

3.6.1.1.4   Seasonal fluke transmission patterns

Under favourable conditions in certain years a minor autumn transmission will occur in association with the emergence of infected aestivating snails surviving the summer drought. Based on data from sentinel calves, herd prevalence rates, and snail populations, the major transmission of F. hepatica in Louisiana starts in February and is terminated by the summer drought in June and July; metacercaria survive for only short periods in the summer drought (Ross and McKay, 1929; Olsen, 1947) while they can survive for 30 days in water at 32°C (Shirai, 1927). The largest number of flukes are acquired in mid- and late spring; this period is earlier in the more southern Texas coastal ranges (Olsen, 1944, Craig and Bell, 1978). Using step-wise regression, the average number of flukes found in calves for each 45-day sentinel period was best forecast by the cumulative surplus water over the prior 4.5-month period, followed by soil moisture storage (depletion) in the top 1.4 cm zone. This statistical relationship was logical as it reflected standing water in habitats and the flood dispersal of snails. The major late winter-spring and minor autumn (over summer) infection in Texas and Louisiana corresponded to the major late summer and minor early summer (over winter) transmission pattern of Europe (Armour, 1975). In these semi-tropical areas the fluke strategy is to survive the (hot) dry summers. It should be noted that Malone, et al., (1984a) over three years of snail collecting recovered only two infected snails, while Olsen (1947) found three infected snails among 449 examined and in 1941, 5.1 infected snails per 1,000 (1944).

An interesting parallel study is that of Owen (1984) in Papua New Guinea, where there were no temperature restraints on transmission and animals shed Fasciola eggs all year. However, sentinel calves failed to become infected during the driest part of the year between July and October.

The optimum control strategy in the semi-tropics of the US Gulf States appears to be to remove the mature F. hepatica infection in cattle during the autumn and induce a sustained reduction of egg shedding. This should increase the herd performance by reducing the loss of condition during the winter nutritional and environmental stress, that would otherwise be excacerbated by fluke-related anaemia and protein loss (Malone, et al., 1982b, 1984b). New drugs effective against immature stages may be best given in the spring to produce their benefit during the summer stress period when grazing is limited and temperatures high. Sheep, with their lower resistance to fascioliasis, require treatment of the immature fluke and therefore early diagnosis is essential to prevent serious losses.

3.6.1.1.5   Fasciola gigantica

Possibly one of the most complete reviews of the epidemiology of this African trematode is by Schillhorn van Veen (1980a). This parasite of African ruminants has a water snail, Lymnaea natalensis, as its intermediate host. In West Africa this snail is fairly common in any region with an annual rainfall of over 1,000 mm. Northwards in the lower rainfall areas the distribution is erratic as snails are only found in permanent waters, mainly adjacent to the river Niger, the Lake Chad basin, and increasingly in man-made lakes. In the savannah, the snails are most abundant during the beginning of

the dry season, but decrease as most pools and streams dry up. There is some controversy about the ability of L. natalensis to survive under drought conditions. Generally, a limited drought survival of a few weeks, especially of juvenile snails, is recorded. A survival time of 30–90 days has been recorded under fairly humid conditions, which are not usually characteristic of African dry seasons. However, in Kenya, Preston and Castelino (1977) observed an apparent aestivation period of eleven months by adult snails in dried mud. In the same country Bitakaramire (1968a and 1968b) noted a survival of six months in the absence of surface water in both field and laboratory studies. Aestivation has not been noted in Uganda (Coyle, 1958) or Tanzania (Hammond, 1965).

In aquarium studies, Prinsloo and van Eeden (1969) noted that at 25°C and 27°C the mean generation times were 13 to 9 weeks respectively, but the net reproductive rate was so low as to preclude survival in such artificially constant conditions. While its incubation period shortened with warmer temperatures to a constant value of 13–14 days at 25°C, egg survival fell off precipitously above 27°C. The reproduction rate was maximal at 18°C when the generation time was 22 weeks and it was even longer at 15°C: 25 weeks. The snail's optimum production seemed to be in the range of 18°C to 21°C. In some stream studies in Northern Nigeria, Schillhorn van Veen (1980b) regularly observed that egg masses during the dry season were below 19°C to 21°C but also the water pH was at, or above, 7. With the onset of the rains, the water temperatures rose to 25°C–26°C, the pH fell progressively to 6.5 and egg masses were seldom observed; also many developing snails and eggs were washed away in torrential streams after heavy rains. The adult snail and also juveniles were apparently susceptible to pH with catastrophic mortalities below 6.4 pH (Preston and Castelino, 1977). pH itself is an integration of reduced photosynthetic activity by water flora, the breakdown of organic matter, disturbance of salts in the lower water levels, and the leaching of various salts. Thomas and Tait (1984) have observed that Lymnaeds were intolerant of low oxygen levels and that Lanistes libycus and Aplexa waterloti were efficient predators of eggs and juvenile snails. The natural dynamics of L. natalensis is clearly more complex than can be ascribed to simply rainfall and temperature. On the preferred snail habitat, readers are referred to van Someren (1946).

L. natalensis populations peak during the rainy seasons. While Fasciola-infected snails can be found throughout the year, most infections are acquired at the end of the wet season, so that 25% of snails might be infected by the beginning of the following dry season (Schillhorn van Veen, 1980a, 1980b). Depending on the availability of cattle the epidemiological pattern appears to be that the first generation of Lymnae snails becomes infected at the beginning of the rainy season. The numbers of miracidia are fairly high as the egg excretion by infected cattle is the most at this time. Moreover, egg survival and miracidium-snail contact is higher during the rainy season. Redial infected snails are found during this time and cercarial shedding occurs from the middle of the wet season as the incidence falls. Dinnik and Dinnik (1963, 1964) have shown that at temperatures of 16°C or less the rediae do not produce cercariae. The first generation rediae and all the rediae of subsequent generations produce only daughter rediae and do not switch over to the production of cercaria until the water reaches 20°C. Sporocysts and first generation rediae are very susceptible to cold temperatures. In Sind 28% – 80% of snails were infected (Sheikh, et al., 1984). During the dry season, only those eggs dropped in or near water developed as the faecal pats rapidly dried out and fluke eggs do not survive more than three weeks in dry pats;

accordingly juvenile snails are seldom infected at this time. Cercarial shedding during the dry (cool Nigerian) season coincides with the lowering of the flood plains. (In Samarkand, the vector, L. bacteriana, sheds the cercaria at night [Salimov and Azimov, 1983,]). Metacercarial viability is not significantly longer than one month in the dry season. Cattle moving into the recently dried-up flood plain risk infection and may show signs of chronic fascioliasis within 3-4 months. While sentinel animal studies indicate that most animal infections are acquired up to and by the middle of the dry season, it does depend on the grazing habits of the stock. Patent animal infections develop by the end of the dry and beginning of the wet season.

The prevalence of Fasciola gigantica infections in West African cattle is very variable but is highest in areas with permanent streams and sedentary livestock and lowest in dry areas. The prevalence can be low in humid areas if, because of vegetation, the cattle are denied access to riversides.

3.6.1.2   Ostertagiasis

In temperate climates, larvae acquired during late autumn and early winter become inhibited in development at the early fourth larval stage. This state can persist for up to three to five months and allows the parasite to survive through the cold, hostile winter (see Section 2.6.1). In areas with mild winters and hot summers, whether in the southern hemisphere (Anderson, 1972; Hotson, 1967), or the northern, such as the US states facing the Caribbean (Williams, 1983), the larvae acquired from pasture in late winter and early spring become inhibited for similar durations. This inhibition carries the infection through hot and dry summer conditions. The mechanisms responsible for inducing inhibition are as yet undefined but clearly include environmental factors, and possibly host immunity, endocrine changes, management practices, and density dependence of the worm population (Smeal, et al., 1977, 1980). It seems that the climatic factors responsible for the conditioning of larvae on semi-tropical pastures begin in February in the northern hemisphere, (August in the southern hemisphere) and have an increasing effect on pasture larvae during March and April; September and October in the south. The temperate conditioning of free-living larvae for survival as inhibited larvae through the cold winter begins in August in the north and in February in the south. Michel, et al. (1974) reported that the longer larvae are exposed the greater the proportion becoming inhibited. The variable winter conditions each year affecting the reversal of the proportions of normally developing and inhibited larvae would explain the different proportions appearing the following spring (Michel, et al., 1978). Similarily, multiple factors affect the duration of inhibition, which largely parallels the period of climatic conditions adverse to the development and survival of the free-living stages. The resumption of development or maturation of previously inhibited worms is likely to have multiple factors, such as environmental effects, number of adult worms and their anthelminthic removal, host conditions such as parturition and lactation, and other host factors suchs as endocrine signals, resistance, and deficient nutrition. Resumption can occur as follows: (a) a few worms mature each day from a finite pool of inhibited larvae in the abomascal mucosa; (b) inhibited larvae may resume development earlier in younger or weaker stock; (c) the inhibited larvae may resume development en masse at the end of climatic adversity.

The development of the free-living stages of Ostertagia ostertagi have been described by Young and his colleagues (1980) at constant

temperatures from 5° to 27°C and in faecal pats in the fields. This is largely dependent on temperature and can be accurately modelled using an Arhenius equation. Readers are strongly recommended to refer to this paper which explains the larval reservoir capacity of faecal pats.

The following discussion on the epidemiology and development of Ostertagia in the semi-tropics is based largely on the work of Williams (1983) and Williams, et al. (1983, 1984).

Type I ostertagiasis is the clinical condition resulting from the rapid acquisition of large numbers of larvae that complete their development to the adult stage in a mere three to four weeks after ingestion. It occurs in beef cattle from weaning to 18 months of age, it responds well to anthelminthics, and at necropsy large numbers of worms will be found and almost all of these adults. In the southern temperate environment the condition is seen during the winter and spring.

Pre-type II ostertagiasis, which precedes type II ostertagiasis, is characterized by large populations of O. ostertagi in thousands or even hundreds of thousands, and the majority are inhibited early fourth-stage larvae. There is no apparent clinical impact though individual animals may show signs of ill-thrift, depending on the numbers of adult worms present. In Louisiana the peak acquisition of inhibition-prone larvae occurs in March to May inclusive, and few after early June; this is the period from the last winter frost up to the onset of the summer droughts. In northern California, USA, (Baker, et al., 1981), Georgia, USA, (Vegors, 1958), the North Island, New Zealand (Brunsdon, 1972), and southern Australia (Bowen, 1979; Smeal, et al., 1980) inhibition-prone larvae are also acquired during the spring. The inhibited larvae persist in massive numbers throughout the summer. Until recently, the inhibited larvae were not susceptible to anthelmintics but can now be cleared with fenbendazole, albendazole, oxfendazole, and ivermectin.

In late summer and autumn large scale maturation is observed with clinical parasitism (type II ostertagiasis) occurring in yearling stock. Autumn is normally a drier period of the year when parasitic gastroenteritis is generally unsuspected and stock for a time removed from the acquisition of infection. In many areas, the declining nutritional value of late summer and autumn grazing will enhance the impact of worm burdens. Type II disease is fairly common in yearling steers and pregnant heifers may be particularily susceptible; cases in older stock are not uncommon. Incidence of severe type II clinical disease is usually low but the fatality rate may reach 20% despite frequent anthelminthic treatments at short intervals. The poor prognosis in type II disease may be associated with the maturation of succeeding waves of inhibited larvae, often following the removal of adult worms by anthelminthics. Type II disease can be complicated by the simultaneous appearance of type I infections. New Zealand veterinarians (Chalmers, 1980) believe that a number of factors are involved in outbreaks, such as stocking rates and nutrition, weather, calving, trace-element deficiencies, and debilitation by other diseases.

In most warmer areas, grazing seasons are long and a year-around availability of larvae is possible. Exceptions are summers with either high temperatures and dryness or alternating wet and dry weather which is adverse to larval survival. Thus pasture contamination progressively accumulates from autumn to spring. It is essential that, through a combination of pasture management and appropriately timed anthelminthics, the number of adult worms

is reduced just before a season that would otherwise enhance larval build-up on pasture. Therefore in warmer areas cattle should be treated in middle to late winter to remove adult worms that give rise to inhibition-prone larvae on pasture during spring and early summer. Effective control of adult worms and inhibited larvae (Williams, et al., 1984) during the previous autumn might make this treatment unnecessary. However, despite the high average efficiency of fenbendazole against inhibited larvae, a small and ill-defined proportion of cattle may harbour substantial numbers of Ostertagia (Anderson and Lord, 1979). The risk that these residual infections pose for the rest of the herd has yet to be quantified and cannot at this time be dismissed. Injectable ivermectin gives more consistent results, and because of its activity against Dermatobia larvae and Boophilus ticks is particularily useful in the tropics.

3.6.1.3   Haemonchosis

This is a problem of sheep and cattle, with evidence that local strains adapt to one or the other depending on host availability (Horak and Louw, 1978). It is most commonly caused by either Haemonchus contortus (in sheep or cattle) or H. placei (in cattle only). It can have an inhibited phase as Ostertagia, but in areas with a year-round rainfall, i.e. Paraguay, the proportion of inhibited larvae may be very low (Benitez-Usher, et al., 1984). The impaired oviposition by adult parasites probably reflects the host's development of immunity (Michel, 1967), which is also related to self-cure and the expulsion of adult worms. In calves the stress of weaning considerably increases disease risks.

There is a wealth of descriptive literature on haemonchosis and Haemonchus in the tropics and semi-tropics. Potentially dangerous weather for the development of larvae in pasture would be a well spaced monthly rainfall in excess of 50 mm together with a mean maximum temperature over 20°C (Pullar, 1953; Rosa, et al., 1971). During such a period two or even three distinct waves of egg-laying activity can be noted in infected stock, but it is more common to see two waves, one at the beginning of the wet season and a smaller one towards the end (Rivera, et al., 1983). It is the length of the hypobiotic period that would determine whether there are one, two or three generations of worms during the year.

The mechanism that produces an increasing number of inhibited larvae is obscure, as it is with Ostertagia. Studies with tracer animals (such as Blitz and Gibbs, 1972; Horak, 1978a; Biggs and Anthonissen, 1982) indicate that peak burdens of adult and immature stages are present within 2-3 months of the beginning of the wet season. At about this time, self-cure of the animal takes place. Thereafter the acquisition of new infections falls off as well as the larval ability to mature to the adult form, and the percentage of fourth stage larvae increases to over 90% by the beginning of the dry season. Careful reading of a number of studies shows that this tranformation to the inhibited form is coincident with falling environmental temperatures, though sometimes with wide diurnal variations, and the direct development with rising temperatures (Biggs and Anthonissen, 1982; Rivera, et al., 1983). This fall in temperature may occur late in the wet season and when it does the number and percentage of acquired inhibited larvae markedly increases (Horak, 1978b) which would parallel the northern temperate trigger of chilling (Armour and Bruce, 1974) but in a warmer temperature range. This temporal pattern of inhibition occurs even when pastures are irrigated (Horak and Louw, 1977, 1978). In the Transvaal there is a delay between the maximum pasture contamination and maximum larval availability of some weeks. The

increased availability of larvae follows the decline of pasture with the oncoming dry season and the consequent increase of larvae per volume of herbage consumed, allied to the improved survivability of larvae in the cooler conditions, until the cold weather of the Transvaal high veld dry season stops further development altogether.

During the dry season faecal egg counts are minimal (Hart, 1964). The egg and the developing larvae are very susceptible to drying and so little or no transmission occurs during dry seasons (Sprent, 1946; Lee, et al., 1960; Horak, 1978b). But outbreaks of clinical haemonchosis can occur in a drought when stock congregate on wetter areas with available grazing (Benitez-Usher, et al., 1985). Okon and Akinpelu (1982) working with experimentally infested unshaded pasture, demonstrated in Calabar, Nigeria, that over a period of constant temperature fluctuation, the time taken to develop to the infective stage (7 to 5 days) and larval survival (6 to 10 weeks) were a function of humidity or rainfall during the relatively dry season of this humid, coastal area of West Africa. When stock is grazing irrigated pastures, some transmission will occur (Pullan and Megadmi, 1983) though most of the parasites may be in the hypobiotic state in the intestine waiting for the wet season.

This reservoir of inhibited larvae in the host allows Haemonchus to successfully survive the dry season, in much the same way as Ostertagia does in the tropics. At the end of the dry season the majority will be young adult worms, five to ten times the number that could have been recovered from infected stock at the beginning of the season, with an increasing number of eggs within the females, and even in fourth stage larvae, and an increasing number of eggs being shed (Hart, 1964). The appearance of mature parasites and eggs together corresponds to the northern latitude "spring rise". After months of strong sunlight and negligible rainfall this major change can only occur through the resumed development of the inhibited, immature stages, and the decline in the host's immune status, probably augmented by the nutritional and environmental stresses due to the dry season.

In eastern Nigeria, where the dry seasons are hot and relatively short, Chiejina and Emehelu (1984) and Chiejina and Fakae (1984) have shown that significant larval development may occur during the last two months of the dry season as a result of the small amount of rain falling at that time. During these two months significant contamination can occur with the release of third stage larvae from faecal pats by the early rains. These early rains not only provide humidity but also encourage herbage growth, which encompasses a supportive even-tempered microclimate for the developing larvae, protecting it from solar irradiation, and providing an appetizing substrate for the infective larvae.

In Paraguay, where there is usually year-round non-seasonal rainfall, L$_3$ larvae can be recovered from the pasture as long as the faecal pats can be identified (Benitez-Usher, et al., 1985). Hatching and larval survival is optimal during the cooler months. Maximum numbers of infective larvae emerge two to four weeks after faecal deposition. The L$_3$ infective larvae disappear eight weeks after the disintegration of the pats, which may persist naturally for 14-20 weeks. The faecal pat is therefore an important reservoir of infective and potentially infective larvae.

3.6.1.4   Some pig parasites

Some of the 16 helminths in swine in the United States are cosmopolitan, others spotty in distribution. The prevalence of some,

especially those requiring an intermediate host, is largely influenced by type of management, housing, and other environmental conditions. The climatic conditions of the south-eastern states are in general more conducive for the transmission and survival of parasites than other areas, as in the more temperate areas of the south-east pastures are used to maintain breeding sows and frequently to provide grazing for the sows and nursing pigs.

Observations in southern Georgia, USA, (Stewart, unpublished data) indicate that the acquisition of internal parasites by suckling pigs differs according to pasture management and weather. The highest and most consistent statistical correlations involved precipitation and temperature extremes. Rainy days and total rainfall had high positive correlations with numbers of Hyostrongylus rubidus and Oesophagostomum dentatum found at necropsy in pigs farrowed in the spring (r = 0.52 and 0.77). Hot days, of over 33°C, had a negligible effect on the number of these nematodes (r = -0.05 and -0.10) during the three-month period before necropsy, but cold days, of less than 1°C, significantly reduced their numbers (r = -0.63 and -0.64). With the spirurid worms, Ascarops strongylina and Physocephalus sexalatus, which require the dung beetle as intermediate host, high positive correlations (r = 0.61 and 0.77) were observed between the number of rainy days during the three-month period before slaughter and the number of worms in spring pigs. Hot days had no appreciable effect but cold days during the three-month period had high negative correlations with the number of worms (r = -0.77).

Strongyloides ransomi and Stephanurus dentatus are largely found in the south-eastern USA and are of great economic importance. S. ransomi is a major pathogen of newborn and suckling pigs. Infective larvae are found in the belly fat of sows and are incorporated in the colostrum prior to pasturation. Newborn pigs become infected with the first nursing as the majority of larvae are passed in within the first 12 hours after parturition. In farrowing pastures, transmission occurs but rarely in high numbers. Faeces are dispersed on pasture so that there are no concentrated sites of infected larvae and eggs. Conversely, in pens in farrowing houses, faecal droppings are concentrated in bedding which tends to be damp and thus an excellent medium for development and transmission through the direct cycle as well as multiplication through the indirect sexual cycle. Unless pens are thoroughly cleaned every three days, S. ransomi will be transmitted from pig to pig and to the sow where the larvae will lodge in the fat in readiness for the next lactation.

Prenatal infection has been reported as a problem in Brazil (Pimento Neto, et al., 1965). Prenatal infection occurs in first litters, but not in subsequent litters from the same sow (Stewart, et al., 1976). However, the transcolostral passage of larvae will take place in virtually all sows from the larvae lodged in the sows' belly fat and larval survival can be long-term (Stewart, et al., 1973). Of the three routes of infection, prenatal, transcolostral, and skin penetration, transcolostral appears to be the most frequent, though all three routes can occur in a single litter (Moncol and Batte, 1966; Stewart, et al., 1969).

Poor management, far too common in the southern states, is associated with severe outbreaks of strongyloidosis. The use of fine moisture-absorbing bedding in farrowing stalls, the presence of corn shucks and other debris in outside lots or pig yards, access to shady, damp areas, and introduction of breeders are incriminating factors. Sanitation in the farrowing area is very important since pigs can pass S. ransomi eggs at 2 1/2 days of age and the ensuing larvae can become infective in three days or less.

Stephanurus dentatus, the swine kidney worm, has been a major cause of financial loss through poor weight gain in pigs and the condemnation of viscera and loins. Before effective drugs were available the south-eastern prevalence was 78% to 95% (Stewart, et al., 1964). A more recent survey (Batte, et al., 1976) showed that three-quarters of all liver lesions in slaughtered swine were from S. dentatum. The liver condemnation rate was 38% in North Carolina, 65% in South Carolina, and 78% in Georgia. The "gilt-only" mangement system was publicized in North Carolina.

For many years Oesophagostomum dentatum, the common nodular worm, was the only Oesophagostomum species with a recognized world-wide distribution. Other species such as O. quadrispinulastum (= O. longicaudum), O. brevicaudum, and O. georgianum were only reported from tropical, sub-tropical, and warmer temperate areas such as Georgia, but they are tending to spread. O. granatensis, originally described as from Spain, is now reported from France. From the literature, Oesophagostomum species are more numerous in the warmer parts of the world, though adaptable to cooler geographical areas. These parasites are more important in breeding stock than in young animals and can maintain sizeable populations in housed stock.

3.6.2   Ectoparasites of livestock

3.6.2.1   Introduction

Environmental factors known to influence parasitism of farm animals in cold climates have comparable or even greater effects on the epidemiology of parasites in hot climates. In contrast with cold climates, annual cycles of hot climates are characterized primarily by changes in moisture and to a lesser extent by temperature. In tropical regions they are usually defined by wet and dry seasons and are increasingly determined by changes in temperature with distance from the Equator. Extremes in hot climates are associated with dry seasons when intense solar radiation contributes to intolerably desiccating high temperatures. This is in contrast with wet seasons when temperatures are subject to relative moderation by cloud cover in moist air. The significance of hot climates in the distribution, abundance, and survival of ectoparasites, especially during free-living phases of life cycles, is determined primarily by extremes in environmental desiccation. Thus, time of arrival, intensity, extent and persistence of wet seasons, often of a "monsoon" type, are major influences in the distribution and abundance of all forms of ectoparasites.

3.6.2.2   Etiology of parasites in tropical climates

The influence of weather and climate on animal parasitism has been relatively unstudied in livestock production for tropical environments in comparison with that of north temperate regions. Although there are clear indications that meteorological factors generally interact to varying degrees with life cycles of most parasites, information available from tropical studies has not been sufficiently comprehensive to develop definitive applications for meteorological services. Nevertheless, studies of host parasite relations of animals in wide ranging climatic environments identify some features of the etiology of parasites that require consideration for future applied research and development for meteorological services in hot climates.

An annual cycle in the immunological resistance of host animals to parasites is a significant underlying factor of the influence of weather and

climate on infestations in temperate and sub-arctic regions. Evidently this cycle is a physiological response to temperature in which cold stress is detrimental to the normal effectiveness of host immunity in resisting infestation by parasites in cold winter seasons. Weather and climate may be expected to have little or no significant influence on immunity in tropical regions unless immune processes are also found to be sensitive to stress of animals exposed to extreme heat.

Some studies in temperate climates have indicated that the impact of ectoparasites on productivity of cattle is accentuated under conditions of drought (Haufe, 1974). These observations may be extrapolated to tropical environments to imply significant influence of meteorological conditions during dry seasons when infested animals are subject to the stress of inadequate nutrition combined with extremes of heat.

3.6.2.3   Meteorological implications in management of ectoparasites

Information on direct assessments to define or quantify the influence of meteorological factors on host/parasite relations are generally unavailable for tropical regions. Consequently, there are no demonstrated examples of practical applications for meteorological services in the management of ectoparasites on domestic animals in hot climates. Nevertheless, some environmental studies of parasites provide good evidence that meteorological conditions have significant implications for the behaviour, abundance, and survival of ectoparasites. The implications are evident both for the fully adapted obligate and also the free-living types of ectoparasites common to livestock. It only remains for much needed technology in applied biometeorology to fully evaluate the application of meteorological services to management systems.

3.6.2.4   Fully adapted obligate ectoparasites

Fully adapted obligate ectoparasites usually live within the pelage of their host so that exposure to climate depends to a large extent on host behaviour. Large animals such as sheep, goats, and cattle are capable of carrying sufficient pelage to insulate them from the extremes of cold climates. This pelage is shed or shorn in the spring and replaced by a thinner summer coat. Whereas the micro-climate of these ectoparasites (e.g. lice) is buffered within the pelage in cool winters, it is more variable in warm summers. The temperature at the tip of the fleece of Merino sheep can reach 70°C within 10 minutes in Australia, and the usual temperature gradient from 39°C at the skin to, say, 20°C at the fleece tip is reversed from 40°C at the skin to 70°C at the tip within 30 minutes. Temperatures in much of the fleece may become lethal, and louse populations are considerably reduced on domestic sheep in hot climates, particularly when shade is absent. Thus, "summer" is a time of severe stress and the shorter "winter" limits the time available for population growth.

Changes in the micro-climate are less dramatic on cattle and, with the loss of the thick winter coat, grooming efficiency is greatly increased. Factors influencing shedding of the pelage are important, especially climate when it influences the nutritional state of the host. Seasonal delays in the shedding of pelage increase the time available to ectoparasites for population growth. Consequently, only those species of lice with high reproductive potentials are found on livestock in hot climates. Immunological responses of the host appear to be dominant in cold climates where the favourable period for population growth is longer, but their effects are probably complementary

to the direct effects of climate and grooming behaviour. Treatment with pesticides will usually further reduce the pest population when it is low and already under stress, so as to remove or reduce the potential of the ecto-parasite to increase rapidly in the more favourable cooler period, or "winter".

3.6.2.5   Free-living ectoparasites

Free-living ectoparasites, such as blood-sucking diptera and ticks, comprise the major pests of hot climates, and their economic importance is frequently associated with their role as vectors of protozoan, helminth, or virus diseases. Obvious relationships exist between their abundance and climate, and the management of livestock by some nomadic tribes is known to be influenced by an appreciation of such relationships.

Several relationships may be identified as examples in which climate is an important influence on the economic impact of free-living ectoparasites:

(a)   The arrival of monsoon rain initiates cycles of changing abundance of species correlated with the soaking and subsequent drying of the land. These changes can be determined by conventional survey techniques.

(b)   Floods cause a sudden release from diapause of simulids in Australia, and the resultant "plagues" cause great distress to livestock. An understanding of the patterns of flooding can enable trouble areas to be predicted. Similar problems are found in most tropical regions.

(c)   The predisposing cause of myiasis of sheep by Lucilia and Calliphora spp. is moisture in the fleece. Around the crotch of ewes this derives from urine and faeces. Heavy rain from thunderstorms can penetrate the fleece on the back of sheep, leading to lesions of bacterial origin, which attract ovipositing flies and favour survival of eggs and first instar larvae. Rain under these conditions may lead to near disastrous situations if sheep cannot be mustered for treatment. Graziers rapidly learn to interpret local weather conditions so as to avoid these disasters, particularly since blowflies have become resistant to insecticides which previously gave protection for several weeks.

(d)   Synoptic winds follow predictable paths across continents according to the position, speed, and intensity of the pressure cells with which they are associated. There is an increasing awareness of the role that such winds play in the dispersion of insects within and from regions of hot climate. An example of this meteorological influence was the spread of ephemeral cattle fever over more than 1500 km in Australia during two months in 1968. The regions into which this vector-borne disease finally progressed were predicted and appropriate warnings made to the authorities (Murray, 1970). Another example, the bush fly Musca velustissima, over-winters in northern Australia and each year invades the south on northerly winds (Hughes and Nicholas, 1974). Bluetongue sheep disease has probably spread to southern Europe by Culicoides spp. from Africa and the Middle East (Sellers, et al., 1978, 1979).

Distribution and abundance of free-living ectoparasites are highly dynamic under the influence of meteorological systems. Populations in hot climates frequently spread into more temperate regions. These movements may be associated with long-term climatic changes or annual events. They are of major importance when the ectoparasite is a vector and can introduce a disease into susceptible populations of livestock. These problems are best examined at the continental level. The simplest approach would be to summarize a study in progress in Australia.

3.6.2.6   Distribution and abundance of free-living ectoparasites in Australia

Australia is a continent some 2500 kilometres from west to east. There is no large land mass to the south in a cold climate zone and the only substantial mountain range is on the east coast of the continent. There is a good network of weather stations across the continent to monitor meteorological systems as they traverse from west to east. The northern two-thirds of the continent receives rain in the summer and the southern one-third in the winter months.

Studies to determine which areas of Australia are potentially suitable for exotic crops have led to a dynamic classification of floristic zones that reflects the basic influence of temperature. These zones have been defined as megathermic, mesothermic, and microthermic (Fitzgerald and Nix, 1970). Models which have been described for selected species of crops, incorporate thermal characteristics of plant growth, summation of daily hours suitable for growth, the effect of rainfall measured as moisture retention by soil type, the influence of light intensity, etc. These models are being found to have considerable application in faunistic studies since they can give a measure of food availability (Nix, 1976) and a measure of the quality of habitat in the ecosystem of which many of the flora and fauna are a part.

The distribution and abundance of disease vectors are also found to correspond with bioclimatic classification of floristic zones. Culicoides brevitarsis, a biting midge, is a vector of many arboviruses of cattle in Australia, including those causing foetal deformities such as Akabane virus and bluetongue virus. Major epidemics of Akabane disease occur along its southern limits of distribution.

The distribution of C. brevitarsis is classically megathermic (Fig. 3.25) as are those of the cattle tick, Boophilus microplus, and the buffalo fly, Siphona exigua. Slight modification of a botanical model gives an accurate representation of the known distribution of C. brevitarsis, and monthly changes in the calculated "growth indices" reflect observed changes in its abundance. Since this insect does not diapause, a mean southern limit has been calculated against which annual change can be measured. This limit, which is called "the brevitarsis line", is of considerable epidemiological significance as it represents the southern limit of one of the major epidemiological systems in Australia (Murray, 1982). The main variable to be measured is weekly rainfall. Useful predictions from meteorological information include changes in this southern limit, the areas where vector densities are likely to favour arbovirus transmission, and the areas favourable to vector survival should the vector disperse extensively.
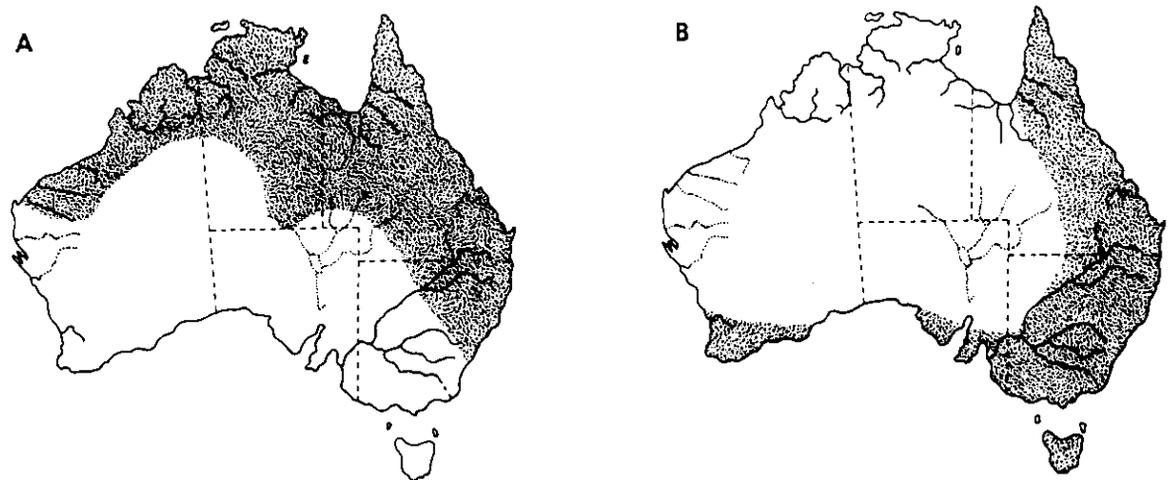
Figure 3.25 – Distribution of <u>Culicoides</u> spp. in Australia: (A) Megathermic for <u>C. brevitarsis</u>; (B) Mesothhermic for <u>C. Victoriae</u>

        Initial studies to develop appropriate computer programmes are intensive and extensive and a considerable knowledge of the biology of the ectoparasite is needed. Easy access to reliable on-line data from good meteorological networks is essential to accommodate verifiable forecasts in field assessments.

        Meteorological services have potentially significant application to present-day problems of pests and vectors of disease. Once the basic data bank has been assembled, it can be applied extensively to diverse floristic and faunistic groups. Such approaches have promising applications in hot climates, particularly to ectoparasites which have no resting or diapause state. However, preliminary investigations have shown that even life cycles with stages of arrested development may be amenable to sophisticated biometeorological application methods. Biometeorological approaches enable quantitative handling of environmental data and a definition of biological information essential for the interpretation of the distribution of ectoparasites. A spin-off will be the facility to predict epidemiological change leading to the practical control of infestations.

*

*   *

C H A P T E R   4

EFFECTS OF ENVIRONMENTAL REQUIREMENTS

4.1   SOME EFFECTS OF AMBIENT CONDITIONS ON THE SURVIVAL AND DISPERSAL
        OF PATHOGENIC ORGANISMS IN AIR

        The ability of diseases to spread in air was recognized even
before the existence of micro-organisms was demonstrated; within the past
seventy-five years, the airborne transmission (within human populations) of
the causal organisms of diseases such as the common cold, influenza,
poliomyelitis, measles, smallpox and rubella has been demonstrated
repeatedly. Some dangerous or economically important diseases of man and
animals may be spread more frequently by airstreams than either by ingestion
of the organism, by contamination of wounds or by fomites. Contagious bovine
pleuro pneumonia (CBPP), infectious bovine rhinotracheitis (IBR), foot and
mouth disease (FMD), rinderpest, hog cholera, Newcastle disease (ND), avian
infectious bronchitis and Marek's disease are now known to be spread
frequently by air, and recently Aujesky's disease (Donaldson, et al., 1983).
Even the virus of African swine fever, previously thought to be spread either
by close contact or the bites of certain "soft" ticks (Ornithodorus spp.), has
been proven recently to be able to survive as an aerosol, and to infect pigs
under experimental conditions (Wilkinson and Donaldson, 1977; Wilkinson,
et al., 1977).

        Clearly, the great potential of the atmosphere for diluting out
the agent, the relatively low densities of human populations over much of the
Earth's surface beyond the main urban areas, and variations in innate or
acquired resistance all play important roles in determining the
epidemiological impact of airborne infection. Therefore, it is mainly among
the viral diseases of animals that one encounters reasonably sound evidence
that agents are carried for quite long distances as aerosols. The progress of
an airborne epizootic may sometimes be readily detected and traced because the
lifespan of domestic livestock is short, and modern husbandry methods favour
intensive systems in which large numbers of young susceptible animals are
concentrated in close proximity. Production units of this type, scattered
across the countryside, and often the subject of carefully controlled entry
and exit, may not only serve as ideal indicators of the arrival of airborne
disease but, subsequently, may also amplify contamination of the environment
when their effluent air is carried downwind.

4.1.1   Generation of aerosols

        Multiple factors contribute to pollution of the environment by
airborne viruses, mycoplasmata and bacterial L-forms. Probably the most
frequent natural cause is the exhalation of organisms (especially by coughing,
sneezing, etc.), following secretion into the respiratory tract during periods
of viremia or following localized multiplication of viruses or bacteria. With
FMD the upper respiratory tract is the major release site during the
preclinical stages; as the disease advances the lower respiratory region
becomes the major source of organisms and changes from producing small
particles to particles of all sizes, like the upper region (Donaldson and
Ferris, 1980). However, the atmospheric burden may be increased by the
release of micro-organisms in other bodily secretions and excretions (such as

rabies virus aerosols from urinating cave bats [Constantine, 1962]), by re-entrainment of contaminated liquid droplets or dust particles, by rainsplash, or by certain methods of sewage disposal. Rabies aerosols have been generated from the over-enthusiastic washing of animal cages. Special dangers exist when sewage from infected premises is transferred under pressure from one holding unit to another, when sewage or slurry tanks are opened, when ambient pressure changes result in sudden venting of inadequately protected sewage system, or when untreated manure is sprayed onto pastures by apparatus such as "rain-guns" (Hyslop, 1970). Poxviruses, some herpesviruses, and bacteria of innumerable species, may be disseminated by incautious handling of bedding contaminated by discharges or epithelial fragments. Vigorous sweeping of floor-dust may be particularly important source of local airborne infection. Adenoviruses infecting both man and animals may be distributed extensively by the exhalations of apparently normal "carriers" and enteroviruses and bacteria achieve aerial suspension in a variety of ways.

4.1.2   Persistence of aerosols

Air currents carry fine particulate matter across vast distances; dust and even small insects have been transported from the region of the Sahara Desert to Northern Europe. However, as a result of virtually limitless aerial dilution, exposure to change in relative humidity (RH), ultraviolet or other radiation, and long transit times, microbial aerosols at high altitudes may be considered to be relatively unimportant because of the improbability of viable particles ever reaching a susceptible host.

By far the highest concentration of particles of the largest size and the greatest diversity of sources are found in a shallow layer of air, usually extending only a few hundred feet above ground level and part of the planetary boundary layer. In this zone, natural clearing occurs continuously. Factors which influence the distribution of low-altitude environmental aerosols include:

(a)   The rate of aerosol generation (in our particular field by infected individuals or by artificial processes);

(b)   Growth of particles by heterogeneous or homogeneous gaseous reactions;

(c)   Increase or decrease in local particle counts resulting from convection between adjacent air currents;

(d)   Coagulation of particles caused by Brownian motion (this increases with increments of temperature, but is small in comparison with convective or turbulent mixing; it is of greatest importance in closed rooms containing aerosols of high concentration);

(e)   Continuing natural diffusion of particles (it should be noted that this will be related to the period of suspension rather than to the distance from the source of the aerosol);

(f)   Aggregation resulting from local turbulence;

(g)   Increase in particle mass caused by hygroscopicity and high relative humidity, or decrease in mass caused by drying;

(h)   Some slight collection of small particles by larger particles
            during gravitational deposition;

(i)   A resultant steady gravitational sedimentation usually offset to
            varying degrees by thermal convection and turbulence currents;

(j)   Electrostatic attraction, usually to man-made surfaces, that may
            increase the rate of removal of charged aerosols under suitable
            circumstances;

(k)   Impaction losses on surfaces, including buildings, water surfaces,
            trees, grassland and crops;

(l)   Losses by collection into water vapour droplets in clouds
            (rainout);

(m)   Losses from sub-cloud layers during periods of rain, snow or hail
            (washout).

        Of course, the need to distinguish between organisms and particles
will be readily understood; a single droplet or particle may carry several
(200 or more) organisms of the same or of different types. It must also be
remembered that filter-passing organisms and even large bacteria seldom exist
in the "naked" state.

        Of all the components listed, (k) to (m) above are probably the
most important for long-lived micro-organisms that attack their hosts via the
alimentary tract. By contrast, when the predilection site for primary
multiplication is in the respiratory tract, deposition clearly tends to
diminish the hazard of infection unless re-entrainment of droplets or
re-suspension of dusts occurs.

        Mathematical models for some of the influences listed have been
reviewed (Hidy, 1972; Boisdron, 1972; Pasquill, 1974). However, it is evident
that we are dealing with immensely complicated and highly variable situations,
and that the concepts of earlier workers (Pasquill, 1961) must be extended by
further concentrated research. This is especially true in respect of aerosol
distribution beyond a few hundred metres downwind from a point source. It is
clear that topographical factors, operating in both the horizontal and
vertical planes, play an important part in the disruption of the downwind
"plume", and also in the subsequent coalescence of smaller air-masses
containing clusters of particles which, in suitable meteorological conditions,
do not diffuse rapidly to occupy an almost infinitely large air volume, but
rather tend to travel in "packets" of relatively high concentration.

        Most naturally-generated aerosols consist of particles that are
present initially in a wide range of sizes. The largest of these do not
travel far before falling to the ground. The smaller the mean particle size
the more stable the aerosol becomes, though the contained organisms suffer a
progressive decline in viability. Particles which remain airborne for long
periods are predominantly smaller than 5-10 microns in diameter. However, in
still air at high RH, increase in size may occur as a result of gradual
coalescence, the rate of clearance being enhanced under conditions that favour
aggregation.

4.1.3   Theoretical implications of air movement

        Further modification of the aerial distribution pattern results
from changes in wind velocity or direction, thermal currents, altitude,
weather andlocal turbulence. Temperature inversions and down-draughts may
have a profoundly enhancing effect on the concentration of airborne material
close to ground level, whereas thermal or mechanical turbulence tends to
re-suspend and to dilute aerosols. During an epizootic in Switzerland,
increased incidence of FMD was observed on mountain ridges below the cloud
ceiling, but not in the valleys, where prevailing winds travelled at about 90°
to the valley systems (Primault, 1972). Down-draughts on the reverse slopes
of hilly country may also be associated with concentration of infection as a
result of "lee-wave effects" (Tinline, 1970). On several occasions a new
series of outbreaks has followed changes in wind direction.

        In the case of the filter-passing organisms, the rate of "fallout"
in "dry" airstreams is determined more by characteristics of the carrier
particles (dusts, micro-droplets, droplet nuclei), and by the prevailing
meteorologic conditions, than by the microscopic or sub-microscopic size of
the agents themselves. It is evident that rainfall may remove significant
amounts of larger infective particles from the air, to cause a localized
increase in contamination of drinking water, crops and pastures. Furthermore,
herbage acts as a filter, removing particles from the boundary-zone air and
collecting material from raindrops (Hyslop, 1970; Hidy, 1972; Chamberlain,
1970).

4.1.4   Survival of airborne organisms

        Factors which influence the survival of airborne organisms include
light (especially UV light), oxygen tension and RH (Hyslop, 1971a). The
effects of these factors are variably inter-related, depending on the
particular organism, but RH, and especially change in RH, usually exerts the
most profound effect. Although generalizations are dangerous, adenoviruses
and some picornaviruses often survive better at high RH, whereas influenza,
parainfluenza, measles and poxviruses tend to survive longer at lower RH, but
some may reveal the shortest viability periods at about 50-60 per cent RH.

        Inactivation of lipid-containing viruses that show poor survival
when airborne at high RH may be the result of surface changes. Thus, the
hydrophobic nature of enveloped viruses may cause them to accumulate at the
air-liquid interface of droplets, where physical stresses may result in the
re-arrangement of the molecular structure (Troubworst, et al., 1974).
Donaldson and Ferris (1976) reported the survival pattern of ten specific
animal viruses at different values of RH; readers are referred to that
publication for details.

        In protein-free media some herpesviruses and other enveloped
viruses appear to be especially sensitive to light. Viability in aerosols
generated experimentally from viruses suspended in culture media may differ
from the viability observed in "natural" aerosols of the same virus derived
from infected faeces or oral and nasal secretions. The proteinaceous nature
of the latter may afford considerable protection against denaturation, but
this protection may be offset by the presence of traces of both specific and
non-specific viral inhibitors, probably a partly secretory antibody (and
possibly a cross-reacting antibody), that may be present during the overt
phase of infection (Hyslop, 1965). Salts, polyhydroxy-compounds and the

protein content of suspending fluids affect the survival of several viruses in the airborne state (Benbough, 1971); and a salivary factor, stable at 60°C but not at 70°C, reduced the longevity of FMD-virus (Strain O_1 - BFS 1860) at high RH (Barlow and Donaldson, 1973).

There is increasing evidence that a still ill-defined "open-air factor" (May, et al., 1969; Harper, 1972; Hood, 1974; Donaldson and Ferris, 1975; de Mik and de Groot, 1977) exerts an adverse effect on certain bacteria and viruses. This is probably a chemical airpollutant formed by reaction between ozone and olefin-products liberated by petrochemical installations, and automobiles. It is also associated with pine woods. Thus, the day-to-day influence of this toxic factor will depend on those meteorological conditions (especially the development of photochemical "smog", or turbulence which mixes high-altitude ozone with ambient air) that tend to enhance the concentration of ozone at lower levels.

4.1.5   Environmental "fallout" of infective agents

Contamination of airstreams by viable viruses and bacteria is usually greatest during cool, cloudy weather (Donaldson and Ferris, 1976) and at night, when the more fragile or sensitive airborne micro-organisms are protected from the effects of insolation and drying. Periods of nocturnal rainfall may cause increased contamination of surfaces, and it has been suggested that an "epidemic intensity index" might be calculated, which would be weighted in favour of rainfall at night (Smith and Hugh-Jones, 1969), and more significantly of higher RH. While washout of organisms in large particles by showers does occur, there is no appreciable removal from the air by rain of small particles containing virus over distances of a few tens of kilometers (Gloster, et al., 1981). Therefore, because of diurnal fluctuations, continuous sampling is essential if the results of air monitoring are to have any real meaning. At a reasonable distance from the source, the concentration of aerosols of FMD virus, and possibly of other organisms, tends to be greater outside than inside. However, there is some evidence that aerosols persist longer indoors, where mean particle sizes are smaller and more uniform, and the possibility of being inhaled is higher.

4.1.6   Respiratory infection

When only the respiratory route of infection is of epidemiologic significance, particle size is of great importance, since it controls the site at which the greater part of the aerosol will be deposited after inhalation. The larger particles, which tend to be removed in the upper respiratory tract, will be especially dangerous in diseases for which the portal of entry is the naso-pharynx, whereas particles of about 0.6 microns in diameter will evoke a greater infection rate for those organisms which gain access principally through the bronchioles and alveoli. It must be ralized that segregation of particles of different sizes by the respiratory tract is relative rather than absolute; a few large particles penetrate as far as the alveoli and some small particles may be deposited in the naso-pharynx.

The matter of total volumetric flow is of greater importance in relation to airborne organisms than is the case when food or water is contaminated. Whereas the intake of water may be only a few litres per person per day, or a few tens of litres per day for the larger animals, the respiratory tract represents an extremely sensitive multi-stage device for the sampling and extraction of air - one which may sample as much as 100 litres

per minute in domestic livestock. Moreover, the lining epithelium of the tract constitutes a predilection site for the proliferation of certain organisms.

4.1.7 Epizootiologic studies

Carefully recorded observations on airborne diseases of animals in the field are becoming more numerous, as the factors important for the survival of airborne pathogens are elucidated in laboratory studies.

For example, a very severe epizootic of FMD occurred in Great Britain in 1967-1968. The early outbreaks were identified on farms in a wedge shaped zone within a densely stocked agricultural district; all were situated downwind from the premises on which the disease was first diagnosed. Subsequently, an additional small number of outbreaks developed along a divergent axis, after a temporary veering of the wind. Despite some peripheral spread, by various agencies (including the possible involvement of the escape of virus during distribution of infected milk), the geographic pattern remained evident some four months after the primary outbreak. In later years, distributional factors in the epizootic have been analysed by many authors as more information became available (Smith and Hugh-Jones, 1969; Hugh-Jones and Wright, 1970; Hyslop, 1970; Tinline, 1971; Hugh-Jones and Tinline, 1976). Detailed analysis of another epizootic during 1967, in Hampshire, England, revealed a probability that airborne virus had spread into areas of high livestock density in a manner that could be correlated closely with wind speed and direction (Smith and Hugh-Jones, 1969; Sellers and Forman, 1973). Somewhat similar conclusions incorporating observations on the effects of altitude and sunlight have been reached as the result of careful investigations in Switzerland (Primault, 1974).

Studies on the survival of FMD virus in aerosols produced from milk or from faecal slurries (both of which are likely to be generated freely by modern agricultural procedures) indicated that viral longevity, under meteorological conditions that may be expected in temperate countries (and particularly at night), was adequate to permit dissemination of infection downwind over quite long distances (Hedger and Dawson, 1970; Dawson, 1970). Although the actual spread of the disease by milk aerosols and milk collectors has been suggested (Hedger and Dawson, 1970; Dawson, 1970), subsequent studies indicated that this risk may be significantly smaller than originally claimed (Hugh-Jones, 1976). Recently, it has been shown that pigs, cattle and sheep which have been vaccinated may release airborne virus for variable periods after exposure to infection (Seller, et al., 1977a). This confirms previous similar results with ND in vaccinated poultry (Hugh-Jones, et al., 1973).

The prediction methods of Smith (1970) have been superseded by the more accurate prediction models for short distance spread (Gloster, et al., 1981) and long distance spread (Gloster, et al., 1982). Both had been tested on historical outbreaks, but proved their worth in the very successful immediate control of the spread of FMD in the Isle of Wight in 1981 when the infection risk had been pre-identified from outbreaks in pig herds in Brittany (Donaldson, et al., 1982) and helped confirm the chances of future spread. The short distance prediction model is based on: (a) an estimate of the daily virus output from infected animals; (b) hourly or three-hourly observations of source outbreak; (c) latitude; and (d) topography. The factors controlling long distance spread are high virus output, low virus dispersion, high virus survival, and large numbers of susceptible livestock exposed to the virus for

a number of hours; these conditions are most commonly satisfied when the downwind plume is over water, and especially when there is a wide difference between the air and water temperatures.

An additional danger to farm livestock is emerging because of the progressive spread of swine vesicular disease (SVD), a malady clinically indistinguishable from FMD, but caused by a completely different viral agent. The virus possessed considerable stability in the airborne state at RH in excess of 55 per cent (Donaldson and Ferris, 1974) and, when aerosolized from porcine faecal slurry, remained relatively stable in the presence of "open air factor" for periods of at least 60 minutes. However, although airborne SVD virus is even more stable than FMD virus, it is probable that SVD virus is disseminated from animal to animal principally as the result of direct contamination of farmland, feedstuffs, fomites, etc. with detritus from lesions, skin or faeces, rather than by airborne infection, and it can remain viable in organic matter despite a wide range of meteorological conditions.

Numerous investigators' reports on the importance of airborne virus in the epizootiology of Newcastle disease (ND) and Marek's disease of poultry are equally convincing. Thus, there was evidence that ND virus had been transported at least 3-10 miles by the wind (Drummond and Hunnam, 1966), while the geographic distribution of outbreaks in England revealed a generally decreasing incidence of disease as distances from primary foci increased (Smith, 1964). Furthermore, as in the case of other diseases, changes in wind direction were associated with additional outbreaks in a new sector. Aggregation of results over two years suggested that dispersion of the virus by wind might account for as many as 85 per cent of all the outbreaks recorded. Experimental studies by staff of the Central Veterinary Laboratory, Weybridge, and the Microbiological Research Establishment, Porton, have confirmed this earlier work, not only by recovering about $10^{2 \cdot 5} ELD^{50}$ in 200 litres of air from poultry houses, and $10^{5 \cdot 7} ELD^{50}$ in 33,000 litres of air, but also by detecting viable virus in the open air downwind from infected premises (Hugh-Jones, et al., 1973). For a more complete discussion of airborne ND, readers should refer to Gloster (1983).

The ultimate significance of aerial dissemination of small infective agents depends partly on their longevity under conditions prevailing at the particular time, and partly on the presence of an adequate population of susceptible hosts or mechanical vector systems. Furthermore one cannot emphasize too strongly that the duration of infectivity in the airborne state may be dependent, to a surprising degree, on the species or strain of the agent itself. Thus, Mucoplasma mycoides var. mycoides, the type species of the pleuropneumonia-like organisms, which evokes clinical disease in cattle by the respiratory route, is capable of persisting in aerosols for somewhat longer than the similar organism M. mycoides, var. capri, whereas certain venereally transmitted mycoplasmata of bovine origin have a very much shorter lifespan in aerial suspension, though they appear to be disseminated occasionally in this way as opportunistic pathogens (Hyslop, 1972). Similar variability between strains under different conditions has been reported for influenza virus (Mitchell, et al., 1968; Mitchell and Guerin, 1972), for poliovirus of type 1 and 2 (Youngner, 1957; Hyslop, 1964), and for the viruses of FMD (Hyslop, 1971b; Donaldson, 1972), rinderpest (Hyslop, 1979), and ND (Hugh-Jones, et al., 1973).

4.1.8   Survival of pathogens in airborne arthropod vectors

        In addition to being transported within droplets or micro-droplets
(or "dry" on droplet nuclei), micro-organisms pathogenic to animals may be
carried through the air by arthropods either mechanically or as an event in a
true vectorial cycle. The prevalence of the diseases caused by such
micro-organismsmay be influenced directly by climatic and micro-climatic
factors. These factors modify life cycles of relatively static populations of
arthropods, or modify the persistence of the organisms (a) by which the
vectors may breed, (b) by determining the time required for the arthropod
vector to pass through successive instars of its life cycle, before it reaches
the important disease-transmitting state(s), (c) by limiting or enhancing
vector longevity, (d) by modifying its hunting and feeding periods or
frequency, (e) by influencing the movement of susceptible animals into range
of the infected insect populations, or of infected animals into areas
containing both vectors and susceptible host populations. An example of these
effects is provided by the fluctuations of the distribution of African horse
sickness in the Middle East, where year-to-year changes in the amount of
surface water may cause, through variation in the population of Culicoides and
other biting flies, a considerable variation in the boundaries of endemic
areas. In the same way, it is generally considered that the distribution and
occasional extension of endemic bluetongue disease of sheep and cattle (and
several other arborvirus diseases) may well be dependent, at least in part,
not only on the existence of conditions suitable for the persistence and
expansion of populations of certain defined species of arthropods, but also on
the continued habitation of the area by susceptible animal populations, or the
introduction into the area of new infected animals.

        Equally important, however, is the influence of meteorologic
factors in physical dispersion, in flight, of disease-bearing arthropods,
which may be carried for very long distances by moving airstreams. The
serious problems in forecasting the spread of disease, and possible preventive
action by the authorities responsible for disease control, are well
illustrated by a series of epizootics of bovine ephemeral fever in Australia
(Murray, 1970; Standfast, et al., 1973). During outbreaks in 1967-1968 and
1970-1971, the disease was carried dramatically eastward and southward on
fronts of up to 500-1800 km for distances as great as 2,000 km. This spread
was apparently attributable to transportation on the prevailing winds of
several species of midges, during periods when climatic conditions favoured
survival of the virus.

        More recently, analysis of the spread of African horse sickness
from Senegal to the Cape Verde Islands, from Turkey to Cyprus and from Morocco
to Spain has provided evidence of an association between new outbreaks and
transportation of the virus to susceptible horses by infected Culicoides
midges during periods of unusual winds (Sellers, et al., 1977b). A similar
sudden extension of the disease was noted in relation to meteorological
disturbances in the Middle East and India. It was inferred that flight
occurred principally when the temperature was likely to have been 15-20°C (at
night) or 20-40°C (during the day), and that flight ranges of up to 700 km
might be possible. There is evidence that this virus can multiply in the body
of the midge.

4.1.9   Conclusions

        Many considerations render the forecasting of the extent of aerial
dissemination of a disease a matter of difficulty. Moreover, all the serious

outbreaks of disease, cited as being attributable to aerial spread, appear to have been dependent upon an infortuitous coincidence of a potent source of infection with geographic, meteorologic, immunologic and population factors which were all favourable to the development of an epidemic situation. The relative inefficiency of existing monitoring systems of biological agents leads to further caution regarding the feasibility of predicting accuratelythe aerial spread of viral diseases for more than a few kilometres or so, beyond a primary outbreak. The success of Gloster and his co-workers would indicate that further research will be fruitful.

4.2   EFFECT OF ENVIRONMENT ON NUTRITION REQUIREMENTS OF DOMESTIC ANIMALS

The term animal environment may be defined as any external factor that influences the physiological or psychological response of farm animals. This would include, but is not limited to, effective ambient temperature (EAT), photoperiod, sound, altitude, environmental contaminants, psychological restraint, and management system. In respect to nutrient requirements of domestic animals little is known of the impact of most environmental variables with the exception of effective ambient temperature. This is to be expected since the climatic environment alters the rate of energy transfer between the animal and the environment and therefore directly influences the energy requirement. The fact that most nutrient requirements are the function of energy requirement suggests that all nutrient requirements may be indirectly affected by environment.

Despite the general awareness that energy demands are increased by cold and that the magnitude of those demands is moderated by total body insulation, few quantitative data exist relating environment, energy need, and productive efficiency. Energetic efficiency is of particular importance because of the "feed vs. fuel" tradeoff that must be considered when environments are modified to improve rate of performance. In other words, the creation of more optimum environment may not be cost effective and, in fact, it may be more sensible to settle for something less than maximum performance.

Figure 4.1 schematically illustrates the partitioning of feed energy within animals and is a useful basis for identifying the modes of influence of the environment on animals' nutrient requirements. Voluntary energy intake (IE) is directly affected by the climatic environment. Depressed intake during heat stress and increased intake (within physical limits) during cold stress are widely reported. Energy loss in the faeces is also directly influenced by ambient temperature with this effect on digestibility more evident for roughages than concentrates. Recent findings have indicated an increase of approximately 0.18 per cent digestibility per 1°C rise in ambient temperature. Maintenance functions involving the utilization and oxidation of metabolizable energy for basal metabolism, voluntary activity for obtaining nutrients, or in combating external stress are all affected by ambient temperature.

Both rate of animal performance (i.e., daily weight gain, milk production) and efficiency of converting feedstuffs are functions of metabolizable energy intake and the energy required for maintenance. The thermal environment affects both intake and maintenance requirement and, in turn, alters both the rate and the efficiency of animal performance. Numerous trials with swine (Heitman, et al., 1958; Mangold, et al., 1967), sheep (Ames and Brink, 1977), beef cattle (Milligan and Christison, 1974), and dairy

cattle (McDowell, et al., 1976) showed a reduced rate of performance by animals when exposed to adverse environments. In each case, the maintenance requirement increased and less energy was available for production; during cold, animals increased their intake but the maintenance energy requirement usually increased more rapidly than their energy intake. When exposed to heat
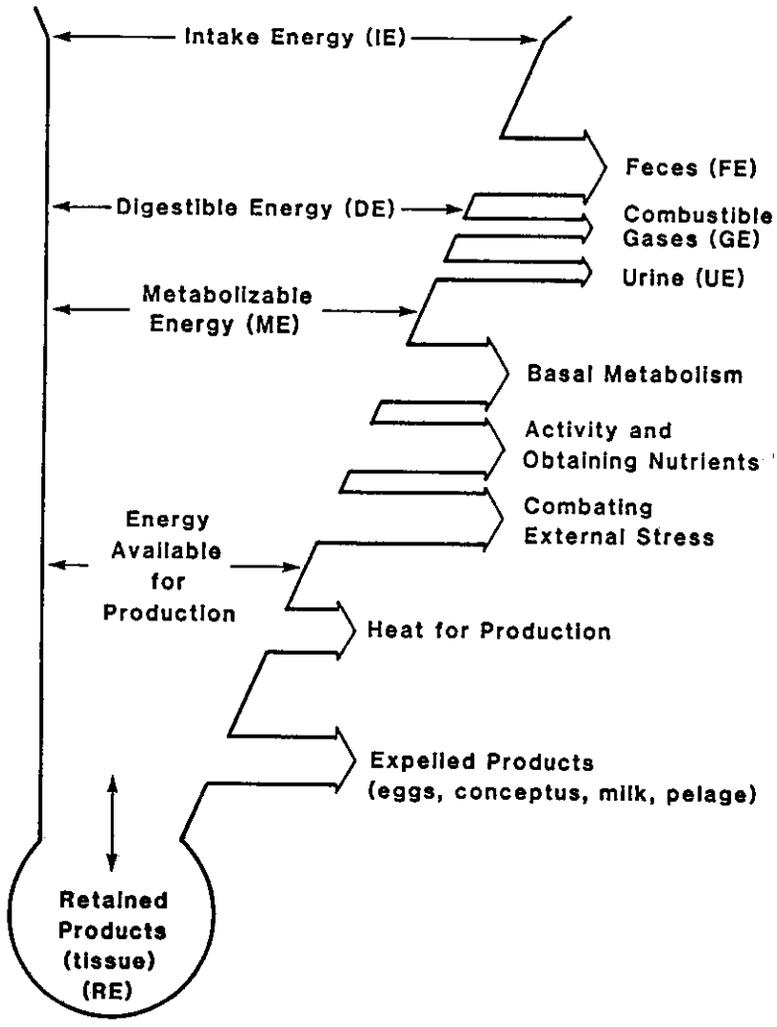

Figure 4.1 – Partition of feed energy within the animal (after Young, 1975). Heat increments of voluntary activity, fermentation, digestion, absorption, and nutrient metabolism for maintenance and productive functions contribute to body heat and in cold environments aid in maintenance of body temperature. However, in hot conditions these heat increments may be a liability to the thermal balance of the animal

stress, animals reduced intake; in the short-term their maintenance requirement increased as the animal responded physiologically in order to cope but the long-term requirements decreased. Reduced performance results in both instances.

The impact of climatic environment on energy flow in terms of both energy intake and energy available for growth may directly affect the utilization of other nutrients because in many cases nutrient requirement is a function of available energy. For example, in sheep, protein efficiency ratio (grams of gain per grams of crude protein) is lowered during both heat and cold stress. Lower nitrogen retention during thermal stress has been reported in swine and cattle (Ames and Brink, 1977; Fuller and Boyne, 1971). These examples emphasize the need to consider available energy in the light of environmental stress and to consider only energy available for production when developing rations to enhance efficient utilization of all nutrients.

4.2.1   Ration adjustment

Published nutrient requirements for livestock do not reflect differences imposed by the environment. Data sources used to establish nutrient requirements are gathered from animals protected from environmental extremes. The basis of changes in nutrient requirements during exposure to environmental extremes is energy balance, i.e., the relative changes in energy intake compared with energy required for maintenance. Increased maintenance energy requirement, altered digestibility, and rate of feed intake are directly affected by climatic environment and all have a distinct impact on the interrelationship of dietary nutrients. Logically, nutrient requirements and therefore ration formulation should be altered in response to changes in the climatic environment.

Several schemes have been proposed to accomplish energy adjustment for animals exposed to cold. The simplest is to increase feed intake. For animals on maintenance level rations or restricted diets, this is usually accomplished by permitting the animals to consume additional feed. The options for increasing energy intake for ad libitum fed animals are more limiting. In this situation, increasing caloric density by the addition of fat or increasing the relative amounts of feedstuffs with higher caloric densities (replacing roughages with concentrates) are possibilities. The latter approach, however, is contrary to the notion of increasing heat increment of feeding during cold. The fact that the heat increment is a much higher percentage of the digestible energy of roughages compared to concentrates is important when it is considered that calories from heat increment in roughage may be less expensive than that from a concentrate source. It has been reported that feeds with high heat increments fed during cold have a sparing effect on their energy production; thus, allowing their use for gain (Moose, et al., 1969). The economic advantage of altering heat increments depends upon the relative ingredient prices, magnitude of cold, and the effect of cold on intake.

The adjustment of rations during heat stress remains a practical solution to minimize the effect of heat; though the increased maintenance requirement during heat is difficult to quantify. McDowell (1972) reports nonlinear increases in maintenance energy during heat when maintenance is calculated as the difference between intake and production. With a similar procedure, a nonlinear increase in maintenance energy requirement during heat exposure has been noted (Brink and Ames, 1978). The validity of formulating rations for relief of heat stress by adjusting the heat increment of the ration while keeping net energy constant has been confirmed (Lofgreen, 1974). This adjustment was accomplished by reducing the roughage content of the ration and increasing the fat content.

While energy remains the basis for adjusting rations in response to environment, the need for other nutrients is often altered because of their relationship with energy. This is particularly true for protein. Calorie to protein ratio is frequently used in the formulation of animal diets. When environment increases requirement or reduces availability of energy, calorie to protein ratio above maintenance levels should remain constant. No effect of thermal environment on protein for maintenance has been reported (Brink and Ames, 1978). Consequently, to maintain the calorie to protein ration one must either increase energy or decrease protein for growing animals exposed to thermal stress. A system of protein removal from growing rations for both sheep and cattle fed ad libitum has been reported (Ames, et al., 1980). This

system removes protein above maintenance in proportion to expected reduction in performance. Results indicated no effect on daily gain but a significant improvement in gain per unit of protein intake (protein efficiency ratio). A different approach for egg-laying hens has been suggested. During heat stress when feed intake is reduced a concurrent reduction in protein intake is avoided by increasing the protein content of the diet (Davis, et al., 1972). The difference in this approach compared with that described for growing animals stems from the difference of expelled daily product in the form of eggs compared with tissue growth. In the former case, body energy stores serve as the energy source when intake is reduced. But protein for egg production is needed in the face of decreasing feed intake. Ration adjustment is one of supplying adequate dietary protein as opposed to reduced protein in growing animals whose daily weight gain may be highly variable. It has been suggested that when formulating rations in respect to environment both energy and protein should be included "in their own right" and that calorie to protein ratio be forgotten (Filmer and Curran, 1977). This approach is particularly applicable when more is known about the growth of production response to environment than of the variation in the energy requirement.

Data indicating the direct effect of environment on vitamin and mineral requirements are limited. Although a climatic influence on thiamin, vitamin A, and calcium and potassium excretion has been reported (Peng and Heitman, 1974; Hidiroglou and Lessard, 1971; Holmes and Grace, 1975). There is, in essence, not enough data to indicate a need for adjusting mineral or vitamin requirements for environmental differences. However, if vitamin and mineral requirements are assumed to be constant over wide ranges of temperature there is due concern that differences in intake can in turn have a major effect on the intake of vitamins and minerals. When these nutrients represent a constant percentage of the diet, it is obvious that reduced intake may lead to deficiency. Care must be taken (and adjustments made when necessary) to meet the animal's requirement when intake varies. The same consideration must be made for various non-nutrient additives (antibiotic premixes, growth promotants, etc.) when they are to be ingested at a constant rate daily. Obviously, ration adjustments for components that are not directly affected by the thermal environment should be altered in proportion to the changes in rate of intake. For this reason, it is important that accurate estimates of voluntary intake during thermal stress be established.

In summary, both direct and indirect influences of the environment have been shown to have a major impact on nutrient intake, requirement, and utilization. Effect of environment on rates of energy flow is important in all cases and is the basis for most observed nutrition-environmental interactions. Clearly, the role of environment in livestock nutritional management is a valid consideration.

*

*   *

CHAPTER 5

SOME ASPECTS OF THE METEOROLOGICAL FORECASTING OF ANIMAL HEALTH AND DISEASES

5.1   INTRODUCTION

Twenty-five years ago it could have been said that a research effort to forecast animal diseases using weather data was an esoteric interest of dubious value. Since that time a large and rapidly increasing number of valuable research and extension applications have been developed.

Under the auspices of WMO, questionnaires for information on research projects on weather and animal health and disease, were prepared by the CAgM VI Working Group on Weather and Animal Health and sent to all WMP Permanent Representatives for distribution to relevant institutions, and separately by working group members to many veterinary and agricultural colleges and research institutes world-wide. Also, a computerized search was made of the Smithsonian Science Information Inc. files for relevant research projects. A total of 112 projects wre identified for 1977-1978. Approximately a third were of parasitic conditions, a third on physiological and environmental aspects of animal production and management, and a third on animal diseases. The majority (71%) were only peripherally concerned with meteorological applications, with, however, the exception of parasite research in which meteorology was an integral part of 50% of the projects. In a significant number of instances when meteorological applications were noted or even encountered, they were pursued. Fifty per cent of the studies were being made in the UK, long a leader in research on the impact of weather on parasites and parasite diseases, while the USA has been more active in animal physiology and weather.

While forecasts of parasitic conditions are the most numerous for historical and ecological reasons at present, the spectrum of forecasts covers many microbial and metabolic diseases, as well as problems arising from animal management. The situation is well exemplified by the relevant WMO Technical Note on "Weather and Parasitic Animal Disease" edited by T.E. Gibson (WMO, 1978). This was commissioned in order to bring together existing knowledge in the belief that there was little more to be some in forecasting parasite disease. Instead it was discovered that this was a burgeoning area of study and relevant applications. Unfortunately the publication date was too early for a number of papers that have since appeared, some in particular, on the mathematical relationship of weather to the hatching and dispersal of flying insects acting as vectors for a number of animal and human diseases.

Weather is the major external factor affecting agriculture, both on the individual farm and at the national level (Capstick, 1970). Thompson and Taylor (1975) of the Economic Service of the New Zealand Meat and Wool Board stated that, "The unpredictability of supply at whatever level disrupts the smooth flow of products from farm gate to consumer, increasing costs to all involved. Weather effects on flow patterns of major products within seasons can be significant, with implications for the scheduling of auctions, in the case of wool and meat, and for the arrangement of shipments to export markets." This affect was given emphasis by Omar (1978) who indicated that of all the potential gains in the USA that would accrue as a result of better meteorological forecasts, 77%, or $570 million, would be to agriculture. This

was for an agriculturally advanced country and did not include the hard-to-quantify benefits to the individual farmer. Disease forecasting forms only a small, but at present unknown, percentage of that potential gain. The measurement of animal disease costs, and health benefits, is a developing discipline among veterinary epidemiologists. We have just begun to emerge from the period of overstating estimates of disease costs into a period of more modest analysis. It is clear that while the direct costs (of lost production and increased managerial and medical costs) of disease are not slight, the major cost to the farmer is that disease constrains his managerial and entrepreneural abilities. This he values more highly than the cash-flow embarrassments caused by disease.

5.2   FORECASTING AIMS AND OBJECTIVES

The aim of forecasting animal disease should be to provide a service to national animal health programme managers and to farmers to aid them in the making of the strategic and tactical decisions necessary to their respective responsibilities.

Forecasting is not possible without the initial research. The continual need to refine a model uncovers more and more of the components of disease and so makes a significant contribution to a deeper understanding of the interactions between the disease processes and the weather. It seems a paradoxical but common experience that applied research produces more basic and objective research results than basic research alone.

The objectives in achieving the priority aim of a service are to provide accurate and reliable forecasts that will allow enough time for appropriate action to be taken in order to prevent or ameliorate conditions that might be of economic consequence.

Accuracy can be used in regard to either relative rates or to the absolute numbers of outbreaks of disease or of affected animals. The former, for example, is found in a forecast for fascioliasis (Ollerenshaw and Rowlands, 1959). The forecast for the future is compared to the current or recent years(s) in the region, i.e. the present experience of those concerned. The advice therefore takes the form that the future will be "better" or "worse" than the present and action should be taken accordingly. The use of terms such as high, low or moderate, though imprecise, are well understood. Forecasts in absolute terms are more common, which allows for retrospective analyses on the causes of success or failure of the forecast. Unfortunately the use of numbers can imply a greater precision than the underlying research may support. Whether the forecast is in relative or absolute terms, its precision should be indicated by the supporting documentation and the manner in which it is presented. Accuracy and precision should not be confused. Lack of precision is more acceptable than lack of accuracy, but this should not be taken as license to fudge the forecast. A 50% accuracy is the equivalent of tossing a coin; at any lower rate a coin is both cheaper and more accurate. It must be remembered that the forecast is provided so that action can be taken by others; the forecaster is neither making the decision nor initiating action. It is up to the decision-maker whether or not to accept the forecast and what action to take.

Reliable forecasts are the result of hard work and some luck. If the forecasts are made as a result of efficient data collection and analysis,

issued at regular intervals and consistent in quality, they will engender confidence and can withstand the occasional spectacular miss. If the reports appear erratically with variable content and are sometimes "right" and sometimes "wrong" the work will be disregarded and eventually dismissed.

Timeliness is important. Action on the forecast should be completed before the expected disease process. Sometimes by the very nature of the disease the interval between the contributing weather factor and the disease is very short, as in temperature stress-related conditions. Here prevention has to be emphasized. With foot-and-mouth disease the minimum incubation period is four to six days. Efficient foot-and-mouth control teams can respond almost on an hour-to-hour basis, and certainly day-by-day, so the lead time is sufficient to relocate search teams. Omar (1978) has shown that the maximum economic gain is on 90-day forecasts. Unfortunately not all weather/disease complexes allow such a long lead time. It must be remembered that the action to control or prevent a disease will include not only staff logistics and the use of drugs and vaccines, but also pulicity. It takes time and skill to persuade farmers.

The economic importance of the disease(s) being forecast must be considered when setting the priorities. A highly accurate forecast for a trivial livestock disease may be regarded as irrelevant though expensive in terms of staff effort and time.

Lastly the disease should be preventable or respond favorably to whatever action is taken. Livestock management, if warned in time, can take action to minimize economic loss. Forecasting the reduction, as a result of disease, in the quality and the quantity of animals and animal products which will come on the market sometime in the future, is of advantage to economic services within national departments of agriculture. While it is difficult to think of a livestock disease that is neither preventable, nor treatable, nor lessened by management, forecasting priority should be given to the conditions whose prevention will give the highest economic return.

5.3   ADVANTAGES AND DISADVANTAGES OF FORECASTING DISEASE

Agricultural meteorology gives some very tangible direct advantages to the livestock industry. For example:

(a) Strategic: The planning and direction of health programmes are aided by seasonal forecasts such as for fascioliasis and nematodiriasis, and for airborne infections such as foot-and-mouth disease.

(b) Tactical: Knowing the dates for maximum larval hatch and tick rise can optimize the use of helminthicides and acaricides reducing the volume of unnecessary dosing and animal handling and thus the cost.

(c) Additive: In areas with chronic problems, such as mineral deficiencies, farmers become less attentive to the provision of minerals and other prophylactic measures. Warnings of increased numbers of outbreaks of these conditions such as swayback, due to congenital copper deficiency, add to and reinforce advice being given by others.

(d) Supportive: Specific warnings and advice on preventive care will reduce helplessness and increase agricultural/meteorological contacts.

(e) Research: The research effort to produce forecasts or implement existing systems will clarify disease cycles and identify specific questions for which answers are needed; this is especially fruitful when analyzing why forecasts were not correct. Because the research frequently uses existing data, its costs are minimal while the benefits, if properly utilized, are very large. The work of J. Donnelly on Ixodes (WMO, 1978) briefly quoted in the WMO Technical Note, exemplifies these advantages.

(f) Data base: While meteorological data are usually available in adequate detail, regular reports of animal diseases are sparse and frequently biased. The development and use of disease monitoring systems produce benefits far beyond those initially concerned, both in the provison of improved veterinary care as well as in epidemiological control and knowledge of disease.

There are, however, some disadvantages:

(a) Unavoidable mistakes: One hundred percent accuracy as to the time, place and volume of future disease outbreaks is impossible. Some diseases will never be forecastable with reasonable reliability or adequate lead-time.

(b) Increased costs: Although the work is inexpensive, it does have a cost which has to be met though the cost:benefit ratio is far in excess of parity. For example, the initial United Kingdom government research effort on the meteorological aspects of foot-and-mouth disease involved approximately 2 1/2 man-years. Out of this research came the development of a simple search technique for new outbreaks based on recent weather. This technique needed to prevent only one outbreak in order to pay for all the research that had been done. Subsequent research refined this forecasting facility with spectacular success (Donaldson, et al., 1982).

(c) Probabilities: The problem takes two forms. Firstly a forecast is an expression of probabilities, which can be misunderstood. Secondly, once the future is known there is a commitment to "do something", even if masterly inactivity would be the best economic decision.

5.4   COMMITMENT

      Enough forecasting systems exist and many have been published (WMO, 1978; Smith, 1970) and they cover techniques that can be adapted for

other diseases as well as systems which can be used in whole or in part. In most instances all that is needed to start is the desire.

Any project to forecast animal disease should start with conditions covered by the priorities discussed above. Obviously it is wise to start with a known disease of recognized importance with a proven forecasting system. The team appointed to a project should be adequately trained and given a stated number of years to properly test and demonstrate the advantages, if any, of disease forecasts. If at the end of the period the team has succeeded the period can more easily be extended, and vice versa.

At the same time the team should be given access to adequate data and to a data-gathering capacity (reliable disease data may not be readily available). Searches should first be made for existing and usable data which is more commonly available than is realized. Many herd managers keep more than adequate records and some local animal health officers and assistants and private veterinary practitioners keep scrupulous records of animals diagnozed and treated. If records do not exist against which the accuracy of forecasts can be made, systems will have to be constructed with the help of veterinarians and animal health officers. The simpler the system the better the data it will contain. These data can also provide information on the economic importance and advantages of the work.

The team should also be supported by communications experts who can relay the forecasts to the agricultural community. Communication should be into two directions with feed-back from the community as to the usefulness of the forecasts. Similarly, recognition must be given to the importance of communication with and through local and government authorities and community leaders.

5.5   CONCLUSIONS

The forecasting of parasitic conditions will undergo a continuing process of quantification and increasing precision. It is also necessary that parasitic conditions should be examined for sensitivity and specificity so that their economic consequences can be better understood. As successful forecasting systems for mosquitoes and midges have emerged, parasites (and also ticks) should be more widely studied in spite of the extensive capture data-base needed. More research groups should examine the aerobiological aspects of diseases disseminated through the spreading of viruses and other organisms, the role of rainfall on anthrax and salmonella, also on mycoses and myotoxioses, reproduction efficiency and production, growth and neonatal mortality.

Current studies on lactation, growth, fertility and egg production largely relate to climatic, seasonal adaptive physiology, but there is an increasing need for relating short-term weather events to the incidence of animal disease. At this time, it is possible to predict meteorological seasonal effects, and, with some moderate effort, the effects of events on animal production processes. For an animal production expert to provide a valid meteorological prediction on production, the qualifying multi-factorial influences of disease, parasites, nutrition, and management must be expressed quantitatively, and the meteorological predictions must be reliable. In general, one may find that the prediction of the effects of weather events on animal production is only as valid as the ability to assess or predict the

disease or parasite influence on the production unit. Whether the environmental stresses are bio-meteorological or due to disease, management, nutrition, or trauma, the responsive and adaptive physiological mechanisms must be known. Further research is needed to clarify and relate animals' functions and responses to weather events.

It is opportune to quote Maunder (1977): "Of prime importance is the continuing need to assess and present impacts of weather in terms of production figures, costs or similar measures, which can be used _directly_ by decision makers -- including economists, agriculturalists (in which we include farmers), planners and politicians. The world is becoming much more sensitive -- in economic, social and political senses -- to the resources of the atmosphere. For this reason, any variation in these resources, which in the past were easily accommodated, have now become very significant factors in both short- and long-term planning."

C H A P T E R   6

REFERENCES

1.1 ANIMAL HEALTH - A ROLE FOR AGRICULTURAL METEOROLOGISTS

Smith, C.V. 1983. Airborne infection and livestock disease control: a meteorological appreciation.
Phil. Trans. R. Soc. London B 302:543-557.

1.4 "CRITICAL TEMPERATURES" - A DISCUSSION

Hahn, G.L., A. Nygaard and E. Simensen. 1983. Towards establishing rational criteria for selection and
design of livestock establishments. Paper 83-4517, 1983 Winter Meeting, Am. Soc. Agric. Engineers,
December 13-16. Chicago, Illinois.

2.1.1 CATTLE - COLD STRESS: General Observations

Ames, D.R. 1980. Livestock nutrition in cold weather. Anim. Nutri. and Health, Oct. 1980. p 6.

Ames, D.R. and L.W. Insley. 1975. Wind-chill effect for cattle and sheep. J. Anim. Sci. 40:161-165.

Blaxter, K.L. 1967. The Energy Metabolism of Ruminants. Hutchinson Ltd., London.

McDowell, R.E. and J.E. Johnston. 1971. Research under field conditions. Pages 306-359 in A Guide to
Environmental Research on Animals. National Academy of Sciences, Washington.

Milligan, J.D. and G.I. Christison. 1974. Effects of severe winter conditions on performance of feedlot
steers. Can. J. Anim. Sci. 54:605-610.

Webster, A.J.F. 1974. Heat loss from Animals and Man. Proceedings, 20th Easter School, University of
Nottingham, School of Agriculture. London, Butterworths. p. 205.

2.1.2 CATTLE - COLD STRESS: Reproduction and Lactation

Agriculture Canada. 1974. Rules governing the Canadian record of performance for dairy cattle.
Agriculture Canada, Ottawa.

ASHRAE. 1984. Handbook of Fundamentals. American Society of Heating, Refrigerating and Air-Conditioning
Engineers, Inc., New York.

Bath, D.L., F.N. Dickinson, H.A. Tucker and R.D. Appleman. 1978. Dairy cattle: principles, practices,
problems, profits. 2nd ed. Lea and Febiger, Philadelphia.

Brody, S., A.C. Ragsdale, R.G. Yeck and D.M. Worstell. 1955. Milk production, feed and water consumption,
and body weight of Jersey and Holstein cows in relation to several diurnal temperature rhythms. Res.
Bull. Mo. Agr. Exp. Sta. No. 578.

Bryson, R.A. and F.K. Hare. 1974. World survey of climatology. Vol. II: Climates of North America.
Elsevier Scientific Publ. Co., Amsterdam.

Cobble, J.W. and H.A. Herman. 1951. The influence of environmental temperatures on the composition of
milk of the dairy cow. Res. Bull. Mo. Agr. Exp. Sta. No. 485.

Dice, J.R. 1940. The influence of stable temperature on the production and feed requirements of dairy
cows. J. Dairy Sci. 23:61-69.

REFERENCES

Heizer, E.E., V.R. Smith and C.E. Zehner. 1953. A summary of studies comparing stanchion and loose housing barns. J. Dairy Sci. 36:281-292.

Johnson, H.D. 1965. Environmental temperature and lactation (with special reference to cattle). Int. J. Biometeor. 9:103-116.

Kibler, H.H. and S. Brody. 1953. Influence of humidity on heat exchange and body temperature regulation in Jersey, Holstein, Brahman and Brown Swiss cattle. Res. Bull. Mo. Agr. Exp. Sta. No. 522.

MacDonald, M.A. and J.M. Bell. 1958a. Effects of low fluctuating temperatures on farm animals. II. Influence of ambient air temperature on water intake of lactating Holstein-Fresian cows. Can. J. Anim. Sci. 38:23-32.

MacDonald, M.A. and J.M. Bell. 1958b. Effects of low fluctuating temperatures on farm animals. III. Influence of ambient air temperature on feed intake of lactating Holstein-Fresian cows. Can. J. Anim. Sci. 38:148-159.

MacDonald, M.A. and J.M. Bell. 1958c. Effects of low fluctuating temperatures on farm animals. IV. Influence of temperature on milk yield and milk composition. Can. J. Anim. Sci. 38:160-170.

McDowell, R.E., N.W. Hooven and J.K. Camoens. 1976. Effect of climate on performance of Holsteins in first lactation. J. Dairy Sci. 59:965-973.

Oosterlee, C.D. 1959. The influence of climatic factors on the heat balance and milk yield of the cow in relation to the design of farm buildings in the Netherlands. Int. J. Biometeor. Bioclimatol. III(III) C:1-10.

Ragsdale, A.C., H.J. Thomson, D.M. Worstell and S. Brody. 1950. Milk production and feed and water consumption responses of Brahman, Jersey and Holstein cows to changes in temperature, 50° to 105°F and 50° to 8°F. Res. Bull. Mo. Agr. Exp. Sta. No. 460.

Ragsdale, A.C., H.J. Thompson, D.M. Worstell and S. Brody. 1953. The effect of humidity on milk production and composition, feed and water consumption, and body weight on cattle. Res. Bull. Mo. Agr. Exp. Sta. No. 521.

Ragsdale, A.C., D.M. Worstell, H.D. Thompson and S. Brody. 1959. Influence of temperature, 50° to 0°F and 50° to 95°F, on milk production, feed and water consumption and body weight in Jersey and Holstein Cows. Res. Bull. Mo. Agr. Exp. Sta. No. 449.

Rako, A. and F. Dumanovsky. 1956. Effect of low temperatures on milk production on the cow. Vet. Bull. 26:169.

Williams, C.M. and J.M. Bell. 1964. Effects of low fluctuating temperatures on farm animals. V. Influence of humidity on lactating dairy cows. Can. J. Anim. Sci. 44:114-119.

Witzel, S.A. and E.E. Heizer. 1946. A 5 year summary of dairy barn research. Agr. Eng. 27:449-505, 508.

Yeck, R.E. and R.E. Stewart. 1959. A ten year summary of the psychroenegetic laboratory dairy cattle recorded at the University of Missouri. Trans. Am. Soc. Agr. Eng. 2:71-77.

2.13 CATTLE - COLD STRESS: Neonatal Survival and Growth

Alexander, G., J.W. Bennett and R.T. Gemmell. 1975. Brown adipose tissue in the newborn calf (Bos taurus). J. Physiol. (Lond) 244:223.

Ames, D.R. 1980. Livestock nutrition in cold weather. Anim. Nutr. Health. 35: 6, 7, 38, 39.

Anderson, J.F. and D.W. Bates. 1982. Interaction of environment and genetics. pp. 298-302, Livestock Environment II. SP 03-82, Am. Soc. Agric. Engrs, St. Joseph, MI.

2.14 CATTLE - COLD STRESS: Acclimatization, Chronic and Acute Exposures and Reactions

Blaxter, K.L. 1967. The energy metabolism of ruminants. Hutchinson Ltd, London.

Christopherson, R.J. 1976. Effects of prolonged cold and the outdoor winter environment on apparent digestibility in sheep and cattle. Can. J. Anim. Sci. 56:201-212.

Christopherson, R.J., R.J. Hudson and M.K. Christopherson. 1979. Seasonal energy expenditures and thermoregulatory responses of bison and cattle. Can. J. Anim. Sci. 59:611-617.

Holmes, C.W. 1971. Local cooling of the mammary gland and milk production in the cow. J. Dairy Res. 38:3-7.

Kelley, K.W., C.A. Osborne, J.F. Evermann, S.M. Parish and C.T. Gaskins. 1982. Effects of Chronic heat and cold stressors on plasma immunoglobulin and mitogen-induced blastogenesis in calves. J. Dairy Sci. 65:1514-1528.

Kennedy, P.M. and L.P. Milligan. 1978. Effect of cold exposure on digestion, microbial synthesis and nitrogen transformation in sheep. Br. J. Nutr. 35:105-117.

NRC. 1981. Effect of Environment on Nutrient Requirements of Domestic Animals. National Academy of Science, Washington, D.C.

Robertshaw, D. 1981. The environmental physiology of animal production. Pages 3-17 in Environmental aspects of housing for animal production. J. A. Clark, ed., Butterworths, London.

Thompson, G.E. and E.M. Thomson. 1977. Effect of cold exposure on mammary circulation, oxygen consumption and milk secretion in the goat. J. Physiol. 272:187-196.

Young, B.A. 1975. Effects of winter acclimatization on resting metabolism of beef cows. Can. J. Anim. Sci. 55:619-625.

Young, B.A. and A.A. Degan. 1981. Thermal influences on ruminants. Pages 167-180 in Environmental Aspects of Housing for Animal Production, J. A. Clark (ed), Butterworth, London.

Young, B.A., A.A. Degen and J.F. Francis. 1979. Will cows eat snow? 58th Annual Feeders Day Report, Univ. of Alberta.

Whittow, G.C. 1962. The significance of the extremities of the ox (Bos taurus) in thermoregulation. J. Agric. Sci. Camb. 58:109-120.

2.21 SHEEP AND COLD STRESS: General Comments

Armstrong, D.G., K.L. Blaxter, N. McC Graham and F.W. Wainman. 1959. The effect of environmental conditions on food utilisation by the sheep. Anim. Prod. 1:1-12.

Davis, G.B. 1973. Effect of weather on conception rates of sheep. NZ Vet. J. 21:54-59.

Hugh-Jones, M.E. 1976. Effects of heat and cold on diseases in sheep. In: Progress in Biometeorology. Vol 1, part 1, (ed. J.D. Johnson), Switz and Zeitlinger, Amersterdam, pp 480-489.

Macfarlane, W.V. 1976. Water metabolism in sheep in relation to heat and cold. In: Progress in Biometeorology. Vol 1, part 1, (ed. J.D. Johnson), Switz and Zeitlinger, Amsterdam, pp. 49-57.

Smith, L.P. 1970. Weather and animal disease. WMO, Geneva, Technical Note 113.

Webster, W.M. 1939. Review of the past season's work upon the problem of infertility in Romney Rams. Proc. 8th Annual Meeting of Sheep Farmers. 85-94 (quoted by Davies, 1973).

2.22 SHEEP AND COLD STRESS: Neonatal Survival

Alexander, G. 1962. Temperature regulation in the newborn lamb: V. Summit metabolism. Aust. J. Agric. Res. 13:100-121.

Alexander, G. 1964. Lamb survival: Physiological considerations. Proc. Aust. Soc. Anim. Prod. 5:113-122.

Alexander, G. 1974a. Heat loss from sheep. In: "Heat loss from animals and man." (ed. J.L. Monteith and L.E. Mount.) Butterworths, London. pp 173-203.

Alexander, G. 1974b. Birthweight in lambs: Influences and consequences. CIBA Symposium #27 (new series), "Size at Birth". Associated Science Publishers, Amsterdam.

Alexander, G. and A.W. Bell. 1975a. Quantity and calculated oxygen consumption during summit metabolism of brown adipose tissue in newborn lambs. Biol. Neonat. 26:214-220.

Alexander, G. and A.W. Bell. 1975b. The maximum thermogenic response to cold in relation to the proportion of brown adipose tissue and skeletal muscle in the body and to other parameters in young lambs. Biol. Neonat. 26:182-194.

Alexander, G., J.J. Lynch, B.E. Mottershead, and J.B. Donnelly. 1980. Reduction in lamb mortality by means of grass windbreaks: Results of a five year study. Proc. Aust. Soc. Anim. Prod. 13:329-332.

Alexander, G. and J.E. Peterson. 1961. Neonatal mortality in lambs. Aust. Vet. J. 37:371-381.

Alexander, G. and D. Williams. 1966. Teat-seeking activity in lambs during the first hours of life. Anim. Behav. 14:166-176.

Blaxter, K.L. 1964. Problems of cold environments. Proc. 6th Int. Congr. Nutrition, Edinburgh, 1963, pp 227-242.

Burroughs, W. 1978. Cold winters and the economy. New Scientist, January 19th, pp. 146-148.

Dalton, D.C., T.W. Knight, and D.L. Johnson. 1980. Lamb survival in sheep breeds on New Zealand hill country. NZ J. Agric. Res. 23:167-173.

Dennis, S.M. and M.E. Nairn. 1970. Perinatal lamb mortality in a Merino flock in Western Australia. Aust. Vet. J. 46:272-276.

Eales, F.A. 1982. Detection and treatment of hypothermia in newborn lambs. In Practice. 4:20-22.

Eales, F.A. and J. Small. 1980. Summit metabolism in newborn lambs. Res. Vet Sci. 29:211-218.

Eales, F.A., J.S. Gilmour, R.M. Barlow and J. Small. 1982a. Causes of hypothermia in 89 lambs. Vet. Rec. 110:118-120.

Eales, F.A., J. Small and J.S. Gilmour. 1982b. Resuscitation of hypothermic lambs. Vet. Rec. 110:121-123.

Egan, J.K., R.L. Thompson and J.S. McIntyre. 1976. An assessment of overgrown Phalaris tuberosa as a shelter for newborn lambs. Proc. Aust. Soc. Anim. Prod. 11:157-160.

Haughey, K.G. 1980. The role of birth in the pathogenesis of meningeal haemorrhage and congestion in newborn lambs. Aust. Vet. J. 56:49-56.

Hight, G.K. and K.E. Jury. 1969. Lamb mortality in hill country flocks. Proc. NZ Soc. Anim. Prod. 29:219-232.

Houston, D.C. and J.G. Maddox. 1974. Causes of mortality amoung young Scottish Blackface lambs. Vet. Rec. 95:575.

Johnston, W.S. 1977. Caithness sheep loss survey: Perinatal lamb losses. In: "Symposium on Perinatal Losses in Lambs", East of Scotland College of Agriculture, Stirling. 1975, pp. 10-13.

Kilgour, R. and H. de Langen. 1980. Neonatal behaviour in easy care sheep. Revs. in Rural Sci. IV:117-121.

McCutcheon, S.N. 1982. A study of some factors affecting the resistance of newborn lambs to cold stress with particular reference to starvation and exposure mortlity. PhD Thesis, Massey University, New Zealand.

McDonald, J.W. 1966. Variation in perinatal mortality of lambs with age and parity of ewes. Proc. Aust. Soc. Anim. Prod. 6:60-62.

Mercer, J. 1974. Thermoregulation in the newborn lamb. PhD Thesis, Trinity College, Dublin.

Obst, J.M. and H.R. Day. 1968. The effect of inclement weather on mortality of Merino and Corriedale lambs on Kangeroo Island. Proc. Aust. Soc. Anim. Prod. 7:239-242.

Obst, J.M. and J.V. Ellis. 1977. Weather, ewe behavious and lamb mortality. Agricultural Record. 4:44-49.

Olaechea, F.V., J.P. Bellati, M. Suarez, J.M. Puyo and C. Robles. 1981. Mortalidad perinatal de corderos en el oeste de la Provincia de Rio Negro. Rev. Med. Vet. (Bs As). 62:128-134.

Purser, A.F. 1967. Is lamb birthcoat type related to hardiness? Animal Breeding Research Organisation Report, pp. 26-34.

Purser, A.F. and G.B. Young. 1964. Mortality amoung twin and single lambs. Anim. Prod. 6:321-329.

Samson, D.E. 1982. Genetic and physiological aspects of resistance to hypothermia in relation to neonatal lamb survival. PhD Thesis, University of Edinburgh.

Samson, D.E. and J. Slee. 1981. Factors affecting resistance to induced body cooling in newborn lambs of 10 breeds. Anim. Prod. 33:59-65.

Sidwell, G.M., D.O. Everson and C.E. Terrill. 1962. Fertility, prolificacy and lamb viability of some purebreeds and their crosses. J. Anim. Sci. 21:875-879.

Slee, J. 1976. Cold stress and perinatal mortality in lambs. 16th Vet. Ann, Wright, Bristol, pp. 66-69.

Slee, J. 1978. The effects of breed, birthcoat, and bodyweight on the cold resistance of newborn lambs. Anim. Prod. 27:43-49.

Slee, J. 1979. Mortality and resistance to hypothermia in young lambs. Biomet. Survey 1, Part B, pp. 60-65.

Slee, J. 1981. A review of genetic aspects of survival and resistance to cold in newborn lambs. Livestock Prod. Sci. 8:419-429.

Slee, J., R.G. Griffiths, D.E. Samson and S.B. Wilson. 1979. Animal Breeding Research Organisation Report, pp. 39-40.

Slee, J., R.G. Griffiths and D.E. Samson. 1980. Hypothermia in newborn lambs induced by experimental immersion in a water bath and by natural exposure outdoors. Res. Vet. Sci. 28:275-280.

Starr, J.R. 1981. Weather and lamb mortality in a commercial lowland sheep flock. Agric. Meteorol. 24:237-252.

Sykes, A.R., R.G. Griffiths and J. Slee. 1976. The influence of breed, birthweight and weather on the body temperature of newborn lambs. Anim. Prod. 22:395-402.

Wiener, G., F.K. Deeble, J.S. Broadbent and M. Talbot. 1973. Breed variations in lambing performance and lamb mortality in commercial sheep flocks. Anim. Prod. 17:229-243.

Wiener, G., C. Woolliams and N.S.M. Macleod. 1983. The effects of breed, breeding system and other factors on lamb mortality: (1) Causes of death and effects on the incidence of losses. J. Agric. Sci. (Camb.). 100:539-551.

Wilson, A.L. 1954. The feeding of farm animals for health and production. BVA Publication. 24:117-122.

2.31 PIGS - COLD STRESS: General Observations

Mount, L.E. 1975. The assessment of thermal environment in relation to pig production. Livestock Prod. Sci. 2:381-392.

2.32 PIGS - COLD STRESS: Reproduction and Lactation

Baxter, S.H. 1978. Some general principles of housing for reproductive efficiency in pigs. Vet. Annual 18:126-133.

Baxter, S.H. and A.M. Robertson. 1980. Pig housing - the last 10 years. Pig News and Info. 1:21.

Fahmy, M.H. 1975. Effects of temperature and level of feeding during rearing on carcass and reproductive traits of pigs. Livestock Prod. Sci. 2:247:259.

Hacker, R.R., M.P. Stefanovic and T.R. Batra. 1973. Effects of cold exposure on growing pigs: growth, body composition and 17-ketosteroids. J. Anim. Sci. 37:739-744.

Ministry of Agriculture, Fisheries and Food. 1972. Housing the pig. Reference No. (0 11 24060 X) H.M.S.O., London, England.

Randall, G.C.B. and R.H.C. Penny. 1970. Stillbirth in the pig: an analysis of the breeding records of five herds. Br. vet. J. 126:593-603.

Sainsbury, D.W.B. 1976. Pig Housing 4th Edition. Farming Press., Ipswich.

Swierstra, E.E. and G.W. Rahnefield. 1972. Effect of cold stress and repeated mating on reproductive performance of swine. Can. J. Anim. Sci. 52:309-316.

2.33 PIGS - COLD STRESS: Neonatal Survival and Growth

Blecha, F. and K.W. Kelley. 1981. Cold stress reduces the acquisition of colostral immunoglobulin in piglets. J. Anim. Sci. 52:594-600.

Hendrix, W.F., K.W. Kelley, C.T. Gaskins and D.J. Hinrichs. 1978. Porcine neonatal survival and serum gamma globulins. J. Anim. Sci. 47:1281-1286.

Kelley, K.W., F. Blecha and J.A. Regnier. 1982. Cold exposure and absorption of colostral immunoglobulins by neonatal pigs. J. Anim. Sci. 55:363-368.

LeDividich, J. and J. Noblet. 1981. Colostrum intake and thermoregulation in the neonatal pig in relation to environmental temperature. Biol. Neonate 40:167-174.

NRC. 1981. Effect of environment on nutrient requirements of domestic animals. National Academy of Sciences - National Research Council, Washington, D.C.

Parker, R.O., P.E.V. Williams, F.X. Aherne and B.A. Young. 1980. Serum concentration changes in protein, glucose, urea, thyroxine and triiodothyronine and thermostability on neonatal pigs farrowed at 25°C and 10°C. Can. J. Anim. Sci. 60:503-509.

Verstegen, M.W.A., H.A. Brandsma and G. Mateman. 1982. Feed requirements of growing pigs at low environmental temperatures. J. Anim. Sci. 5:88-94.

2.34 PIGS - COLD STRESS: Acclimatization, Chronic and Acute Exposure and Reactions

Blecha, F. and K.W. Kelley. 1981a. Cold stress reduces the acquisition of colostral immunoglobulin in piglets. J. Anim. Sci. 52:594-600.

Blecha, F. and K.W. Kelley. 1981b. Effects of cold and weaning stressors on the antibody-mediated immune response of pigs. J. Anim. Sci. 53:439-447.

Curtis, S.E., D.A. Kingston, J. Simon and J.G. Brummond. 1976. Effects of age and cold on pulmonary bacterial clearance in the young pig. Am. J. Vet. Res. 37:299-301.

Kelley, K.W. 1983. Immunobiology of domestic animals as affected by hot and cold weather. Trans. Am. Soc. Agr. Eng.

Kelley, K.W., F. Blecha and J.A. Regnier. 1982. Cold exposure and absorption of colostral immunoglobulins by neonatal pigs. J. Anim. Sci. 55:363-368.
LeDividich, J. and J. Noblet. 1981. Colostrum intake and thermoregulation in the neonatal pig in relation to environmental temperature. Biol. Neonate. 40:167-174.

Mersmann, H. 1974. Metabolic patterns in the neonatal swine. J. Anim. Sci. 38:1022-1030.

2.4   POULTRY - COLD STRESS

Arieli, A., A. Meltzer and A. Berman. 1979. Seasonal acclimatization in the hen. Br. Poult. Sci. 20:505-513.

Aulie, A. 1976. The shivering pattern in an artic (willow Ptarmigan) and a tropical bird (Gantam hen). Comp. Biochem. Physiol. 53A:347-350.

Bobek, S., J. Niezgoda, M. Pietras, M. Kacienska and Z. Ewy. 1980. The effect of acute cold and warm ambient temperatures on the thyroid hormone concentration in blood plasma, blood supply, and oxygen consumption in Japanese quail. Gen. Comp. Endocrinol. 40:201-210.

Brandley, C.A., H.E. Moses, E.R. Jones and E.L. Jungherr. 1946. Epizootiology of Newcastle disease of poultry. Am. J. Vet. Res. 7:243-249.

Cogburn, L.A., P.C. Harrison and D.E. Brown. 1976. Scotophase-dependent thermoregulatory dysfunction in pinealectomized chickens. Proc. Soc. Exp. Biol. Med. 153:197-201.

Davison, T.B. 1973. Metabolite changes in the neonate fowl in response to cold stress. Comp. Biochem. Physiol. 44A:979-989.

REFERENCES

Davison, T.F. and P.A. Lickiss. 1979. The effect of cold stress on the fasted, water deprived, neonate chicken (Gallus domesticus). J. Therm. Biol. 4:113-120.

Ensor, D.M. 1975. Prolactation and adaptation. In: Avian Physiology. (ed. M. Peaker). Symp. Zool. Soc. Lond. No. 35:129-148. London, Academic Press.

Farrell, D.J. and S. Swain. 1977. Effects of temperature treatments on the heat production of starving chickens. Br. Poult. Sci. 18:725-734.

Freeman, B.M. 1971. Body temperature and thermoregulation. In: Physiology and Biochemistry of the Domestic Fowl. (ed. D.J. Bell and B.M. Freeman). Vol. 2, Ch. 48, 1115-1151. London, Academic Press.

Glaja, J. and S. Gelineo. 1933. Sur la resistance an fraid de quelques homeothermes. Arch. Int. Physiol. 37:20-34.

Hagan, A.A. and J.E. Heath. 1976. Metabolic responses of white Pekin ducks to ambient temperature. Poult. Sci. 55:1899-1905.

Hensel, H. 1981. Neural processes in long-term thermal adaptation. Fed. Proc. 40:2830-2834.

Keller, J.S. 1980. Role of glucagon in the regulation of metabolism in growing chickens. Proc. 8th EAAP Symp. on Energy Metabolism (ed. L.E. Mount). 417-419. London, Butterworths.

Kitchell, R.L., L. Strom and Y. Zolterman. 1959. Electrophysiological studies of thermal and taste reception in chickens and pigeons. Acta physiol. scand. 46:133-151.

Lin, Y-C. and P.D. Sturkie. 1968. Effect of environmental temperatures on the catecholamines of chickens. Am. J. Physiol. 214:237-240.

Mission, B.H. 1976. The effects of temperature and relative humidity on the thermoregulatory response of grouped and isolated neonate chicks. J. agric. Sci. Camb. 86:35-43.

Moreng, R.E. and C.S. Shaffner. 1951. Lethal internal temperatures for the chicken, from fertile egg to mature bird. Poultry Sci. 30:355-266.

Nichelmann, M., S. Ellerkamp, I. Herntrich and L. Lyhs. 1976. Untersuchungen zum Warmehaushalt von Puten. 1 Mitteilung: Warmeproduktion. Mh. Vet-Med. 31:213-217.

Palokangas, R., V. Vihko and I. Nuuja. 1973. The effects of cold and glucagon on lipolysis, glycogenolysis and oxygen consumption in young chicks. Comp. Biochem. Physiol. 45A:489-495.

Poczopko, P. 1981. The environmental physiology of juvenile animals. In: Environmental Aspects of Housing for Animal Production (ed. J.A. Clark). 109-130. London, Butterworths.

Ram, T. and F.B. Hutt. 1955. The relative importance of body temperature and lymphocytes in genetic resistance to Salmonella pullorum in fowl. Am. J. Vet. Res. 16:437-449.

Rautenberg, W. 1971. The influence of skin temperature on the thermoregulatory system of pigeons. J. Physiol. Paris. 63:396-398.

Regnier, J.A. and K.W. Kelley. 1981. Heat and cold stress suppresses in vivo and in vitro cellular immune responses of chickens. Am J. Vet. Res. 42:294-299.

Simensen, E., L.D. Olson and G.L. Hahn. 1980. Influence of Pasteurella multocida and high and low environmental temperatures on adrenals and bursa of Fabricius in turkeys. Avian Dis. 24:844-867.

Soerjadi, A.S., J.H. Druitt, A.B. Lloyd and R.B. Cumming. 1979. Effect of environmental temperature on susceptibility of young chickens to Salmonella typhimurium. Aust. Vet. J. 55:413-417.

Sykes, A.H. 1977. Nutrition environment interactions in poultry. In: Nutrition and the Climatic Environment (ed. W. Haresign, H. Swan and D. Lewis). 17-29. London, Butterworths.

Tauson, R. and S.A. Svenson. 1980. Influence of plumage condition on the hen's feed requirement. Swed. J. agric. Res. 10:35-39.

Untergasser, G. and J.S. Hayward. 1972. Development of thermoregulation in ducklings. Can. J. Zool. 50:1243-1250.

Wathes, C.M. and J.A. Clark. 1981. Sensible heat transfer from the fowl: thermal resistance of the pelt. Br. Poult. Sci. 22:175-183.

2.51 COLD EXPOSURE AND IMMUNE FUNCTION: General Observations

Ferguson, W. and D. Branagan. 1972. Meteorological effects on animal disease. In Progress in Biometerology. Ed. S.W. Tromp. Div. A, Vol. 1, Part III. Biometerological aspects of plants, trees and animals in human life. Chapter 10, pp. 93-108.

2.52 COLD EXPOSURE AND IMMUNE FUNCTION: Disease

Armstrong, W.D. and T.R. Cline. 1977. Effects of various nutrient levels and enviromental temperatures on the incidence of colibacillary diarrhea in pigs: intestinal fistulation and titration studies. J. Anim. Sci. 45:1042-1050.

Hamdy, A.H., A.L. Trapp, C. Gale and N.B. King. 1963. Experimental transmission of shipping fever in calves. Am. J. Vet. Res. 24:287-294.

Jennings, A.R. and R.E. Glover. 1952. Enzootic pneumonia in calves. II. The experimental disease. J. Comp. Path. 62:6-22.

Jericho, K.W. and E.V. Langford. 1978. Pneumonia in calves produced with aerosols of bovine herpes-virus I and Pasteurella haemolytica. Can. J. Comp. Med. 421:269-277.

Kelley, K.W. 1980. Stress and immune function: A bibliographic review. Ann. Rech. Vet. 11:445-478.

Shimizu, M.,Y. Shimizu and Y. Kodama. 1978. Effects of ambient temperatures on induction of transmissible gastroenteritis in feeder pigs. Infect. Immun. 21:747-752.

Shope, R.E. 1955. The swine lungworm as a reservoir and intermediate host for swine influenza virus. V. Provocation of swine influenza by exposure of prepared swine to adverse weather. J. Exp. Med. 102:567-572.

Soerjadi, A.S., J.H. Druitt, A.B. Lloyd and R.B. Cumming. 1979. Effect of environmental temperature on susceptibility of young chickens to Salmonella typhimurium. Aust. Vet. J. 55:413-417.

Weiss, H.S., J.F. Pitt, K.M. Kerr, A.G. Hakaim, S.E. Weisbrode and J.B. Daniel. 1981. Sensitivity of mice to benzo (a) pyrene skin cancer following acclimation to cool and warm temperature. Proc. Soc. Exp. Biol. Med. 167:122-128.

2.53 COLD EXPOSURE AND IMMUNE FUNCTION: Non-specific resistance

Curtis, S.E., D.A. Kingdon, J. Simon and J.G. Drummond. 1976. Effects of age and cold on pulmonary bacterial clearance in the young pig. Am. J. Vet. Res. 37:299-301.

Donald, K.J. 1972. Elevated rates and altered characteristics of the clearance of carbon from the blood of rabbits with variations in environmental conditions. J. Pathol. 107:73-85.

Green, G.M. and E.H. Kass. 1965. The influence of bacterial species on pulmonary resistance to infection in mice subjected to hypoxia, cold stress, and ethanolic intoxication. Br. J. Exp. Pathol. 46:360-366.

Rodbard, D.R. 1981. The role of regional body temperature in pathogenesis of disease. New Eng. J. Med. 305:808-814.

Tizard, I.R. and D.I. Ellicott. 1974. Cell-mediated hypersensitivity to Pasteurella hemolytica in normal and cold-stressed mice. Can. J. Comp. Med. 38:44-48.

Woodard, L.F., W.P. Eckblad, D.P. Olson, R.C. Bull and D.O. Everson. 1980. Hemolytic complement levels of neonatal calves delivered from proteinenergy malnourished dams and subjected to cold stress. Cornell Vet. 70:266-271.

2.54 COLD EXPOSURE AND IMMUNE FUNCTION: Passive Immunity

Blecha, F., R.A. Barry and K.W. Kelley. 1982. Stress-induced alterations in delayed-type hyprsensitivity to SRBC and contact sensitivity to DNFB in mice. Proc. Soc. Exp. Biol. Med. 169:239-246.

Kelley, K.W., F. Blecha and J.A. Regnier. 1982. Cold exposure and absorption of colostral immunoglobulins by neonatal pigs. J. Anim. Sci. 55:363-368.

LeDividich, J. and J. Noblet. 1981. Colostrum intake and thermoregulation in the neonatal pig in relation to environmental temperature. Biol. Neonate. 40:167-174.

Slee, J. 1981. A review on genetic aspects of survival and resistance to cold in newborn lambs. Livestock Prod. Sci. 8:419-429.

2.55 COLD EXPOSURE AND IMMUNE FUNCTION: Active Immunity

Blecha, F., and K.W. Kelley. 1981. Effects of cold and weaning stressors on the antibody-mediated immune response of pigs. J. Anim. Sci. 53:439-447.

Eiseman, B., R.S. Wotkyns and H. Hirose. 1964. Hypothermia and infection: three mechanisms of host protection in type III pneumcooccal peritonitis. Ann. Surg. 160:994-1006.

Kelley, K.W., M.T. O'Conner and D.L. Oldenkamp. 1981. Immunosuppression in calves caused by alternating heat and cold stress. J. Anim. Sci. 53: (Suppl. 1): 184.

Kelley, K.W., C.A. Osborne, J.F. Evermann, S.M. Parish and C.T. Gaskins. 1982. Effects of chronic heat and cold stressors on plasma immunoglobulin and mitogen-induced blastogenesis in calves. J. Dairy Sci. 65:1514-1528.

Klorpes, A.L. and T.M. Yuill. 1975. Environmental modification of western equine encephalomyelitis infection in the snowshoe hare (Lepus americanus). Infect. Immun. 11:986-990.

Regnier, J.A., K.W. Kelley and C.T. Gaskins. 1980. Acute thermal stressors and synthesis of antibodies in chickens. Poult. Sci. 59:985-990.

Sabiston, B.H. and J.E. Ste. Rose. 1976. Effect of cold exposure on the metabolism of immunoglobulins in rabbits. J. Immunol. 116:106-111.

Sabiston, B.H., J.E. Ste. Rose and B. Cinader. 1978. Temperature stress and immunity in mice: effects on environmental temperature on the antibody response to human immunoglobulin of mice differing in age and strain. J. Immunogenetics. 5:197-212.

Ste. Rose, J.E. and B.H. Sabiston. 1971. Effect of cold exposure on the immunologic response of rabbits to human serum albumin. J. Immunol. 107:339-343.

REFERENCES

Shimizu, M.,Y. Shimizu and Y. Kodama. 1978. Effects of ambient temperatures on induction of transmissible gastroenteritis in feeder pigs. Infect. Immun. 21:747-752.

Subba Rao, D.S. and B. Glick. 1977. Effect of cold exposure on the immune response of chickens. Poult. Sci. 56:992-996.

Trapani, I.L. and D.H. Campbell. 1959. Passive antibody decay in rabbits under cold or altitude stress. J. Appl. Physiol. 14:424-426.

2.56 COLD EXPOSURE AND IMMUNE FUNCTION: Cell-mediated immunity

Blecha, F., R.A. Barry and K.W. Kelley. 1982a. Stress-induced alterations in delayed-type hyprsensitivity to SRBC and contact sensitivity to DNFB in mice. Proc. Soc. Exp. Biol. Med. 169:239-246.

Blecha, F., K.W. Kelley and D.G. Satterlee. 1982b. Adrenal involvement in the expression of delayed-type hypersensitivity to SRBC and contact sensitivity to DNFB in stressed mice. Proc. Soc. Exp. Biol. Med. 169:247-252.

Kelley, K.W., R.E. Greenfield, M.T. O'Connor and C.T. Gaskins. 1981a. Acute cold stress in zeranol-implanted steers. J. Anim. Sci. 53: (Suppl. 1): 184.

Kelley, K.W., M.T. O'Conner and D.L. Oldenkamp. 1981b. Immunosuppression in calves caused by alternating heat and cold stress. J. Anim. Sci. 53: (Suppl. 1): 184.

Kelley, K.W., R.E. Greenfield, J.F. Evermann, S.M. Parish and L.E. Perryman. 1982a. Delayed-type hypersensitivity, contact sensitivity, and PHA skin-test responses of heat- and cold-stressed calves. Am. J. Vet. Res. 43:775-779.

Kelley, K.W., C.A. Osborne, J.F. Evermann, S.M. Parish and C.T. Gaskins. 1982b. Effects of chronic heat and cold stressors on plasma immunoglobulin and mitogen-induced blastogenesis in calves. J. Dairy Sci. 65:1514-1528.

Kelley, K.W., H.J. Mertsching, B.A. Mathison and C.R. Kissinger. 1982c. Physiologic concentrations of cortisol suppress mitogenesis of porcine lymphoid cells. J. Anim. Sci. 55: (Suppl. 1): 191.

Regnier, J.A. and K.W. Kelley. 1981. Heat- and cold-stress suppresses *in vivo* and *in vito* cellular immune responses of chickens. Am. J. Vet. Res. 42:294-299.

2.61   ANIMAL PARASITES IN COLD CLIMATES: Internal Parasites and Cold Weather Stress

Anderson, N., J. Armour, R.M. Eadie, F.W. Jarrett, F.W. Jennings, J.S.D. Ritchie and G.M. Urquhart. 1966. Experimental *Ostertagia ostertagi* infections in calves: Results of single infections with five graded dose levels of larvae. Amer. J. Vet. Res. 27:1259-1265.

Armour, J., K. Bairden, J.L. Duncan, F.W. Jennings and J.J. Parkins. 1979. Observations on ostertagiasis in young cattle over two grazing seasons with special reference to plasma pepsinogen levels. Vet. Rec. 105:500-503.

Armour, J. and C.P. Ogbourne. 1981. Bovine ostertagiasis, a review and annotated bibliography. Miscellaneous Publication No. 7 of the Commonwealth Institutes of Parasitology.

Doyle, J.F. 1972. Evidence of an acquired resistance in calves to a single experimental infection with *Fasciola hepatica*. Res. Vet. Sci. 13:456-459.

Gettinby, G., K. Bairden, J. Armour and C. Benitez-Usher. 1979. A prediction model for bovine ostertagiasis. Vet. Rec. 105:57-59.

Gettinby, G. and G. Paton. 1981. The role of temperature and other factors in predicting the pattern of bovine *Ostertagia* spp. infective larvae on pasture. J. Therm. Biol. 6:395-402.

Gibson, M. 1980. The effect of constant and changing temperatures on the development of the free-living stages of Ostertagia ostertagi (Stiles 1892). M.Sc. Thesis. Trinity College, Dublin.

Hope-Cawdery, M.J. 1981. Changing temperatures and prediction models for the liver fluke. (Fasciola hepatica). J. Therm. Biol. 6:403-408.

Hope-Cawdery, M.J., G. Gettinby and J.N.R. Grainger. 1978. Mathematical models for predicting the prevalence of liver fluke disease and its control from biological and meteorological data. In Weather and Parasitic Animal Disease. T.E. Gibson (ed.) Wrld. Meteror. Org., Geneva, WMO-Tech. Note No. 159 p. 21-38.

Jacobs, D.E. 1966. The peri-parturient egg rise of the sow. Vet. Rec. 79:272-273.

Jacobs, D.E. and M.T. Fox. 1985. Relationship between date of spring turnout and lungworm infection in calves. Vet. Rec. 116:75-76.

Michel, J.F. 1969. Observations on the epidemiology of parasitic gastro-enteritis in calves. J. Helminth. 43:111-133.

Michel, J.F., M.B. Lancaster and C. Hong. 1979. The effect of age, acquired resistance, pregnancy and lactation on some reactions of cattle to infection with Ostertagia ostertagi. Parasit. 79:157-168.

Nansen, P., R.J. Jorgensen, J.W. Hansen and K.R. Sejrsen. 1978. Some Factors influencing the exposure of grazing cattle to trichostrongylid infection. Veterinary Science Communications. 2:193-205.

Nice, N.G. and R.A. Wilson. 1974. A study of the effect of temperature on the growth of Fasciola hepatica in Lymnaea truncatula. Parasit. 66:47-56.

Ollerenshaw, C.B. 1974. Forecasting liver fluke disease. In The effects of meteorological factors upon parasites. Symp. Brt. Soc. Parasit. 12:33-52.

Ollerenshaw, C.B. and W.T. Rowlands. 1959. A method of forecasting the incidence of fascioliasis in Angelsey. Vet. Rec. 71:591-598.

Ollerenshaw, C.B. and L.P. Smith. 1969. Meteorological factors and forecasts of helminth disease. Adv. Parasit. 7:283-323.

Over, H. 1982. Personal communication.

Pandey, V.S. 1972. Effects of temperature on development of the free-living stages of Ostertagia ostertagi. J. Parasit. 58:1037-1041.

Ross, J.G. 1970. The Stormont 'wet-day' forecasting system for fascioliasis. Brit. Vet. J. 126:401-408.

Salih, N.E. 1981. A brief review of the development of strongylid nematode eggs and larvae under constant and changing temperature conditions - I. Egg Development. J. Therm. Biol. 6:287-295.

Smeal, M.G., G.C. Fraser, and G.G. Robinson. 1980. Seasonal changes in the structure of nematode populations of cattle in New South Wales in relation to inhibited larval development. Aust. Vet. J. 56:80-86.

Smeal M.G., and A.D. Donald. 1982. Inhibited development of Ostertagia ostertagi. (I) In relation to production system for cattle; (II) Effects of temperature on the infective larval stage. Parasit. 85:(I)21-25; (II)27-32.

Smith, G. 1982. An analysis of variations in the gae structure of Fasciola hepatica populations in sheep. Parasit. 84:49-61.

REFERENCES

Smith, G. 1984. Density-dependent mechanisms in the regulation of <u>Fasciola hepatica</u> populations in sheep. Parasit. 88:449-461.

Smith, G. and R.A. Wilson. 1980. Seasonal variations in the microclimate of <u>Lymnaea truncatula</u> habitats. J. Appl. Ecol. 17:329-342.

Wilson, R.A., G. Smith and M.R. Thomas. 1982. Fascioliasis. The Population Dynamics of Infective Diseases: Theory and Applications. Pp 262-319, edited by R.M. Anderson, Chapman and Hall, London.

Starr, J.R. (Under publication). Weather and climate and animal performance.

2.6   ANIMAL PARASITES IN COLD CLIMATES: Internal Parasites and Cold Stress

Alicata, J.E. 1935. Early developmental stages of nematodes occurring in swine. U.S.D.A. Tech. Bull. 489.

Ames, E.R. 1976. Strategies for the control of swine parasites. Internat. Pig Vet. Soc. Cong. Proc. 1976:0.4.

Avery, J.L. 1942. Survival on soil of oocysts of two species of swine coccidia of <u>Eimeria debliecki</u> and <u>E. scabra</u>. Parasit. 28:Suppl. 28.

Beer, R.J. and J.M. Rutter. 1972. Spirochaetal invasion of the colonic mucosa in a syndrome resembling swine dysentery following experimental <u>Trichuris suis</u> infections in weaned pigs. Res. Vet. Sci. 13:593-595.

Blecha, F. and K.W. Kelley. 1981a. Cold stress reduces the acquisition of colotral immunoglobulins in piglets. J. Anim. Sci. 52:594-600.

Blecha, F. and K.W. Kelley. 1981b. Effects of cold and weaning stressors on the antibody-mediated immune response of pigs. J. Anim. Sci. 53:439-447.

Burden, D.J. and N.C. Hammet. 1979. Development and survival of <u>Trichuris suis</u> ova on pasture plots in the south of England. Res. Vet. Sci. 26:66-70.

Connan, R.M. 1971. <u>Hyostrongulus rubidus</u>: the size and structure of worm populations in adult pigs. Vet. Rec. 89:186-191.

Fayer, R. 1980. Epidemiology of protozoan infections: The Coccidia. Vet. Parasit. 6:75-103.

Frankel, J.K., A. Ruiz, and M. Chinchilla. 1975. Soil survival of <u>Toxoplasma</u> oocysts in Kansas and Costa Rica. Am. J. Trop. Med. & Hyg. 24:439-443.

Gaafar, S.M. and H.W. Jones. 1965. Effect of housing on ascariasis in growing-finishing swine. J.A.V.M.A. 146:358-360.

Hill, C.H. 1957. The survival of swine whipworm eggs in hoglots. J. Parasit. 43:104.

Hoefling, D. and K.S. Todd. 1981. Coccidiosis and toxoplasmosis. Pages 590-597 in A.D. Leman, et al., eds. Diseases of swine. Iowa State University, Ames, Iowa.

Morriss, C.C. 1979. <u>Hyostrongylus rubidus</u> in the domestic pig. N.Z. J. Zool. 6:651.

Potskhveriya, SH. O. 1979. The seasonal dynamics in the prevalence of <u>A. suum</u> larvae in earthworms in the Georgias SSR. Byulleten' Vsesoyuznago Instituta Gel ' mintologii Im. K.I. Skryabina. 24:40-42.

Rose, J.H. 1959. <u>Metastrongylus apri</u> the pig lungworm. Observations on the free-living embryonated eggs and the larvae in the intermediate host. Parasit. 49:439-447.

Rose, J.H. and A.J. Small. 1980a. Transmission of <u>Oesophagostomum</u> spp. among sows at pasture. Vet. Rec. 107:223-225.

Rose, J.H. and A.J. Small. 1980b. Observations on the development and survival of the free-living stages of Oesophagostomum dentatum both in their natural environments out-of-door and under controlled conditions in the laboratory. Parasit. 81:507-517.

Smith, H.J. 1979. Transmission of Oesophagostomum spp. on pasture in the maritime provinces of Canada. Can. Vet. J. 20:184-185.

Smith, H.J. 1981. Resistance of Trichinella nativa to low temperatures. A.A.V.P. Proc. 26th Annual Mtg. p.33.

Soulsby, E.J.L. 1965. Textbook of Veterinary Clinical Parasitology. Vol. I - Helminths. Blackwell Scientific Publications, Oxford, England. p.1120.

Stevenson, P. 1979. The influence of environmental temperature on the rate of development of A. suum in Great Britain. Res. Vet. Sci. 27:193-196.

Stewart, T.B. and O.M. Hale. 1975. Swine parasite transmission in relation to housing. J. Anim. Sci. 40:192-193.

Svenson, R. 1955. On the resistance of heating and cooling of Balantidium coli in culture and some observations regarding conjugation. Expt. Parasit. 4:502-525.

Sweatman, G.K. and R.J. Williams. 1963. Survival of Echinococcus granulosus and T. hydatigena eggs in two extreme climatic regions of New Zealand. Res. Vet. Sci. 4:199-216.

Wade, W.F. and S.M. Gaafar. 1982. Personal communication.

2.62 ANIMAL PARASITES IN COLD CLIMATES: Ectoparasites of Livestock in Cold Climates

Gibson, T.E. (Editor). 1978. Weather and parasitic animal diseases. Technical Note 159. World Meteorological Organization. Geneva, Switzerland.

Gordon, D.V., W.O. Haufe and K.K. Klein. 1984. Determination of economic thresholds for horn fly control in Western Canada: A farm level simulation approach. Cdn. J. of Agric. Economics 32:399-421.

Haufe, W.O. 1965. Synoptic correlation of weather with mosquito activity. Proc. 3rd Int. Congr. Biomet. (1963). Biometeorology 2(II):532-40.

Haufe, W.O. 1976. Systems analysis in biometeorology and its relevance to the biological sciences. Proc. 7th Int. Congr. Biomet. (1975). Biometeorology 6(II):82-92.

Haufe, W.O. 1978. Reduced productivity of beef cattle infested with horn flies. Pp. 61-63 in Research Highlights - 1978. Agriculture Canada Research Station, Lethbridge, Alberta, Canada.

Shifrine, M., N. Taylor, L.S. Rosenblatt and F.D. Wilson. 1980. Seasonal variation in cell-mediated immunity of clinically normal dogs. Exp. Haematol. 8:318-26.

3.1.1 CATTLE - HEAT STRESS: General Observations

Finch, V.A. 1976. An assessment of the energy budget of Boran cattle. J. Thermal Biol. 1:143-148.

Frisch, J.E. 1981. Changes occurring in cattle as a consequence of selection for growth rate in a stressful environment. J. Agric. Sci., Camb. 96:23-38.

Frisch, J.E. and J.E. Vercoe. 1976. Maintenance requirement, fasting metabolism and body composition in different cattle breeds. In Energy Metabolism of Farm Animals, Proc. of the 7th Symposium. (Ed. M. Vermorel). Clermont-Ferrand: de Bussac (Eur. Ass. Anim. Prod., Publ. No. 19).

Gibson, E.A., B.J. Shreeves, E.D. Borland, F.G. Clegg and D.F. Collings. 1982. The effect of the drought of 1976 on the health of farm livestock in England and Wales. Vet. Rec. 111:407-411.

Hahn, G.L., A. Nygaard and E. simensen. Towards establishing national criteria for selection and design of livestock environments. ASAE Paper 83-4517. 26 pp. Am. Soc. Agric. Engr, St. Joseph, MI, USA.

Hahn, G.L. 1985. Management and housing of farm animals in hot environments. Chapter 11 in Stress Physiology in Livestock, Vol. 11, (ed M.K. Yousef). CRC Press, Boca Raton, FL., USA.

Johnson, H.D. 1965. Responses of animals to heat. Meteorological Monograph, Vol. 6, No. 28:109-122. Amer. Meteorological Soc., Boston, MA, USA.

Jones, T.O. and D.R. Jones. 1977. Nitrate/nitrite poisoning of cattle from forage crops. Vet. Rec. 101:266-67.

da Silva, R.G. 1973. Improving tropical beef cattle by simultaneous selection for weight and heat tolerance. Heritabilities and correlations of the traits. J. Anim. Sci. 37:637-42.

Thom, E.C. 1959. The Discomfort Index. Weatherwise. 12:57.

Turner, H.G. 1982. Genetic variation of rectal temperature of cows and its relationship to fertility. Anim. Prod. 35:401-412.

Vercoe, J.E. and J.E. Frisch. 1970. The effect of increased rectal temperature on nitrogen metabolism in Brahman cross and Shorthorn x Hereford steers fed on a low nitrogen roughage. Aust. J. Agric. Res. 21:857-63.

3.12 CATTLE - HEAT STRESS: Reproduction and lactation

Ansell, R.H. 1981. Extreme heat stress in dairy cattle and its alleviation: A case report. pp. 285-306 in J.A. Clark (Ed.), Environmental Aspects of Housing for Animal Production. Butterworths: London.

Casady, R.B., R.M. Myers, and J.E. Legates. 1953. The effect of exposure to high ambient temperature on spermatogenesis in the dairy bull. J. Dairy Sci. 36:14-23.

Gwazdaukas, F.C., W.W. Thatcher, and C.J. Wilcox. 1973. Physiological, environmental, and hormonal factors at insemination which may affect conception. J. Dairy Sci. 56:873-877.

Gibson, E.A., C.B. Ollerenshaw, B.J. Shreeve, E.D. Borland, F.G. Clegg, and D.F. Collings. 1982. Effect of the drought of 1976 on the health of cattle, sheep and other farm livestock in England and Wales. Vet. Rec. 111:407-411.

Hahn, G.L. 1981. Housing and management to reduce climatic impacts on livestock. J. Anim. Sci. 52:176-186.

Ingraham, R.H. 1973. Conception rate, milk production, and endocrine responses of lactating Holstein cows to subtropical climates in Hawaii and Mexico. Unpublished Ph.D. Thesis, Iowa State University, Ames, Iowa.

Ingraham, R.H. 1974. Discussion on the influence of environmental factors on reproduction of livestock. Livestock Environment: Proc. of Int'l Lvstk Envir. Symp., pp. 55-61. SP-01-74, Amer. Soc. Agr. Engr., St. Joseph, Michigan.

Ingraham, R.H., R.W. Stanley, and W.C. Wagner. 1976. Relationship of temperature and humidity to conception rate of Holstein cows in Hawaii. J. Dairy Sci. 59:2086-2090.

Kelly, M.S. and V. Hurst. 1963. The effect of season on fertility of the dairy bull and dairy cow. J. Am. Vet. Med. Assoc. 143:40-43.

REFERENCES

Long, C.R., W.A. Nipper, and C.K. Vincent. 1969. Body temperature and estrous control of beef cattle. J. Anim. Sci. 28:146. (Abstract)

McDowell, R.E., E.E. Moody, P.J. Van Soest, R.P. Lehmann, and G.L. Ford. 1969. Effect of heat stress on energy and water utilization of lactating cows. J. Dairy Sci. 52:188-194.

Rhynes, W.E. and L.L. Ewing. 1973. Testicular endocrine function in Hereford bulls exposed to high ambient temperature. Endocrinology 92:509-515.

Skinner, J.D. and G.N. Louw. 1966. Heat stress and spermatogenesis in Bos indicus and Bos taurus cattle. J. Appl. Physio. 21:1784-1790.

Stott, G.H. and R.J. Williams. 1961. Female and breed associated with seasonal fertility variation in dairy cattle. J. Dairy Sci. 4:1-7.

Stott, G.H. and R.J. Williams. 1962. Causes of low breeding efficiency in dairy cattle associated with seasonal high temperatures. J. Dairy Sci. 45:1369.

Zakari, A.Y., E.C.I. Molokwu, and D.I.K. Osori. 1981. Effects of rectal and ambient temperatures and humidities on conception rates. Theriogeneology. 16:331-336.

3.13 CATTLE - HEAT STRESS: Neonate Survival and Growth

Baccari, F.H., D. Johnson and G.L. Hahn. 1980. Compensatory growth of young dairy heifers subjected to heat stress. J. Anim. Sci. 51:4 (Abstr.).

Balling, R.C. 1982. Weight gain and mortality in feedlot cattle as influenced by weather conditions: refinement and verification of statistical models. CAMaC Prog. Rept. 82-1. Center for Agric. Meteorology and Climatology, Univ. of Nebraska-Lincoln, NE.

Hahn, G.L. 1982. Compensatory performance in livestock: influences on environmental criteria. Proc. 2nd Int'l. Livestock Environment Symposium: 285-294. SP0382. Amer. Soc. Agric. Engr., St. Joseph, MI, USA.

Hahn, L., N. Meador, G.B. Thompson and M.D. Shanklin. 1974. Compensatory growth of beef cattle in hot weather and its role in management decisions. Proceedings of the Int'l. Livestock Environment Symposium: 228-295. SP0174. Amer. Soc. Agric. Engr., St. Joseph, MI, USA.

McDowell, R.E. 1977. Performance of lactating cows and heifers on tropical grass pastures with and without supplement. Proc. 11th Conf. on Livestock and Poultry in Latin America. Univ. of Fla., Gainesville. p. C4.

NRC. 1981. Effect of environment on nutrient requirements of domestic animals. Report of the Sub-Comm. on Environ. Stress, Comm. on Animal Nutrition. Nat'l. Academy Press, Washington, D.C.

Ragsdale, A.C., Chu Shan Cheng and H.D. Johnson. 1957. Effects of constant environmental temperature of 50° and 80°F on the growth responses of Brahma, Santa Gertrudis and Shorthorn calves. Mo. Agr. Exp. Sta. Res. Bul. 642.

Strachan, R.T. and W.F. Marnson. 1963. Growth of Jersey calves in the tropics. Anim. Br. Abstr. 31:186. Art. 1035.

Turner, H.G. 1972. Selection of beef cattle for tropical Australia. Austr. Vet. J. 48(4):162-166.

Yazman, J.A. 1980. Feeding systems for lactating cows and growing heifers in a tropical environment. Unpublished PhD Thesis. Cornell University, Ithaca, NY.

3.14 CATTLE - HEAT STRESS: Acclimatization, chronic and acute exposures and reactions

Colditz, P.J. and R.C. Kellaway. 1972. The effect of diet and heat stress on feed intake, growth and nitrogen metabolism in Friesian, F1 Brahman x Friesian, and Brahman heifers. Aust. J. Agric. Res. 23:717-725.

Curtain, C.C. 1971. The influence of environment upon serum immunoglobulin levels in cattle. Brit. Vet. J. 127:442-450.

Hahn, G.L., W.D. Clark, D.G. Stevens and M.D. Shanklin. 1978. Interaction of temperature and relative humidity on shrinkage of fasting sheep, swine, and beef cattle. Paper No. 78-6010. Amer. Soc. Agric. Engr., St. Joseph, MI.

Harston, C.R. 1959. Cattle shrinkage depends on how you market. Montana AES Circular 221.

Ingraham, R.H. 1968. Milk production and rectal temperature responses of Holstein cattle to cooled drinking water in a subtropical climate. Unpublished Master's thesis. Iowa State University, Ames, Iowa.

Ingraham, R.H. 1974. Discussion on the influence of environmental factors on reproduction of livestock. Livestock Environment: Proc. of Int'l. Lvstk Environ. Sympos., pp. 55-61. SP-01-74, American Society of Agricultural Engineers, St. Joseph, MI.

Ingraham, R.H. 1979. Seasonal effects of tropical climate on shaded and non-shaded cows as measured by rectal temperatures adrenal cortex hormones, thyroid hormones, and milk production. Am. J. Vet. Res. 40:1792-1797.

Kelley, K.W., R.E. Greenfield, J.F. Evermann, S.M. Parish and L.E. Perryman. 1982a. Delayed-type hypersensitivity, contact sensitivity and PHA skin test responses of heat- and cold-stressed calves. Am. J. Vet. Res. (In Press).

Kelley, K.W., C.A. Osborne, J.F. Evermann, S.M. Parish and C.T. Gaskins. 1982b. Effects of chronic heat and cold stressors on plasma immunoglobulin and mitogen-induced blastogenesis in calves. J. Dairy Sci. 65(8):1514-1528.

Kibler, H.H. 1964. Environmental physiology and shelter engineering with special reference to domestic animals: LXVII. Thermal effects of various temperature-humidity combinations on Holstein cattle or measured by eight physiological responses. MO AES Res. Bul. 862.

LCI. 1970. Patterns of transit losses. Information release by Livestock Conversation, Inc., Omaha, NE.

NRC. 1981. Effect of environment on nutrient requirements of domestic animals. Dept. of the Sub-Committee on Environmental Stress, Comm. on Animal Nutrition. National Academy Press, Washington, DC.

Oliver, J.C., H.M. Hellman, S.E. Bishop, C.L. Pellisier and L.F. Bennett. 1979. Heat stress survey. Calif. Agr. 33(3):6-8.

Soper, F.F., C.C. Muscoplat and D.W. Johnson. 1978. In vitro stimulation of bovine peripheral blood lymphocytes: Analysis of variation of lymphocyte blastogenic response in normal dairy cattle. Am. J. Vet. Res. 39:1039-1042.

Stott, G.H., F. Wiersman, B.E. Menefee and F.R. Radwanski. 1976. Influence of environment on passive immunity in calves. J. Dairy Sci. 59:1306-1311.

Warren, W.P., F.A. Martz, K.H. Asay, E.S. Hilderbrand, C.G. Payne and J.R. Vogt. 1974. Digestibility and rate of passage by steers fed tall fescue, alfalfa and orchardgrass hay at 18 and 32°C ambient temperatures. J. Anim. Sci. 39:93-96.

3.2.1 SHEEP AND GOATS - HEAT STRESS: General Observations

Jindal, S.K. 1980. Effect of climate on goats. A Review. Ind. J. Dairy. Sci. 33:285-293.

Jindal, S.K. 1984. Goat Production. Cosmo Publications, New Delhi. 334 pp.

Johnson, K.G. 1976. Evaporative temperature regulation in sheep. Progress in Biometeorology Vol. 1:140-147.

Kamal, T.H. 1976. Indices for heat adaptability of domestic animals. Progress in Biometeorology Vol. 1:470-475.

Macfarlane, W.V. 1964. Terrestrial animals in dry heat: Ungulates. Chapter 33 in Handbook of Physiology, Sec. 4, Adaptation to Environment, Dill, D.E., E.F. Adolph, and C.H. Wilbur (editors). Amer. Physiol. Soc., Washington, DC.

Macfarlane, W.V. 1976a. Water metabolism in sheep in relation to heat and cold. Progress in Biometeorology Vol. 1:49-57.

Macfarlane, W.V. 1976b. Adaptation to climatic zones and the ecophysiology of ruminants. Progress in Biometeorology Vol. 1:425-433.

Macfarlane, W.V. 1982. Concepts in Animal Adaptation. Proc. Third Int. Conf. on Goat Production and Disease. Dairy Goat Publ. Co., Scottsdale, AR, pp 375-385.

Singh, M. and A.K. Rai. 1981. Adaptation of sheep to hot environment. 1st Proc. Soc. and Nat'l. Seminar for Sheep and Goat Production and Utilization. Compiled by A.K. Bosuthakur and R.M. Acharya, Jaipur, India.

Terrill, C.E. 1968. Adaptation of sheep and goats. In Adaptation of Domestic Animals, E.S.E. Hafez (Ed.). Lea and Febiger, Philadelphia. pp. 246-263.

3.22 SHEEP AND GOATS - HEAT STRESS: Heat Tolerance

Appleman, R.D. and J.C. Delouche. 1958. Behavioral, physiological, and biochemical responses of goats to temperature, 0° to 40°C. J. Anim. Sci. 17:326-335.

Bianca, Von W. and P. Kunz. 1978. Das Verhalten von Ziegen in Kalte, Hitze and Hohe. Z. Zierzuchtg. Zuchtgsciol. 94:276-290.

Bligh, J. 1961. The synchronous discharge of apocrine sweat glands of the Welsh mountain sheep. Nature (Lond.). 189:582-583.

Bligh, J. 1963. The receptors concerned in the respiratory response to humidity in sheep at high ambient temperature. J. Physiol. 168:747-763.

Dmi'el, R., A. Prevulotzky and A. Shkolnik. 1980. Is a black coat in the desert a means of saving metabolic energy? Nature (Lond.). 283:761-762.

Finch, V.A., R. Dmi'el, R. Boxman, A. Shkolnik and C.R. Taylor. 1980. Why black goats in hot deserts? Effects of coat color on heat exchanges of wild and domestic goats. Physiol. Zool. 53:19-25.

Hutchinson, J.C.D. and G.B. Brown. 1969. Penetrance of cattle coats by radiation. J. of Appl. Physiol. 36:454-464.

Lee, D.H.K. 1950. Studies of heat regulation in the sheep with special reference to the Merino. Austr. J. Agric. Res. 1:200-216.

Maskrey, M., J.R.S. Hales and A.A. Fawcett. 1981. Effect of constant arterial CO_2 tension on respiratory pattern in heat stressed sheep. J. Appl. Physiol. 50:315-319.

Parer, J.T. 1963. Wool length and radiant heating effects in sheep. J. Agr. Sci. 60:141-144.

Robertshaw, D. 1982. Concepts in animal adaptation - thermoregulation of the goat. Proc. Third. Int. Conf. on Goat Production and Diseases. Dairy Goat Pub. Co., Scottsdale, AR. pp 395-397.

Robinson, D.W. 1969. Preliminary observations on the heat tolerance of shorn and nutritionally depleted sheep in a tropical environment. Brit. Vet. J. 125:112-120.

Shkolnik, A., A. Borut and J. Choshniak. 1972. Water economy of the Bedouin goat. Symp. of Zool. Soc. of London. 31:229-242.

Singh, M. and A.K. Rai. 1981. Adaptation of sheep to hot environment. 1st Proc. Soc. and Nat'l. Seminar for Sheep and Goat Production and Utilization. Compiled by A.K. Bosuthakur and R.M. Acharya, Jaipur, India.

Singh, M., A.K. Rai and S.A. Karim. 1983. Relative heat tolerance of Indian Nali and Soviet Merino X Nali halfbred sheep to continuous S3S and 40 celsius temperature conditions. 2. Tierphysiol. Tierernahrg. U. Futtermittelkde. 49:18-23.

Tsuda, T., S. Yamamoto, Y. Sasaki, Y. Shoji, N. Yamagishi and Y. Ootomo. 1973. Effects of environmental temperatures and carbon dioxide levels on physiological responses of sheep. Tohoku J. of Agr. Res. 23:196-206.

Waites, G.M.H. 1962. The effect of heating on the scrotum of the ram on respiration and body temperature. Quar. J. Exper. Physiol. 47:314-323.

Walsberg, G.E., G.S. Campbell and J.R. King. 1978. Animal coat color and radiative heat gain; a re-evaluation. J. Comp. Physiol. 126:211-222.

3.23 SHEEP AND GOATS - HEAT STRESS: Temperature Regulation

Baker, M.A. and J.N. Hayward. 1968a. Intracranial heat exchange and regulation of brain temperature in sheep. Life Sci. 7:349-354.

Baker, M.A. and J.N. Hayward. 1968b. The influence of the nasal mucosa and the carotid rete upon hypothalmic temperature in sheep. J. Physiol. (Lond.). 198:561-579.

Daniel, P.M., J.K.D. Dawes and M.L. Pritchard. 1953. Studies of the carotid rete and its associated arteries. Phil. Trans. Roy. Soc. B. 237:173-208.

Degen, A.A. and B.A. Young. 1981. Effect of air temperature and feed intake on live weight and water balance in sheep. J. Agric. Sci. (Camb.). 96:493-496.

Hales, J.R.S. 1973. Effects of heat stress on blood flow in respiratory and nonrespiratory muscles in the sheep. Pflugers Arch. 345:123-130.

Johnson, K.G. 1976. Evaporative temperature regulation in sheep. Progress in Biometeorology Vol. 1:140-147.

Macfarlane, W.V. 1976a. Water metabolism in sheep in relation to heat and cold. Progress in Biometeorology Vol. 1:49-57.

Rawson, R.W. 1976. Temperature regulation in sheep. Progress in Meteorology Vol. 1:81-87.

3.24 SHEEP AND GOATS - HEAT STRESS: Water Metabolism

Guerrini, V.H., N. Koster and H. Bertchinger. 1980. Effect of ambient temperature and humidity on urine output in sheep. Amer. J. Vet. Res. 41:1851-1853.

Macfarlane, W.V. 1964. Terrestrial animals in dry heat: Ungulates. Chapter 33 in Handbook of Physiology, Sec. 4, Adaptation to Environment, Dill, D.E., E.F. Adolph, and C.H. Wilbur (editors). Amer. Physiol. Soc., Washington, DC.

Macfarlane, W.V. 1976b. Adaptation to climatic zones and the ecophysiology of ruminants. Progress in Biometeorology Vol. 1:425-433.
Macfarlane, W.V. 1982. Concepts in Animal Adaptation. Proc. Third Int. Conf. on Goat Production and Disease. Dairy Goat Publ. Co., Scottsdale, AR, pp 375-385.

Macfarlane, W.J., R.J.H. Morris and B. Howard. 1958. Heat and water in tropical Merino sheep. Aust. J. Agr. Res. 9:217-228.

More, T. and K.L. Sahni. 1980. Effect of intermittent watering on milk production and lamb growth in Chokla ewes under semi arid conditions. Indian Vet. J. 57:562-566.

Robertshaw, D. 1982. Concepts in animal adaptation - thermoregulation of the goat. Proc. Third. Int. Conf. on Goat Production and Diseases. Dairy Goat Pub. Co., Scottsdale, AR. pp 395-397.

Taneja, G.C. 1965. Effect of varying the frequency of watering during summer on the temperature, respiration, body weight, and packed cell volume of sheep. Indian J. Expt. Biol. 3:259-262.

3.25 SHEEP AND GOATS - HEAT STRESS: Energy Metabolism

Ames, D.R. and D.R. Brink. 1977. Effect of temperature on lamb performance and protein efficiency ratio. J. Anim. Sci. 44:136-140.

Bhattacharya, A.N. and F. Hussain. 1974. Intake and utilization of nutrients in sheep fed different levels of roughage under heat stress. J. Anim. Sci. 38:877-886.

Knox, K.L. 1976. Effects of heat on energy metabolism of sheep. Progress in Biometeorology Vol. 1:189-194.

Valtorta, S., L. Hahn and H.D. Johnson. 1982. Effect of high ambient temperature and feed intake on phasma T_4 levels in sheep. Proc. Soc. Exp. Biol. Med. 169:260-265.

3.26 SHEEP AND GOATS - HEAT STRESS: Reproduction

Alexander, G. and A. Williams. 1966. Heat stress and growth of the conceptus in sheep. Proc. Aust. Soc. Anim. Prod. 6:102-105.

Alexander, G. and D. Williams. 1971. Heat stress and development of the conceptus in domestic sheep. J. Agric. Sci., Camb. 76:53-72.

Alliston, C.W. 1976. Effects of heat on reproduction of sheep. Progress in Biometeorology Vol. 1:381-386, 574-575.

Brown, D.E., P.C. Harrison, F.C. Hinds, J.A. Lewis and M.H. Wallace. 1977. Heat stress effects on fetal development during late gestation in the ewe. J. Anim. Sci. 44:442-446.

Brown, D.E. and P.C. Harrison. 1981. Central sympathetic control of uterine blood flow during acute heat stress. J. Anim. Sci. 52:1114-1121.

Cartwright, G.A. and C.J. Thwaites. 1976a. Foetal stunting in sheep. 1. The influence of maternal nutrition and high ambient temperature on the growth and proportions of Merino foetuses. J. Agric. Sci. (Camb.). 86:573-580.

REFERENCES

Cartwright, G.A. and C.J. Thwaites. 1976b. Foetal stunting in sheep. 2. The effects of high ambient temperature during gestation on wool follicle development in the foetal lamb. J. Agric. Sco. (Camb.). 86:581-585.

Dutt, R.H. 1954. Fertility and embryonic death loss in ewes early in the breeding season. J. Anim. Sci. 13:464-473.

Dutt, R.H. 1960. Temperature and light as factors in reproduction among farm animals. J. Dairy Sci. 43:123-144.

Dutt, R.H. and P.T. Hamm. 1957. Effect of exposure to high environmental temperatures and shearing on semen production of rams in winter. J. Anim. Sci. 16:328-334.

Goode, L. 1964. Effects of summer confinement upon ewe productivity. J. Anim. Sci. 23:906, Abstr.

Hopkins, P.S., C.J. Nolan and P.M. Pepper. 1980. The effect of heat stress on the development of the foetal lamb. Aust. J. Agric. Res. 31:763-771.

McKenzie, F.F. and V. Berliner. 1937. The reproductive capacity of rams. Mo. Agr. Exp. Res. Bul. 265.

Mishra, M.S. and B.P. Sengupta. 1965. Semen quality of ram and goat in autumn. Indian Vet. J. 42:742-744.

Moule, G.R. and G.M.H. Waites. 1963. Seminal degeneration in the ram and its relation to the temperature of the scrotum. J. Reprod. Fert. 5:433-446.

Ramamurty, A. 1963. Breeding performance of Nilgiri, Romney Marsh, and Kibishow rams. Indian Vet. J. 40:621-623.

Roman-Ponce, H., W.W. Thatcher, D. Caton, D.H. Barron and C.J. Wilcox. 1978. Effects of thermal stress and epinephrine on uterine blood flow in ewes. J. Anim. Sci. 46:167-174.

Sahni, K.L. and S.B. Tiwari. 1981. Reproductive performance of buck and ram under hot arid and hot humid conditions. 1st Proc. Soc. and Nat'l Seminar on Sheep and Goat Production and Utilization. Compiled by A.K. Basuthakur and R.M. Acharya, Jaipur, India.

Sawyer, G.J. 1979. The influence of radiant heat load on reproduction in the Merino ewe. II The relative effects of heating before and after insemination. Aust. J. Agr. Res. 30:1143-1149.

Shelton, M. 1964. Relation of environmental temperature during gestation to birth weight and mortality in lambs. J. Anim. Sci. 23:360-364.

Shelton, M. and J.E. Huston, 1968. Effects of high temperature stress during gestation on certain aspects of reproduction in the ewe. J. Anim. Sci. 27:153-158.

Shukla, D.D. and P. Bhattacharya. 1952. Seasonal variation in reaction time and semen quality of goat. Indian J. Vet. Sci. 22:179-190.

Singh, B. and A. Roy. 1963. Studies on certain aspects of sheep and goat husbandry. IV Seasonal variation in semen quality of Corriedale and Bikaneri rams. Indian J. Vet. Sci. 33:211-214.

Sinha, N.K., G.M. Wani and K.L. Sahni. 1979. Observations on reproductive behavior and semen quality of rams reared under tropical conditions. Indian J. Anim. Sci. 49:916-920.

Yeates, N.T.M. 1956. The effect of high air temperature on pregnancy and birth weight in Merino sheep. Aust. J. Agr. Res. 7:435-439.

Yeates, N.T.M. 1958. Foetal dwarfism in sheep - an effect of high atmospheric temperature during gestation. J. Agr. Sci. (Camb.). 51:84-90.

3.27 SHEEP AND GOATS – HEAT STRESS: Breed Adaptation and Disease

Acharya, R.M. 1982. Sheep and goat breeds of India. F.A.O. Animal Production and Health Paper 30. Rome.
    190 pp.

Devendra, C. and M. Burns. 1970. Goat production in the tropics. Tech. Commun. No. 19 of Commonwealth
    Bureau of Animal Breeding and Genetics. Edinburgh. 184 pp.

Hugh-Jones, M.E. 1976. Effects of heat and cold on diseases in sheep. Progress in Biometeorology. Vol.
    1:480-486.

Shkolnik, A., E. Maltz and S. Gordin. 1980. Desert conditions and goat milk production. J. Dairy Sci.
    63:1749-1754.

Singh, M. and A.K. Rai. 1981. Adaptation of sheep to hot environment. 1st Proc. Soc. and Nat'l. Seminar
    for Sheep and Goat Production and Utilization. Compiled by A.K. Bosuthakur and R.M. Acharya, Jaipur,
    India.

Singh, J. and R.M. Acharya. 1977. A note on the mode of heat dissipation in different types of sheep.
    Indian J. Anim. Sci. 47:367-368.

Singh, R. and A. Roy. 1963. Studies on certain aspects of sheep and goat husbandry. V. Effect of season
    on respiration rate of Corriedale and Bikaneri sheep and Jamurapari goat. Indian J. Vet. Sci.
    33:128-141.

Taneja, G.C. 1966. Physiological responses of sheep to an arid environment. I Comparative responses of
    indigenous and exotic breeds. Indian J. Physiol. & Allied Sci. 20:20-49.

Terrill, C.E. 1982. Applications of experimental results of crossbreeding of sheep in the United States
    of America. Proc. of World Congress on Sheep and Beef Cattle Breeding (1980). Dunmore Press Ltd.,
    Palmerston North, New Zealand.

Thompson, G.E., P.E. Hartmann, J.A. Goode and K.S. Lindsay. 1981. Some effects of fasting and climatic
    stresses upon milk secretion in Friesland sheep. Comparative Biochemistry and Physiology. A.70:13-16.

3.28 SHEEP AND GOATS – HEAT STRESS: Management

Morgan, P.D., G.W. Arnold and C.A.P. Boundy. 1972. The behavior of ewes and their newborn lambs in a hot
    climate. Proc. Aust. Soc. Anim. Prod. 9:371-375.

Smith, I.E. 1961. Thermoregulation in the newborn lamb. Aust. Vet. J. 37:205-210.

Stephenson, R.G.A., R.D. Hooley, J.K. Findlay and P.S. Hopkins. 1980. Effects of heat stress on the
    lactation performance of ewes accustomed to tropical conditions and the total fluid intake of their
    lambs. Aust. J. Biol. Sci. 33:449-456.

Yeates, N.T.M. 1976. Breeding management of sheep. Progress in Biometeorology Vol. 1:387-392.

3.29 SHEEP AND GOATS – HEAT STRESS: Sheep and Goat Comparisons

McDowell, R.E. and A. Woodward. 1982. Proc. Third Int. Conf. on Goat Production and Disease. Dairy Goat
    Publishing Co., Scottsdale, AR. pp 387-393.

3.31 PIGS – HEAT STRESS: General Observations

Beckett, R.E. 1965. Effective temperature as a means of evaluating or designing hog environments. Trans.
    Am. Soc. Agr. Eng. 8:163-166.

REFERENCES

Bond, T.E., C.F. Kelly and H. Heitman. 1967. Physiological response of swine to cycling environmental conditions. Anim. Prod. 9:453-462.

Close, W.H. and L.E. Mount. 1978. The effects of plane of nutrition and environmental temperatures on the energy metabolism of the growing pig. 1. Heat loss and critical temperature. Br. J. Nutr. 40:413-421.

Ingram, D.L. 1965. The effect of humidity on temperature regulation and cutaneous water loss in the young pig. Res. Vet. Sci. 6:9-17.

Morrison, S.R., T.E. Bond and H. Heitman, Jr. 1968. Effect of humidity on swine at high temperature. Trans. Am. Soc. Agr. Eng. 11:526-528.

Morrison, S.R., H. Heitman, Jr. and T.E. bond. 1969. Effect of humidity on swine at temperatures above optimum. Int. J. Biometeor. 13:135-139.

Roller, W.L. and R.F. Goldman. 1969. Responses of domestic swine to acute heat exposure. Trans. Am. Soc. Agr. Eng. 12:164-169 and 174.

3.32 PIGS - HEAT STRESS: Reproduction and Lactation

Alvarez, M.B. and H.D. Johnson. 1973. Environmental heat exposure on cattle plasma catecholamine and glucocorticoids. J. Dairy Sci. 56:189-194.

Bazer, F.W. 1975. Uterine protein secretion: Relationship to development of the conceptus. J. Anim. Sci. 41:1376-1382.

Beier, H.M., W. Kuhnel and G. Petry. 1971. Uterine secretion proteins as extrinsic factors in preimplantation development. Adv. Biosci. 6:165-189.

Chang, M. C. 1966. Effects of oral administration of medroxyprogesterone acetate and ethinyl estradiol on transportation and development of rabbit. Endocrinology. 79:939-948.

Christenson, R.K. 1980. Environmental influences on the postpartum animal. J. Anim. Sci. 51 (Suppl. II):53-67.

Christenson, R.K., H.S. Teague, A.P. Grifo, Jr. and W.L. Roller. 1972. The effect of high environmental temperature on the boar. Res. Summ 61, Ohio Agr. Res. and Develop. Center, Wooster, p. 19.

Christenson, R.K., R.R. Maurer, G.L. Hahn and J.A. Nienaber. 1984. Unpublished data.

Corteel, J.M., J.P. Signoret and F. du Mesnil du Buisson. 1964. Variations saisonnieres de la reproduction de la Truie et facteurs favorisant l'anoestrus temporale. V Internat'l. Cong. Anim. Reprod. Artif. Insem., Trento. 3:536.

d'Arce, R.D., H.S. Teague, W.L. Roller, A.P. Grifo, Jr. and W.M. Palmer. 1970. Effect of short-term elevated dry-bulb and dew-point temperature on the cycling gilt. J. Anim. Sci. 30:374-377.

Dutt, R.H., E.F. Ellington and W.W. Carlton. 1959. Fertilization rate and early embryo survival in sheared and unsheared ewes following exposure to elevated air temperature. J. Anim. Sci. 18:1308-1318.

Edwards, R.L., I.T. Omtvedt, E.J. Turman, D.F. Stephans and G.W.A. Mahoney. 1968. Reproductive performance of gilts following heat stress prior to breeding and in early gestation. J. Anim. Sci. 27:1634-1637.

Ford, S.P. and R.K. Christenson. 1979. Blood Flow to uteri of sows during the estrous cycle and early pregnancy: local effect of the conceptus on the uterine blood supply. Biol. Reprod. 21:617-624.

Gwazdauskas, F.C., W.W. Thatcher and C.J. Wilcox. 1972. Adrenocorticotropin alteration of bovine peripheral plasma concentrations of cortisol, corticosterone and progesterone. J. Dairy Sci. 55:1165-1169.

Hawk, H.W. and H.H. Conley. 1971. Loss of spermatozoa from the reproductive tract of the ewe and intensification of sperm 'breakage' by progestagen. J. Reprod. Fertil. 27:339-347.

Herak, M. and M. Sviben. 1974. Variation of farrowing rate of sows covered during various months of the years 1965-1968. Proc. 3rd Internat'l. Pig Vet. Soc. Cong., Lyon, France. P. G6.

Herak, M., M. Sviben and I. Vukovic. 1974. Correlation of farrowing rate of sows covered during various months of the economic year with temperature relative atmospheric moisture and pressure of the air. Proc. 3rd Internat'l. Pig Vet. Soc. Cong., Lyon, France, p. G5.

Howarth, B., Jr., C.W. Alliston and L.C. Ulberg. 1965. Importance of uterine environment on rabbit sperm prior to fertilization. J. Anim. Sci. 24:1027-1032.

Hurtgen, J.P. 1976. Seasonal anestrus in Minnesota swine breeding herd. Proc. 4th International Pig Vet. Soc. Cong., Ames, IA. PD22.

Hurtgen, J.P. and A.D. Leman. 1978. Seasonal breeding patterns in parous sows: A slaughter-house survey. Proc. 5th Internat'l. Pig Vet. Soc. Cong., Zabreb, Yugoslavia, P. M50.

Knight, J.W., F.W. Bazer, H.D. Wallace and C.J. Wilcox. 1974. Dose-response relationships between exogenous progesterone and estradiol and porcine uterine protein secretions. J. Anim. Sci. 39:747-751.

Love, R.J. 1978. Definition of a seasonal infertility problem in pigs. Vet. Rec. 103:443-446.

Madan, M.L. and H.D. Johnson. 1972. Environmental heat effects on bovine luteinizing hormone and reproductive cycle. Int. J. Biometeor. 16 (Suppl. 5):75.

Maurer, R.R., R.K. Christensen and J.J. Ford. 1984. Unpublished data.

Mills, A.C., W.W. Thatcher, S.E. Dunlap and C.K. Vincent. 1972. Influence of postbreeding thermal stress on peripheral plasma progestin concentrations in heifers. J. Dairy Sci. 55:400 (Abstr.)

Omtvedt, I.T., R.E. Nelson, R.L. Edwards, D.F. Stephens and E.J. Turman. 1971. Influence of heat stress during early, mid and late pregnancy of gilts. J. Anim. Sci. 32:312-317.

Paterson, A.M., I. Barker and D.R. Lindsay. 1978. Summer infertility in pigs: Its incidence and characteristics in an Australian commercial piggery. Australian J. Exp. Agr. Anim. Husb. 18:698-701.

Reese, D.E., B.D. Moser, E.R. Peo, Jr., A.J. Lewis, Dwane R. Zimmerman, J.E. Kinder and R.K. Johnson. 1980. Influence of dietary energy intake during lactation on the interval to first postweaning estrus in swine. J. Anim. Sci. 51 (Suppl. 1):217 (Abstr.).

Stevenson, J.S., N.M. Cox and J.H. Britt. 1981. Roll of the ovary in controlling luteinizing hormone, follicle stimulating hormone, and prolactin secretion during and after lactation in pigs. Biol. Reprod. 24:341-353.

Stork, M.G. 1979. Seasonal reproductive inefficiency in large pig breeding units in Britain. Vet. Rec. 104:49-52.

Sviben, M., M. Herak and I. Vukovie. 1974. Rate of farrowing variation of sows covered during various months of the year and estimation of the number of coverings required for a constant number of farrowings. Proc. 3rd Internat'l. Pig Vet. Soc. Cong., Lyon, France.

Teague, H.S., W.L. Roller and A.P. Grifo, Jr., 1968. Influence of high temperature and humidity on the reproductive performance of swine. J. Anim. Sci. 27:408-411.

Tompkins, E.C., C.J. Heidenreich and M. Stob. 1967. Effect of post-breeding thermal stress on embryonic mortality in swine. J. Anim. Sci. 26:377-380.

Wettemann, R.P., M.E. Wells, I.T. Omtvedt, C.E. Pope and E.J. Turman. 1976. Influence of elevated ambient temperature on reproductive performance of boars. J. Anim. Sci. 42:664-669.

Wettemann, R.P., M.E. Wells and R.K. Johnson. 1979. Reproductive characteristics of boars during and after exposure to increased ambient temperatures. J. Anim. Sci. 49:1501-1505.

Wagner, W.C., R.E. Strohbehn and P.A. Harris. 1972. ACTH, corticoids and luteal function in heifers. J. Anim. Sci. 35:789-793.

Warnick, A.C., H.D. Wallace, A.Z. Palmer, E. Sosa, D.J. Duerre and V.E. Caldwell. 1965. Effect of temperature on early embryo survival in gilts. J. Anim. Sci. 24:89-92.

Wells, B. 1977. The Pig Farmer. 12(2):73.

3.33 PIGS - HEAT STRESS: Neonatal Survival and Growth

Addis, P.B., H.R. Johnson, C.J. Heidenreich, H.W. Jones and M.D. Judge. 1967. Effects of humidity level in a warm growing environment on porcine carcass composition and quality. J. Anim. Sci. 26:705-707.

Bond, T.E., C.F. Kelly and H. Heitman, Jr. 1952. Heat and moisture loss from swine. Agr. Eng. 33:148-152, 154.

Bond, T.E., H. Heitman, Jr. and C.F. Kelly. 1963. Effect of diurnal temperature on heat loss and well being of swine. Trans. Am. Soc. Agr. Eng. 6:132-135.

Bond, T.E., H. Heitman, Jr., and C.F. Kelly. 1965. Effects of increased air velocities on heat and moisture loss and growth of swine. Trans. Am. Soc. Agr. Eng. 8:167-174.

Close, W.H. 1978. The effects of plane of nutrition and environmental temperature on the energy metabolism of the growing pig. Br. J. Nutr. 40:423-431.

Close, W.H. and L.E. Mount. 1978. The effects of plane of nutrition and environmental temperature on the energy metabolism of the growing pig. 1. Heat loss and critical temperature. Br. J. Nutr. 40:413-421.

Comberg, B., H.F. Wolfermann and H. Runfeldt. 1967. The influence of temperature and relative humidity on weight gains and food conversion of fattening pigs. Z. Tierzuch. Zucht. Biol. 83:240.

Fuller, M.F. 1965. The effect of environmental temperature on the nitrogen metabolism and growth of the young pig. Br. J. Nutr. 19:531-542.

Fuller, M.F., W.R.H. Duncan and A.W. Boyne. 1974. Effect of environmental temperature on the degree of unsaturation of depot fats of pigs given different amounts of food. J. Sci. Fd. Agric. 25:205-210.

Hahn, G.L. 1982. Compensatory Performance in Livestock: In Thesis on Environmental Criteria. pp. 285-294, in Proc. 2nd Int'l Livestock Environ. Symp. SP-03-82. Am. Soc. Agric. Engrs., St. Joseph, MI, USA.

Hale, O.M., R.L. Givens, J.C. Johnson, Jr. and B.L. Southwell. 1966. Effectiveness of movable shades and water sprinklers for growing-finishing swine. J. Anim. Sci. 25:756-759.

Heitman, H., Jr., C.F. Kelly and T.E. Bond. 1958. Ambient air temperature and weight gain in swine. J. Anim. Sci. 17:62-67.

Holme, D.W. and W.E. Coey. 1967. The effects of environmental temperature and method of feeding on the performance and carcass composition of bacon pigs. Anim. Prod. 9:209-218.

Holmes, C.W. 1971. Growth and backfat depth of pigs kept at a high temperature. Anim. Prod. 13:521-527.

Holmes, C.W. 1973. The energy and protein metabolism of pigs growing at a high ambient temperature. Anim. Prod. 16:117-133.

Holmes, C.W. 1974. Further studies on the energy and protein metabolism of pigs growing at a high ambient temperature, including measurements with fasting pigs. Anim. Prod. 19:211-220.

Houghton, T.R., M.H. Butterworth, D. King and B. Goodyear. 1964. J. Agric. Sci. (Bamb). 63:43-51.

Mangold, D.W., T.E. Hazen and V.W. Hays. 1967. Effect of air temperature on performance of growing -finishing swine. Trans. Amer. Soc. Agr. Eng. 10:370.

Morrison, S.R., R.L. Givens and H. Heitman, Jr. 1976. Effects of air movement on swine at high temperature. Int. J. Biometeor. 20:337-343.

Morrison, S.R., L. Hahn and T.E. Bond. 1970. Predicting summer production losses for swine. U.S. Dept. of Agr. Prod. Res. Report No. 118.

Morrison, S.R., H. Heitman, Jr. and T.E. Bond. 1969. Effect of humidity on swine at temperatures above optimum. Int. J. Biometeor. 13:135-139.

Morrison, S.R., H. Heitman, Jr. and R.L. Givens. 1975. Effect of diurnal air temperature cycles on growth and food conversion in pigs. Anim. Prod. 20:287-291.

Mount, L.E. 1959. The metabolic rate of the newborn pig in relation to environmental temperature and age. J. Physiol. 247:333-345.

Mount, L.E. 1963. Environmental temperature preferred by the young pig. Nature. 199:1212-1213.

Nelson, D.P., C.H. Read, G.J. Barfield, J.N. Walker, V. Hayes and G. Cromwell. 1972. The performance of swine under warm environments. Trans. Am. Soc. Agr. Eng. 15:133-136.

Restrepo, G., M.D. Shanklin and L. Hahn. 1977. Heat dissipation from growing pigs as a function of floor and ambient temperature. Trans. Am. Soc. Agr. Eng. 20:145-147.

Smith, W.C. and H.M. Tonks. 1966. The effect of a high-temperature, high-humidity indoor environment on the performance of bacon pigs. 9th International Congress Anim. Prod. 51.

Sorenson, P.H. and J. Moustgaard. 1961. The influence of sty climate on growth rate, food conversion and carcass quality of pigs. 8th International Congress Anim. Prod. 104.

Stahly, T.S. and G.L. Cromwell. 1979. Effect on environmental temperature and dietary fat supplementation on the performance and carcass characteristics of growing and finishing swine. J. Anim. Sci. 49:1478-1488.

Straub, G., J. H. Weniger, E.S. Tawfik and D. Steinhauf. 1976. The effect of high environmental temperatures on fattening performance and growth of boars. Livestock Prod. Sci. 3:65-74.

Sugahara, M., D.H. Baker, B.G. Harmon and A.H. Jensen. 1970. Effect on ambient temperature on performance and carcass development in young swine. J. Anim. Sci. 31:59-62.

Tonks, H.M., W.C. Smith and J.M. Brice. 1972. The influence of high temperatures, high humidity, indoor environment on pig performance under farm conditions. Vet. Rec. 90:531-537.

Vajrabukka, C., C.J. Thwaites and D.J. Farrell. 1981. In "Recent Advances in Animal Nutrition in Australia", pp. 99-114. ed., D.J. Farrell, University of New England Publishing Unit.

Wagenbach, H. 1961. The influence of housing temperature and season on fattening of pigs at three German litter testing stations. Zuchtungskunde. 33:133.

3.34 PIGS - HEAT STRESS: Acclimatization, Chronic/Acute Exposures and Reactions

Blaxter, K.L. 1967. The Energy Metabolism of Ruminants. 2nd Ed. Hutchinson, London.

Bond, T.E., C.F. Kelly and H. Heitman. 1959. Hog house air conditioning and ventilation data. Trans. Am
    Soc. Agric. Engrs. 2:1-4.

Bond, T.E., C.F. Kelly and H. Heitman. 1967. The influence of environmental temperature and plane of
    nutrition on heat losses from goups of growing pigs. Anim. Prod. 9:452-462.

Close, W.H., L.E. Mount and B. Start. 1971. The influence of environmental temperature and plane of
    nutrition on heat losses from growing pigs. Anim. Prod. 13:285-294.

Holmes, C.W. 1971. Growth and backfat depth of pigs kept at high temperature. Anim. Prod. 13:521-527.

Holmes, C.W. and D.W. Close. 1977. In "Nutrition and the Climatic Environment", pp. 51-73. eds. W.
    Haresign, H. Swan, and D. Lewis, Butterworths, London.

Ingram, D.L. 1965. Physiology - Evaporative cooling in the pig. Nature (London). 207:415-416.

Ingram, D.L. 1974. In "Heat Loss from Animals and Man". p. 233-354. eds. J.L. Monteith and L.E. Mount,
    Butterworths, London.

Ingram, D.L. and L.E. Mount. 1965. The metabolic rates of young pigs living at high ambient temperatures.
    Res. Vet. Sci. 6:300-306.

Kelly, C.F., G. Heitman and J.R. Morris. 1948. Effect of environment on heat loss from swine. Agric.
    Eng. 29:525-529.

Morrison, S.R. and L.E. Mount. 1971. Adaptation of growing pigs to changes in environmental temperature.
    Anim. Prod. 13:51-57.

Mount, L.E. 1962. Evaporative heat loss in the newborn pig. J. Physiol. 164:274-281.

Mount, L.E. 1963. Responses to thermal environment in newborn pigs. Fed. Proc. 22:818-823.

Mount, L.E. 1968. "The Climatic Physiology of the Pig". Edward Arnold, London.

Mount, L.E., C.W. Holmes, W.H. Close, S.R. Morrison and I.B. Start. 1971. A note on the consumption of
    water by the growing pig at several environmental temperatures and levels of feeding. Anim. Prod.
    13:561-563.

Robinson, D.W. and D.H.K. Lee. 1947. The effect of the nutritional plane upon the reactions of animals to
    heat. J. Anim. Sci. 6:182-194.

Roller, W.L. and R.F. Goldman. 1969. Response of domestic swine to acute heat exposure. Trans. Am. Soc.
    Agr. Eng. 12:164-170.

Straub, G., J.H. Weniger, E.S. Tawfik and D. Steinhauf. 1976. The effect of high environmental
    temperature on fattening performance and growth of boars. Livestock Prod. Sci. 3:65-74.

Tonks, H.M., W.C. Smith and J.M. Brice. 1972. The influence of high temperatures, high humidity, indoor
    environment on pig performance under farm conditions. Vet. Rec. 90:531-537.

Vajrabukka, C., C.J. Thwaites and D.J. Farrell. 1981. In "Recent Advances in Animal Nutrition in
    Australia", pp. 99-114. ed., D.J. Farrell, University of New England Publishing Unit.

3.4 POULTRY - HEAT STRESS

Arima, Y., F.B. Mather and M.M. Ahmad. 1976. Response of egg production and shell quality to increase in environmental temperature in two age groups of hens. Poultry Sci. 55:818-820.

Assenmacher, I. 1973. The peripheral endocrine glands. In: Avian Biology, Vol. 3, p. 210. ed. D.S. Farner and J.R. King. Academic Press, NY.

Bouchillon, C.W., F.N. Reece and J.W. Deaton. 1970. Mathematical modeling of thermal homeostasis in a chicken. Trans. Am. Soc. Agr. Eng. 13:648-652.

Brown, K.I., D.J. Brown and R.K. Meyer. 1958. The effect of surgical trauma, ACTH and adrenal cortical hormones on electrolytes and gluconeogenesis in male chickens. Am. J. Physiol. 192:43-50.

Calder, W.A. and J.R. King. 1974. Thermal and caloric relations of birds. In: Avian Biology, Vol. 4, pp. 259-413. eds. D.S. Farner and J.R. King. Academic Press, NY.

Carr, L. and T. Carter. 1985. Housing and management of poultry in hot and cold climates. In: Stress Physiology in Livestock. Vol III. M.K. Yousef, ed. pp. 73-110. CRC Press, Boca Raton, Florida.

Dale, N.M. and H.L. Fuller. 1979. Effect of diet composition on feed intake and growth of chicks under heat stress. I. Dietary fat levels. Poultry Sci. 58:1529-1534.

Dale, N.M. and H.L. Fuller. 1980. Effect of diet composition on feed intake and growth of chicks under heat stress. II. Constant vs cycling temperatures. Poultry Sci. 59:1434-1441.

de Andrade, A.N., J.C. Rogler, W.R. Featherstone and C.W. Alliston. 1977. Interrelationships between diet and elevated temperatures (cyclic and constant) on egg production and shell quality. Poult. Sci. 56:1178-1188.

De Shazer, J.A., L.L. Olson and F.B. Mather. 1974. Heat losses of large white turkeys - 6 to 36 days of age. Poultry Sci. 53:2047-2054.

Edens, F.W. and H.S. Siegel. 1975. Adrenal responses in high and low response lines of chickens during acute heat stress. Gen. Comp. Endocrinol. 25:64-73.

El-Halawani, M.E., P.E. Walbel, J.R. Appel and A.R. Good. 1973. Effects of temperature stress on catecholamines and corticosterone of male turkeys. Am. J. Physiol. 224:384-388.

Fox, T.W. 1951. Studies on heat tolerance in the domestic fowl. Poultry Sci. 30:477-483.

Harrison, P.C. 1976. Effects of heat stress on cardiovascular responses in birds in "Progress in Biometeorology". (S.W. Tromp, Editor-in-chief). Vol. 1, Part 1. Swets and Zertlinger, B.V. Amsterdam. pp. 301-304.

Heald, P.F., P.M. McClachlan and K.A. Rookledge. 1965. The effect of insulin, glucagon and adrenocorticotrophis hormone on plasma glucose and free fatty acids of the domestic fowl. J. Endocrinol. 33:83-87.

Heller, E.D., D.B. Nathan and M. Perek. 1979. Short heat stress as an immunostimulant in chicks. Avian Pathol. 8:195-203.

Huggins, G. and R.W. Lewis. 1980. Precisely controlled temperature variations: Effect of edible yield of laying and broiler stock. Poultry Sci. 59:456-458.

Jeffers, T.K. 1974. Eimeria tenella: Incidence, distribution, and anticoccidial drug resistance of isolants in major broiler-producing areas. Avian Dis. 18:74-84.

Jones, G.E. and T.M. Huston. 1967. The effects of environmental temperature upon deomestic fowl deprived of feed and water. Poultry Sci. 46:1389-1395.

Keshavarz, K. and H.L. Fuller. 1980. The influence of widely fluctuating temperatures on heat production and energetic efficiency of broilers. Poultry Sci. 59:2121-2128.

Krueger, W.F., J.W. Bradley, C.R. Creger and D. Thompson. 1976. Pullet condition and protein level on cage layer performance. Poultry Sci. 55:1598.

Lee, R.P. 1978. Parasitism. In: Introduction to Animal Husbandry in the Tropics. 3rd ed., G.W. Williamson and W.J.A. Payne, eds. Longman, London. Pp 43-61.

McNaughton, J.L., L.F. Kubena, J.W. Deaton and F.N. Reece. 1977. Influence of dietary protein and energy on the performance of commercial egg-type pullets reared under summer conditions. Poultry Sci. 56:1391-1398.

Mueller, W.J. 1961. The effect of constant and fluctuating environmental temperatures on the biological performance of laying pullets. Poultry Sci. 40:1562-1571.

Oluyemi, J.A. and Adetowun Adebanjo. 1979. Measures applied to combat thermal stress in poultry under practical tropical environment. Poultry Sci. 58:767-773.

Ota, H. and E.H. McNally. 1965. Preliminary broiler heat and moisture data for designing poultry houses and ventilating systems. Am. Soc. Agric. Engr. Summer meeting, paper no. 65-411.

Reece, F.N., J.W. Deaton and F.W. Harwood. 1976. Roof insulation and its effect on broiler chicken mortality in hot weather. Trans. Am. Soc. Agr. Eng. 19:733-735.

Reece, F.N., J.W. Deaton and L.F. Kubena. 1972. Effects of high temperature and humidity on heat prostration of broiler chickens. Poultry Sci. 51:2021-2025.

Reece, R.N. J.W. Deaton and B.D. Lott. 1970. Use of evaporative cooling for broiler chicken production in areas of moderate and high relative humidity. Am. Soc. Agr. Eng. paper No. 70-412.

Regnier, J.A., K.W. Kelley and C. T. Gaskins. 1980. Acute thermal stressors and synthesis of antibodies in chickens. Poult. Sci. 59:985-990.

Scott, M.L. 1976. Effects of heat on vitamin metabolism in "Progress in Biometeorology" (S.W. Tromp, Editor-in-chief), Vol. 1, Part 1. Swets and Zertlinger B. V. Amsterdam, pp. 275-284.

Shane, S.M. 1984. Design and operation of poultry facilities under conditions of high temperature. Trop. Vet. J. 1(2):804-807.

Siegel, H.S. 1976. Effects of heat on energy metabolism in birds in "Progress in Biometeorology" (S.W. Tromp, Editor-in-chief), Vol. 1, Part 1. Swets and Zertlinger B.V. Amsterdam. pp. 204-209.

Siegel, H.S. and L.N. Drury. 1968a. Physiological responses of chickens to variations in air temperature and velocity. Poultry Sci. 47:1120-1127.

Siegel, H.S. and L.N. Drury. 1968b. Physiological responses to high lethal temperature and air velocity in young fowl. Poultry Sci. 47:1230-1235.

Siegel, H.S. and M. van Kampen. 1981. Metabolic responses of growing chickens to chronic corticosterone injections. Poultry Sci. 60:1733.

Simenson, E. 1985. Diseases of poultry in hot and cold environments. Chapter 5, pp 137-146 In Stress Physiology in Livestock, Vol. III Poultry (ed. M.K. Yousef). CRC Press, Boca Raton, FL, U.S.A.

Snedecor, J.G., D.B. King and R.C. Hendrikson. 1963. Studies on the chick glycogen body: Effects of hormones and normal glycogen turnover. Gen. Comp. Endocrinol. 3:176-183.

Swain, S. and D.J. Farrell. 1975. Effects of different temperature regimens on body composition and carry-over effects on energy metabolism of growing chickens. Poultry Sci. 54:513-520.

van Kampen, M. 1976. Evaporative temperature regulation in birds. In: Progress in Biometeorology, Division B, Progress in Animal Biometeorology, pp. 158-166. C.W. Tromp and J.J. Bouma, eds. Swets and Zeitlinger, Amsterdam.

Vo, K.V., M.A. Boone and W.E. Johnston. 1978. Effect of three lifetime ambient temperatures on growth, feed and water consumption, and various blood components in male and female Leghorn chickens. Poultry Sci. 57:798-803.

Warren, D.C. and R.L. Schnepel. 1940. The effect of air temperatures on egg shell thickness in the fowl. Poultry Sci. 19:67-72.

Whittow, G.C. 1976. Regulation of body temperature in "Avian Physiology" (P.D. Sturkie, Editor). Third Edition, Springer-Verlag, New York. pp. 146-173.

Wilson, W.O., C.F. Kelley, R.T. Lorenzen and A.E. Woodard. 1957. Effect of wind on growth of fryers after two weeks of age. Poultry Sci. 36:978-989.

3.51 HEAT EXPOSURE AND IMMUNE FUNCTION: Introduction

Kelley, K.W. 1980. Stress and immune function: A bibliographic review. Ann. Rech. Vet. 11:445-478.

Webster, A.J.F. 1981. Weather and infectious disease in cattle. Vet. Rec. 108:183-187.

3.52 HEAT EXPOSURE AND IMMUNE FUNCTION: Antibody-mediated Immunity

Curtain, C.C. 1971. The influence of environment upon serum immunoglobulin levels in cattle. Brit. Vet. J. 127:442-450.

Heller, E.D., D.B. Nathan and M. Perek. 1979. Short heat stress as an immunostimulant in chicks. Avian Pathol. 8:195-203.

Joshi, B.C., V.K. Goel and S.K. Sexena. 1979. A note on the effect of acute heat exposure on the serum gamma-globulin content in zebu and zebu cross cattle. Indian J. Anim. Sci. 49:145-147.

Kelley, K.W., R.E. Greenfield, J.F. Evermann, S.M. Parish and L.E. Perryman. 1982a. Delayed-type hypersensitivity, contact sensitivity and PHA skin test responses of heat- and cold-stressed calves. Am. J. Vet. Res. 43:775-779.

Kelley, K.W., C.A. Osborne, J.F. Evermann, S.M. Parish and C.T. Gaskins. 1982b. Effects of chronic heat and cold stressors on plasma immunoglobulin and mitogen-induced blastogenesis in calves. J. Dairy Sci. 65:1514-1528.

Norman, L.M., W.D. Hohenboken and K.W. Kelley. 1982. Genetic differences in concentrations of immunoglobulins G_1 and M serum and colostrum of cows and in serum of neonatal calves. J. Anim. Sci. 53:1465-1472.

Regnier, J.A., K.W. Kelley and C.T. Gaskins. 1980. Acute thermal stressors and synthesis of antibodies in chickens. Poult. Sci. 59:985-990.

Stott, G.H. 1980. Immunoglobulin absorption in calf neonates with special considerations of stress. J. Dairy Sci. 63:681-688.

Stott, G.H., F. Wiersman, B.E. Menefee and F.R. Radwanski. 1976. Influence of environment on passive immunity in calves. J. Dairy Sci. 59:1306-1311.

Subba Rao, D.D. and B. Glick. 1970. Immunosuppressive action of heat in chickens. Proc. Soc. Exp. Biol. Med. 133:445-448.

Thaxton, P., D.R. Sadler and B. Glick. 1968. Immune response of chickens following heat exposure or injections with ACTH. Poult. Sci. 47:264-266.

Thaxton, P. and H.S. Siegel. 1970. Immunodepression in young chickens by high environmental temperature. Poult. Sci. 49:202-205.

Thaxton, P. and H.S. Siegel. 1972. Depression of secondary immunity by high environmental temperature. Poult. Sci. 51:1519-1526.

Thaxton, P. and H.S. Siegel. 1973. Modification of high temperature and ACTH induced immunodepression by metyrapone. Poult. Sci. 52:618-624.

3.53 HEAT EXPOSURE AND IMMUNE FUNCTION: Cell-mediated Immunity

Blecha, F., R.A. Barry and K.W. Kelley. 1982. Stress-induced alterations in delayed-type hypersensitivity to SRBC and contact sensitivity to DNFB in mice. Proc. Soc. Exp. Biol. Med. 169.

Kelley, K.W., M.T. O'Conner and D.L. Oldenkamp. 1981. Immunosuppression in calves caused by alternating heat and cold stress. J. Anim. Sci. Abstract 144.

Kelley, K.W., R.E. Greenfield, J.F. Evermann, S.M. Parish and L.E. Perryman. 1982a. Delayed-type hypersensitivity, contact sensitivity and PHA skin test responses of heat- and cold-stressed calves. Am. J. Vet. Res. 43:775-779.

Kelley, K.W., C.A. Osborne, J.F. Evermann, S.M. Parish and C.T. Gaskins. 1982b. Effects of chronic heat and cold stressors on plasma immunoglobulin and mitogen-induced blastogenesis in calves. J. Dairy Sci. 65:1514-1528.

Pitkin, D.H. 1965. Effect of physiological stress on the delayed hypersensitivity reaction. Proc. Soc. Exp. Biol. Med. 120:350-351.

Regnier, J.A. and K.W. Kelley. 1981. Heat- and cold-stress suppresses in vivo and in vitro cellular immune responses in chickens. Am. J. Vet. Res. 42:294-299.

Soper, F.F., C.C. Muscoplat and D.W. Johnson. 1978. In vitro stimulation of bovine peripheral blood lymphocytes: Analysis of variation of lymphocyte blastogenic response in normal dairy cattle. Am. J. Vet. Res. 39:1039-1042.

3.54 HEAT EXPOSURE AND IMMUNE FUNCTION: Possible Mechanisms for Heat-Induced Changes in the Immune System

Blecha, F., R.A. Barry and K.W. Kelley. 1982. Stress-induced alterations in delayed-type hypersensitivity to SRBC and contact sensitivity to DNFB in mice. Proc. Soc. Exp. Biol. Med. 169.

Christison, G.I. and H.D. Johnson. 1972. Cortisol turnover in heat-stressed cows. J. Anim. Sci. 35:1005-1010.

Crabtree, G.R., A. Munch and K.A. Smith. 1980. Glucocorticoids and lymphocytes. I. Increased glucocorticoid receptor levels in antigen-stimulated lymphocytes. J. Immunol. 124:2430-2435.

Friedman, S.B., L.A. Glasgow and R. Ader. 1969. Pyschosocial factors modifying host resistance to experimental infections. N.Y. Acad. Sci. 164:381-393.

Gillis, S., G.R. Crabtree and K.A. Smith. 1979. Glucocorticoid induced inhibition of T cell growth factor production. I. The effect on mitogen-induced lymphocyte proliferation. J. Immunol. 123:1624-1631.

Heller, E.D., D.B. Nathan and M. Perek. 1979. Short heat stress as an immunostimulant in chicks. Avian Pathol. 8:195-203.

Karmall, R.A., I. Lauder and D.F. Horrobin. 1974. Prolactin and the immune response. Lancet 2:106-107.

Maiti, B.R. and S. Chakroborty. 1979. Mitogenic action of prolactin on some endocrine organs of chickens. Canc. Tr. Rep. (Meeting) 63:1174.

Marple, D.N., E.D. Aberle, J.C. Forrest, W.H. Blake and M.D. Judge. 1972. Effects of humidity and temperature on porcine plasma adrenal corticoids, ACTH and growth hormone levels. J. Anim. Sci. 34:809-812.

Riley, V. 1981. Psychoneuroendocrine influences on immunocompetence and neoplasia. Science 212:1100-1109.

Stephens, D.B. 1980. Stress and its measurement in domestic animals: A review of behavioral and physiological studies under field and laboratory situations. Adv. Vet. Sci. Comp. Med. 24:179-210.

Tache, Y., P. Du Ruisseau, J.R. Ducharme and R. Collu. 1978. Pattern of adenohypophyseal hormone changes in male rats following chronic stress. Neuroendocrinology 26:208-219.

Tucker, H.A. and R.P. Wettemann. 1976. Effects of ambient temperature and relative humidity on serum prolactin and growth hormone in heifers. Proc. Soc. Exp. Biol. Med. 151:623-626.

Wettemann, R.P. and H.A. Tucker. 1974. Relationship of ambient temperature to serum prolactin in heifers. Proc. Soc. Exp. Biol. Med. 146:908-911.

3.61 ANIMAL PARASITES IN HOT CLIMATES: Internal Parasites and Hot Weather Stress

Anderson, N. 1972. Ostertagiasis in beef cattle. Vict. Vet. Proc. 30:36-38.

Anderson, N. and V. Lord. 1979. Anthelminthic efficacy of oxfendazole, fenbendazole, and levamisole against naturally acquired infections of Ostertagia ostertagi and Trichostrongylus axel in cattle. Aust. Vet. J. 5:158-162.

Armour, J. 1975. The epidemiology and control of bovine fascioliasis. Vet. Rec. 96:198-201.

Armour, J. and R.G. Bruce. 1974. Inhibited development in Ostertagia ostertagi - a diapause phenomenon in a nematode. Parasitology. 69:161-174.

Baker, N.F., R.A. Fisk and R.B. Busnell. 1981. Seasonal occurrence of infective nematode larvae on irrigated pasture grazed by cattle in California. Am. J. Vet. Res. 42:1188-1191.

Batte, E.G., R.D. McLamb and V.J. Vestal. 1976. Pathology and control of swine kidneyworms. Pig Vet. Soc. Cong, Ames, Iowa. Abstract #0.2.

Benitez-Usher, C., S. Maciel, C. Rebollo and J. Armour. 1984. A study of bovine parasitic gastro-enteritis in Paraguay. Prev. Vet. Med. 2:295-308.

Biggs, H.C. and M. Anthonissen. 1982. The seasonal incidence of helminth parasites and of Oestrus ovis in Karakul sheep in the Kalahari region of South West Africa/Namibia. Onersterpoort J. Vet. Res. 49:73-77.

Bitakaramire, P.K. 1968a. Bovine fascioliasis in Kenya. Bull. Epizoot. Dis. Afr. 16:107-113.

Bitakaramire, P.K. 1968b. The survival of Lymnaea natalensis in drought conditions. Bull. Epizoot. Dis. Afr. 16:473-475.

Blitz, N.M. and H.C. Gibbs. 1972. Studies on the arrested development of Haemonchus contortus in sheep. I. The induction of arrested development. Int. J. Parasit. 2:5-12.

Bowen, F.L. 1979. Nematode parasite control in weaner cattle on the northern tablelands of New South Wales. Aust. J. Exp. Agric. and Anim. Husb. 19:269-275.

Brunsden, V.R. 1972. Inhibited development of Ostertagia spp. and Cooperia spp. in naturally acquired infections in calves. New Zealand Vet. J. 10:183-189.

Chalmers, K. 1980. Ostertagiasis survey: Summary and discussion. N. Z. Vet. J. 28:27-34.

Chiejina, S.N. and C.O. Emehelu. 1984. Seasonal changes in pasture populations of infective larvae of gastrointestinal nematodes of cattle in eastern Nigeria. Res. Vet. Sci. 37:144-147.

Chiejina, S.N. and B.B. Fakae. 1984. Development and survival of infective larvae of gastrointestinal nematode parasites of cattle on pasture in eastern Nigeria. Res. Vet. Sci. 37:148-153.

Coyle, T.J. 1958. Experiments in the diagnosis and treatment of fascioliasis in Ugandan cattle. Bull. Epizoot. Dis. Afr. 6:255-272.

Craig, T.M. and R.R. Bell. 1978. Seasonal transmission of liver fluke to cattle in the Texas Gulf Coast. J. Am. Vet. Med. Assoc. 173:104-107.

Dinnik, J.A. and N.N. Dinnik. 1963. Effect of the seasonal variations of temperature on the development of Fasciola gigantica in the snail host in the Kenya Highlands. Bull. Epiz. Dis. Afr. 11:197-207.

Dinnik, J.A. and N.N. Dinnik. 1964. The influence of temperature on the succession of redial and cercarial generations of Fasciola gigantica in a snail host. Parasit. 54:59-65.

Hammond, J.A. 1965. Observations on fascioliasis in Tanganyika. Bull. Epizoot. Dis. Afr. 13:55-65.

Hart, J.A. 1964. Observations on the dry season strongyle infestations of Zebu cattle in northern Nigeria. Brit. Vet. J. 120:87-94.

Horak, I.G. 1978a. Parasites of domestic and wild animals in South Africa. V. Helminths in sheep on dryland pasture on the Transvaal highveld. Ondersterpoort J. Vet. Res. 45:1-6.

Horak, I.G. 1978b. Parasites of domestic and wild animals in South Africa. XI. Helminths in cattle on natural pastures in the northern Transvaal. Ondersterpoort J. Vet. Res. 45:229-234.

Horak, I.G. and J.P. Louw. 1977. Parasites of domestic and wild animals in South Africa. IV. Helminths in sheep on irrigated pasture on the Transvaal highveld. Ondersterpoort J. Vet. Res. 44:261-270.

Horak, I.G. and J.P. Louw. 1978. Parasites of domestic and wild animals in South Africa. IV. Helminths in calves on irrigated pasture on the Transvaal highveld. 45:23-28.

Hotson, I.K. 1967. Ostertatiasis in cattle. Aust. Vet. J. 43:383-388.

Lee, R.P., J. Armour and J.G. Ross. 1960. The seasonal variations of strongyle infestations of Nigerian zebu cattle. Brit. Vet. J. 116:34-46.

Lindsay, M.G. 1979. Dynamics of Fasciola hepatica infected snail populations in south Louisiana. M S Thesis, Louisiana State University, Baton Rouge, LA 70803, 53 pp.

Malone, J.B., A. Loyacano, D.A. Armstrong and L.F Archbald. 1982a. Bovine fascioliasis: economic impact and control in Gulf Coast cattle based on seasonal transmission. Bovine Practitioner. 17:126-133.

Malone, J.B., P.H. Smith, A.F. Loyacano, F.G. Hembry and L.T. Brock. 1982b. Efficacy of albendazole for treatment of naturally acquired Fasciola hepatica in calves. Am. J. Vet. Res. 43:879-881.

Malone, J.B., A.F. Loyacano, M.E. Hugh-Jones and K.C. Corkum. 1984a. A three-year study on seasonal transmission and control of Fasciola hepatica of cattle in Louisiana. Prev. Vet. Med. 3:131-141.

REFERENCES

Malone, J.B., R.T. Ramsey and A.F. Loyacano. 1984b. Efficacy of clorsulon for treatment of mature naturally acquired and 8-week-old experimentally induced Fasciola hepatica infections in cattle. Am. J. Vet. Res. 45:851-854.

Michel, J.F. 1967. Regulation of egg output of populations of Ostertagia ostertagi. Nature, Lond. 215:1001-1002.

Michel, J.F., M.B. Lancaster and C. Hong. 1974. Studies on arrested development of Ostertagia ostertagi and Cooperia onophora: Effect of temperature at the free-living third stage. J. Comp. Path. 84:539-554.

Michel, J.F., M.B. Lancaster and C. Hong. 1978. Arrested development of Ostertagia ostertagi and Cooperia oncophora: Effect of the time of year on the conditioning and deconditioning of infective larvae. J. Comp. Path. 88:131-136.

Moncol, D.J. and E.G. Batte. 1966. Transcolostral infection of new-born pigs with Strongyloides ransomi. VM/SAC. 61:583-586.

Okon, E.D. and A.I. Akinpelu. 1982. Development and survival of nematode larvae on pasture in Calabar, Nigeria. Trop. Anim. Hlth. Prod. 14:23-25.

Olsen, O.W. 1944. Bionomics of the lymnaeid snail, Stagnicola bulimoides techella, the intermediate host of the liver fluke in southern Texas. J. Agric. Res. 69:389-403.

Olsen, O.W. 1947. Longevity of metacercariae of Fasciola hepatica on pastures in the upper coastal region of Texas and its relationship to liver fluke control. J. Parasit. 3:36-42.

Owen, I.L. 1984. Production effects of the liver fluke Fasciola hepatica on weaner cattle in Papua New Guinea. Trop. Anim. Hlth. Prod. 16:158-160.

Pimento Neto, M., R.P. Mello, R.V. Nascimento and A.C.S. Moreira. 1965. Ocorrencia de larvas de Strongyloides ransomi Schwartz, and Alicata, 1930, em figado de suino natimorto no municipio de Itaguai. Atas Soc Biol Rio de Janeiro. 9:40-42.

Preston, J.M. and J.B. Castelino. 1977. A study of the epidemiology of bovine fascioliasis in Kenya and its control using N-tritylmorpholine. Br. Vet. J. 133:600-608.

Prinsloo, J.F. and J.A. van Eeden. 1969. Temperature and its bearing on the distribution and chemical control of freshwater snails. So. Afr. Med. J. 43:1363-1365.

Pullan, N.B. and K. Megadmi. 1983. Haemonchosis in a winter rainfall area in Libya. Trop. Anim. Hlth. Prod. 15:237-238.

Pullar, E.M. 1953. The epidemiology of helminthosis in sheep in winter rainfall regions of Australia. Australian Vet. J. 29:357-364.

Rivera, B., D. Parra, O. Garcia and E. Aycardi. 1983. Gastro-intestinal parasites in calves in Colombia. Trop. Anim. Hlth Prod. 15:107-114.

Rosa, W.A.J., R. Lukovich and R. Niec. 1971. Parasitismo gastrointestinal de los ovinos y bovinos en la zona sur de la provincia de Buenos Aires. Rev Inv Agropec, INTA Series 4, Patalogia Animal. 8:1-12.

Ross, I.C. and A.C. McKay. 1929. The bionomics of Fasciola hepatica in New South Wales and of the intermediate host Limnea brazeiri (Smith). Australian Council Sci. and Indus. Res. Bull. 43:1-62.

Salimov, B.S. and Z.A. Azimov. 1983. The epizootiology and biology of Fasciola gigantica. Veterinariya. 10:55-56.

Schillhorn van Veen, T.W. 1980a. Fascioliasis (Fasciola gigantica) in West Africa: A review. Vet. Bull. 50:529-533.

Schillhorn van Veen, T.W. 1980b. Dynamics of _Lymnaea natalensis_ populations in the Zaria area (Nigeria) and the relation to _Fasciola gigantica_ infections. Acta Trop. 37:183-194.

Sheikh, B.A., G.B. Isani and N.N. Ansari. 1984. Occurrence of fascioliasis in relation to bio-climatic conditions in Sind. Pakistan Vet. J. 4:37-41.

Shirai, Mitsuji. 1927. The biological observation on the cysts of _Fasciola hepatica_ and the route of migratiion of the young worms in the final host. Sci. Repts. Govt. Inst. Infectious Dis. Tokyo Imp. Univ. 6:511-523.

Smeal, M.G., I.K. Hotson, P.J. Mylrea, A.R. Jackson, N.J. Campbell and H.C. Kirton. 1977. Studies on nematode infections of beef cattle in New South Wales. Aust. Vet. J. 53:566-574.

Smeal, M.G., G.C. Fraser and G.G. Robinson. 1980. Seasonal changes in the structure of nematode populations of cattle in New South Wales in relation to inhibited larval development. Aust. Vet. J. 56:80-86.

Smith, G. and R.A. Wilson. 1980. Seasonal variations in the microclimate of _Lymnae truncatula_ habitats. J. Appl. Ecol. 17:329-342.

Sprent, J.F.A. 1946. Some observations on the bionomics of _Bunostomum phlebotomum_, a hookworm of cattle. Parasitology. 37:202-210.

Stewart, T.B., O.M. Hale and J.S. Andrews. 1964. Eradication of the swine kidneyworm, _Stephanurus dentatum_, from experimental pastures by herd management. Am. J. Vet Res. 25:1141-1150.

Stewart, T.B., W.N. Smith and W. M. Stone. 1969. Strongyloidiasis: Perinatal infection of pigs with _Strongyloides ransomi_. Georgia Vet. 21:13-18.

Stewart, T.B., W.M. Stone and O.G. Marti. 1973. _Strongyloides ransomi_: Detecting drug action on larvae in porcine tissue. Am. J. Vet. Res. 34:191-193.

Stewart, T.B., W.M. Stone and O.G. Marti. 1976. _Strongyloides ransomi_: Prenatal and transmammary infection of pigs of sequential litters from dams experimentally exposed as weanlings. Am. J. Vet. Res. 37:541-544.

Thomas, J.D. and A.I. Tait. 1984. Control of the snail hosts of schistosomiasis by environmental manipulation: A field and laboratory appraisal in the Ibadan area, Nigeria. Phil Trans Roy Soc London, B 305:201-253.

Thornthwaite, C.W. and J.R. Mather. 1957. Instructions and tables for computing potential transpiration and water balance. Publ. Climatol. 10:185-311.

Van Someren, V.D. 1946. The habitats and tolerance ranges of _Lymnaea_ (Radix) _caillaudi_, the intermediate snail host of liver fluke in East Africa. J. Anim. Ecol. 15:170-197.

Vegors, H.H. 1958. Observations on inhibited development of cattle nematodes. Proc. Helminth Soc. Washington. 25:86-90.

Williams, J.C. 1983. Ecology and control of gastrointestinal nematodes of beef cattle. Vet. Clinics of N. America: Large Animal Practice. 5:183-205.

Williams, J.C., J.W. Knox, B.A. Baumann, T.G. Snider, M.D. Kimball and T.J. Hoerner. 1983. Seasonal changes of gastrointestinal nematode populations in yearling beef cattle in Louisiana with emphasis on prevalence of inhibition in _Ostertagia ostertagi_. Int. J. Parasit. 13:133-143.

Williams, J.C., J.W. Knox, K.S. Marbury, M.D. Kimball, S.W. Scheide, T.G. Snider and M.U. David. 1984. Efficacy of fenbendazole against inhibited larvae of _Ostertagia ostertagi_ in yearling cattle. Am. J. Vet. Res. 45:1989-1993.

Young, R.R., R.M. Nicholson, R.L. Tweedle and H.J. Schuh. 1980. Quantitative modelling and prediction of development times of the free-living stages of Ostertagia ostertagi under controlled and field conditions. Parasit. 81:493-505.

3.62 ANIMAL PARASITES IN HOT CLIMATE: Ectoparasites of Livestock

Fitzgerald, E.A. and H.A. Nix. 1970. The climatic factor in Australian grassland ecology. In Australian Grassland (Ed. R.M. Moore), pp. 3-26. Aust. Nat. Univ. Press, Canberra.

Haufe, W.O. 1974. Haematophagous flies - a component in the impact of environment on health and productivity of cattle. Proc. Int. Livestock Environment Symposium, University of Nebraska, Lincoln, Apr. 17-19, 1974. American Society of Agricultural Engineers, St. Joseph, Michigan. SP-0174, pp. 203-208.

Hughes, R.D. and W.L. Nicholas. 1974. The spring migration of the bushfly (Musca velustissima): evidence of displacement provided by natural population markers including parasitism. J. Anim. Ecol. 43, pp. 411-428.

Murray, M.D. 1970. The spread of ephemeral fever of cattle during the 1967-68 epizootic in Australia. Aust. Vet. J. 46, pp. 77-82.

Murray, M.D. 1982. The "brevitarsis line" (the southern distribution of Culicoides brevitarsis). Arbovirus Research in Australia: Proc. 3rd Symposium. (Eds. T.D. St. George and B.H. Kay), p. 152.

Nix, H.A. 1976. Environmental control of breeding, post-breeding dispersal and migration of birds in the Australian region. In Proc. Int. Ornithol. Cong. 16th, Canberra, Australia, 1974 (Eds. H.J. Frith and J.H. Calaby), pp. 272-305. Australian Academy of Science, Canberra.

Sellers, R.F., D.E. Pedgley and M.R. Tucker. 1978. Possible windborne spread of bluetongue to Portugal, June-July 1956. J. Hyg., Camb., 81, pp. 189-196.

Sellers, R.F., E.P.J. Gibbs, K.A.J. Herniman, D.E. Pedgley and M.R.F. Tucker. 1979. Possible origin of the bluetongue epidemic in Cyprus, August 1977. J. Hyg., Camb., 83, pp. 547-555.

4.1 SOME EFFECTS OF AMBIENT CONDITIONS ON THE SURVIVAL AND DISPERSAL OF PATHOGENIC ORGANISMS IN AIR

Barlow, D.F. and A.I. Donaldson. 1973. Comparison of the aerosol stabilities of foot and mouth disease virus suspended in cell-culture fluid or natural fluids. J. Gen. Virol. 20:311-318.

Benbough, J.E. 1971. Some factors affecting survival of airborne viruses. J. Gen. Virol. 10:209-220.

Boisdron, Y. and J.R. Brock. 1972. Particle growth processes and initial particle size distributions. In: Assessment of Airborne Particles. Springfield, Ill.: C. Thomas & Co., P. 129.

Chamberlain, A.C. 1970. Deposition and uptake by cattle of airborne particles. Nature, Lond. 225:99-100.

Constantine, D.G. 1962. Rabies transmission by the non bite route. Publ. Hlth. Rep. 77:287-289.

Dawson, P.S. 1970. The involvement of milk in the spread of foot-and-mouth disease: an epidemiological survey. Vet. Rec. 87:543-548.

Donaldson, A.I. 1972. The influence of relative humidity on the aerosol stability of different strains of foot and mouth disease virus suspended in saliva. J. Gen. Virol. 15:25-33.

Donaldson, A.I. 1973. Influence of relative humidity on the stability of foot and mouth disease virus in aerosols from milk and faecal slurry. Res. Vet. Sci. 15:96-101.

Donaldson, A.I. and N.P. Ferris. 1974. Airborne stability of swine vesicular disease virus. Vet. Rec. 95:19-21.

Donaldson, A.I. and N.P. Ferris. 1975. The survival of foot and mouth disease virus in open air conditions. J. Hyg. Camb. 74:409-416.

Donaldson, A.I. and N.P. Ferris. 1976. The survival of some airborne animal viruses in relation to relative humidity. Vet. Micro. 1:413-421.

Donaldson, A.I. and N.P. Ferris. 1980. Sites of release of airborne foot-and-mouth disease virus from infected pigs. Res. Vet. Sci. 29:315-319.

Donaldson, A.I., J. Gloster, L.D.J. Harvey and D.H. Deans. 1982. Use of prediction models to forecast and analyse airborne spread during foot-and-mouth disease outbreaks in Brittany, Jersey, and the Isle of Wight in 1981. Vet. Rec. 110:53-57.

Donaldson, A.I., R.C. Wardley, S. Martin and N.P. Ferris. 1983. Experimental Aujesky's disease in pigs:excretion, survival and transmission of the virus. Vet. Rec. 113:490-494.

Drummond, D.J. and R. Hunnam. 1966. Personal communication.

Gloster, J. 1983. Factors influencing the airborne spread of Newcastle disease. Br. Vet. J. 139:445-451.

Gloster, J., R.M. Blackall, R.F. Sellers and A.I. Donaldson. 1981. Forecasting the airborne spread of foot-and-mouth disease. Vet. Rec. 108:370-374.

Gloster, J., R.F. Sellers and A.I. Donaldson. 1982. Long distance transport of foot-and-mouth disease virus over the sea. Vet. Rec. 110:47-52.

Harper, G.J. 1972. The influence of urban and rural air on the survival of micro-organisms exposed on microthreads. Proc. IV Int. Symp. on Aerobiology, Utrecht, Oostholk, p. 151-154.

Hedger, R.S. and P.S. Dawson. 1970. Foot-and-mouth disease virus in milk: an epidemiological survey. Vet. Rec. 87:186-188, 213.

Hidy, G.M. 1972. The dynamics of aerosols in the lower troposphere. In Assessment of Airborne Particles. Springfield, Ill.: C. Thomas, p. 81.

Hood, A.M. 1974. Open air factor in enclosed systems. J. Hyg. Camb. 72:53-60.

Hugh-Jones, M.E. 1976. A simulation spatial model of the spread of foot and mouth disease through the primary movement of milk. J. Hyg. Camb. 77:1-9.

Hugh-Jones, M.E., W.H. Allen, F.A. Dark and G.J. Harper. 1973. The evidence for the airborne spread of Newcastle disease. J. Hyg. Camb. 71:325-339.

Hugh-Jones, M.E. and R. Tinline. 1976. Studies on the 1967-68 foot and mouth disease epidemic: Incubation period and herd serial interval. J. Hyg. Camb. 77:141-153.

Hugh-Jones, M.E. and P.B. Wright. 1970. Studies on the 1967-68 foot and mouth disease epidemic. J. Hyg. Camb. 68:253-271.

Hyslop, N.St.G. 1964. Report. A.V.R.I., Pirbright

Hyslop, N.St.G. 1965. Secretion of foot and mouth disease virus and antibody in the saliva of infected and immunized cattle. J. Comp. Path. Therap. 75:111-117.

Hyslop, N. St. G. 1970. The epizootiology and epidemiology of foot and mouth disease. In Advances in Veterinary Science, Vol. 14. New York and London: Academic Pres, p. 261-307.

REFERENCES

Hyslop, N.St.G. 1971a. Factors influencing the epidemiology and epizootiology of airborne disease. J.A.V.M.A. 159:1500-1507.

Hyslop, N.St.G. 1971b. Observations on pathogenic organisms in the airborne state. Trop. Anim. Hlth. Prod. 4:28-40.

Hyslop, N.St.G. 1972. Epizootiological and serological studies on strains of Mycoplasma bovigenitalium. In: Proc. VII Int. Kong. F. Tierische Fortpflanzung, Munich, p. 375.

Hyslop, N.St.G. 1977. Proceedings, International Microbial Environment Symposium, Dunedin, New Zealand.

Hyslop, N. St. G. 1979. Observations on the survival and infectivity of airborne rinderpest virus. Int. J. Biomet. 23:1-7.

May, K.R., H.A. Druett, and L.P. Packman. 1969. Toxicity of open air to a variety of microorganisms. Nature, London 221:1146-1147.

de Mik, G. and I. de Groot. 1977. The germicidal effect of open air in different parts of the Netherlands. J. Hyg. Camb. 78:175-185.

Mitchell, C.A. and L.F. Guerin. 1972. Influenza A of human, swine, equine and avian origin: comparison of survival in aerosol form. Can J. Comp. Med. 36:9-11.

Mitchell, C.A., L.F. Guerin and J. Robillard. 1968. Decay of influenza A viruses of human and avian origin. Can. J. Comp. Med. 32:544-546.

Murray, M.D. 1970. The spread of ephemeral fever of cattle during the 1967-68 enzootic in Australia. Aust. Vet. J. 46:77-82.

Pasquill, F. 1974. Atmospheric diffusion: the dispersion of windborne material from industrial and other sources. 2nd Edition, Ellis Horwood Ltd, Chichester, England.

Primault, B. 1972. Les effets des conditions meteorologiques exterieures sur l'extension de la fievre aphteuse. Biometeorology 5, 100 (and personal communication).

Primault, B. 1974. La propagation d'une epizootie de fievre aphteuse: depend - elle des conditions meteorologiques? Schw. Arch. Tierheilk. 116:7-9.

Sellers, R.F. and A.J. Forman. 1973. The Hampshire epidemic of foot and mouth disease. J. Hyg. Camb. 71:15-34.

Sellers, R.F., K.A.J. Herniman and I.D. Gumm. 1977a. The airborne dispersal of foot and mouth disease virus from vaccinated pigs, cattle and sheep after exposure to infection. Res. Vet. Sci. 23:70-75.

Sellers, R.F., D.E. Pedgley and M.R. Tucker. 1977b. Possible spread of African horse sickness on the wind. J. Hyg. Camb. 79:279-298.

Smith, C.V. 1964. Some evidence for windborne spread of fowl pest. Met. Mag. 93:257.

Smith, L.P. 1970. Weather and Animal Diseases. World Meteorological Organization. Technical Note No. 113, WMO, Geneva.

Smith, P. and M.E. Hugh-Jones. 1969. The weather factor in foot and mouth disease. Nature, London 223: 712-715.

Standfast, H.A., M.I. Murray, A.L. Dyce and T.D. St. George. 1973. Report on ephemeral fever in Australia. Bull. Off. Int. Epiz. 79:615-625.

Tinline, R. 1970. Lee-wave hypothesis for the initial pattern of spread during the 1967-68 foot and mouth epizootic. Nature, London 227:860-862.

Tinline, R. 1971. Ph.D. Thesis, University of Bristol: A simulation study of the 1967-68 foot and mouth epizootic in Great Britain.

Troubworst, T., Sjoukje-Kuyper, J.C. de Jong and A.D. Plantinga. 1974. Inactivation of some bacterial and animal viruses by exposure to liquid-air interfaces. J. Gen. Virol. 24:155-165.

Wilkinson, P.J. and A.I. Donaldson. 1977. Transmission studies with African swine fever virus, the early distribution of virus in pigs infected by airborne virus. J. Comp. Path. 87:497-501.

Wilkinson, P.J., A.I. Donaldson, A. Greig and W. Bruce. 1977. Transmission studies with African swine fever virus. J. Comp. Path. 87:487-495.

Youngner, J.S. 1957. Thermal inactivation studies with different strains of poliovirus. J. Immunol. 78, 282-290.

4.2 EFFECT OF ENVIRONMENT ON NUTRITION REQUIREMENTS OF DOMESTIC ANIMALS

Ames, D.R. and D.R. Brink. 1977. Effect of temperature on lamb performance and protein efficiency ratio. J. Anim. Sci. 44:136-140.

Ames, D.R., D.R. Brink and C.'. Williams. 1980. Adjusting protein in feedlot rations during thermal stress. J. Anim. Sci. ...:1-6.

Brink, D.R. and D.R. Ames. 1978. Effect of thermal stress on protein for maintenance. 70th ASAS, p. 407.

Davis, R.H., O.E.M. Hassan and A.H. Sykes. 1972. The adaptation of energy utilization in the laying hen to warm and cool ambient temperatures. J. Agric. Sci. (Camb.) 79:363-369.

Filmer, D.G. and M.K. Curran. 1977. Climatic environment and practical nutrition of the growing pig, p. 25. In W. Haresign, H. Swan, and C. Lewis, eds., Nutrition and the Climatic Environment. Butterworth, London.

Fuller, M.F. and A.W. Boyne. 1971. The effectsof environmental temperature on the growth and metabolism of pigs given different amounts of food. I. Nitrogen metabolism, growth, and body composition. Br. J. Nutr. 25:259-272.

Heitman, H., C.F. Kelley and T.E. Bond. 1958. Ambient air temperature and weight gain in swine. J. Anim. Sci. 17:62-67.

Hidiroglou, M. and J.R. Lessard. 1971. Some effects of fluctuating low ambient temperatures on beef cattle. Can. J. Anim. Sci. 51:111-120.

Holmes, C.W. and N.D. Grace. 1975. A note on the metabolism of Ca, P, Mg, Na and K by pigs growing at a high ambient temperature. Anim. Prod. 21:341-343.

Lofgreen, G.P. 1974. Ration formulation for relief from heat stress, p. 81. In 13th Calif. Feeders Day, Imperial Valley. Exp. Stn., El Centro, Calif.

Mangold, D.W., T.E. Hazen and V.W. Hays. 1967. Effect of air temperature on performance of growing-finishing swine. Trans. Am. Soc. Agr. Eng. 10:370.

McDowell, R.E. 1972. Improvement of livestock production in warm climates. W.H. Freeman and Co., San Francisco, CA, U.S.A.

McDowell, R.E., N.W. Hooven and J.K. Camoens. 1976. Effect of climate on performance of Holsteins in first lactation. J. Dairy Sci. 59:965-973.

Milligan, J.D. and G. Christison. 1974. Effects of severe winter conditions on performance of feedlot steers. Can. J. Anim. Sci. 54:605-610.

Moose, M.G., C.V. Ross, W.H. Pfanders. 1969. Nutritional and environmental relationships with lambs. J. Anim. Sci. 29:619-627.

Peng, C.L. and H. Heitman. 1974. The effect of ambient temperature on the thiamine requirement of growing-finishing pigs. Br. J. Nutr. 32:1-9.

Young, B.A. 1975. Effect of winter acclimitisation on resting metabolism of beef cows. Can. J. Anim. Sci. 55:619-625.

5.1 SOME ASPECTS OF THE METEOROLOGICAL FORECASTING OF ANIMAL HEALTH AND DISEASES: Introduction

Capstick, M. 1970. The Economics of Agriculture. Studies in Economics #3, Editor: Charles Carter. London: Allen & Unwin.

Omar, M.H. 1978. Economic Value of Improved Weather Forecastes. FAO/WMO Technical Conference on the Potential Economic Benefits of Agricultural Meteorology. FAO, Rome.

Thompson, R.T. and N.W. Taylor. 1975. The application of meteorological data to supply forecasting in the meat and wool industry. Proc. Symposium on Meteorology and Food Production. N.Z. Meteorological Service, pp. 217-230.

World Meteorological Organization, 1978. Weather and Parasitic Animal Disease. WMO Technical Note #159, Geneva.

5.2 SOME ASPECTS OF THE METEOROLOGICAL FORECASTING OF ANIMAL HEALTH AND DISEASES: Forecasting Aims and Objectives

Ollerenshaw, C.B. and W.T. Rowlands. 1959. A method for forecasting the incidence of fascioliasis in Anglesey. Vet. Rec. 71:591-598.

Omar, M.H. 1978. Economic Value of Improved Weather Forecastes. FAO/WMO Technical Conference on the Potential Economic Benefits of Agricultural Meteorology. FAO, Rome.

5.3 SOME ASPECTS OF THE METEOROLOGICAL FORECASTING OF ANIMAL HEALTH AND DISEASE: Advantages and Disadvantages of Forecasting Disease

Donaldson, A.I., J. Gloster, L.D.J. Harvey and D.H. Dean. 1982. Use of prediction models to forecast and analyse airborne spread during the foot-and-mouth disease out-breaks of Brittany, Jersey, and the Isle of Wight in 1981. Vet. Rec. 110:52-57.

World Meteorological Organization, 1978. Weather and Parasitic Animal Disease. WMO Technical Note #159, Geneva.

5.4 SOME ASPECTS OF THE METEOROLOGICAL FORECASTING OF ANIMAL HEALTH AND DISEASES: Commitment

Smith, L.P. 1970. WMO Technical Note No. 113 "Weather and Animal Diseases". Geneva.

World Meteorological Organization, 1978. Weather and Parasitic Animal Disease. WMO Technical Note #159, Geneva.

5.5   SOME ASPECTS OF THE METEOROLOGICAL FORECASTING OF ANIMAL HEALTH AND DISEASES: Conclusions

Maunder, W.T. 1977. Weather and climate as factors in forecasting national dairy production in management
    of dynamic systems in New Zealand agriculture. Proceedings of a Symposium, DSIR, Lower Hutt, 28 June
    1977. DSIR Information Series. 129:101-126.

Starr, J.R. (Under publication). Weather and climate and animal performance.

Recent WMO Technical Notes