

# symposium

AT THE ZOOLOGICAL SOCIETY OF LONDON

## IMPACTS OF ENVIRONMENTAL CHANGE ON REPRODUCTION AND DEVELOPMENT IN WILDLIFE

Thursday 15 and Friday 16 October 2009

Organised by

**William V. Holt**  
**Stuart R. Milligan**  
**Rhiannon E. Lloyd**

The Meeting Rooms  
The Zoological Society of London  
Regent's Park  
London NW1 4RY

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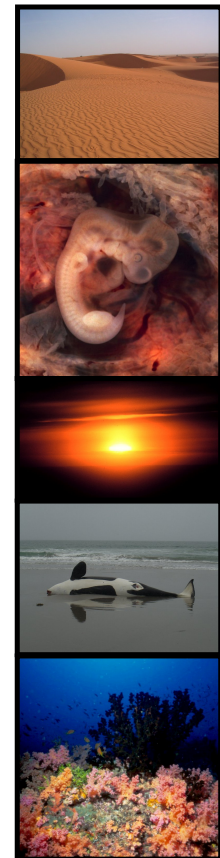


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9.15–9.30 **Welcome from Ralph Armond, Director General, ZSL**

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**SESSION I: ENVIRONMENTAL CHANGE AND REPRODUCTION: SETTING THE SCENE**

Chairs: William V. Holt and Rhiannon Lloyd (ZSL)

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9.30–10.15 **Keynote paper: Climate change and seasonal reproduction in mammals**  
*Franklin Bronson, University of Texas at Austin, USA*

Seasonal reproduction is commonly seen in mammals at all latitudes, even in the deep tropics. The objectives of this paper are three-fold: first, to consider what laboratory experimentation has taught us about the neuroendocrine pathways that link seasonal factors to the reproduction of mammals; second, to relate the knowledge gained in the laboratory to the kinds of challenges mammals actually face in natural habitats; third, to use the available information to consider how mammals might adapt seasonally to global climate change.

The two environmental factors of most concern when considering seasonal breeding are foraging conditions as they determine energy balance and predictive cues like photoperiod. The neuroendocrine links between these two factors and the neurons secreting gonadotropin-releasing hormone will be explored.

Food availability and the ambient temperature encountered during foraging determine energy balance and that, in turn, is the ultimate cause of seasonal breeding in all mammals and the proximate cause in many. Low temperatures experienced during foraging are particularly important for small mammals. Photoperiodic cueing is common among long-lived mammals from the highest latitudes to the mid-tropics; it is much less important for shorter-lived mammals, many of which exhibit great individual variation in photoresponsiveness and thus mixed strategies for adapting to winter conditions. An unidentified predictive cue, possibly a secondary plant compound, triggers reproduction after rains spur plant growth in some desert and dry grassland rodents.

The available information suggests that as our climate changes the small rodents of the world will probably reproduce successfully wherever they can survive the higher temperatures. Their short generation time is an asset and most species are opportunistic generalists to at least some degree. The situation may be quite different for longer-lived mammals at the mid and higher latitudes, particularly those whose reproduction is rigidly controlled by photoperiod. Mismatches between the time of birth and the peak of food availability are already being recorded in some of these animals. A major gap in our knowledge concerns the tropics. That is where most species live but it is where we have the least understanding of how reproduction is influenced by environmental factors.

10.15–11.00 **The impacts of climate change on the annual cycles of birds**  
*Cynthia Carey, Department of Integrative Physiology, University of Colorado,  
Boulder, USA*

The annual cycles of birds, including breeding, are typically coordinated with environmental events, such as the advent of spring in temperate and Arctic and Antarctic regions, and with periodic rainfall or dry periods in tropical regions. Some species of birds, however, breed irregularly or at times of specific food supplies. The timing of annual cycles has been under stringent natural selection to ensure that young are being raised at times of optimal food supplies. Considerable variation exists in the role of photoperiod in the timing of annual cycles. Annual cycles of birds that winter above about 20 °N or S latitude are usually controlled to varying degrees by photoperiod, the most reliable cue signaling the time of year.

While migration and breeding of many birds are controlled by photoperiod, their food supplies are usually temperature-dependent. That is, the growth of insect larvae, leaves, and other food supplies in the spring is caused by rising air and soil temperatures. As climates warm, spring time food supplies are reaching optimal levels several days to weeks earlier than in previous decades. However, many photoperiodically controlled species are still arriving on breeding grounds at the same time each year as in the past. These factors are contributing to mismatches between food availability and breeding. In response, a few species have been noted to migrate earlier than they historically had, but mismatches in food supplies on migratory stop-over sites and upon arrival of the adults may cause problems for reproductive success.

Other climate-driven changes can also affect food supplies during the post-breeding season when birds are molting and fattening in preparation for fall migration. In some areas, early growth of plants in the spring can lead to frost damage, causing failure to set seed. Birds reliant on these seeds for molting and fattening may find that food supplies restriction appropriate preparation for migration.

11.00–11.30 **POSTER SESSION (TEA/COFFEE)**

11.30–12.15 **Developmental programming: lessons from human and animal studies**  
*Susan Ozanne, University of Cambridge, UK*

Epidemiological studies have revealed a relationship between poor early growth and the development of type 2 diabetes, insulin resistance and other features of the metabolic syndrome. The mechanistic basis of this relationship is not known. However, compelling evidence suggests that early environmental factors, such as nutrition, play an important role. Studies of individuals *in utero* during a period of famine have shown a direct relationship between maternal nutrition and glucose tolerance. Further evidence has come from studies of monozygotic twins who were discordant for type 2 diabetes. Nutrition during the early postnatal period has also been shown to have long-term consequences on metabolic health. Excess nutrition and accelerated growth during the neonatal period has been suggested to be particularly detrimental. Animal models, including that of maternal protein restriction, have been developed to elucidate mechanisms linking the early environment and future disease susceptibility. Maternal protein restriction during pregnancy leads to a low birth weight and development of many features of the metabolic syndrome including type 2 diabetes. The

glucose intolerance is associated with  $\beta$  cell dysfunction and insulin resistance. The insulin resistance is associated with changes in expression of key components of the insulin-signalling cascade in muscle and adipocytes. Similar changes are observed in tissue biopsies from young men with a low birth weight. These proteins may be molecular markers of early growth restriction and disease risk, and thus in the long-term may make targeted intervention strategies a realistic possibility. Further analysis in both human cohorts and animal models has suggested that the effects of *in utero* growth restriction are exaggerated if it is followed by rapid postnatal growth. Studies using rodent models have demonstrated that animals that are born small due to maternal protein restriction during pregnancy but which then grow quickly by suckling a normally fed dam during lactation gain more weight post-weaning, are more susceptible to diet-induced obesity and have a reduced life span. The increased adiposity is apparent in very young adult life. Growing evidence from humans and animal models that obesity during pregnancy is associated with increased risk of metabolic diseases such as type 2 diabetes, insulin resistance and obesity in the offspring. This therefore provides a potential mechanism by which the effects of nutrition during pregnancy can be transmitted to many subsequent generations without any further nutritional insult.

12.15–13.00 **Epigenetic responses to environmental change: Lamarckian ideas and molecular mechanisms**

*Bryan M. Turner, University of Birmingham, UK*

Chromatin is a complex of DNA, RNA, histones and non-histone proteins and provides the platform on which genes are expressed or silenced in all eukaryotes. The structure and composition of chromatin are manipulated by families of enzymes, some catalyzing the dynamic addition and removal of chemical ligands to selected protein amino acids and some directly reconfiguring chromatin structures. The activities of these enzymes are sensitive to environmental and metabolic cues and they can serve as sensors through which environmental agents can alter gene expression. Altered patterns of gene expression can, in turn, precipitate changes in cellular physiology or behaviour. I will present recent data to show that, in the pre-implantation mouse embryo, epigenetic change induced by an environmental agent can be passed on, through mitosis, from one cell generation to the next, even in the absence of the inducing agent; this constitutes an epigenetic mutation. It is possible, though yet to be proven, that the induced epigenetic change could persist into the germ cell lineage and thereby be transmitted to the next generation. Plausible mechanisms now exist through which an epigenetic change might give rise to a localized change in DNA sequence exerting the same functional effect, thereby converting an epigenetic to a genetic mutation. If the induced genetic change has phenotypic effects on which selection can act, then this hypothetical chain of events constitutes a potential Lamarckian route through which the environment might directly influence evolutionary change.

13.00–14.00 **LUNCH**

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**SESSION II: ACUTE AND CHRONIC EFFECTS OF THE ENVIRONMENT ON  
DEVELOPMENT AND REPRODUCTION**

Chair: Sir Brian Heap (Cambridge)

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14.00–14.45 **Environmental consequences of altered nutrient supply during oocyte  
and embryo development**

*Cheryl Ashworth<sup>1</sup>, Luiza Toma<sup>2</sup> & Morag Hunter<sup>3</sup>*

*<sup>1</sup>The Roslin Institute, University of Edinburgh, UK; <sup>2</sup>Scottish Agricultural  
College, UK; <sup>3</sup>University of Nottingham, UK*

The environment in which a breeding female lives prior to conception and during the early stages of her pregnancy has striking effects on oocytes developing in the ovarian follicle and on early embryos in the reproductive tract. Of the various environmental factors known to affect oocyte and embryo development, altered nutrition during this critical period has been particularly well studied. Alterations in the quantity of food consumed or the composition of the diet imposed solely during the pre-mating period affect oocyte maturity, blastocyst yield, prenatal survival and the number of offspring born alive. Importantly, nutrition at this time also affects the quality of embryos and resultant offspring, with increasing evidence from a variety of species showing that periconception nutrition can alter behaviour, cardiovascular function and reproductive function throughout post-natal life. In livestock species it is important to devise nutritional strategies that improve reproductive efficiency and the quality of offspring but which do not add to the environmental footprint of the production system and which recognise likely changes in feedstuff availability arising from predicted changes in climate.

14.45–15.30 **Early life programming of fecundity**

*David Gardner, University of Nottingham, UK*

The early-life developmental environment is instrumental in shaping our overall adult health and well-being. Early-life diet and endocrine exposure may independently, or in concert with our genetic constitution, induce a pathophysiological process that amplifies with age and leads to premature morbidity and mortality. Recently, this has become known as 'programming' but is akin to 'maternal effects' described for many years in the biological sciences and is defined as any influence that acts during critical developmental windows to induce long-term changes in the organisms' phenotype. To date such delayed maternal effects have largely been characterised in terms of susceptibility to cardiovascular or metabolic disease. I will review evidence from experimental animal species, non-human primates and man for an effect of the early-life nutritional environment on adult fecundity and fertility. In addition, I shall present new data from a database of pedigree sheep, in which the hypothesis that being born small-for-gestational age with or without postnatal growth acceleration directly programs fertility can be tested. In conclusion, there appears to be a lack of compelling evidence to suggest prenatal undernutrition may directly reduce adult fecundity and fertility but may exert some effects secondarily via an increased incidence of 'Metabolic Syndrome'. However, possible effects of being born relatively large on subsequent fecundity and fertility warrant further investigation.

15.30–16.00 **TEA/COFFEE**

16.00–16.45 **Effects of heat stress on mammalian reproduction**

*Peter Hansen, University of Florida, USA*

Heat stress can have large effects on most aspects of reproductive function in mammals. These include disruptions in spermatogenesis and oocyte development, oocyte maturation, early embryonic development, fetal and placental growth and lactation. These deleterious effects of heat stress are the result of either the hyperthermia associated with heat stress or the physiological adjustments made by the heat-stressed animal to regulate body temperature. Many effects of elevated temperature on gametes and the early embryo involve increased production of reactive oxygen species. Artificial selection for production of milk, meat and fiber has increased the susceptibility of some breeds of farm animal species to heat stress. Thus, the impact of global warming on body temperature regulation and reproduction may be more severe for domestic animals than for wild mammalian species. Nonetheless, there is allelic variation in genes controlling body temperature regulation and cellular resistance to heat shock so that genetic adaptation to increasing global temperature will be possible for many wild and domesticated species.

16.45–17.30 **Genomic analysis of the impacts of EDCs: converting information to understanding**

*Peter Kille<sup>1</sup>, Alex Ford<sup>2</sup> & Charles Tyler<sup>3</sup>*

*<sup>1</sup>Cardiff School of Biosciences, University of Cardiff, UK; <sup>2</sup>School of Biological Sciences, University of Portsmouth, UK; <sup>3</sup>School of Biosciences, University of Exeter, UK*

There is wide ranging evidence for the effects of Endocrine Disrupting Chemicals (EDCs) in wild populations of several animal phyla, including reptiles, amphibians, fish, birds, mammals and a range of invertebrates. The consequences of EDC exposure range from developmental abnormalities to sexual dysfunction leading to the occurrence intersex phenotypes.

However, attributing occurrence of intersex directly to anthropogenic inputs, such as EDCs, can be problematic within invertebrates such as in arthropods where biological factors, including parasitism, can cause intersex. Studies on the inter-tidal crustacean *Gammarus marinus*, which exhibits both male and female intersex phenotypes, as determined by morphometry, has shown a direct impact of intersex on fecundity. Modelling indicates that the increased occurrence of intersex at highly polluted sites will result in significant population-level consequences. Transcriptomic investigations are starting to reveal the complex molecular endocrinology underlying the occurrence of the intersex phenotype and may provide novel insight into the targets of EDCs in arthropods and show whether these molecules are mechanistically related to those identified in vertebrates.

The incidence of intersex within wild populations of fish is now well documented. A study of wild roach populations identified intersex at 86% of sites sampled, with 23% of all males examined displaying intersex, with similar observations recorded for other UK fish species. The consequences of intersex for individual fecundity have been demonstrated both by direct perturbations in sperm density and motility, and through the reduced proportion of eggs that result in viable offspring. Competitive breeding studies also have demonstrated that severely

intersex individuals have a reduced competitive success. The intersex phenotypes can be induced through controlled exposure to acute and chronic water effluents which correlate with the uptake of oestrogens mixtures entering the fish. The issues of modelling the effects of mixtures combined with observations of the possible synergistic effect of anti-androgenics reinforce the need for better understanding of the complex endocrine pathways that drive sexual differentiation.

Exploitation of targeted molecular analysis has allowed the identification of chemical-specific signatures within the transcriptome indicative of alterations that may lead to endocrine disruption. Harnessing genome wide transcript and informatic data has facilitated the characterisation of the global processes underlying gonadal differentiation and of novel EDC targets within gonadal tissues. Exploitation of microsatellites to determine parentage and dominance in the presence of a known EDC reveals a disruption in the normal fish behaviour, and an approach involving brain-area specific transcriptomic analysis may reveal the role of the endocrine system in controlling aggressive behaviour patterns. Significant challenges remain in mining the current molecular data and to resolve this information into an understanding of how exposure EDCs results in the phenotypic and behavioural changes observed within vertebrates and invertebrates. In addition to novel environmental diagnostics the ultimate objective of this work should result in predictive models allowing environmental management to change from a reactive to a preventative mode.

*17.30–18.45    **POSTER SESSION with cash bar***

*18.45            **End of Day One***

*19.00–21.00    **Symposium dinner for speakers and guests with tickets***

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**SESSION III: ENDOCRINE DISRUPTION: A CAUSE FOR CONCERN?**

Chair: Stuart Milligan (King's College London)

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9.15–10.00     **Endocrine disruption and British rivers, why were we hit so hard?**  
*Andrew C. Johnson, Centre for Ecology & Hydrology, Wallingford, UK*

Surveys based on trout published in 1994, and on wild roach in 1998 and 2006, revealed widespread endocrine disruption in English rivers. The endocrine disruption raised concerns about fertility and reproduction effects in wild fish. The severest forms of endocrine disruption were usually linked to proximity to large sewage treatment plants (STP) where up to two-thirds of the male fish were affected. Further studies strongly suggested natural and synthetic steroid oestrogens excreted by the human population and discharged from STPs were the causative agents. These findings prompted many research programmes which revealed endocrine disruption and estrogens in water were indeed a worldwide issue. However, studies indicated endocrine disruption was variable depending on where and when you took the fish. Furthermore, whilst most countries found hot spots of endocrine disruption, no country reported that the problem was as extensive as it appeared to be in England. Why was endocrine disruption so unpredictable and why did England appear to have more problems than anyone else?

The research pursued by CEH followed the principal that effects in aquatic wildlife caused by chemicals discharged by the human population would be a function of load versus dilution. Thus, a knowledge of geography and hydrology should accurately guide predictions on where endocrine disruption would be most, or least, likely to occur. This approach can be used to guide which parts of river networks, regions, or indeed nations would be most at risk. Thus, a densely populated island, such as the UK (England in particular) with limited river dilution would be expected to be in the high risk category. The UK and Japan are both densely populated islands but Japan appears to have less endocrine disruption. We argue that differences here are due to the location of cities and rainfall patterns. More recently there have been some arguments that endocrine disruption in fish in rivers is in decline. But is this due to changes in sewage treatment performance, or the weather? Many climate change predictions for the UK suggest lower spring, summer and autumn flows in our rivers. Far from going away, endocrine disruption in our fish may be about to get worse.

10.00–10.45     **Anthropogenic pollutants - a threat to ecosystem sustainability**  
*Stewart M. Rhind, Macaulay Land Use Research Institute, Craigiebuckler, Aberdeen, UK*

Endocrine disrupting chemicals (EDCs) include a wide range of organic and inorganic chemicals derived from many different anthropogenic sources. They are present in many everyday items, including plastics, detergents, electrical equipment, food-can linings, paints, adhesives and pesticides and are also produced as a result of incomplete combustion of hydrocarbon fuels. Unlike more conventional pollutants which tend to be found at high concentrations close to the source, they are largely invisible, highly dispersed and present throughout the environment. Physiological processes in animals of all taxa can be affected by exposure to EDCs, and particularly to mixtures, even when environmental concentrations are very low. Effects of prolonged exposure to multiple EDCs at environmental concentrations

have been investigated using sheep exposed to pastures fertilised with sewage sludge, which contains a mixture of anthropogenic pollutants. Effects of exposure are diverse and fetuses are particularly susceptible. For example, disruption of kisspeptin expression in the fetal hypothalamus and pituitary has been demonstrated and may contribute to the observed perturbations of fetal ovary and testis structure and gene and protein expression. In addition to effects on the reproductive system, perturbations of mammary and bone structure, immune, thyroid and cardiovascular function, obesogenic mechanisms and offspring behaviour in response to EDC exposure have all been reported in sheep or other species. It is possible to extrapolate from these studies the effects on wildlife because many of the mechanisms of action involve fundamental processes that have been highly conserved throughout evolution. However, responses are likely to differ with patterns of exposure, with compounding effects of other physiological stressors and with differences in metabolism between species and individuals. Requirements for future work include identification of the most potent pollutants, assessment of exposure rates and mixture effects and identification of relevant end points and sentinel species. It is concluded that these pollutants can compromise ecosystem sustainability through effects on health and reproductive capacity of key species and that all species in all ecosystems are at risk.

**10.45–11.15 POSTER SESSION (TEA/COFFEE)**

**11.15–12.00 Endocrine disruptors and transgenerational epigenetics**  
*Carlos Guerrero-Bosagna, Washington State University, USA*

Certain natural agents can induce changes in the physiology of pregnant mammals, leading to alterations in DNA methylation patterns of the developing fetus and to the emergence of new phenotypes. Nevertheless, in order for this process to occur and to lead to evolutionary changes it would require (1) certain key periods in the ontogeny of the organism where the environmental stimuli could produce effects, (2) particular environmental agents to act as such stimuli, and (3) that a genomic persistent change be consequently produced in a population. This persistent change can be achieved by exposing animals to certain compounds available in the environment. Endocrine disrupting chemicals are compounds known to alter reproductive related phenotypes.

It has recently been shown that this class of compounds can also alter DNA methylation patterns in mammals. Exposure to endocrine disruptors can produce persistent changes in DNA methylation in two ways. One is by repeated exposures inducing an altered DNA methylation pattern in each generation (extrinsic process). This may occur, for example, after exposure to nutritional phytoestrogens, which can produce both changes in DNA methylation and in reproductive features. Another way in which endocrine disrupting chemicals can produce transgenerational changes is by affecting intrinsic features (intrinsic process) in the DNA that allow for epigenetic transgenerational transmission of characters. For example, exposure of the germ line to the endocrine disruptor vinclozolin during the critical period of sex differentiation produces changes in DNA methylation patterns in several future generations of rats. This process occurs even in the absence of the initial trigger stimulus after the first exposure. In addition to transgenerational reproductive alterations, this transient exposure to vinclozolin can also cause transgenerational behavioral changes. The implications for the action of endocrine disrupting chemicals are usually limited to physiological, toxicological or epidemiological studies, but this recent epigenetic transgenerational evidence poses new

questions about the role of environment as an inducer of changes that are relevant from an evolutionary perspective.

12.00–13.00 **LUNCH**

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**SESSION IV: ECOLOGICAL AND EVOLUTIONARY CONSEQUENCES OF ENVIRONMENTAL CHANGE**

Chair: Nathalie Pettorelli (ZSL)

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13.00–13.45 **The endocrine control of reproduction in Nereidae: a new multi-hormonal model with implications for their functional role in a changing environment**

*Andrew Lawrence, University of the West Indies, Trinidad and Tobago*

Nereidae are vital to the functioning of estuarine ecosystems and major components in the diets of over-wintering birds and commercial fish. They use environmental cues to synchronise reproduction. Photoperiod is the proximate cue, initiating vitellogenesis in a temperature compensated process. In Nereidae the prevailing paradigm is of a single “juvenile” hormone controlling growth and reproduction. However, a new multi-hormone model is presented here which integrates the environmental and endocrine control of reproduction. This is supported by evidence from *in vitro* bioassays. The juvenile hormone is shown to be heat stable and cross reactive between species. In addition, a second neuro-hormone, present in mature females, is found to promote oocyte growth. Furthermore, dopamine and melatonin appear to switch off the juvenile hormone whilst serotonin and oxytocin promote oocyte growth. Global warming is likely to uncouple the phase relationship between temperature and photoperiod with significant consequences for Nereidae that use photoperiod to cue reproduction during the winter in northern latitudes. Genotypic adaptation of the photoperiodic response may be possible but significant impacts on fecundity, spawning success and recruitment are likely in response to short-term extreme events. Endocrine disrupting chemicals may also impact on putative steroid hormone pathways in Nereidae with similar consequences. These impacts may have significant implications for the functional role of Nereidae and highlight the importance of comparative endocrinology studies in these and other invertebrates.

13.45–14.05 **Reproductive anatomy of a murine rodent from Australia, The Spinifex Hopping Mouse (*Notomys alexis*): adaptations for life in the arid environment**

*Bill Breed, University of Adelaide, Australia*

In Australia about 25% of the extant mammalian fauna is composed of native mice and rats (subfamily Murinae) whose earliest ancestors arrived around 4 million years ago. Since that time there has been a modest radiation into around 60 species of 12 genera one of which is the genus *Notomys*, the Hopping mice, three of five species of which occur in the arid zone-an environment that is characterised by high unpredictability and prolonged droughts. What is their reproductive biology that enables these animals flourish in this environment? To

investigate this we have selected the Spinifex hopping Mouse, *Notomys alexis*, that has a widespread distribution throughout much of arid Australia.

Females have a mean litter size of about four (there are four nipples), normal gestation length of around 32 days, and maturation age at around 65 days; these parameters are similar to those of many other close relatives. By contrast, males have a highly derived reproductive anatomy. The testes are minute (*ca.* 0.1% body mass) and there is comparatively low sperm production per gram testis mass with highly pleiomorphic sperm being produced. Comparatively low sperm numbers are stored in the excurrent ducts albeit that the distal vas deferens acts as an accessory storage site. These data suggest absence of intermale sperm competition but observations on social behaviour question this, hence there may be other functional cause(s). Observations of the distal reproductive tract show that males have a highly spinous penis and females a coevolved narrow vaginal lumen, thick surrounding muscle coat, and small cervix- morphological features that relate to locking during copulation. Sperm transport in the female tract is highly efficient and an adult male can fertilise up to three females within a few days. These data indicate that, in *N. alexis*, there are a suite of coadapted features of the reproductive system whereby males have evolved a strategy of low energy expenditure in sperm production and insemination and females a highly efficient system for sperm transport. Females, furthermore, have slightly greater body mass, and are more aggressive than males although males have typical, markedly higher, levels of circulating testosterone in peripheral blood; they also exhibit extensive paternal care including the retrieval of pups when they wander from the nest. Such a suite of highly derived features of the male and female reproductive system of *Notomys alexis* have no doubt evolved for maximising reproductive success in the arid environment in which the animals occur.

14.05–14.30 **Trophic imbalances in phenological change for marine, freshwater and terrestrial environments**

*Stephen Thackeray, Centre for Ecology and Hydrology, UK*

Recent changes in the seasonal timing (phenology) of familiar biological events have been one of the most conspicuous signs of climate change. However, the lack of a standardised approach to analysing change has hampered assessment of consistency in such changes among different taxa and trophic levels and across freshwater, terrestrial and marine environments. We present a standardised assessment of 25,532 rates of phenological change for 726 UK terrestrial, freshwater and marine taxa. The majority of spring and summer events have advanced, and more rapidly than previously documented. Such consistency is indicative of shared large-scale drivers. Furthermore, average rates of change have accelerated in a way that is consistent with observed warming trends. For the first time we show a broad scale signal of differential phenological change among trophic levels; across environments advances in timing were slowest for secondary consumers, thus heightening the potential risk of temporal mismatch in key trophic interactions. If current patterns and rates of phenological change are indicative of future trends, future climate warming may exacerbate trophic mismatching, further disrupting the functioning, persistence and resilience of many ecosystems and having a major impact on ecosystem services.

14.30–15.00 **POSTER SESSION (TEA/COFFEE)**

15.00–15.45 **Effects of environmental change on wildlife health**

*Karina Acevedo-Whitehouse<sup>1</sup> & Amanda L. J. Duffus<sup>1,2</sup>*

*<sup>1</sup>Institute of Zoology, Zoological Society of London, UK; <sup>2</sup>School of Biological and Chemical Sciences, Queen Mary, University of London, UK*

The current rate of environmental change is unprecedented and has negatively affected most biological systems on Earth. These changes are becoming of increasing concern for the health and survival of many species. At the level of the organism, effects encompass not only endocrine disruptions, changes in sex ratios, and decreases in reproductive parameters, but include genotoxic effects, immunosuppression, and the impairment of other immune components, which can lead directly to disease or increase the risk of developing disease. Living organisms attempt to maintain health by recognising and resolving problems such as the presence of microorganisms or foreign peptides, abnormal cell growth and DNA damage. Unfortunately, with the fast rate of environmental change, additional pressure on the maintenance of health and immunocompetence may seriously impact population viability and persistence. Here we outline the importance of a functional immune system for survival and examine the potential effects of the rapidly changing environment on immunocompetence. We then explore the different levels at which anthropogenic environmental change may impact wildlife health in a series of examples. These examples serve to demonstrate the complex and dynamic nature of the effects of environmental change on wildlife health, which can manifest itself at multiple levels. We also emphasize the necessity for an environmentally-explicit research paradigm to adequately study how the changes interact to fully understand the multitude of ways in which the rapidly changing environment is impacting wild animal health.

15.45–16.30 **Large scale population effects, genetics and environmental change**

*Nils Stenseth, Centre for Ecological and Evolutionary Synthesis (CEES), University of Oslo, Norway*

This contribution will provide a review of how climate affect both ecological and population genetic structuring in both marine and terrestrial systems. It will include a summary of how time series analysis of long-term monitoring data can be used for deducing the underlying population dynamics model, and how this subsequently can be used for asking population genetic questions (relating to geographic structuring). It will be argued that climate affect the ecological dynamics which subsequently influences the population genetic structuring.

16.30–16.45 **Closing remarks**

*Sir Brian Heap (Cambridge, UK)*

16.45 **End of Symposium**