



SCIENTIFIC MEETING

## ENVIRONMENTAL INFLUENCES ON DEVELOPMENT

Tuesday, 13 January 2009

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

**Chair: Professor Sir Patrick Bateson FRS, University of Cambridge & President, ZSL**

### **The different effects of early experience on development**

*Professor Sir Patrick Bateson FRS, University of Cambridge*

The disruption of normal development by a variety of environmental agents has been well known since the thalidomide scandal. More subtle environmental influences can arise when an organism is deprived of necessary resources; the organism will 'make the best of a bad job'. Organisms can also respond to environmental cues that enable them to direct their development of their bodies, metabolisms and behaviour towards what are likely to be adaptive responses to local conditions. This can go wrong if the environment does not turn out to be as was originally forecast by the cues that influenced the individual's development. Finally, a variety of different mechanisms, ranging from the simple to the highly complex, enable animals to produce learnt responses to the conditions in which they live.

### **Parental influences on offspring growth and survival in the seahorse *Hippocampus kuda***

*Professor William V. Holt, Institute of Zoology, ZSL*

The over-exploitation of seahorses is known to be causing global declines in abundance. Catch sizes reported by the five major seahorse fishing countries are 15–75% smaller than 10 years ago, but even so the global catch in 2002 was estimated to have been 24.5 million individuals. Anecdotal reports have suggested that the size of harvested individuals is decreasing year by year but this is difficult to substantiate. Here we present experimental evidence to support the possibility. In a planned seahorse breeding experiment the growth rates and postnatal mortality of offspring produced by couples that had only just reached maturity were significantly poorer than those of offspring produced by more mature adults. Growth rates during the first 6 weeks after birth were negatively correlated with the absolute number of offspring in broods, but this was only true for juveniles produced by the older, and larger, parents. Examination of the internal pouch structures in the older adults suggested that this effect might be attributable to the

considerable differences in size and depth of the implantation sites, with the larger and deeper implantation sites providing the developing embryos with superior metabolic support during pregnancy. Pouches of the smaller seahorses did not show these differences. These results support the possibility that if seahorse fishing depletes the stocks of larger individuals, the next generation might in turn show slower growth and greater mortality. Functional parallels between the nature of mammalian and seahorse pregnancies also suggest that the seahorse might be used as an interesting general model for studying mechanisms of developmental plasticity.

### **Developmental plasticity and risk of chronic disease**

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The heritable or familial components of susceptibility to chronic non-communicable diseases such as type 2 diabetes, obesity and cardiovascular disease are well established, but have until recently been thought to be largely genetic (on the basis of e.g. family linkage analysis, but see<sup>1</sup>) or environmental (in terms of shared lifestyle risk factors). There is now increasing evidence that some elements of such heritability are transmitted non-genomically and that the processes whereby environmental influences act during early development to shape disease risk in later life can have effects beyond a single generation<sup>2</sup>. Such heritability may operate through epigenetic mechanisms involving regulation of either imprinted or non-imprinted genes and involve changes in DNA methylation, histone structure and in miRNAs. These produce changes in the phenotype of the offspring<sup>3</sup>. Other processes relate to parental physiology or behaviour such as uteroplacental blood flow or suckling<sup>4,5</sup>. Common environmental factors can of course also play a role. The potential mechanisms for non-genomic transgenerational inheritance of 'lifestyle' disease suggest that the DOHaD phenomenon is a consequence of an ancestral mechanism of developmental plasticity, which may have had adaptive value in the evolution of generalist species such as *Homo sapiens*. However our current exposure to high energy and fat content foods, and low levels of physical activity leaves today's children and adolescents mismatched<sup>6</sup>. They have inappropriate responses to environmental challenges and risks of chronic disease such as obesity, cardiovascular disease and type 2 diabetes are increased. These in turn increase risk in the next generation. The effects are exacerbated by demographic changes, and those in reproduction, such as the tendency for women to have children at the extremes of their reproductive age and for more primiparous pregnancies. The effects will be very great in economic terms, particularly in countries undergoing socioeconomic transition. Because recent animal data reveal that the changes induced by dietary change or endocrine challenges in pregnancy can be passed to the grand-offspring (F<sub>2</sub>) without additional challenge in the F<sub>1</sub>, and affect blood pressure, endothelial dysfunction, HPA responses and left ventricular hypertrophy<sup>7-9</sup> the social consequences of DOHaD will be far-reaching. We have now shown that epigenetic changes are central to induction of these effects and to their transmission between generations<sup>10,11</sup>. Understanding these processes holds hope for future detection of those at risk and design of appropriate interventions<sup>12</sup>.

## References

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## **Early life experience and reproductive timing in humans**

*Dr Daniel Nettle, Newcastle University*

Where mortality rates are high, it is generally adaptive to begin reproductive effort sooner in life than when they are low. Across human populations, there are very clear relationships between mortality and fertility, with high-mortality regimes producing earlier, and often more, reproduction. These differences are also mirrored within the British population, where the groups living in the most dangerous environments reproduce half a decade or more earlier than others living in the same cities. It seems likely that early life experiences act as cues of the local ecological context and affect the schedule of sexual maturation and reproductive motivation. I present recent data on teenage pregnancy in British women, focussing on the predictive value of birthweight and early family circumstances.

### **Further reading**

Pesonen, A-K., K. Raikonen, K. Heinonen, E. Kajantie, T. Forsen & J.G. Eriksson (2008): Reproductive traits following a parent-child separation trauma during childhood: A natural experiment following World War II. *American Journal of Human Biology* **20**: 345–51.

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