



Keynotes

Keynote 1: Mervyn Singer, UK

Adaptation to critical illness – a conserved approach that is increasingly appreciated.

One hundred years ago Cannon described the fight and flight stress response, and bodily adaptations to fear, rage, pain and hunger. The theories of stress have been since extended to, among others, the General Adaptation Syndrome and the concept of allostasis and allostatic overload. Here, the body can protect itself from excess stress by inducing a state of metabolic shutdown that recovers once the stressful situation has abated. This response is preserved through nature, up to and including man during critical illness. Sepsis represents a dysregulated host response to infection resulting in organ dysfunction. Yet these functionally failing organs have a (usually) preserved architecture and a remarkable ability to recover performance if the patient survives. Paradoxically, many established therapies, such as the use of catecholamines and antipyretics, run counter to this adaptive response. Even the use of oxygen is being re-examined in the light of this increased awareness of iatrogenic harm.

Keynote 2: Jonathan Wells, UK

Maternal capital and the inter-generational transmission of health and disease

There is now compelling evidence that multiple aspects of human phenotype are strongly imprinted by experience in early life - the “developmental origins of health and disease” hypothesis. Most of this evidence relates to pregnancy and early infancy, meaning that the primary environmental influences on offspring derive from the phenotype of the mother. Consequently, maternal phenotype is a key factor shaping health and disease susceptibility in the next generation. This talk will discuss how both physical and social components of maternal phenotype can profoundly impact the health and development of the next generation.

Keynote 3: Sylvia Cremer, Austria

Social immunity: protecting the colony against disease

Social insect colonies fight disease as a collective: in addition to their individual immune systems, they have evolved sophisticated disease defences that they perform together to achieve social immunity. As a first line of defence, they perform nest hygiene and mutual sanitary care to prevent

pathogen uptake and establishment of infections in the colony. If these defences fail and a colony member develops an infection, a second line of social immune defences is employed to prevent disease spread within the colony, which can involve the elimination of infected colony members. This care-kill dichotomy arises as selection acts at the level of the colony rather than the individual. Social immunity in insect colonies therefore parallels organismal immunity, where infected cells are sacrificed to protect the remaining body.

I will give an overview of the behavioural repertoire of ant colonies, both before and after infection. First, I will discuss how ants use antimicrobials for self-medication and the treatment of colony members, showing that these measures are highly effective in reducing the probability of infection and that they can even completely break pathogen lifecycles. Secondly, our research has shown that close contact between ants during sanitary care can actually promote the transmission of low levels of infectious propagules through the colony. Rather than causing disease, I show that this transmission induces a protective immune stimulation that leads to the social immunisation of many colony members. Colony-level protection against infectious diseases therefore relies on a tight interaction between individual and social immunity.

Keynote 4: Francisco Ubeda, UK

The evolution of sex-specific virulence in infectious diseases

Fatality rates of infectious diseases are often higher in men than women. Although this difference is often attributed to a stronger immune response in women, we show that differences in the transmission routes that the sexes provide can result in evolution favouring pathogens with sex-specific virulence. Because women can transmit pathogens during pregnancy, birth or breast-feeding, pathogens adapt, evolving lower virulence in women. This can resolve the long-standing puzzle on progression from Human T-cell Lymphotropic Virus Type 1 (HTLV-1) infection to lethal Adult T-cell Leukaemia (ATL); a progression that is more likely in Japanese men than women, while it is equally likely in Caribbean women and men. We argue that breastfeeding, being more prolonged in Japan than in the Caribbean, may have driven the difference in virulence between the two populations. Our finding signifies the importance of investigating the differences in genetic expression profile of pathogens in males and females.

Keynote 5: Peer Bork, Germany

(Microbiome)

Keynote 6: Linda Partridge, Germany

Evolutionary biology and healthy ageing

Ageing results in increased likelihood of death and loss of fecundity, and is hence an individually maladaptive trait. Ageing is not inevitable, since some organisms seem not to age at all, yet it is routinely seen in nature and occurs at very different rates in different kinds of organisms. This evolutionary paradox is largely explained by the decreasing power of natural selection to determine the fate of mutations that exert phenotypic effects at later stages in the survival schedule determined by external hazards. As far as we know, no genetic variants have been selected to cause somatic damage, infertility, pathology and death, and ageing evolves as a side-effect of mutations with deleterious effects at late ages (mutation accumulation) or that are advantageous in youth (pleiotropy). Captive animals and humans are largely protected from the external hazards

encountered in nature, and hence show marked effects of ageing, although whether these are attributable to mutation accumulation or pleiotropy remains unclear. Ageing nonetheless shows some evolutionarily conserved hallmarks, and both genetic and environmental interventions have proved capable of slowing the process in laboratory animals, raising the prospect of a broad-spectrum, preventative medicine for the diseases of ageing. The mechanisms leading to natural diversity in rate of ageing remain largely unknown and unexploited to improve human health.

Keynote 7: Svante Pääbo, Germany

A Neandertal Perspective on the Human Genome

Over 30 years, we have worked on the development of methods to study the tiny amounts of DNA that can sometimes be found in ancient remains of extinct organisms. In particular, we have focused on reconstructing the genome of Neandertals, the closest extinct relatives of present-day humans. Analyses of these genomes show that Neandertals contributed about 2.0% of the genomes of people today living outside Africa while Denisovans, a hitherto unknown group of Asian hominins distantly related to Neandertals, contributed about 4.8% of the genomes of people living in Oceania as well as small amounts to people elsewhere in Asia. Together, these findings suggest a 'leaky replacement' scenario of human origins in which anatomically modern humans emerged out of Africa and received some degree of gene flow from archaic human populations. Work from several laboratories has shown that these genetic contributions have consequences today for the immune system, for lipid metabolism, for adaptation to life at high altitudes in the Himalayas, and for susceptibility for diseases such as diabetes.

The Neandertal and Denisova genomes also allow the identification of novel genomic features that appeared in modern humans since their divergence from a common ancestor shared with Neandertals. A future challenge is to identify the subset of these features that contributed to that modern humans developed complex culture, technology and art.

Keynote 8: Stephen Stearns, USA

The relations between evolution and medicine, journals and societies

Evolutionary biology and evolutionary medicine share ideas but emphasize them differently. Ideas very important in evolutionary medicine – such as mismatch, clonal evolution, and evolution-proof interventions – are either given other names, play lesser roles, or are mostly ignored in evolutionary biology. There is opportunity for bright young evolutionary biologists to make foundational contributions to evolutionary medicine. The timing is right, and the need is great. Examples will be discussed.

Societies and journals also have complex relations. Founding a society with a journal is easier when there is pent-up demand: initial growth is rapid, and financial security is more easily attained. When the idea for a society with a journal resides with a small number of visionaries, growth is slower, no matter how solid the long-range vision. Moreover, the direction taken by a journal is determined in important part by a process of self-assembly. Paleontologists did not get involved with JEB; mainstream immunobiologists, oncologists, and infectious disease specialists have not yet contributed heavily to EMPH. The reasons for that will be analyzed.

Scientists also have diverse talents: some for research, some for teaching, and some for building the infrastructure that supports the entire community. The rewards – and costs – of those activities differ in how rapidly they are delivered and how intimately they are felt. Since all are important, the

key for an individual choosing among them is to find the happy intersection of what one is good at, what one enjoys, and what one will get paid for – not always easy.