Evolutionary Approaches to Disease and Health

Darwin Room, Brunel University, 19th March 2010

Part of the ESRC Darwin's Medicine Seminar Series
Funding provided by the ESRC and the Brunel University School of Social Sciences
Affiliated with the Brunel Centre for Culture and Evolutionary Psychology

Programme

Speaker presentations should last about 30 minutes, with the remaining 10 minutes left for audience questions; for the keynote address, this split should be 45/15 minutes

9.00-9.50	Registration
9.50-10.00	Opening remarks by conference organiser Michael Price
10.00-10.40	Martin Brüne (Ruhr-Universität Bochum): "Understanding schizophrenia in evolutionary perspective: "Disease" versus symptom-based approach"
10.40-11.10	Tea
11.10-11.50	Rick Maizels (University of Edinburgh): "Host-parasite co-evolution - to whose advantage?"
11.50-12.30	Gillian Bentley (Durham University): "Reproductive pathologies: Perspectives from evolutionary medicine"
12.30-1.30	Lunch (catered on location)
1.30-2.10	William Hanage (Imperial College): "Infectious disease evolution: It's Darwin, but not quite as we know him"
2.10-2.50	Paul Gilbert (University of Derby): "An evolutionary approach to depression: The role of social rank and compassion"
2.50-3.20	Tea
3.20-4.00	Mervyn Singer (University College London): "Coping with critical illness... and the maladaptive forces of modern medicine"
4.00-5.00	Keynote address by Randolph Nesse (University of Michigan): "Medicine without evolution is like engineering without physics"
5.00-5.30	Concluding panel discussion (all speakers): The future of evolutionary approaches to medical research and practice

Abstracts

Martin Brüne: Understanding schizophrenia in evolutionary perspective: "Disease" versus symptom-based approach

The term "schizophrenia" refers to a group of disorders that seem to occur at similar prevalence rates of around 1% in every human culture. The persistence of schizophrenia in human societies can be called an "evolutionary paradox", because patients with schizophrenia are reproductively disadvantaged compared to the general population. Some researchers have therefore proposed that a compensatory advantage must exist in people who are heterozygous for genes that otherwise cause vulnerability to psychosis. This endeavour to detect genes with major effect sizes has, however, been unsuccessful, probably owing to the diversity of schizophrenia phenotypes. Does that mean that evolutionary insights are futile with regard to schizophrenia? In this talk I will argue that, instead of exploring a complex phenotype as if it were a "disease entity", examining individual symptoms or symptom constellations is fruitful, and that evolutionary thinking can be useful in guiding the generation of testable hypotheses. This shall be addressed with regard to 3 levels: social behaviour, social cognition, and social neurons that subserve the execution of the former. This approach largely draws on the "social brain" hypothesis, which suggests that primates including humans have evolved mechanisms that were selected to process social stimuli to maximise an individual's inclusive fitness. Schizophrenia symptoms, in this view, can be seen as the extremes of variation of selected traits – traits that increase inclusive fitness, but become maladaptive if expressed outside normal variation. Examples given include delusional ideation, impaired perspective-taking, mirror neuron activity and reductions of von Economo neurons.

Rick Maizels: Host-parasite co-evolution - to whose advantage?

The vertebrate immune system has evolved in close and constant association with infectious organisms, which form a continuum from harmless commensals to dangerous pathogens. Midway in this spectrum are the helminth parasites, multicellular nematode, trematode and cestode worms which occupy a predominantly extracellular niche in the gut, vasculature or other tissues of the host. These parasites can establish themselves for many months or years, with no untoward effects in a large proportion of the population, reflecting in part the ability of these parasites to down-modulate immune reactivity. After long evolutionary time in which most individuals would have harbored heminth parasites for most of their lives, the last century has seen the effective elimination of these infections from many developed countries, primarily through improved sanitation and housing. In this time of socio-economic transformation, we have also experienced a sharp intensification of immunopathological diseases (autoimmunity and allergy). The idea that our immune system, tuned to optimal fitness in the presence of parasites, may overshoot in their absence, helps underpin the "Hygiene Hypothesis" that infections can protect from allergies and related maladies. Indeed, evidence from helminth-endemic countries, and from laboratory model systems, supports the proposition that helminth parasites condition their hosts in a manner which minimises allergic reactivity. However, different helminth species, and different intensities of infection, exert very different pressures on the host immune system. Perhaps reflecting this, helminth infections may have resulted in immune gene diversification, rather than fixation, giving rise to the modern-day alleles linked to susceptibility to autoimmunity and allergy. Hence, we could now be witnessing the downside to an otherwise beneficial host-parasite co-evolutionary process.

Gillian Bentley: Reproductive pathologies: Perspectives from evolutionary medicine

One of the key concepts in Evolutionary Medicine proposed by Nesse and Williams in their seminal article, The Dawn of Darwinian Medicine, is that certain pathologies arise from a "mismatch" between our current and evolutionary environments. This term has been extended to the study of developmental environments and how a mismatch can arise between the environment experienced during growth and that encountered later in life. Different developmental environments contribute to the degree of plasticity of many traits observable within a species (including humans), referred to as a "reaction norm" in biological parlance. This, in turn, translates into individual and often observable "phenotypic variability" – the blend of genetic and environmental influences that shape individuals. Many of these concepts are at variance with clinical perspectives which necessarily centre on normative functions. In the contemporary world, the processes of modernization, urbanization, industrialization and many other ". . .izations" have led to an increased occurrence of environmental mismatches, and a range of pathologies creating medical nightmares with huge fiscal costs for health care providers. Metabolic disorders such as obesity and type 2 diabetes are two well studied examples. Less well studied from the perspective of evolutionary medicine (barring breast cancer) are a range of reproductive disorders that can arise from specific environmental mismatches, exacerbated in some cases by other environmentally-induced conditions. This talk will present case studies of some of these conditions with consequences for health in later life, such as changes in reproductive hormone levels, focusing on the speaker's own work with migrant Bangladeshis in the UK. The talk will also cover how public health and medicine can be geared to tackle these problems.

William Hanage: Infectious disease evolution: It's Darwin, but not quite as we know him

Infectious diseases are among the most significant selective pressures in nature. The evolutionary arms race between host and parasite is often held up as an example of evolution in action. The evolution of parasites shows a number of differences from 'higher' organisms, including enormous population sizes, frequently high mutation rates, and the ability to import evolutionary innovation (like drug resistance) in the form of genes from distantly related organisms. While the fundamentals of Darwin's theory remain unchanged, we will discuss how they influence pathogen populations and the evolutionary roots of virulence, and how we can exploit them to track diseases and estimate epidemiological parameters using DNA sequence.

Paul Gilbert: An evolutionary approach to depression: The role of social rank and compassion

Depression-like behaviours are recognised throughout the animal kingdom. This suggests that the capacity to lower negative affect and drive and increase threat based affects has adaptive value. This talk will argue that evolutionary approaches can help identify the natural regulators of mood and emotion, (e.g. major defeats and attachment losses). It will also suggest that therapies can be directed towards activating 'anti-depressant mechanisms' that are linked to experiences of social affiliation and compassion. Special attention will be given to the way in which humans can activate these mechanisms through their own styles of thinking and ruminating.

Mervyn Singer: Coping with critical illness... and the maladaptive forces of modern medicine

For thousands of years man has been confronted by four major external stressors – infection, trauma, starvation and temperature extremes. We have evolved to adapt and thus cope with these insults through modifications in both genotype and phenotype, allowing the stronger individuals to survive and procreate. Rapid advances in general health and nutrition, and in medical technologies, have however upset this equilibrium as we are living far longer than nature probably intended. Furthermore, we can also maintain an artificial and unnatural existence for patients during periods of critical illness through mechanical and drug supports that may last weeks, if not months. While certainly saving some lives, it has become increasingly apparent that our attempts to modify physiological and biochemical variables to 'normal' values may, in fact, be deleterious. In addition, many of our therapies are probably counter-adaptive. For example, catecholamines are used to increase blood pressure in shock states yet, at the same time, these increase exogenous stress on an already intrinsically stressed individual with further potential for harm. Patients may thus survive in spite rather than because of our best efforts. Indeed, modern medicine has overlooked impressive survival rates from injuries sustained during major battles before the advent of 'life-saving' techniques such as blood transfusion, antibiotics and sophisticated surgery. We need to reappraise how we treat critical illness, with greater awareness of how to intervene in line with natural adaptive processes.

Randolph Nesse: Medicine without evolution is like engineering without physics

It is perfectly possible to practice medicine without understanding evolutionary biology; millions of doctors do it every day. Learning evolutionary biology would, however, make many physicians more effective, and more satisfied with their work. While evolution offers some direct applications in medicine, its more powerful utility is the same as what physics offers for engineering—a foundation in basic principles that explain why things are the way they are. Larger investments in evolution education for physicians pay off in three ways. First, researchers who already use some evolution find greater power as they have opportunities to learn the details; infectious disease and genetics offer good examples. Second, new answers come from asking new evolutionary questions about why the body is the way it is; studies of gout, bilirubin, sex differences in mortality and depression are good examples. Third, and perhaps most important, is replacing the outmoded metaphor of the body as a machine with a more biological model of the body as a bundle of tradeoffs shaped by natural selection to maximize Darwinian fitness. This change in perspective is as fundamental for medicine as atomic theory is for engineers. Medicine can be practiced without evolution, and engineering without physics, but only at a great loss in depth of understanding.
