Evolutionary Psychiatry (EPSiG)

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Notes from the editor

This is our 10th EPSiG newsletter and we are in our third year of existence as a SIG.

We are looking at plans for the future. All those interested are welcome to attend or scientific meeting and AGM at the College on May 18th. There will also be presentations including Dr Nikhil

Chaudhary, a Research Associate at University College London Human Evolutionary Ecology Group and a presentation on the evolutionary aspects of Alzheimer's and ageing. Places can be booked free of charge by emailing: jonathan.bennett@rcpsych.ac.uk . However, places are limited and will be allocated on a first come first served basis.

We have feedback from Dominic Sohotra-fuge who brought his A-level students to our last symposium. This was very exciting for us as we did not realise we were even remotely reaching such a valuable target audience.

Here is his feedback:-

Nearing a decade since Nesse *et al.* (2010) wrote about the new applications of evolutionary biology in medicine, as mentioned during the symposium, there is still a significant lack of emphasis on Darwinian evolution during medical and subsequent psychiatric training. Unsurprisingly, the same can be said for A-Level and school curricula, in subjects where a focus on evolutionary theory should be essential – such as in Biology or Psychology. It is fantastic to have events organised by groups such as the Evolutionary Psychiatry Special Interest Group (EPSIG), to bridge this unfortunate gap in syllabus-led student knowledge, who not only allow admittance to practicing psychiatrists, academics and interested professionals but also to A-Level students who are considering moving on from school to potentially read medicine.

Not only was attending the symposium a valuable experience within itself – rubbing shoulders with doctors and being exposed to university-style lectures – but the symposium opened the minds of the students to the usefulness of evolutionary psychiatry and how this rather young field of academic discipline can enhance our understanding of psychopathology. With the EPSIG's aim to "…encourage evolutionary inspired research, help produce and distribute teaching material on evolutionary principles, help advocate for the inclusion of evolution into the undergraduate medical curriculum in the UK and elsewhere…" (Abed and St John-Smith, 2016), perhaps engaging with A-Level students before the next step of university can be one of the many useful avenues in which to disseminate evolutionary principles.

For instance, one student mentioned how she was surprised that "Darwin's theory can have such an impact on medical application and our understanding of the brain". As I have seen from being a teacher of Psychology and one of the school's supervisors for the Extended Project Qualification (EPQ), one of my supervisees, who attended the symposium, has already decided to focus her research project on the evolutionary origins of human behaviour. Thus, the consequences of exposing young people to principles within Darwinian evolution – before they start their academic careers – have the potential to be far-reaching. As a school, we would like to thank the EPSIG for this unique and valuable

opportunity and would very much like to keep in touch. Extended thanks also to those who met with the students and took the time to answers some of their questions and gave them an insight into the medical profession. Contact: dominic.sohotra-fuge@burlingtondanes.org

Future meeting dates :

Half-Day Scientific Meeting and AGM, RCPsych, London 18 May, 2018, 1-5pm

https://i.emlfiles4.com/cmpdoc/9/4/5/6/7/1/files/23861_flyer-for-half-day-scientific-meeting-may-18.pdf?dm_i=3S85,D4RE,2GOCN2,1DM5D,1

EPSIG 3rd Symposium will take place on 22 March, 2019 at the RCPsych.

Advice to potential authors

We have had a number of people write in to our newsletter with articles whereby the author attempts to refute or demonstrate the error of evolutionary thinking about psychiatric illnesses. They are generally labouring under one or more misunderstandings that evolutionary psychiatry ALWAYS maintains that Psychiatric disorders are adaptive. We highly recommend that any authors consult the actual existing Evolutionary psychiatry literature first and show an actual understanding and knowledge of the field before approaching us with this very basic type of error. The argument goes that (name illness) decreases survival and reproduction therefore cannot have an explanation with an evolutionary component.

They also assume evolutionary psychiatry explanations are given as total explanations i.e. complete alternatives to proximate causes and that Evolutionary psychiatrists are trying to replace immediate models of understanding of disease pathophysiology. This shows a lack of familiarity with the basic Tinbergen framework and what we are trying to do!

We do not intend to publish articles containing these basic fallacies, errors and showing a poor grasp of the evolutionary literature in our newsletter.-

Putting false premise and straw man arguments and articles in the newsletter then demolishing them could look like mockery. We do try to explain these issues politely to such contributors. There is, of course, legitimate room for debate in this field but it must be about the field, not articles demonstrating a lack of knowledge of the field, making false assumptions and getting the basic premises wrong.

Here is my straw man example:-
Next time you have a patient with a backache ask yourself, whether the backache can have a partial evolutionary explanation. (The general evolutionary one about bipedality comes to mind), Start with the (false) premise that evolutionists claim backache must have some adaptive value. Thereupon ask:-
1) How is backache good for you?
2) How is is it adaptive?
3) does it increase the chances of reproduction or survival?
If you can demonstrate backache is an illness or disability and therefore disadvantageous, you can subsequently conclude there can be no evolutionary explanation for any back problems.
Similarly our immune system has evolved but autoimmune disease is not considered by evolutionists as adaptive.

Looking at depression/low mood as an EP example, an evolutionary perspective asks a fundamental question: Why has natural selection left all humans so vulnerable to depression? Evolutionary science helps resolve these issues as it recognises two categories of causation, the proximate,

(mechanism and ontogeny) and the ultimate or evolutionary (phylogenetic and function). Evolutionary concepts help make a sharp distinction between defects, disorders and protective responses such as defences (Nesse,). Evolution/nature has selected adaptations (defence mechanisms) that help protect against injuries and infections.

These include: - 1. Pain, sickness, illness behaviour 2. Anxiety, Depression, OCD, 3. Fever, Lethargy, Fatigue 4. Nausea, 5. Itching, 6. Expulsions: - Sneezing, Vomiting, Coughing.

General evolutionary explanations are not alternatives to proximate causes of depression.
General evolutionary mechanisms include problems caused by
1. Mismatch: exposure to evolutionarily mismatched or novel environment
2. Life History factors
3. Excessive defence mechanisms
4. Co-evolutionary considerations: losing the arms race against pathogens
5. Constraints imposed by evolutionary history
6. Sexual Selection and its consequences
7. Balancing selection: maintaining an allele that raises disease risk
8. Demographic history and its consequences
9. Selection favours reproductive success at the expense of health

There are some adaptationist aspects to depression however. Ethological perspectives emphasise the adaptive function of behaviour. Depressive behaviours, withdrawal, reduced activity, reduced appetite, reduced motivation, frowning, downcast gaze etc. occur in many higher mammals.

These behaviours have adaptive consequences (Nesse, 2000): Depressive symptoms may elicit caring from others. After loss of an attachment figure, depression inhibits searching and protest, when it has become futile. In situations of social conflict the depressive state operates as an unconscious, involuntary losing strategy, enabling the individual to accept defeat and accommodate to what would otherwise be unacceptably low social rank (Price et al, 1994). A theory of the functions of depression, helps to distinguish between those depressions which are adaptive, i.e. the depression is fulfilling a function and may be considered in some way adaptive, and those situations where it has become dysfunctional, and is threatening the long term survival of the individual (Wakefield, 2007). This has implications for therapy.

A paper by Rantala et al does considerable justice to the critical appraisal of these various ideas and models (discussed below).

Finally, sometime when we get a chance we will write an article about the common misconceptions in detail! In the meantime the following extract is taken from David M. Buss' book Evolutionary Psychology: The New Science of the Mind.

MISCONCEPTION 1: CURRENT MECHANISMS ARE OPTIMALLY DESIGNED

The concept of adaptation, the notion that mechanisms have evolved functions, has led to many outstanding discoveries over the past century. This does not mean, however, that the current collection of adaptive mechanisms that make up humans is in any way "optimally designed." An engineer might cringe at some of the ways that our mechanisms are structured, which sometimes appear to be assembled with a piece here and a bit there. In fact, many factors cause the existing design of our adaptations to be far from optimal. Let's consider two of them. One constraint on optimal design is evolutionary time lags. Recall that evolution refers to change over time. Each

change in the environment brings new selection pressures. Because evolutionary change occurs slowly, requiring hundreds or thousands of generations of recurrent selection pressure, existing humans are necessarily designed for the previous environments of which they are a product. Stated differently, we carry around a Stone Age brain in a modern environment. In other words, "we are walking archives of ancestral wisdom" (Cronin, 1991).

A strong taste preference for fat and sugar, adaptive in a past environment of scarce food resources, now leads to clogged arteries, Type 2 diabetes, and heart attacks. The lag in time between the environment that fashioned our mechanisms (the hunter-gatherer past that formed much of our selective environment) and today's environment means that our some of our existing evolved mechanisms may not be optimally designed for the current environment.

A second constraint on optimal design pertains to the costs of adaptations. Consider as an analogy the risk of being killed while driving a car. In principle, we could reduce this risk to near zero if we imposed a five-mile-per-hour speed limit and forced everyone to drive in armoured trucks with ten feet of padding on the inside. But we consider the costs of this solution to be ridiculously high.

Similarly, we might consider a hypothetical example in which natural selection built into humans such a severe terror of snakes and spiders that people never ventured outdoors. Such a fear would surely reduce the incidence of snake and spider bites, but it would carry a prohibitively high cost. Further, it would prevent people from solving other adaptive problems, such as gathering fruits, plants, and other food resources necessary for survival. In short, the existing fears of snakes and spiders that characterize humans are not optimally designed—after all, thousands of people do get bitten by snakes every year, and some die as a result. But it works reasonably well, on average.

All adaptations carry costs. Selection favours a mechanism when its benefits outweigh the costs relative to other designs existent at the time. Humans have evolved mechanisms that are reasonably good at solving adaptive problems efficiently, but they are not designed as optimally as they might be if costs were not a constraint. Evolutionary time lags and the costs of adaptations are just two of the many reasons why adaptations are not optimally designed.

MISCONCEPTION 2. HUMAN BEHAVIOUR IS GENETICALLY DETERMINED

Genetic determinism is the doctrine that argues that behavior is controlled exclusively by genes, with little or no role for environmental influence. Much of the resistance to applying evolutionary theory to the understanding of human behavior stems from the misconception that evolutionary theory implies genetic determinism. Contrary to this misunderstanding, evolutionary theory represents a truly interactionist framework. Human behavior cannot occur without two ingredients: (1) evolved adaptations and (2) environmental input that triggers the development and activation of these adaptations.

Consider calluses as an illustration. Calluses cannot occur without an evolved callus-producing adaptation, combined with the environmental influence of repeated friction to the skin. Therefore to invoke evolutionary theory as an explanation for calluses, we would never say "calluses are genetically determined and occur regardless of input from the environment." Instead, calluses are the result of a specific form of interaction between an environmental input (repeated friction to the skin) and an adaptation that is sensitive to repeated friction and contains instructions to grow extra new skin cells when the skin experiences repeated friction. Indeed, the reason that adaptations evolve is that they afford organisms tools to grapple with the problems posed by the environment. So notions of genetic determinism—behaviours caused by genes without input or influence from the

environment—are simply false. They are in no way implied by the evolutionary theory or by evolutionary psychology.

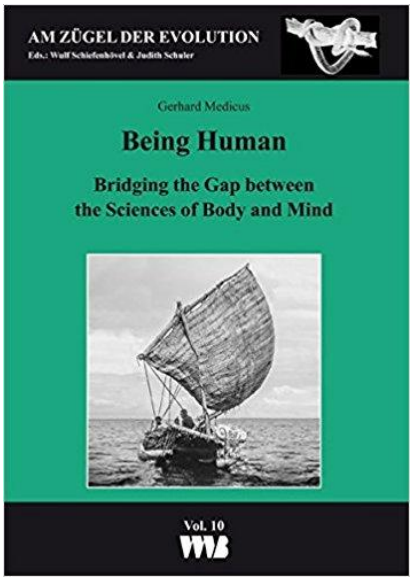
MISCONCEPTION 3: IF IT'S EVOLUTIONARY, WE CANNOT CHANGE IT

Another misunderstanding is that evolutionary theory implies that human behavior is impervious to change. Consider the simple example of calluses again. Humans can and do create physical environments that are relatively free of friction. These friction-free environments mean that we have designed change—a change that prevents the activation of the underlying callus-producing mechanisms. Knowledge of these mechanisms and the environmental input that triggers their activation give us the power to decrease callus production. In a similar manner, knowledge of our evolved social psychological adaptations along with the social inputs that activate them gives us power to alter social behavior, if that is the desired goal.

Knowledge about our evolved psychological adaptations along with the social inputs that they were designed to be responsive to, far from dooming us to an unchangeable fate, can have the liberating effect of paving the way for changing behavior in areas in which change is desired. This does not mean that changing behavior is simple or easy. More knowledge about our evolved psychology, however, gives us more power to change.

New Books. Being Human: Bridging the Gap between the Sciences of Body and Mind Hardcover – 2015 by Gerhard Medicus

I was privileged that I was given an authors' copy of this excellent compendium of ideas and lecture notes How are body and mind connected, and how are the sciences of the body connected with the sciences of the mind? The book carefully considers how evolution has left its traces on both the body and the soul. For this reason, Medicus argues that accumulated evolutionary knowledge is a useful and indispensable underpinning for a better understanding of humans: social behaviour, moral consciousness, aggression and the inhibition of aggression, attachment behaviour, learning and intellect, political judgment and activity, as well as behavioural differences due to gender. He maintains the interlacing of nature, culture and mind is visible in all realms of humanness/human nature/humanity and that this knowledge can help to expand our behavioural freedom, and with that, our freedom to act responsibly.



Response to the first English edition: "Medicus has shown us both breadth and depth in his far-reaching synthesis of the physical and mental aspects of our humanity. Using classical ethology as a starting point, he then ranges widely in the natural and social sciences, especially Psychology, and beyond, for example, Philosophy. Few scholars are in a position to provide us with such a satisfying compendium on human nature." William McGrew, University of Cambridge, UK, 2015. *The current book is a well done English translation of the* German version published in 2012. It translates lots of material published over the past century and previously available only in German. This book is not "pop" science book nor easy or light reading for the non-specialist. It is definitely not a trade book. However, for those persons willing to put in the effort, the material covered in the book rests on a much firmer empirical foundation (i.e., what things are) than many other such publications.

The book's twelve chapters are divided into three main Parts. There is an adequate Bibliography, Subject and Author Index. Part I contributions to the epistemology of interdisciplinary in the human sciences. Part II is: Behavioural phylogeny in relation to the higher-level systematics of vertebrates. Part III is: Contributions to the ethology of specific behavioural areas and capacities, with an emphasis on universal traits in higher mammals and unique traits in apes and humans.

The two chapters in Part I address Medicus' own contributions to bridging the gap and creating dialogue between the sciences and the humanities. The six chapters in Part II explore aspects of human behaviour from a perspective that probably will be new information for many readers: phylogeny, or the behaviour's evolutionary history. Topics addressed include human cognition, phylogenetic and ontogenetic (within the lifetime of the individual) knowledge gain, social behaviour, morality, resource acquisition and ownership, gender differences and then whether our ontogenetic behavioural development recapitulates our behavioural phylogeny, which has potential applicability to the roles of genes and cultural learning. Part III addresses more general issues from the same cross-disciplinary, biological and anthropological perspective. Topics covered are an evolutionary and epistemological critique [and deconstruction] of "constructivism" (i.e., the world is merely constructed in our minds without reference to the external world), attachment, aggression and lastly, the biopsychology of political behavior. Of particular interest is the in depth discussion of "verisimilitude" (the appearance of being true), which has many applications, such as how religious beliefs function in religions.

Gerhard Medicus' book, a reflection of his lifelong work as a psychiatrist and human ethologist, is a good example of such approaches, of triangulations which have their point of reference outside but whose effects unfold inside the disciplines . His book invites us to: "Try this bridge."

Finally, we are delighted that Dr Medicus has agreed to speak at our 3rd Evolutionary Psychiatry Symposium in March 2019.

<u>An interesting paper</u> P ST J Smith ed.

Rantala, M.J., Luoto, S., Krams, I., Karlsson, H.,

Depression subtyping based on evolutionary psychiatry: proximate mechanisms and ultimate functions. Brain, Behavior, and Immunity (2017)

In a really helpful contribution to contemporary evolutionary psychiatry, Rantala et al have produced an evolutionarily informed classification or model, for depression. Depression is often purely seen by many biological psychiatrists as a disease even a unitary process, especially those that stick to a strong biomedical model. This is conversely vehemently disputed by those who take social or psychological perspectives. This is a false dichotomy. This disparate state of affairs also results in heated arguments about the failures of antidepressants, their side effects, and the problems of meta-analysis of antidepressants and the consequent whole antipsychiatry stance on depression and myth of the chemical cure etc.etc ad nauseam.

As evolutionarily informed psychiatrists, we have generally considered that depression treatments should be made more sophisticated by identifying which subtype the depressive episode belongs to. If it appears to be a response to an adverse life event, it should be evaluated as to whether the symptoms are adaptive or whether the depressive episode has exacerbated into pathological depression, coupled with features of sickness behaviour. Symptomatic as well as cause altering or curative regimens can be seen within this context

Rantala considers that major depressive disorder is not a unitary disease; it is a heterogeneous syndrome, with patients differing remarkably in symptom profile, pathophysiology and treatment responsiveness. Previous attempts to subtype major depressive disorder have shown limited clinical applicability. Rantala et al. thus present a classification of major depressive disorder episodes based on the proximate mechanisms that led to the original mood change that caused the depressive episode.

They identify discrete depression subtypes that are induced by: 1) infection, 2) long-term stress, 3) loneliness, 4) traumatic experience, 5) hierarchy conflict, 6) grief, 7) romantic rejection, 8) postpartum events, 9) the season, 10) chemicals, 11) somatic diseases and (12) starvation.

The authors further examine the ultimate functions of these subtypes and show that not all types of mood changes that trigger depression are adaptive. Instead, some are clearly maladaptive and some are by-products of other adaptations. In modern societies, low mood after adverse life events may turn into a pathological depressive state.

Subtyping depression enables an effective and intelligent long-term treatment of patients in each subtype by treating the underlying causes of depression. They consider the main reason for the failure of evolutionary explanations to provide an adequate theoretical framework for depression is that most of the authors who have suggested or criticized evolutionary explanations for depression have seen it as a single disorder. Likewise, major depressive disorder is routinely diagnosed based on the number of reported symptoms and a threshold score of their sum-score. This diagnostic tool in itself is based on the assumption that depression is a single condition and that all symptoms are interchangeable and equally good indicators of one underlying disorder.

Evolutionary explanations for a trait or a behaviour focus on two different levels:

1) What is the proximate mechanism underlying the trait: how does it work?—and

2) what is the ultimate reason it evolved: what fitness benefit, if any, does the trait provide for the organism?

In their article they suggest a novel evolutionary subtyping of depression based on proximate mechanisms and ultimate functions underlying specific depression episodes. Because of the novelty of the model presented in this article, it is obvious that at this stage there are no studies systematically testing whether depression subtypes based on evolutionary psychiatry differ from each other in treatment responsiveness with regard to commonly used interventions (antidepressant or psychotherapy). It is expected that the 12 depression subtypes vary in treatment responsiveness due to stress hormone and immune function differences in each subtype.

Instead, the evaluation should be based on whether the intervention helped the individual to cope with the adverse life event which caused the lowered mood. Thus, the focus of a treatment regime based on evolutionary psychiatry focuses on an individual's long-term mental and physical well-being instead of myopically fixating on the short-term alleviation of symptoms. We hope that the present subtyping based on an evolutionary and immunological approach to depression will prove its practical utility, helping to develop more effective therapeutic treatments and drugs that are targeted to the specific areas of and subtypes of depression identified here.

We are delighted that Dr Rantala has also agreed to speak at our 3rd EP Symposium in March 2019.

For those who wish to compare this paper to existing models I suggest reading the following introduction to models of depression using an evolutionary basis. This is a synopsis of the extensive composite pages of a Wikipedia entry :-
https://en.wikipedia.org/wiki/Evolutionary_approaches_to_depression

Evolutionary models of depression use the theory of evolution as a starting point to illuminate the problems (pathogenesis, symptoms, prevalence etc) of mood disorders. In psychiatry, depression is thought of as disorder or dysfunction, but there is a problem because as a symptom, it is often considered a cultural universal (although there are clearly cultural variants and influences) in some form and can occur potentially in everybody. Depression is also much more common than other mental conditions except anxiety, such as schizophrenia, or autism, which only have prevalence rates about one tenth that of depression, or less. Also the prevalence of clinical depression as a disorder does not increase with age the way dementia and other organic dysfunction commonly does. Physical or organ dysfunctions usually increase with age, with low rates in adolescents and young adults, and the highest rates in the elderly, consistent with theories about selection and ageing, which posit that selection against dysfunctional traits decreases with age especially post reproductive age.

In contrast to these patterns, prevalence of clinical depression is high in all age categories, including otherwise healthy adolescents and young adults. The common occurrence and persistence of a trait like clinical depression with such negative effects early in life is difficult to explain, unless it is or was somehow adaptive? Rates of infectious disease are high in young people, of course, but clinical depression is not thought to be just caused by an infection. The following hypotheses attempt to identify a benefit of depression that outweighs its obvious costs. Such hypotheses are not necessarily incompatible with one another and may explain different aspects, causes, and symptoms of depression.

1) Psychic pain hypothesis
2) Behavioural shutdown model
3) Analytical rumination hypothesis
4) Possibilities of depression as a dysregulated adaptation
5) Rank theory
6) Social risk hypothesis
7) Honest signaling theory
8) Bargaining theory
9) Social navigation or niche change theory
10) Prevention of infection

Resources and EPSIG Website

The link to the EPSIG web pages that contain a range of resources is below:

http://www.rcpsych.ac.uk/workinpsychiatry/specialinterestgroups/evolutionarypsychiatry.aspx

Articles for the newsletter

We welcome submissions for future newsletters in the form of articles, reviews and interviews. Please send to me at

paulstjohnsmith@hotmail.com

Correspondence: Replies, suggestions and clarifications on articles are welcomed and may be printed/included in our next newsletter.

Also, we welcome brief reviews of seminal articles where there is an evolutionary or other relevant conceptual angle (please include the weblink if the article is open access).

Please send any submissions to me at: - paul.stjohnsmith@hpft.nhs.uk or paulstjohnsmith@hotmail.com