Evolutionary Special Interest Group of the Royal College of Psychiatrists

Evolutionary Psychiatry (EPSiG)

Newsletter No.6 March 2017

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Notes from the editor

This is our 6th EPSiG newsletter and we are delighted to publish an interview with Jay R. Feierman, M.D. (ret.), Clinical Professor of Psychiatry, University of New Mexico.

Resources and EPSIG Website

Dr Sheftel's powerpoint on the history of psychiatry and in particularly evolutionary thought is now on the website. I would like to thank her for all the work she has done on this topic identifying a whole range of issues and barriers that have beset the field. The link to the EPSIG web pages that contain a range of resources is below:

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

Future SIG meetings

Further dates for meetings were discussed at the AGM in January Future meeting dates include:-

May 19th 2017 Scientific Meeting @RCPsych

January 12th 2018 EPSIG 2nd Symposium

We will also have to book a date for the annual general meeting for 2018

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

The 'Virtual Interview'. Questions from Riadh Abed

Jay R. Feierman, M.D. (ret.), Clinical Professor of Psychiatry, University of New Mexico

1.What triggered off your interest in evolutionary theory in relation to psychiatry/psychology?

A: I was interested in animal behaviour for many years before I had any interest in psychiatry. For about 5 years, starting in high school in the 1950s, I worked 16 hours a week for a veterinarian doing the work that licensed veterinary technicians do today. I developed an interest in animal behaviour at that time. As an undergraduate at Penn State University in the early 1960s I majored in Zoology in general and comparative anatomy in particular, which allowed me to spend my senior year in a joint program between Penn State and the University of Pennsylvania's School of Veterinary medicine. I'm sure that I am one of the few psychiatrists who have dissected a cow and a

horse. I lost my appetite for eating chicken that year because I knew all the anatomical names for what I was eating.

After a year and a half at the veterinary medical school, I changed course (literally) and spent the next two years a few blocks away in the University of Pennsylvania's medical school's Brain Research Laboratory doing neurophysiology research. It was an easy next step to go to medical school at Penn. After graduation from medical school in 1969, and after almost going into a neurology residency at Columbia University in New York, my interest in behaviour won out and led me to chose two psychiatric post-doc residency programs. My psychiatric residency was separated by a two year period in Arizona on the Navajo Indian reservation as an Emergency Room physician and where I also delivered around 300 Navajo Indian babies. My two years with the Navajo were the two happiest years of my life as well as for my wife and two children, for whatever that might tell you about me. As an aside, my daughter is a child/adolescent and adult psychiatrist today in New Mexico.

The two psychiatric residency programs were "biologically" oriented, which really meant neurochemically oriented in those years. They were Washington University, St. Louis (1970-1971) and University of California, San Diego (1973-1975). Both psychiatric chair persons (Eli Robins and Arnold Mandell) were highly respected neurochemists. At U.C., San Diego, for my two years there, I spent 3 days a week in the psychiatry department and two days a week at University of California, Riverside in the biology department, and studying hibernation cycles in *Citellus lateralis* (golden mantled ground squirrel). I used the squirrels as a biological model for manic-depressive disorder. That research included studying tryptophan hydroxylase in awake versus hibernating ground squirrel brains and giving squirrels imipramine, ECT, etc.

My first academic position in 1975 was as Assistant Professor of Psychiatry at University of New Mexico in the medical school, although I also taught an honours course to undergraduates on ethology, using Eibl-Eibesfeldt's *Ethology* (1975) textbook. To satisfy my biological interests in animal behaviour in those days, I started going to the Animal Behaviour Society meetings each year. Those meetings got me interested in applying at least ethology to psychiatric disorders, which I did for the next several decades. I presented a number of ethological studies having to do with psychiatry at the Animal Behaviour Society. There was no other place to present such papers in those years.

In 1985, I organized a Symposium at the Animal Behaviour Society on "Ethological Studies of Psychiatric Disorders," which was published as Supplement 3 to *Ethology and Sociobiology* in 1987, for which I was the Guest Editor. Many of the older, retired, and even deceased (e.g., Michael McGuire) psychiatrists interested in evolutionary psychiatry today had articles in that Supplement, which had a Foreword by Konrad Lorenz. The 1985 Symposium was the first of its kind for evolutionary-oriented psychiatrists to meet, as far as I know.

2. Why would you say is evolution important to the understanding of mental disorder?

A: The only acceptable answer is Dobzhansky's 1973 essay, "Nothing in Biology Makes Sense Except in the Light of Evolution." Psychiatric disorders (which I prefer over "mental disorders") are generated by biological (in the broad sense) mechanisms.

3. Why have psychiatrists (and medics generally) been slow to embrace evolutionary theory?

A. From a research perspective, evolutionary theory is not very good at generating empirically testable hypotheses in which the outcomes can't also be predicted by common sense (i.e., they have to be counter-intuitive) or by simple observation and deductive reasoning. One of the cardinal principles I learned many years ago in medical school is that there are two types of therapeutics: rational therapeutics (do something because according to some theory it should work) and empirical therapeutics (do something because there is empirical evidence that it does work).

To date, evolutionary psychiatry is almost completely theoretical, which is only applicable to rational therapeutics. Rational therapeutics has no place in evidence-based clinical medicine outside of a research setting. Rational therapeutics is only a hypothesis generating mechanism for research. There are few evolutionary-minded psychiatrists in research. Hence, most psychiatrists, who practice clinically using evidence-based medicine, have not embraced evolutionary theory in their work. They are waiting for an effective treatment to actually be generated by evolutionary psychiatry that is evidence based in terms of empirical efficacy rather than just theory based.

4. Is it important to include evolutionary science into the undergraduate and postgraduate curricula and if so what would be the best strategy to achieve this end?

A. I'm not sure what "evolutionary science" means. Evolutionary theory is not like the infectious theory of disease, at least in its application to psychiatry. Evolutionary psychiatrists need to do more work in using evolutionary theory to predict the outcome of empirical studies that can't be predicted by common sense or by simple observation and deductive reasoning. Otherwise, evolutionary theory in psychiatry is really just a new version of Freudian, depth psychology psychodynamics. However, that is not the case in internal medicine, where there is a lot more empirical data about treatments that "fit into" evolutionary theory, even though evolutionary theory is probably not what generated the empirically effective treatment.

I remember having bad diarrhoea on a small island in the hot tropics in Indonesia about 20 years ago where sanitation was less than optimal. When I asked the small town Indonesian physician for antibiotics and something to stop the diarrhoea, after a few questions and examination, I was surprised that he told me it is better to let the body just rid itself of the bacteria through the diarrhoea, as long as I was staying well hydrated. I doubt he knew anything about evolutionary theory to tell me that. His decision was just common sense in Indonesia, and it works empirically. The diarrhoea almost always just eventually goes away, although sometimes it does not with some virulent pathogens.

I think evolutionary theory's application to medicine is more meaningful in an internal medicine lecture for medical students at this stage than in a psychiatry lecture to medical students. But it would not hurt to expose psychiatric residents to evolutionary theory, at least in a lecture. Perhaps such a lecture would stimulate some of them to use evolutionary theory

to generate empirically testable hypotheses whose outcomes can't be predicted by common sense or by simple observation and deductive reasoning. However, psychiatric post-doc residents will learn a lot more about evolution of human behaviour by reading Eibl-Eibesfeldt's 848 page *Human Ethology* (1989), attending Animal Behaviour Society meetings, the International Ethological Congresses, and any primate meetings. They can apply what they learn there to human psychiatric disorders.

5. In your view why are there still no evolutionary psychiatry university departments and no academic journals dedicated to the subject whereas there are many dedicated to evolutionary psychology?

A. Evolutionary theory has not risen to the point of the infectious theory of disease in psychiatry to warrant a whole department with this orientation at the time. We have empirical methods to test the predictive potency of the infectious theory of diseases. What are the methods to test evolutionary theories of psychiatry? Methods are there only in bits and pieces but don't exist as a coherent, agreed upon whole.

In 1982 I published a paper, Nocturnalism: An ethological theory of schizophrenia. In it I wrote that the nocturnalism theory "explained" most of the clinical manifestations of schizophrenia. Niko Tinbergen read the paper and told me that explaining what already exists is not what science is about. He told me that I had to use the nocturnalism theory of schizophrenia to predict that which has not yet occurred and which can't be predicted by common sense or by simple reasoning and deductive reasoning.

I spent many years, including a sabbatical in 1989 at the Max Plank Institute in Andechs, Germany, trying to figure out how to do that, which I finally did and published in *Ethology & Sociobiology* 15:(5/6): 263-282, 1994. By the time it was published, my career had changed and I was no longer in a position in which I could test the predictions made by the theory. However, it took me 12 years to come up with a single, counter-intuitive prediction that also could not be made by common sense or by simple observation and deductive reasoning and that was generated by the nocturnalism theory of schizophrenia. Tinbergen and Lorenz (who also read the paper) had died by the time it was published. I also have a fond remember of Lorenz's answer to me in 1985, when I asked him how he thought the nocturnalism theory of schizophrenia could be tested. He said (with a smile), "I'm too old. That's for you to figure out."

I should add that during those 12 years in which I was trying to figure out how to test the nocturnalism theory empirically, my wife and I spent a number weeks during many summers visiting very remote tribal societies in Africa, Asia and the various islands in the Pacific, trying to see what occurs when someone has schizophrenia in a tribal society in which there is no modern medical care. I thought that might give me a clue as to what schizophrenia really is, not just what causes it. We did find people with schizophrenia in several of these tribal societies. They were not difficult to identify. I gave a series of lectures on these findings that were titled, "Schizophrenia in the Bush."

By contrast, it is a lot easier to do evolutionary psychology. Much of what evolutionary psychologists do is make up adaptive "just so stories" and give evolutionary *post hoc* explanations in their interpretation of their data, when they have data. They also weave evolutionary theory into their Introduction. From my perspective, evolutionary theory in evolutionary psychology is just one of the two paradigms *du jour* that have replaced psychodynamic depth psychology. Darwin just replaced Freud and Jung. There is less enthusiasm for evolutionary psychology than for "cognitive science." Both will be displaced eventually, maybe even in my lifetime. Neither of them will have much longevity if they can't inter-relate with neuroscience, which is where the future lies.

6. How can evolutionary psychiatry fend off the accusations of promulgating 'just so' stories?

A. Stop talking about them. Evolutionary psychiatry needs to generate testable hypotheses about psychiatric disorders, based on evolutionary theory, whose outcome can't be predicted by common sense (i.e., they have to be counter-intuitive) or by simple observation and deductive reasoning. And if a theoretical statement can't be empirically tested in this way, don't articulate it.

7. Why have there been so few interventions in psychiatry based on evolutionary science?

A. Because there is no such thing as "evolutionary science." There is evolutionary theory, which should be used to just generate empirically testable hypotheses whose outcomes can't be predicted by common sense or by simple observation and deductive reasoning. And that should be done in a clinical research setting rather than a non-research clinical setting. Again, rational therapeutics should just be a research tool, not a tool for evidence-based clinical medicine.

8. What would you say is your most important contribution to evolutionary Psychiatry? And given your special interest in the biology of religion and religious experience, do you see a link between this field of enquiry and psychiatry?

A. I made a very modest contribution to evolutionary psychiatry by looking at psychiatric disorders from an ethological (behavioural biology) perspective as early as the 1960s, when few other psychiatrists had that interest. I remember an article in the Albuquerque, New Mexico newspaper about me when I first arrived at University of New Mexico in 1975. The headline said, "Psychiatry Professor Studies Squirrels." That was supposed to make people who saw the headline laugh. But I was serious.

From my perspective, the ground squirrels I was studying went through annual cycles (even in constant light and temperature cold rooms) of hibernating (my model for major depression) and then getting hyperactive and hypersexual for several weeks (my model for manic episodes) when they came out of hibernation. I made predictions and tested them experimentally. Unfortunately, one of the grad students live trapping the squirrels in the Jemez Mountains in New Mexico in 1976 caught plague from them. After that, the dean of

the medical school would not allow any live trapped ground squirrels to be brought back into the medical school, which brought that research program, which I had started as a psychiatric resident in California, to a halt. However, in my current home in New Mexico, we put out seed blocks on the low wall in our backyard. The seed blocks attract birds and antelope ground squirrels from the nature preserve behind our house. Although the ground squirrels are a different species, they look similar and bring back fond memories. I like to observe their behaviour with binoculars, like I used to observe the behaviour of the golden mantled ground squirrels in the mountains with binoculars many years ago.

I also used evolutionary theory to make a prediction about Anorexia Nervosa (AN) in 1984, which in its day was quite counter-intuitive. I had attended a paper session on female choice (over impregnation and reproduction) at a meeting of The American Society of Primatologists in Atlanta Georgia in 1977, which is where I met Eibl-Eibesfeld, one of Lorenz's students and the "Father of Human Ethology" for the first time. This was in the very early days of primatology and there were only a few reports in the literature that lower social rank in group living female primates produced lower reproductive success by unknown mechanisms. There were also early reports of non-self-initiated female reproductive failure by male infanticide among primates, such as in Hrdy's *The Langurs of Abu* (1977). Someone at the female choice paper session said that compared to the well known social reproductive suppression of lower rank group living male primates by higher rank males, in the absence of malnutrition, which physiologically delayed female primate maturation, he was not aware that young and potentially fertile, reproductive age female primates had either social suppression of reproduction (like in the male primates) or any female-choice mechanism to suppress their own ovulation and therefore reproduction. A light went on in my head when I heard that. AN in human females came to mind.

Humans are female primates, and when they have AN, they temporally suppress their reproduction when their body fat goes below 8% and they temporarily stop menstruating. AN looked to me as a reproductive delaying process in (primarily) higher socio-economic status (SES) teenage girls in the industrialized democracies. There was already a biosocial anthropology literature on the breed early versus breed later reproductive strategies in young reproductive age women. A few years later in 1984, I gave a lecture to the International Primate Society in Nairobi, Kenya on that that theme, which ironically was picked up by the Associated Press as a quirky newspaper story. I also had planned to empirically pilot test the AN theory in tribal Sub-Saharan Africa with my wife (who has an anthropology degree) by looking for the absence of AN among teenage girls in the less hierarchically stratified, Sub-Saharan tribal African societies, where delaying reproduction made little sense.

After bouncing around in a 4-wheel drive vehicle on dirt tracks in Kenya for several weeks with my wife looking for AN girls, we both realized that there was a problem. In 1984, there was a severe drought in sub-Saharan Africa and most teenage girls looked anorexic in the drought area from the lack of water and food. If any had AN, they were indistinguishable from all the people who were dying of dehydration and starvation. Food deprivation appeared to do the same thing to tribal girls' weight and reproductive suppression as high achievement expectations did for the higher SES girls in the industrialized democracies.

The effect of getting pregnant during the drought was obvious. My wife and I actually saw a room full of dead and dying babies lying on a concrete floor in a room in a Kenyan government "hydration and feeding station" in the remote African bush. Skin and bone mothers whose babies were barely alive were holding them out to us, pleading with us in body language to take their babies, as they knew they were going to die. They had no breast milk. In the feeding station an African attendant with us took a half dead baby from an already dead mother lying on a cot and put the baby, still barely alive, on the concrete floor to die. He said it was too far gone to even try to revive. One of the worst and saddest things I've ever witnessed in my life. That's not doing evolutionary psychiatry in an armchair.

The importance of all of that work for evolutionary psychiatry was really just the approach. Use evolutionary theory to make empirically testable hypotheses whose outcome can't be predicted by common sense or simple observation and deductive reasoning.

When I retired in 2006, after 30 years on faculty (regular, adjunct, and clinical) at University of New Mexico, I applied my interests in the evolution of behaviour to religious behaviour. It was an easy transition as religion has lots in common with psychiatry. One of the few differences is that religious beliefs are more "contagious" than schizophrenic delusional beliefs in psychiatry. In 2008, I organized a Symposium in Bologna, Italy at the biennial meeting of the International Society for Human Ethology. The Symposium was titled, "The Biology of Religious Behaviour: An Ethological Perspective." Some of those papers became part of the peer reviewed, edited collection, *The Biology of Religious Behaviour: The Evolutionary Origins of Faith and Religion* (Praeter/ABC-CLIO, 2009).

Religion is more important than psychiatry as more people have it and it is dangerously dividing the world. I've published a number of articles and book chapters in that area and have organized six international invited paper sessions on the topic at the yearly meetings of the Society for the Scientific Study of Religion. For what it is worth, there is an order of magnitude more people interested in the evolution of religion than in the evolution of psychiatric disorders. We used to joke in the 1980s that you could put all the evolutionary psychiatrists into one bus and have many seats left over.

8. What aspect of your evolutionary work are you most proud of?

A. That I at least tried to do more than tell "just so stories" using evolutionary theory. I got my hands dirty in the field and lab to empirically test the predictions about psychiatry made by the evolutionary theory.

9. What advice would you like to offer to your fellow evolutionary psychiatrists?

A. Do what I did but be more successful. I pass the baton to a younger generation. And, don't wait until you retire to apply your knowledge of the evolution of human behaviour to religion. A link to the upcoming conference I'm co-organizing in New Mexico, "The Evolution of Religion II: How Biology
and Culture Interact," November 12 - 15, 2017, will be forthcoming. I'll make sure the link reaches the evolutionary psychiatrists.

This month we are going with a review of some recommended reading instead of a special article (Ed),

Neuroscience Needs Behavior: Correcting a Reductionist Bias

John W. Krakauer, Asif A. Ghazanfar, Alex Gomez-Marin, Malcolm A. MacIver, and David Poeppel

Neuron *93*, February 8, 2017

The thorny issue of reductionism has been a festering problem in biological psychiatry and this has deepened with the advances made in neuroscience whereby molecular and neuronal events have been considered to offer a full understanding of behaviour and other psychological responses. This important article elegantly sets out why this kind of reductionism will not and cannot work. However, unlike the challenge posed by the post-modernists who eschew the scientific method and have thus caused greater confusion, this critique is firmly within the scientific tradition.

The authors point out that one reason why detailed examination of brain parts or their selective perturbation is not sufficient to understand how the brain generates behaviour is that we have no prior knowledge of what the relevant level of brain organization is for any given behaviour.

To complicate matters, the same behaviour may result from alternative circuit configurations, from different circuits altogether or the same circuit may generate different behaviours.

The authors describe an interesting and provocative study that applied modern neuroscience techniques to a single computer microprocessor (analogous to the brain) in an attempt to understand how it controls three classic videogames (analogous to behaviours). Crucial to the experiment was that the answer was known a priori: as the processor's operations can be drawn as an algorithmic flow chart and was hence fully understood. The sobering result was that performing interventionist neuroscience on the processor could **no**t explain how the processor worked.

Another salient and striking example cited in this article is the case of the roundworm (*Caeno- rhabditis elegans, also known as C. elegans*). The interesting thing about this extensively studied worm is that it's genome is fully known, and so are all it's the cell types, and the connectome i.e. every cell and its connections. However, despite this wealth of knowledge, our understanding of how all this structure maps onto the worm's behaviour remains frustratingly incomplete.

The authors contend that the question 'How does the brain generate behaviour?' is best answered through precise hypotheses articulated in an a priori conceptual framework, careful task design, and the collection of **behavioural data**.

The first step for developing conceptual frameworks that meaningfully relate neural circuits to behavioural predictions is to design hypothesis-based behavioural experiments.

They quote David Marr's statement in his classic critique of reductionism in neurophysiology ''Trying to understand perception by understanding neurons is like trying to understand a bird's flight by studying only feathers. It just cannot be done'' (Marr, 1982/2010).

As Woese has argued (Woese, 2004), science is driven by both technological advances and a guiding vision. The key is to balance their contributions, ''without the proper technological advances the road ahead is blocked. Without a guiding vision there is no road ahead''. Insofar as the goal of a neuroscience research question is to explain behaviour, be it a phenomenon from vision, communication, motor control, navigation, language, memory, or decision making, the behavioural research must be considered, for the most part, epistemologically *prior*. The neural basis of behaviour cannot be properly characterized without first allowing for independent detailed study of the behaviour itself.

Although the authors only discuss the place of evolution in their framework in passing, their formulation is firmly embedded within evolutionary thinking. Behaviour in this case is a phenotypic trait that has been shaped by selection due to the function that it served the organism in its natural environment. Hence, the neural circuitry and molecular events within the brain are subservient to this end-product. The same principle applies to emotions and cognitions which are all end products shaped by selection and are all emergent properties of lower level neural events. An important message from this article is that a complete understanding of a higher level emergent property cannot be obtained from lower-level events and thus reverse engineering as a strategy is destined to failure.

I would highly recommend this article for discussion in journal clubs (which would be a good anti-dote to the usual choices of antidepressant and antipsychotic trial articles).

References:

Marr, D. (1982/2010). Vision: A Computational Approach (MIT Press)

Woese, C.R. (2004). A new biology for a new century. Microbiol. Mol. Biol. Rev. *68*, 173–186.

Onto the reviews; PSTJS Ed.

Review of "Sapiens: A Brief History of Humankind" by Yuval Noah Harari. Harvill Secker 2014

By Sunjai Gupta

This book describes the evolution of the human species (*Homo Sapiens*), and is built around a series of "Revolutions": Cognitive, Agricultural, Scientific and Industrial. At the same time the book has running through it a number of interconnected themes and, in this review, I will attempt to summarise Harari's views on the nature of some of these and their implications.

One of these themes is the development of the modern food economy. Common to all species of the genus *Homo* is the "big brain" which required large amounts of energy, and placed greater demands on the food supply. Eventually *Sapiens* was catapulted to the top of the food

chain. It acquired, for good or for ill, the ability to change the world around it rather than just to adapt to it, and the revolution in agriculture paved the way for the Industrial Revolution. However, according to Evolutionary Psychology, even after the Agricultural Revolution human brains and minds continued to operate as they did in the pre-agricultural era with a preference for high calorie foods. This may have sown the seeds of modern obesity.

Another noticeable effect of the Agricultural Revolution was the increase in population size, the rise of social hierarchies, and the development of animal husbandry which contributed to disease amongst humans. The domesticated animals were themselves mistreated as they had emotional and social needs that had developed in the wild and were retained even though they no longer had survival value. Harari points out that these needs were now no longer being met even though their material requirements were.

Another key theme is the growth of the special talents that marked the Cognitive Revolution. These included the ability to solve new problems such as how to deal with the freezing temperatures that the early humans encountered as they migrated northwards. The development of the capacity to store and to communicate large amounts of information (including gossip about other humans) was also linked to the advent of a flexible, "fictive" language about things that existed only in the abstract, such as legends. There were further leaps and bounds in the shape of numbers and written scripts. However Harari issues a chilling warning that, in the future, scientific developments might go too far: " What might happen to human memory, human consciousness and human identity if the brain has direct access to a collective memory bank?...Such a cyborg would no longer be human or even organic".

The cognitive changes, including shared myths, also made it possible for *Sapiens* to evolve down a new, more rapid, cultural route. As a result it was feasible to form cooperative networks even though the biological instincts for collaboration did not keep pace. However, emotional evolution lagged even further behind. Harari suggests that evolutionary progress may have been won at the expense of an increase in suffering, and that objective conditions alone do not equate to happiness, which depends on expectations that inevitably rise as conditions improve. There is an interesting discussion on Buddhism and both the similarities and differences between the latter and other theories of wellbeing. One of the key insights, derived from Buddhism, which Harari offers is that happiness can be independent not only of the external world but also of inner emotional states as well.

Though it has relevance to a number of "modern" conditions such as Seasonal Affective Disorder, this book contains few if any explicit references to Evolutionary Psychiatry. Nevertheless, it is a very useful summary of how we got to where we are today, and where we might be heading in the future. Harari argues, in fact, that bioengineering, and the substitution of intelligent design for natural selection, could lead to the end of the human species as we know it.

Special Article on Naturalism This is deferred until the next letter due to lack of space.

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**