

Evolutionary Special Interest Group of the Royal College of Psychiatrists



Evolutionary Psychiatry Special Interest Group (EPSIG)

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Editors:

Dr Paul St John-Smith (Consultant Psychiatrist Single point Of Access Team, Hertfordshire Partnership University foundation Trust);

Dr Annie Swanepoel (Clinical Director CAMHS, Consultant Child and Adolescent Psychiatrist, Potter's Bar Hospital, Elysium Healthcare).

Contact Emails: -

p.stjohnsmith@nhs.net or paulstjohnsmith@hotmail.com

EPSIG Chair: Riadh Abed (Retired Consultant Psychiatrist and Medical Director; currently Medical Member of the Mental Health Tribunal Service in England) abedrt@btinternet.com

World Psychiatric Association (WPA) Evolutionary psychiatry email group moderator: Mohammed Abbas mohdgum@hotmail.com

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1. **A Note from the Outgoing Chair:** I will be stepping down from my role as founding chair of EPSIG next month but I will still be involved in the running of the SIG as its new finance officer. EPSIG was set up in 2016 and during its 4 years of existence has held 3 successful full-day international symposia where leading evolutionary thinkers and authors from around the world have presented their work as well as 3 successful half-day scientific meetings. We have also set up an EPSIG YouTube channel where all the lectures from our 3 symposia have been posted and these have attracted well over 100,000 viewings. This has been an important open

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access evolutionary psychiatry learning/teaching resource available to all. In addition we have published 2 editorials promoting the teaching of evolutionary biology, one in the BJPsych Bulletin (2016) and the second in the BJPsych (2019). We have also published 19 newsletters.

The EPSIG membership currently stands at over 1250 and we are extremely pleased that our membership is truly multi-professional as we have GPs, neurologists, medical students, psychologists, anthropologists and philosophers in addition to psychiatrists (both consultants and trainees) among our members. Our executive committee includes a psychiatric trainee, a postgraduate fellow, an anthropologist and a philosopher as well as 6 consultant psychiatrists. I am sure I speak for all my colleagues when I say that we all feel proud of what EPSIG has achieved in its short existence but we also appreciate the challenges ahead. Our aims are both big and radical: they are for evolution to be accepted as the overarching framework for psychiatry and for evolution to take centre stage in our understanding of mental health and mental disorder. We appreciate that our project is a long term one which will only bear fruit over time but we can feel satisfied that EPSIG has, at least, taken the first few crucial steps toward achieving this. RA

2. **Notes from the editor**

Unfortunately due to the Covid-19 pandemic, our AGM and half day scientific meeting on 'Evolutionary Perspectives on Childhood Trauma' have been cancelled.

EPSIG conferences and meetings in the pipeline (see details below).

Change of college officers; this is required every 4 years. From June 2020 the EPSIG officers will be as follows: Riadh Abed is the new finance officer, taking over from Agnes Ayton, Paul St John-Smith is the new Chair and Annie Swanepoel is the new the newsletter editor.

3. **Meetings:** The October 2020 4th international symposium has now been moved to 29th October 2021 due to Covid-19. Details of the program will be announced in due course. **Enquiries to** Catherine Langley: catherine.langley@rcpsy^{ch}.ac.uk
4. **Video links on the evolutionary biology of where viruses come from and specifically Covid 19**

A) **Where Did Viruses Come From?**

<https://www.youtube.com/watch?v=X31g5TB-MR>

B) **Where do new viruses come from?**

<https://www.youtube.com/watch?v=NJLXdsO1GBI>

C) **Specifically the evolution of SARS-CoV-2**

<https://youtu.be/MHRGPzoFyEM>

D) **Evolutionary psychopathology lecture by Marco Del Giudice**

<https://www.youtube.com/watch?v=HG9OD5rYszg>

The Divided Brain <https://vimeo.com/ondemand/thedividedbrain>

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The Divided Brain had a wonderful premiere in London (see attached comments) last year and then was broadcast by the CBC in Canada. The film has had some “big event” screenings in London, Scotland, Toronto and Washington (American Enterprise Institute) which have been very successful, since the film was followed by discussions with Iain, with participants including John Cleese, Charles Murray, and Drs Jordan Peterson and Norman Doidge .

The film appears to appeal across people of various disciplines and walks of life: public policy (Washington’s AEI) economists, mental health specialists, faith-based organisations, environmental sciences, the advertising industry and the legal profession.

Below is the commercial streaming link. If you like the film, please consider recommending it to your colleagues and friends.

<https://vimeo.com/ondemand/thedividedbrain>

4.Essay and Special Article by Adam Hunt

True stories: A new evidence-based method to avoid the ‘just-so’ criticism of evolutionary theorising.

Readers of the EPSIG newsletter will likely be well aware of the hastily slung, frustratingly recurrent ‘just-so story’ criticism of evolutionary sciences which has migrated from evolutionary biology and psychology to evolutionary psychiatry, most famously outlined in ‘The Spandrels of San Marco’ by Gould & Lewontin (1979). Put simply, the criticism is that the invisibility of the past makes evolutionary theorising a matter of speculative storytelling rather than scientific discovery: researchers are said to be free to propose any evolutionary explanation they like to explain modern bodies and minds, constrained only by their imagination, immune from being disproven because the facts are out of reach. This criticism is omnipresent, and unfortunately occasionally valid.

My training was in philosophy; I completed my masters in 2015 at the University of Bristol with special emphasis on the philosophy of biology, which almost exclusively concentrated on exploring evolutionary theory, its powers and weaknesses. The just-so critique was front and centre of many discussions. At the time I wasn’t particularly invested in a response.

That changed in 2016. I became convinced that evolutionary thinking, and mismatch in particular, would completely rejuvenate the lingering failure of psychiatry to explain, truly explain, its subjects. Here, finally, was a theoretically coherent explanation with great potential, one which could be hugely emancipating to many millions and set psychiatry on a path of true discovery and progress.

Since then I have been working on a theory which proposes long-term, common, heritable cognitive differences are specialised evolved traits – individual differences which would serve a function in the world of our hunter gatherer ancestors. Some of those differences are all-too mismatched to modern life, and so manifest and classify as disorders. ADHD, psychopathy, many cases of autism and other disorders fall into this category; normal Big Five personality traits are manifestations which are orderly rather than disorderly. This work has resulted in a forthcoming book, ‘*The Specialised Mind*’.

This essay is not designed to dwell on the theory and forces behind cognitive specialisation, but the method I developed to make the case, acknowledging the need to pre-empt and overcome the criticisms of ‘speculation’, ‘storytelling’ and ‘untestable’. Facing a discipline

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which struggles for lack of proof and sure truth, a philosophical reassessment of the process of theorising in evolutionary psychiatry seemed timely. Tinbergen's four questions (1963) clarified the levels of causation one can investigate in our evolved faculties. A new heuristic for building hypotheses of function and discerning disease from adaptation could add another level of methodological rigour to our evolutionary approaches to explanation.

The crux of the problem facing evolutionary arguments is their historical nature. Even if the theory of evolution by natural selection is indubitable and illuminating, it leaves us conjuring up stories of past function to explain modern biological characteristics, and that inevitably places the science in an uncertain place. The current best solution is of 'reverse engineering' (Pinker, 1999; Tooby and Cosmides, 2015). The essence of this approach is to appeal to the complexity and specificity of a particular trait, arguing that disease or drift cannot explain such well adapted function; only natural selection could be the cause. Whilst applicable for adaptations which maintain their usefulness (e.g. eyes, sexual desire), this cannot be used to assess adaptations mismatched to the modern environment and not showing their original functional specificity. There is, however, another way to formulate our theories, made practical only in the last few decades as our evidence base has grown.

Rather than concentrating on referencing phenotype characteristics and speculating on how they played a role in ancestral life, the foundation of evolutionary theorising can be based on the biological, uninterpreted, non-historical evidence. In *The Specialised Mind* I rely primarily upon studies of the brain, genetics, age of onset and course, environmental influences, sex differences and prevalence. From these areas of evidence, all directly observable, none open to accusations of just-so storytelling, a solid theory can begin to be built. I shall briefly explain the method here.

The DCIDE Method

A robust method for evolutionary theorising has various requirements. It needs to be simple and intuitive. It needs to be evidence-based, ideally on objectively observable data rather than psychometrics. It needs to be able to account for heterogeneity and loose diagnostic labels. It cannot assume that every psychiatric diagnosis necessarily refers to a dysfunctional brain. It should avoid, as much as possible, conjecture about the unobservable past. My solution, after years of tinkering, cannot be considered complete (in that its criteria can certainly be improved upon, and adapted around new findings and technologies) but seems a highly promising direction. I call it the DCIDE method, an acronym for the five required stages: Describe, Categorise, Infer, Depict, Evaluate. Here I will briefly outline the concepts (but not the full criteria) behind each stage, and in the next section a short example will be made of how this method could be used to analyse autism:

Describe: Identify a trait, condition or cluster of cooccurring symptoms, in the manner of modern psychiatric diagnosis.

Categorise: Reviewing the objective evidence of a particular condition (I use brain studies, genetics, age of onset and course, environmental influences, sex differences and prevalence) consider whether the findings are the expected result of dysfunction or an adaptation: are these clues pointing towards disease or evolution. E.g. late onset is more likely to be a disease that avoided purifying selection, early onset is more likely to be evolved; common complex genetic bases could be evolved, de novo mutations cannot; brain developmental differences could be evolved, brain damage could not.

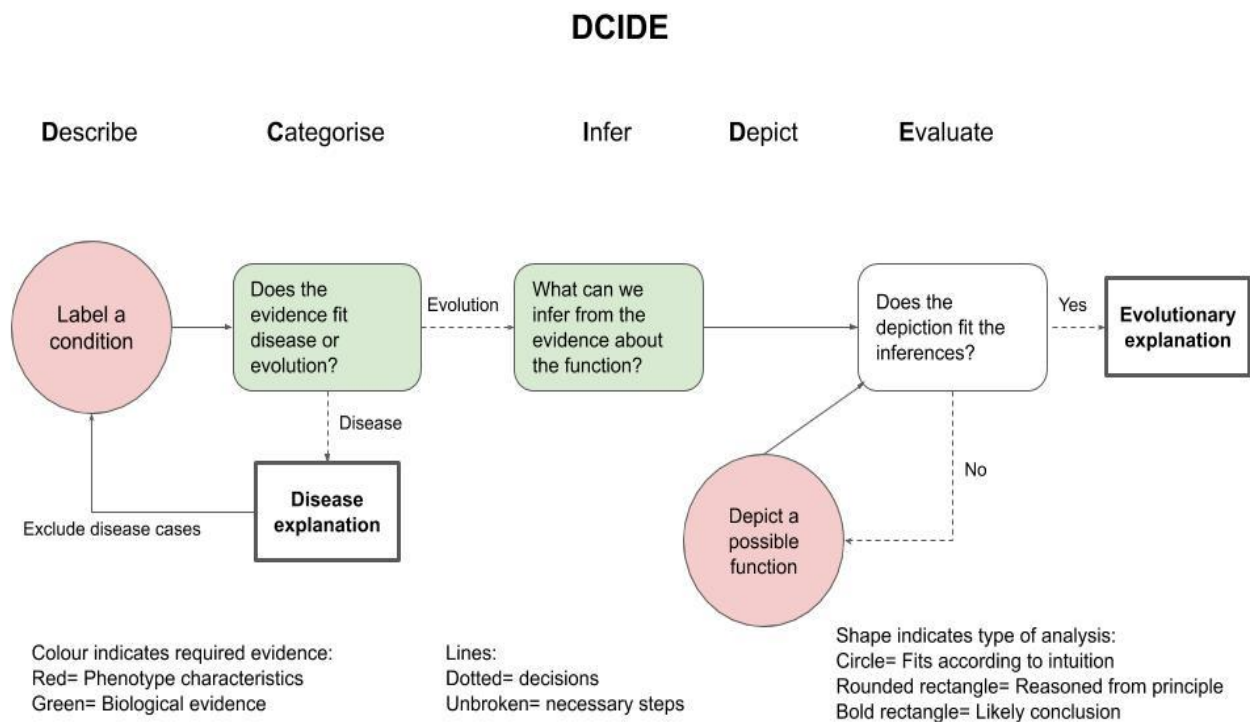
If a subset within the diagnostic category shows signs of disease whilst the remainder does not, distinguish between the groups (as seen in autism example below).

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Infer: Taking the group which categorises as potentially evolved, consider what the same findings imply about the trait if they were functional features. E.g. A trait appearing at adolescence and then receding should function to optimise the time of early sexual reproduction and entering adulthood; a trait more common in males should serve a more male-specific function; a trait appearing in one percent of individuals should be ideally suited to exist once per Dunbar-sized group.

Depict: In the method of traditional evolutionary psychology and psychiatry, relate the characteristics of the condition to ancestral life, and propose a possible function. Here we engage in the ‘storytelling’ which has previously been suspect.

Evaluate: Finally compare the depicted story/s of function to the inferences from the evidence. The better the function accords with the inferences from the evidence, the stronger the proposed theory. E.g. If the condition is far more common in females, and the proposed function is a female-specific strategy, it should be preferred to another proposed function which would apply equally to males and females.



Applying the DCIDE method to autism

To make an abstract process more concrete, it’s worth briefly running through the current evidence for autism, seeing what kind of theory the DCIDE method leads us to.

Autism starts off as a frustrating disorder to discuss, because the spectrum is so wide. The core symptoms are social inability concurrent with obsessive, restricted or repetitive behaviours. These criteria can simultaneously apply to Silicon Valley tech billionaires or carer-dependent non-verbal individuals. One autistic individual could feed a million mouths with their wealth, another cannot feed themselves. Even though the autism label is too wide to expect a single unifying etiology, the DCIDE method automatically accounts for this heterogeneity.

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Taking our initially broad autism label as our **d**escription, we use the **c**ategorisation criteria to identify potentially evolved or diseased forms. Studies have found that at least 3-10% of autism is related to de novo and rare genetic variants (De La Torre-Ubieta, Won, Stein, & Geschwind, 2016). These genetic causes cannot have an ancestral history of function; these cases are confirmed as diseases. Within environmental factors too, we find certain prenatal teratogens, such as valproate or alcohol, can cause syndromes which often classify as autism (Mandy & Lai, 2016). These are, again, not evolved cases.

Once these confirmed disease cases (often individuals with lower IQ and more severe disability) are set aside as explained via disease, a significant proportion of the autism spectrum meets the categorisation criteria we expect of evolved traits: Brains develop differently, but subtly, not obviously biologically dysfunctioning. Genes are common and complex. Onset is early and lifelong. An environmental factor is parental age, and autism is more common in males, but for neither finding is there an explanatory dysfunction, so both could be adaptive developmental strategies (Nettle and Bateson, 2015) related to function. Lastly, autism is prevalent enough that it should have existed in every one of our ancestral hunter gatherer tribes, and individuals on the broad spectrum would exist in every band. (For reviews of autism research see Ecker, Bookheimer, & Murphy (2015); Lai, Lombardo, & Baron-Cohen (2014); Modabbernia, Velthorst, & Reichenberg (2017)). All of these facts are bizarre and paradoxical to a disease explanation, but perfectly fit an explanation of autism as having some specialised evolved function.

Before storytelling about the characteristics of autism in the context of hunter gatherer life, the evidence serves another purpose, in providing inferences about its likely function. Autism is more common in males, would be prevalent about once or twice per a group of Dunbar's number, appears young and is lifelong, and is more likely to appear in children born to older parents. These are the facts, objective, certain, not historical. If autism evolved for a particular function, these features should be linked to that function.

In **d**epicting, we return to the more classic approach of evolutionary sciences. Examine autism's characteristics and consider them in terms of function during our hunter-gatherer past. Accompanying the deficits we define them by, autistics often show outstanding abilities in memory and develop special skills in their area of obsession. Various hypotheses have been proposed of autistic talent lending success in the ancestral world. Here we'll depict autistics as the memory banks and object and system specialists of hunter gatherer tribes, obsessing over systems of nature, lending the tribe their talents and being understandably forgiven for their oddities. For simplicity, this depiction alone will be evaluated.

Before the DCIDE method, this story of autism's evolutionary function would have been wide open to the 'just-so' criticism. No matter how intuitive, no matter how appealing, it's based on imagining modern differences in an ancient world. However, here, in the final step of evaluation, we can ask whether the depicted story of autistic object-obsessed specialists fits with the inferences gained from analysing the evidence. We are, in essence, testing our hypotheses against the available evidence.

Would object-obsession at the cost of social ability be more suitable for males? It seems so, given the importance of mothering for females and the relative importance of social status for males, which can be gained by specialist knowledge (Sugiyama & Sugiyama, 2003; von Rueden, Gurven, & Kaplan, 2008). Would the prevalence of one or two autistic individuals per group be ideal? Again, it fits the function of being the system specialist of the group. Young, lifelong appearance also fits, as autistic skill comes from the many years of obsession with their area of interest (Happé & Vital, 2009). And finally, an autistic child being born to

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older parents makes strategic sense, because they are likely to be born with more respected parents and older siblings, a more protective situation to get through the early years where autism's deficits are most obvious and abilities not yet fully developed (Meilleur, Jelenic, & Mottron, 2015).

Any theory of autism would have to explain all these facts; if a disease explanation can better explain them, it should take precedence, and if another evolutionary function can better explain them, it should take precedence. As it happens, they fit well with an evolutionary explanation of these non-disease cases of autism as object and system cognitive specialisation, creating minds which obsess and revel in the non-social world.

Finishing evaluating autism with the DCIDE method, what has been achieved? Firstly, we have distinguished between a too-wide spectrum, identifying cases of disease and cases which meet all the expectations of purposefully evolved traits. The spectrum is clarified. Then, for the remaining cases, we have made an evolutionary hypothesis about potential function and held that hypothesis up against the physical evidence. From start to finish, this is an evidence-based and evolution-oriented approach, and makes simple sense of a long confusing disorder.

Conclusion: Biologically-based systematic storytelling

This synopsis and overview of the DCIDE method are quick proof of a concept rather than a complete description. Each step leads to its own discussion, each criteria for categorising and inferring from each area of evidence requires its own development.

My book and research have concentrated on applying this method to long-term individual differences in cognition, but it can theoretically be applied to any trait in biology, mental or physical, to discern disease and narrow down stories of function – although the chasm in psychological and psychiatric theorising make it especially desirable in the science of the mind. This approach cannot be considered entirely new in concept (for example, EPSIG's own Riadh Abed produced a sexual-selection hypothesis explaining eating disorders (Abed, 1998) by referring to female predominance and age of onset) but is perhaps better seen as a fleshing out and formalising of evidence-based hypothesising about function.

The core shift which allows the just-so story criticism to be avoided is simple: wait until the objective evidence is fully taken into account before talking about historical function. Categorise as diseased or evolved, and then form evidence-based foundations for hypothesising about ultimate cause. Moving past the days of just-so critiques and relying more than ever on biological, observable evidence might not be the prize of biomarkers, sought so long and found so little, but it seems like it could be the next best thing.

Adam is in talks with the Evolutionary Medicine department at the University of Zurich, and is seeking funding opportunities to develop some aspect of his work within a PhD. 'The Specialised Mind' should be released in 2020. He is currently open to sending out a limited number of manuscripts for review. You can email him at contact@adamhunt.info

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Further topics and links

Dear all, FYI, here's a link to an interview I did for Ricardo Lopes' "The Dissenter" channel on Youtube:

<https://youtu.be/am0fbTEGXQg>

The whole channel is highly recommended if you don't know it--dozens of in-depth interviews on psychological/philosophical/biological topics, and a fantastic range of perspectives.

Marco Del Giudice, Ph.D.

*Associate Professor
Department of Psychology
University of New Mexico*

Website: marcodg.net

ALSO [Russell Powell](#), [Eric Scarffe](#)

‘Rethinking “Disease”’: a fresh diagnosis and a new philosophical treatment’ <https://jme.bmj.com/content/45/9/579.full>

Abstract

Despite several decades of debate, the concept of disease remains hotly contested. The debate is typically cast as one between naturalism and normativism, with a hybrid view that combines elements of each staked out in between. In light of a number of widely discussed problems with existing accounts, some theorists argue that the concept of disease is beyond repair and thus recommend eliminating it in a wide range of practical medical contexts. Any attempt to reframe the ‘disease’ discussion should answer the more basic sceptical challenge, and should include a meta-methodological critique guided by our pragmatic expectations of what the disease concept ought to do given that medical diagnosis is woven into a complex network of healthcare institutions. In this paper, we attempt such a reframing, arguing that while prevailing accounts do not suffer from the particular defects that prominent critics have identified, they do suffer from other deficits—and this leads us to propose an amended hybrid view that places objectivist approaches to disease on stronger theoretical footing, and satisfies the institutional-ethical *desiderata* of a concept of disease in human medicine. Nevertheless, we do not advocate a procrustean approach to ‘disease’. Instead, we recommend disease concept pluralism between medical and biological sciences to allow the concept to serve the different epistemic and institutional goals of these respective disciplines.

Articles for the newsletter

We welcome submissions for future newsletters in the form of articles, reviews and interviews. Correspondence: Replies, suggestions and clarifications on articles are welcomed and may be printed/included in our next newsletter. Also, we welcome brief reviews of

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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: - paulstjohnsmith@hotmail.com

We are now including a student section in future newsletters. This student section of the newsletter would be peer-reviewed with a lighter touch than other articles to encourage contributions. Remember it is a newsletter, so popular science is OK as long as it is science. Articles on evolution and psychology/psychiatry will be the mainstay, though we are open to related topics. Up to 2000 words is fine. Projects and preliminary findings would be suitable too. Any appropriate contribution on human behaviour with an evolutionary slant can be considered. If in doubt, email us:-

abedrt@btinternet.com , paulstjohnsmith@hotmail.com or annie.panzer@gmail.com