Confectionery consumption and violence

Moore et al.1 found a ‘novel and robust’ relationship between confectionery consumption during childhood and conviction for violence in adulthood. However, there are serious methodological concerns, which make the overall findings questionable. As the authors recognise, the number of violent people in their cohort is very low. The lack of descriptive information in the paper, contrary to recommendations on the reporting of observational studies,2 forces the reader to an exercise of reconstruction. What emerges is that only about 33 participants were violent (0.47% of 6942) and of these, only 23 (69% of the 33 violent individuals) had eaten confectionery excessively. With such numbers, it is highly discouraged in the biostatistical literature to model the probability of being violent using as many parameters (8) as the authors did, since the fit is essentially driven by the number of cases and not by the entire sample size.3 The deficiencies of this approach are well known and numerous, affecting all aspects of the modelling process, from variable selection to effect size estimation,4 and are not, generally, accommodated by the adoption of rare-events logistical models, which only provide a fix for bias in estimating regression parameters. With such few cases, no interactions have been considered, even though some may be very intuitive (e.g. confectionery consumption and child-oriented parenting). With no serious attempt at considering interactions in the model, the risk of finding spurious associations is well documented (Simpson’s paradox).5 Unfortunately, no details are provided in the paper concerning distribution of the other seven factors included in the multivariable model (gender, late birth, etc.) between violent and non-violent people, so that it is almost impossible to understand how low the cell frequency is in some such combinations. With these considerations in mind, the conclusions suggesting a relationship between confectionery and violence seem an over-interpretation of the fitted model.


Author’s reply: We take issue with Gregori’s statement that methodological concerns render the overall findings of our report1 questionable. Gregori correctly observes that ‘the number of violent people in their cohort is very low’ and goes on to suggest that reporting results on such small samples should be discouraged. We are interested in life-course factors that predict adult violence in the hope that such research might inform early life-course interventions. We therefore have two options. Either recruit violent offenders and enquire of their childhoods, or follow a cohort of individuals recording information on their circumstances to assess associations with later problem behaviour. Unfortunately, compounding the vagaries of human memory are the particular difficulties many offenders have with recalling what they did the previous day, let alone several decades ago. It is thus unfeasible to conduct retrospective studies; this leaves cohort studies as the only realistic and robust methodology. We are fortunate in the UK to have one of the most highly regarded cohort studies in the world, but despite its large initial sample size the rarity of violence means that only a small number of respondents demonstrate the behaviour of interest. Should we, as Gregori counsels, simply not consider using the British Cohort Study to look into childhood factors predicting adult violence because violence is rare? We suggest that this would be a valuable and informative resource squandered if that advice were followed. Gregori also suggests that models on rare data should not involve too many covariates. In our short paper we reported that we considered various configurations including the unadjusted association between confectionery and violence and that the strength of association was consistent across models – analytically we did as much as we could to test this association. We chose not to report simpler models and hardly mentioned the extensive analyses assessing the impact of attrition simply because we felt this paper suited a short-report format and including this additional information would only detract from what was a perfectly well-articulated finding. We therefore maintain that we analysed some of the best cohort data available to assess childhood predictors of an important outcome and found a robust association. We were honest with regard to the sample size, concluding in the paper that this is one area that should be addressed before firm conclusions can be drawn.


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Structural brain abnormalities in bipolar disorder: what meta-analyses tell us

Findings from Arnone et al’s1 systematic review and meta-analysis of magnetic resonance imaging (MRI) studies suggest that patients with bipolar disorder are characterised, in comparison with healthy controls, by significant reductions of whole-brain and prefrontal lobe volumes and by enlargement of lateral ventricles and globus pallidus, although most of the brain changes detected in bipolar disorder do not seem to be diagnostically specific and some clinical variables, such as patients’ age, duration of illness and pharmacological treatment, appear to be relevant in determining the magnitude of observed effect sizes.

These findings are not completely consistent our own recent meta-analysis2 of MRI studies in first-episode bipolar disorder.
Our study did not evidence whole-brain volume deficits in first-episode bipolar disorder compared with healthy controls. This may indicate that a progressive decrease of whole-brain volume occurs over the course of the disease, and might be detectable only when multi-episode or chronic cases are considered. This is confirmed by the correlation found between gray matter loss and duration of illness in the meta-regression performed by Arnone et al.1 and by the results of longitudinal studies demonstrating gray matter volume loss over time in the prefrontal cortex in young adults with bipolar disorder3 or of cross-sectional comparisons between first- and multiple-episode bipolar disorder showing more severe brain abnormalities in patients with multiple episodes of illness.4

On the other hand, we did find a significant decrease of total white matter volume in first-episode bipolar disorder, while Arnone et al.5 failed to obtain the same finding in their analysis of a larger number of studies mainly conducted in patients with chronic illness. This may indicate that alterations in white matter normal growth may constitute early and primary abnormalities in bipolar disorder, consistent with some preliminary evidence of the association between patterns of disturbed structural white matter integrity in bipolar disorder and genetic liability for the illness.6 In order to explain the lack of white matter volume reduction in chronic illness, it could be hypothesised either that other, more generalised brain changes may override white matter abnormalities over the course of the disease, or that white matter changes may be attenuated by treatment or, again, may be less sensitive to the later effects of ageing. Indirect support for this idea derives from the finding of smaller volumetric differences in the temporal lobes in bipolar disorder with increasing age, duration of illness and use of mood stabilisers,1 the only discrete brain volume including white matter analysed in the meta-regressions performed by Arnone et al.

In conclusion, the finding of different brain abnormalities in chronic v. first-episode bipolar disorder supports the notion of different pathophysiological trajectories of specific brain morphological characteristics over the course of the disease and emphasises the need for further longitudinal studies aimed at addressing specifically the issue of the time of appearance and course of individual brain abnormalities in bipolar disorder, from which may derive a better understanding of the pathogenesis of the disease itself.


Author’s reply: We are grateful to Drs Vita, Peri and Sacchetti, who raise the very important point that morphometric abnormalities detectable in first-onset bipolar disorder appear different from those described in chronic patients. An observation which, as Vita et al suggest, may underpin important information about the pathogenesis of the disorder and would benefit from clarification emerging from longitudinal studies. Prompted by their meta-analysis1 and our own work,2 we have conducted further analyses by including only patients with first-episode bipolar disorder v. healthy controls. Despite methodological differences and different inclusion and exclusion criteria, we are in agreement with Vita et al. We found no evidence of whole-brain volume reduction in the first-episode patients v. healthy controls (effect size –0.23; 95% CI –0.47 to 0.002; I² = 0, P = 0.51; Egger’s P = 0.31). This finding supports Vita et al’s hypothesis that whole-brain volume loss may be occurring with illness progression and/or its epiphenomena (e.g. number of episodes, pharmacological treatment). Similarly we found no evidence of gray matter loss (effect size –0.02; 95% CI –0.40 to 0.37; I² = 0.02, P = 0.36; Egger’s P = 0.16) but significant white matter volumetric reduction in the first-episode patients v. healthy controls (effect size –0.45; 95% CI –0.85 to –0.06; I² = 0.04, P = 0.35; Egger’s P = 0.68). These and other observations3,4 support the possibility that white matter deficits may have a particular relevance to the aetiology of bipolar disorder. However, the paucity of first-episode studies is reflected in the relatively wide confidence intervals around our estimates. Further studies of patients with first-episode bipolar disorder, as well as cohort and high-risk studies, are necessary if we are to improve our understanding of the role of structural changes in the pathogenesis of this condition.

Evolution and psychiatry

If evolution is the missing half of a ‘truly biological psychiatry’,1 the other half being biological reductionism, then value is out of the picture. But this cannot be. We do not deny the gains from biology or those that are to come (millions of people manage to live because of advances in this field). Nor are we pessimistic about the potential gains that evolution claims for mental healthcare. However, these two ‘halves’ do not make a whole. We understand the aspiration for a truly biological psychiatry: life would be easier. Biology (although a big part, or the major part of the picture) cannot (alas!) be the whole, and evolutionary theoretical considerations of disorder, natural function, design and the like cannot fill what is missing. The reason is that even if we accept a value-free account of naturally selected mechanisms, physical as well as mental, these must be considered within the spectrum of individual and social values. Fulford2 explains why values are so feared. Other theorists who have considered

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evolution in terms of disorder also accept the indisputable place of values in psychiatry. Psychiatry is conceptually and empirically more complex than the rest of medicine. Every now and then a claim is made for a concept that will push psychiatry to an exclusively biological future. But psychiatry must be the avant-garde of science, rather than the run-up of medicine, for perspectives which truly add to those complexities (empirical as well as philosophical) to do justice to themselves.1

Psychiatry’s interest in evolutionary theory is not new. The authors will be familiar with the views of Scadding, Kendall and Boorse, who all attempted to define disease in evolutionary terms. Accounts of disorder based on evolutionary theory allegedly offered the epistemological background for a value-free conceptualisation of disease. This is one way out of psychiatry’s embarrassment when comparing itself against the scientific status of the rest of medicine and the medical model. However, this seems to be a misuse of natural selection, dressed in the colours of realism in order to enhance a ‘scientific’ psychiatry.

We do not argue that evolutionary theory has no role to play. Evolutionary psychology may offer new significant ways of approaching mental disease. But let us hope that this interest in Darwin will not be a pretext for blind reductionism and a return to a fact/value dichotomy. Britain is in the front line of value-based and evolution-based considerations with the work of Fulford, Thornton,2 Bolton3 and others. Great heritage, greater present.


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Nesse1 argues that psychiatry requires both proximate and evolutionary explanations to become a fully fledged biological science. He thinks that mental disorders such as schizophrenia and depression would benefit from posing the question of whether low mood and variable social ability were adaptive traits in times long gone and are no longer of evolutionary advantage in our current environment.

I think that Nesse’s approach is as laudable as it is flawed. Evolutionary psychology proposes that most if not all human psychological traits are complex adaptations which have evolved under selective pressures. Richardson convincingly shows that the claim that all our psychological capacities have been selected for the purpose of accomplishing a particular task is too strong and that the empirical evidence required to support this claim is necessarily historical.2 The problem is, however, that the required historical evidence is hard or impossible to come by – we simply do not know what psychological traits were prevalent let alone advantageous to survive in a Pleistocene environment about which we also have little information.

For evolutionary psychology to be regarded as a credible theoretical framework it will have to be examined against standards of scientific enquiry used in other evolutionary fields such as evolutionary biology. Archaeopteryx was thought to be able to fly as it possessed feathers and claws to allow it to perch on trees.3 However, fossil records also showed that archaeopteryx lacked a sternal keel and that its awkward tail would have been likely to impede flying. Further comparative analysis showed that archaeopteryx was neither likely to perch nor be able to fly and refuted the conclusions arrived at by reverse engineering as proposed by Dennett.

Evolutionary psychology relies mainly on reverse engineering as this strategy requires comparatively few historical data but risks arriving at erroneous conclusions. Buller4 shows this to be the case for evolutionary explanations of the existence of cheater detection modules or gender differences in jealousy.

This is not to say that evolutionary psychology cannot offer an exciting and innovative framework for scientific inquiry into common mental disorders such as depression and schizophrenia but that we have to be aware of its current theoretical and methodological shortcomings and the need for further conceptual work. I agree with Geaney5 that further advances to understanding human behaviour and mental disorder would be best served by interdisciplinary cooperation whether based on evolutionary theory or not.


Author’s reply: Douzenis is concerned that adding evolution will make psychiatry narrowly biological in a way that excludes values. However, my article makes no claim that proximate and evolutionary approaches make up the whole of psychiatry, it says only that ‘biological psychiatry is making full use of only one half of biology’.1 Applying this additional biological knowledge to psychiatry should not exclude values. In fact, it offers a scientific foundation for addressing the very difficulties Douzenis mentions. It is fundamentally different from 19th-century evolutionary applications to medicine.2 It is an antidote to mindless reductionism. It helps to solve the problem of defining disease,3 and to explain why psychiatric nosology is inherently problematic.4 Furthermore, profound advances in understanding human moral capacities, with important implications for psychiatry, are coming from evolutionary analyses of their origins and functions. I encourage those who share Douzenis’ concerns to consider how evolutionary approaches can help us better understand our patients as individuals and provide personalised treatments that go far beyond analysing genes and prescribing drugs.

I am delighted that Treffurth finds my approach laudable, but dismayed that she seems to think my article is about evolutionary psychology. Evolutionary biology has vastly more to offer psychiatry than just evolutionary psychology, a field not mentioned in the article. I share Treffurth’s concerns about the
The fields of animal behaviour, behavioural ecology, and evolutionary genetics offer well-developed frameworks for understanding phenomena of core importance to psychiatry, such as the origins and functions of the capacities for emotions, attachment, and social behaviour. Darwinian medicine offers explanations for why natural selection has left us vulnerable to diseases. My argument is simple: basic knowledge from these fields is useful in psychiatry. Unfortunately, they are connected to psychiatry by only a few bridges. I hope readers will explore and build more.


**Correction**

Long-term mental health of Vietnamese refugees in the aftermath of trauma. *BJP*, 196, 122–125. The second sentence of the Method (p.122, col. 1) should read: An interview administered in the respondents’ home (by A.B.V. and T.V.T.) included a self-report questionnaire available in Vietnamese and Norwegian, and a structured face-to-face interview in Vietnamese.

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**Bringing new life into psychiatry**

Rebecca McKnight

I recently spent my elective doing psychiatry in a world-renowned hospital in the USA. I went hoping to confirm my interest in psychiatry as a career, but also as a way of avoiding the practical nature of most placements in low- and middle-income countries. I am not a ‘hands-on’ person, much preferring talking therapies to actually doing anything practical.

During my time on the in-patient unit, a patient with bulimia nervosa was admitted with hypokalaemia secondary to thrice-daily purging. This was not an unusual scenario, but this lady happened to be 34 weeks pregnant. One morning, having arrived on the ward at 6.40am to prepare for the daily rounds, I was asked to review the patient as she was having abdominal pain. From the end of the bed I could see she was sweaty, pale, and looked to be in severe discomfort. I was concerned, and asked the nurse to contact an obstetrician urgently. Moving closer I saw there was bloody fluid on the bedclothes, and the patient starting yelling she could ‘feel something coming out’. I took the plunge and asked for permission to examine her. After the usual psychiatric ward struggle to find a place to perform the examination, I performed a vaginal examination. I was alarmed to feel a head pushing down on my hand, and immediately went into the push... stop... push mode I had learnt during obstetrics. A few moments later and I had delivered the baby, which thankfully started to breathe by itself. I put the baby onto the mother’s chest, and then started to panic as to what to do next. I was saved by the arrival of a paediatrician, swiftly followed by someone with a pair of umbilical cord scissors. Now all I had to do was to sort out the fourth year resident – obstetrics was optional in her training, and witnessing her first delivery left her collapsed in a heap on the floor.

While I hated obstetrics as a student, and complained about most practical specialties, I am extremely glad the UK training system remains for the most part general and all-inclusive. I’m still heading for psychiatry, but perhaps will put a little more effort into honing my practical skills, and encouraging other psychiatrists to do the same.

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Author's reply:
R. Nesse
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