Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

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Magic bullet thinking – why do we continue to perpetuate this fallacy?

We write in response to the editorial on dietary supplementation for the treatment of attention-deficit hyperactivity disorder (ADHD). Although Rajaguru & Cooper adequately reviewed some of the literature,1 they missed an opportunity to challenge current methodologies and they simultaneously perpetuated an outdated model of disease pathophysiology.

These authors promote the idea that one single nutrient can effect a consistent change in ADHD symptoms. However, such magic bullet thinking is at odds with human physiology (which requires the ingestion of many nutrients in balance) and largely explains why the single-nutrient strategy has yielded only modest benefits.2 Supplementing with broad-spectrum formulations ensures the patient’s safety as the combination prevents imbalances, such as one nutrient causing a deficiency in another (e.g. taking zinc alone may cause copper deficiency). Recommending that researchers first understand how one nutrient functions on its own, in isolation, ignores the inevitable changes and potential for harm occurring in other nutrient levels.

In the authors’ brief review of nutrients as part of a complex formula, they failed to highlight that this multi-ingredient method has the potential to be more beneficial for treating mental health symptoms than any one nutrient.3 The concept underlying the use of micronutrients for the amelioration of mental health symptoms is that mental illness may be a manifestation of suboptimal nutrition, relative to genetically mental health symptoms is that mental illness may be a

Authors’ reply: We thank the authors for their comments but would contest the view that our editorial actively promotes the premise that a single nutrient could consistently ameliorate attention-deficit hyperactivity disorder (ADHD) symptoms. We have summarised the literature to date and highlight that the single-nutrient literature is complicated by methodological heterogeneity and inadequately rigorous study designs, making overall interpretation difficult. But before dismissing the potential contribution of single agents in isolation it should be borne in mind that such methodological factors, as well as a potential genuine lack of effect, may also go some way to explaining why single-nutrient approaches have not yielded notable benefits.

Attention-deficit hyperactivity disorder is a complex, multifactorial disorder and we agree with Rucklidge et al that serum levels may not reflect the complex metabolic requirements of the brain. Indeed, we emphasise in our article that it is unclear whether serum levels considered most favourable for general health would be optimal for improving brain function in ADHD, and that it needs to be further understood how we would determine such levels. However, caution should be exercised in dismissing the importance of serum levels, as this lack of clarity and the interlinked action between nutrients that Rucklidge et al highlight, whereby supplementing with one alone may lead to decreases in another, make it even more important to measure a range of serum levels when investigating supplements in the absence of baseline levels below standard reference ranges. Whatever the optimal levels of nutrients are for brain functioning in ADHD, ‘flooding the system with high doses of nutrients’ has the potential to confer risk to physical health. Potentially serious adverse outcomes of nutrient excess are documented1,2 and, although nutritional interventions can be perceived as safer than stimulants, we should first ensure we are doing no harm. There
is no conclusive evidence to date that suboptimal nutrition is an aetiological factor in mental illness in general; however, dietary factors are biologically plausible agents and as such the field would certainly benefit from carefully designed trials, be that of single nutrients or of nutrient complexes.


Correspondence

Miriam Cooper, Child & Adolescent Psychiatry Section, Cardiff University School of Medicine, Cardiff, UK. Email: cooperml1@cardiff.ac.uk; Priya Rajyaguru, Child & Adolescent Psychiatry Section, Cardiff University School of Medicine, Cardiff, UK

doi: 10.1192/bjp.203.2.154a
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Julian J. Rucklidge, Jeanette Johnstone and Bonnie J. Kaplan
Access the most recent version at DOI: 10.1192/bjp.203.2.154

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