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Present and future trajectories towards a possible valid and useful diagnosis of ADHD

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To date, diagnosing Attention Deficit Hyperactivity Disorder (ADHD) remains indeed one of the most controversial issues in contemporary psychiatry and behavioural sciences. Most of the conceptual problems regarding the validity of this diagnostic category arise from the heterogeneity of syndromal pictures and the high rate of comorbidity observed in subjects diagnosed with ADHD at all stages of the longitudinal course of the disorder (Wilens, Biederman and Spencer, 2002). In this regard, DSM 5 increased complexity by allowing a diagnosis of comorbidity between ADHD and autism spectrum disorders while these two diagnoses were mutually exclusive in DSM-IV-TR.

In 2009, a very interesting dialogue on the methodological limits of scientific studies aimed at clarifying the nosographic status of ADHD appeared on this journal (Thurber 2009, Tait 2009, De Morais Ribeiro 2009). In both the original paper (Thurber et al. 2009) and the commentaries (Tait 2009, De Morais Ribeiro 2009) is highlighted the need for a more rigorous "bottom-up" approach based on scientific observation and testing in order to formulate diagnostic entities that can be both empirically valid and replicable. With respect to this, Tait questions the validity of the concept of "scientific method" itself (Tait, 2009), while De Morais Ribeiro and colleagues tackle the very important issue of the practical implications of a debate, yet scientifically pertinent, on whether ADHD is a hypothetic construct or a nosologic condition (De Morais Ribeiro and Cavalheiro da Silveira, 2009).

Indeed, medicine is a practical discipline. Therefore knowledge, within a medical context, should also be applied to practical problems. For this reason, Kendell and Jablensky suggested a distinction between the concepts of "validity" and "utility" of a psychiatric diagnosis. The articulate conclusion of their paper reads:

> "At present there is little evidence that most contemporary psychiatric diagnoses are valid, because they are still defined by syndromes that have not been demonstrated to have natural boundaries. This does not mean, though, that most psychiatric diagnoses are not useful concepts. […] statements about utility must always be related to context, including who is using the diagnosis, in what circumstances, and for what purposes." (Kendell and Jablensky, 2003).

In fact it is clear that, although most diagnostic concepts in psychiatry have not proven valid, many of them are useful for clinicians for the information they are associated with (e.g. about outcome and treatment response). For example, in the case of ADHD a diagnosis in childhood/adolescence is associated with a significantly higher risk of developing a substance use disorder and with significantly worse young adult educational and vocational outcomes (Mannuzza et al., 1993; Capusan et al., 2016). Furthermore, clinical response to stimulant drugs in ADHD is around 70-80% even in adults (Spencer et al., 2005), which is significantly higher than response rates to pharmacological treatments usually observed in all the other neurological or

psychiatric disorders.

However, I reckon that Thurber and colleagues correctly pointed out that the existing brain-based causal models were not adequate to provide rigorous supporting data coming from testing falsifiable hypotheses.

In this context, a re-opening of the dialogue is definitely warranted as new evidence on ADHD was provided since 2009. Of course, a thorough review of such evidence is not possible in a commentary. Therefore, I will focus on some important longitudinal neuroimaging studies that have helped clarify the peculiar way neuro-development unfolds in ADHD compared to typically developing subjects. Such studies, conducted on ADHD as on the other childhood psychiatric disorders, suggest that subjects with a diagnosis of ADHD which is confirmed by repeated longitudinal assessments is associated with a peculiar anomaly of brain cortical developmental trajectories (Shaw, Gogtay and Rapoport, 2010). In particular, ADHD is characterized by a delay in cortical maturation of key brain areas, predominantly the prefrontal regions of the cerebral cortex, and this remains associated with the severity of inattentive symptoms at the transition between late adolescence and young adulthood (Shaw et al., 2007; Shaw et al., 2012; Shaw et al., 2013). Finally, in ADHD remission is associated with convergence to the template of typical development, whereas persistence is accompanied by progressive divergence away from typical trajectories both in developmental age and adulthood (Shaw et al., 2009; Shaw et al., 2013).

The studies providing this evidence have been conducted by clinically assessing and scanning multiple times through childhood, adolescence and young adulthood large samples of subjects diagnosed with ADHD and typically developing subjects over a period of 20 years. Although the above described results display a high grade of "objectivity", this does not allow etiopathogenetic inferences. In fact, given the plasticity of the human central nervous system, it is impossible to conclude whether the peculiar, delayed cortical developmental trajectory seen in ADHD is pre-determined or results from a lifelong and very complex brain-environment interaction.

However, the aforementioned studies are a good example of how an empirically valid approach based on scientific observation can be used to start validating a diagnostic concept and/or providing therapeutic/prognostic indices. In fact, the peculiar shape of the cortical thickening and thinning process as longitudinally measured by magnetic resonance imaging has indeed a negligible probability of being influenced by the formal diagnosis a given subject has received.

Of course these results, yet very promising, need replication, and I am more than inclined to believe that the researchers who are starting to make this possible do not disdain to adopt "Nullius in verba" as their motto.

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