LETTER TO THE EDITOR

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PATHOLOGICAL EVIDENCE SUPPORTING
THE ROLE OF NEURO-HORMONAL REGULATION
IN HUMAN COGNITIVE ABILITIES

The nature of human intelligence and its pathological markers has received considerable attention among evolutionary scientists. One theory purported by Kirschner and Gerhart, (1998, Jones et al. 2007, Gualtieri 2014) is that disorders such as schizophrenia and autism are an outcome of evolvability. During hominin brain evolution there seems to have been considerable genomic reorganisation generating non-uniform neurodevelopmental patterns and duplication rates (Gualtieri 2014). However, such evolutionary neurodevelopment probably had an adverse evolutionary cost (Gualtieri 2014).

Several studies provide convincing evidence to support the notion that intelligence is considerably dependent on neuro-hormonal regulation comes from neuropathology. A strong association between lower intelligence and schizotypal tendencies has been established (Zammit et al. 2004). Similarly, these individuals have been shown to possess relatively lower concentrations of certain neurotransmitters, namely dopamine DA within the mesocortical pathway (Knable, Weinberger 1997). In the prefrontal cortex, the working memory is modulated via the D1 receptors and it has been found that overt or insufficient stimulation of the D1 receptors in the prefrontal cortex can impair working memory (Takahashi et al. 2012), and might cause negative symptoms and cognitive deficits in schizophrenia (Goldman-Rakic et al. 2004). This speculation is further supported by the phenotypic presentation of major depressive disorders. In accordance with the mono-amine hypothesis of depression, the underlying pathophysiological mechanism is attributable to decreased concentrations of serotonin and noradrenaline within the synaptic clefts (Hirschfeld 2000). As dictated by the universally accepted diagnostic and statistical manual of mental
disorders, amongst the clinical manifestations of a depressive episode is an impaired ability to think or concentrate (American Psychiatric Association 2000). Consequently, modest endowments of these crucial stimulatory neurotransmitters can significantly inhibit cognitive function whereas greater concentrations are associated with higher intelligence.

While current research into neuropathological markers is improving, we are still a long way from understanding their evolutionary antecedents and current selective pressures informing the human genome. Such an understanding is vital due to the exponential increase of mental disorders world-wide; second, because of the possible consequences of future genetic engineering (i.e. human/non-human recombinant DNA) which may lead to further irregular neuro-hormonal reorganisation and regulation.

REFERENCES


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