

differences in the relationship between measured IQ and latent intelligence). A lack of predictive bias does not mean there is no measurement bias, and under certain conditions may even imply measurement bias [2].

- (2) Malloy thinks it “unlikely that confounding differences in preadoption experiences” explain the Minnesota Transracial Adoption Study’s results, partly because of a meta-analysis that found no significant effects on IQ from age at adoption or pre-adoptive abuse. In fact it found appreciably different effect sizes (–0.5 vs. 0.4) associated with different adoptive ages. The difference is statistically insignificant, but it’s impossible to say whether that is down to a lack of effect or a lack of statistical power, so the meta-analysis is incapable of ruling out an adoptive age confound. Either way, possible confounds of race with psychological adjustment problems, prenatal environment and nonshared environment remain unaddressed.
- (3) That the Flynn effect “is not a gain in real *g* factor intelligence” may mean only that IQ gains are subtest-specific. Deciding instead that the gains are just meaningless measurement bias is risky, because that concedes that IQ tests do not measure the same traits in the same way over time. In that case comparing or pooling IQ measurements

taken at different times would be dubious, which would suggest Malloy’s mistaken in trying to assess African IQ by averaging estimates taken up to 58 years apart.

- (4) The origins of the Flynn effect and of the European–African IQ difference are not “unrelated questions”. One argument for racial IQ differences having a partly genetic origin is that high heritability of IQ precludes plausible environmental accounts of such differences. The Flynn effect falsifies this argument by revealing large, non-genetic IQ differences between populations [3].

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Vascular endothelial growth factor and its receptor may be one molecular mechanism for androgenetic alopecia

Androgenetic alopecia (AGA), or male pattern hair loss, is the most common form of alopecia in men, affecting approximately 50% of the male population. AGA is an androgen-related condition in genetically predisposed individuals [1]. To date, the exact molecular mechanism is yet to be established. In his article published in the March 2004 issue of *Medical Hypotheses*, Hernandez nicely emphasized the importance of dihydrotestosterone (DHT) in the pathogenesis of androgenetic alopecia and proposed the hypothesis that DHT exerts its effect on the scalp via structural and anatomical changes on the vasculature [2]. We would like to further add that the balding as a result of the direct effects of DHT on the pilosebaceous unit or indirect effects of DHT on the vasculature supplying the scalp may be caused via vascular endothelial growth factor and its receptor (VEGF/VEGFR) on the molecular level.

Vascular endothelial growth factor is one of the most important players that regulate vessel formation during embryonic development, in wound healing and in maintaining vessel homeostasis in adult organisms [3]. In addition, impaired vessel function resulting from defects in VEGF ligands or receptors is the cause of many diseases. Goldman and colleagues demonstrated that the expression of VEGF in human alopecia follicles significantly decreased comparing to the normal follicles [4]. Minoxidil, one of the pharmaceutical treatments approved for the therapy of AGA in men, could promote hair growth through upregulating the expression of VEGF in hair dermal papilla cells (DPC) [5]. Furthermore as an autocrine growth factor for hair dermal papilla cell, VEGF could stimulate the proliferation and migration of DPC [6]. Current data has shown that androgen receptor was expressed not only by vascular endothelial cells but also by DPC from balding as well as non-balding scalp [7].

And in different cell types, androgens may increase androgen receptor protein expression.

By combining the above information, it is logical to propose that DHT may affect hair growth via VEGF/VEGFR. In vessels supplying the scalp, DHT binds to the androgen receptor of endothelial cells after the conversion of testosterone to DHT via 5α -reductase. Afterwards, on the one hand, DHT reduces the production of VEGF resulting in less vascularization of hair follicle; on the other hand, DHT effects structural and anatomical changes in the vasculature, which result in diminished blood flow to the pilosebaceous unit and subsequently diminished hair growth. In hair follicle, the binding of DHT to androgen receptor down-regulate the expression of VEGF in DPC, shortening the duration of anagen and subsequently leading to hair loss.

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The role of serotonin in viral hepatitis – Depletion of plasma tryptophan as a potential option to reduce virus persistence and immunopathology?

According to Lang and colleagues serotonin (5-HT) plays a decisive role in the underlying pathophysiology of viral hepatitis [1]. Platelet-derived vasoactive 5-HT in mice infected with the noncytotoxic lymphocytic choriomeningitis virus, resulting in virus-induced CD8⁺ T cell-dependent immunopathological hepatitis, supported virus persistence in the liver and aggravated virus-induced immunopathology [1].

As regards 5-HT functioning in humans, a modification of the rapid tryptophan depletion (RTD) procedure in accordance with Moja et al. [2] called Moja-De was developed. Moja-De can be administered to humans within an amino acid (AA) drink, enabling a lowering of central nervous 5-HT synthesis by decreasing the central nervous availability of tryptophan (TRP), the physiological precursor AA of 5-HT. The AA then compete with endogenous plasma TRP on the uptake over the blood brain barrier by using the same active transport system [3–5]. The diminished availability of TRP in the central nervous system then results in a reduction of central nervous 5-HT synthesis of approximately 90%. Additionally, endogenous TRP is used for increased protein synthesis induced by the AA by taking TRP from plasma stores, including platelets [6].

Therefore we would like to hypothesise that depletion of peripheral TRP in plasma could contribute beneficially to treatment in virus-induced hepatitis as regards decreased virus persistence in the liver and virus-induced immunopathology. In the newly developed RTD procedure Moja-De the AA of this procedure were administered to body weight of the patients as there was a positive relationship between plasma TRP and body weight [7]. The modifications made to the original RTD procedure by Moja et al. [2], in particular the administration of the AA according to as well as the changed quantities of the AA, resulted in acceptable tolerability of the RTD Moja-De procedure. This finding is of particular significance as in past RTD studies a frequent problem was that side-effects in terms of vomiting and nausea occurred [6]. Supporting this, this newly created RTD procedure Moja-De was already successfully used in children and adolescents with attention-deficit/hyperactivity disorder (ADHD, see [5]), and was also hypothesised to have beneficial therapeutic effects as regards manic symptoms [8]. Because of potential central nervous side-effects to this clinical application of RTD (i.e. mood problems, aggression, for a summary see [6]) we would like to suggest a further RTD modification with potentially reduced central nervous effects but also satisfactory depletion of TRP in plasma, i.e. by changing the AA concentrations thus potentially

resulting in a diminished TRP uptake over the blood brain barrier and increased protein synthesis in the liver.

In summary, dietary administration of AA resulting in a reduction of plasma TRP and therefore peripheral 5-HT concentrations, in particular because of increased protein synthesis in the liver and use of endogenous TRP from plasma stores, could have a beneficial therapeutic effect in patients suffering from infections with viral hepatitis. A RTD modification could provide clinical advantages as regards 5-HT related central nervous side-effects due to reduced 5-HT availability in the brain.

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