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Did human hairlessness allow natural photobiomodulation 2 million years ago and enable photobiomodulation therapy today? This can explain the rapid expansion of our genus's brain

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ABSTRACT

Present hypotheses to explain human hairlessness appear to be inadequate because hairlessness is not accompanied by any immediate benefit. A new, testable, hypothesis is advanced to explain our hairlessness based on photobiomodulation research, also known as low-level light therapy. This shows that red and near infrared radiation has a very beneficial effect on superficial tissues, including the brain. Random mutation/s resulting in complete hairlessness allowed early humans to receive daily doses of red and near infrared radiation at sunset. Photobiomodulation research shows this has a twofold effect: it results in increased mitochondrial respiratory chain activity with consequent ATP 'extrasynthesis' in all superficial tissues, including the brain. It also advantageously affects the expression of over 100 genes through the activation of transcription factor *NFkB* which results in cerebral metabolic and haemodynamic enhancement. It is also possible that melanin can supply electrons to the respiratory chain resulting in ATP extrasynthesis. These effects would start automatically as soon as hairlessness occurred resulting in a selective sweep of the mutation/s involved. This was followed by the very rapid brain evolution of the last 2my which, it is suggested, was due to intelligence-led evolution based initially on the increased energy and adeptness of the newly hairless individuals.

Introduction

Present hypotheses [1,2] attempting to explain human hairlessness seem inadequate; they do not confer any immediate benefit on the hairless, neither do they do anything to explain the explosive increase in volume of the human brain over the last 2my – see Fig. 1. The question of hairlessness has been a problem since the time of Charles Darwin [1].

The present leading theory, proposed by Jablonski [2,3], suggests that the human lineage had become hairless by 1.6mya and that hairlessness allowed the development of additional sweat glands to permit evaporative cooling in times of heat stress. Jablonski envisages hairlessness occurring as a gradual process so that as body hair decreased, the density of sweat glands increased [4]. It could perhaps be seen as a gradual adaptation which mitigated heat stress as hairlessness increased.

Jablonski and Chapman [3,4] have also pointed to evidence for photolysis of the vitamin folate by ultraviolet radiation (UVR). UVR is higher in the tropics and at higher altitudes. A recent investigation in Brisbane, Australia, at latitude 27° S has confirmed a significant, up to 20%, reduction in folate levels by UVR photolysis in women of child-bearing age [5]. This is despite a contrary finding [6] from Oslo (latitude 60° N) – possibly latitudinal UVR differences played a part. Deficiency of folate in pregnancy is linked to neural tube defects and anencephaly at birth, and to megaloblastic anaemia in later life, which has led to the folate fortification of food in developed countries.

When hairlessness occurred in Africa, Jablonski assumes the bare skin to have been a pinkish colour [2] so that the development of a "dark skin was presumably a requisite evolutionary follow-up to the loss of our sun-shielding body hair". If so, it would be protected by immediate pigment darkening, which has been shown to be due to ultra-violet-A-mediated expression of the visual photopigment rhodopsin in skin melanocytes, resulting in the rapid formation of melanin [7]. This is followed by a delayed tanning reaction [4]. The iconic ebony skin of the African was then set in

Abreviations: COX, cytochrome C oxidase; ETC, electron transport chain; LED, light emitting diode; LLLT, low level light therapy; NO, nitric oxide; Oxphos, oxidative phosphorylation; PBM, photobiomodulation; ROS, reactive oxygen species; R&NIRR, red and near infrared radiation; RBC, red blood cell; SAD, seasonal affective depression; SNiP, single nucleotide polymorphism; UVR, ultraviolet radiation; VDR, vitamin D receptor.

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stone by a mutation at the *MC1R* locus which has persisted to this day; but the high number of non-synonymous mutations at the *MC1R* locus in Eurasians compared with Africans is seen as evidence of adaptive evolution to a paler skin in Eurasians [8] to maintain adequate vitamin D synthesis.

Looking for alternative solutions to explain human hairlessness, one might wonder whether vitamin D could be involved. Its synthesis starts when UVB of 297 nm interacts with 7-dehydrocholesterol in the skin so that levels of this essential nuclear hormone would presumably have increased with hairlessness. The vitamin D receptor (VDR) is a ligand activated transcription factor which is widespread throughout the brain, skin and other tissues. Calcitriol, the end product of vitamin D synthesis, binds to the VDR thereby affecting the expression of hundreds of other genes [9,10] particularly in the brain. Vitamin D deficiency has long been known as the cause of rickets, but deficiency can also cause a host of non-classical effects such as painful weakness of the proximal muscles [11] and an almost four times greater chance of requiring Caesarian section in childbirth [12]. Vitamin D tends to suppress innate immunity and early life deficiency may be associated with later autoimmune conditions [13].

Calcitriol stimulates neurogenesis in the developing rat brain and promotes myelination, synaptogenesis and neurotransmitter release through its control of intracellular calcium [14]; VDRs have been identified in oligodendrocytes and Schwann cells, both of which are involved with myelin formation. Through its promotion of neurotrophin secretion and control of intracellular calcium, calcitriol influences development and function of the neocortex as well as the more primitive areas of the brain [14].

Two studies [15,16] found correlations between low vitamin D levels and cognitive decline in the elderly while a recent larger study has extended the correlation to include all forms of dementia [17]. Schizophrenia is significantly more common in dark skinned migrants to higher latitudes, and in urban environments. Infants born in winter and spring are significantly more prone to later schizophrenia, possibly connected to low levels of vitamin D in the last trimester of pregnancy, or to maternal winter viral infections such as influenza. In rats, gestational vitamin D deficiency can cause neurological abnormalities suggestive of schizophrenia [18]. Concluding their in-depth review, McCann and Ames [14] said: "Evidence that vitamin D is involved in brain development and function is strong." Vitamin D has played a part in brain evolution, particularly in the development and connectivity of the huge number of additional myelinated neurons required to form the neocortex of modern humans – see Fig. 1.

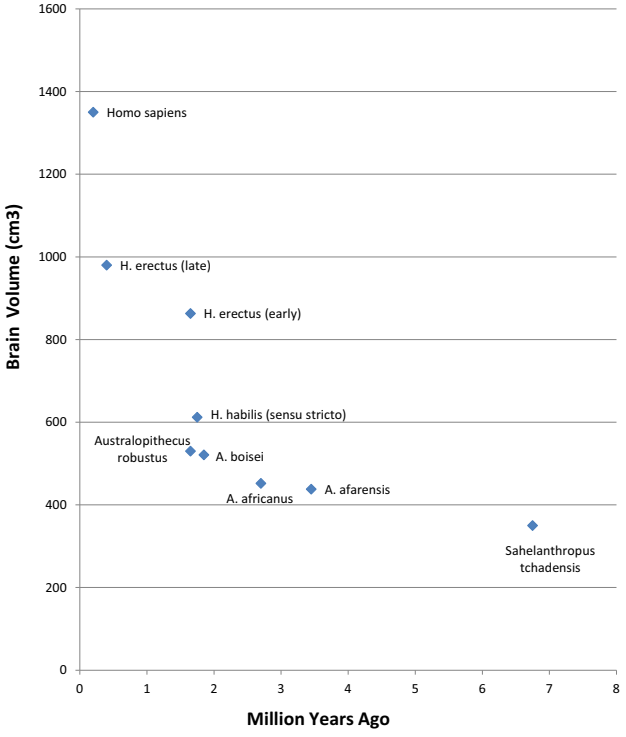

Fig. 1. Data from Table 2 of Leonard et al. [61] expressed graphically using the midpoints of the quoted geological ages for the various species and with the addition of sahelanthropus. With grateful acknowledgment to Prof. W. Leonard.

It is certainly beneficial to have additional mechanisms, such as sweating, to keep the brain temperature within optimal limits. However, the additional eccrine glands were not a direct response to hairlessness but perhaps an adaptive response to overheating on the expanding savannah. Hairlessness itself has downsides (see below) so that such a momentous event would necessarily need to be accompanied by immediate beneficial consequences otherwise the hairless mutation/s would quickly succumb to negative selection. Jablonski suggests [2] that "naked skin itself played a crucial role in the evolution of other characteristic human traits, including our large brain…" but does not explain exactly how this may have occurred. It seems unlikely that an increase in eccrine glands could, in itself, account for the great increase in brain size that occurred in our genus in the past 2my – see Fig. 1. Likewise, although increased levels of vitamin D would be advantageous for building a large brain, it would take many generations before the benefits would be apparent, by which time any evolutionary pressure to maintain hairlessness would have long disappeared. So increased levels of vitamin D were fortuitous and its genetics do not appear to have played any role in the initiation of hairlessness. Neither the evaporative cooling theory nor the vitamin D scenario confers an immediate benefit. A theory is required where some immediate advantage occurs *concomitantly* with hairlessness. Such a theory is elaborated in the rest of this paper; this theory can explain how hairlessness initiated the huge increase in the brain volume of our genus over the past 2my [Fig. 1].

In mammals the energy required to maintain body temperature is generated by the catabolism of energy stores and presumably hair in mammals developed as an adaptation to conserve endothermic heat. So to maintain temperature after the loss of insulating hair will entail an energy deficit when the ambient temperature falls much below 37 °C such as in Africa 2mya during cold nights on the increasingly open savannah – or even in a tree or a cave. How could such an apparently deleterious genetic mutation have been so successful? What beneficial attributes could hairlessness possibly confer?

Could hairlessness perhaps allow a type of light-mediated energy synthesis? This is not such an outlandish idea – indeed it seems the only plausible explanation. It is already known that low levels of red and near infrared radiation (R&NIRR) acting on hairless human tissue can stimulate the production of ATP, the universal energy currency of life on our planet. Low levels of R&NIRR are the basis for the non-invasive medical treatment known as photobiomodulation (PBM) [19], or low-level light therapy (LLLT). PBM should not to be confused with photodynamic therapy or with the burgeoning invasive science of optogenetics. In the correct dosage, PBM appears to be beneficial in a wide range of conditions such as wound healing, osteoarthritis and tendonopathies [19], also myocardial infarction [20] and particularly in neurological conditions such as neck pain [21], and, transcranially, for embolic stroke [22], severe depression [23] and chronic traumatic brain injury [24] – more later. The effects of PBM are however biphasic or hormetic – if the irradiance is increased beyond an optimal point, the benefits can decrease. PBM has similarities with other non-invasive therapies: transcranial magnetic stimulation and

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