Response to Jablonski and Chaplin

Mel Greaves
Centre for Evolution and Cancer, The Institute of Cancer Research, London, UK

The purpose of my paper [1] was to suggest that skin cancer, and particularly squamous cell carcinoma (SCC), is a plausible candidate selective force in the evolution of black pigmentation in early humans. The argument was advanced in part to counter the cursory dismissal of cancer as a selective force by Blum 50 years ago, and by Jablonski and Chaplin and others since. Jablonski and Chaplin now acknowledge that many factors could contribute to the evolution of black pigmentation but assert that skin cancer was not one of them, i.e. they claim it is implausible.

Jablonski and Chaplin restate the Blum argument [2] that skin cancers occur too late in life to impact on reproductive output. The point may be based on Peter Medawar’s well-founded view that evolutionary selection is most effective when it impacts at ages before or during reproductively active life [3], but it ignores the contextual setting we need to consider—age-associated cancer risk of unclothed, unprotected individuals living in the open savannah of Africa with very high, all year round ultraviolet radiation (UVR) exposure. Additionally, natural selection could operate post-reproductively via the ‘grandmother’ effect on kin selection and inclusive fitness [4].

OCA2 albinism is by no means a perfect proxy for the condition of early human skin. But this type of albinism is not a complete loss of melanin pigmentation. OCA2 albinos have some ability for facultative tanning and their skin UVR transmission quality is said to be similar to that of Caucasian white skin. Jablonski and Chaplin assert that in early human evolution, reproductive activity would have largely ceased before lethal cancer could have had an impact, even if the albino model was valid. We know nothing of the reproductive habits of early humans. However, contemporary hunter–gatherer females have a first birth at 19–20 years. The average number of children born is five but, with birth intervals of approximately 4 years, mainly owing to protracted breast feeding and suppressed ovulation, reproductive activity lasts for around two decades [5,6]. Lethal skin cancer if it occurred in early hominins before the age of 40 (as in albinos) would certainly have an impact on reproductive success. Even if an allele had only a modest effect on reproductive output, it could, over time, undergo a selective sweep or reach fixation. Evolutionary selection does not, as Jablonski and Chaplin suggest, require certainty of outcome.

It is certainly possible that early humans paralleled chimpanzees (facial skin) in being born white but capable of facultative tanning on exposure and this could well have been more marked than in OCA2 albinos. Whatever tanning capacity early human skin had, it was clearly insufficient to obviate the imposition of strong selection for the MC1R black pigmentation allele. Jablonski and Chaplin implicitly suggest therefore that death from systemic folate loss is a much more sensitive read-out of UVR exposure of tanned skin than cancer. Where is the evidence?

Jablonski and Chaplin note that apes are not afflicted by skin cancer. The paper quoted [7] is a literature review and refers exclusively to primate species in zoos or research establishments (and rarely as pets) so its relevance is doubtful. However, the paper explicitly refers to malignant skin cancers in primate species including, interestingly enough, melanoma in an albino gorilla. Even if it were true that chimpanzees and other feral ape species in equatorial Africa rarely developed skin cancer, this would not be very informative as evidence; they live in forested areas with only dappled light exposure to part of the face.

SCC, Jablonski and Chaplin suggest, cannot have been a selective force because skin pigmentation is most highly correlated to autumnal, not peak, UVR levels. It is unclear what relevance this has to pigmentation and exposure...
in equatorial regions where UVR exposure is, by the authors’ own studies, non-seasonal and high all year round. I agree with Jablonski and Chaplin that several UVR-associated pathologies could have contributed to the emergence of the MC1R allele determining black pigmentation. For example, deficient DNA repair via folate depletion in the skin, burning, infection and ulceration of tumours could have a compound impact on morbidity and survival. Systemic folate loss and neural tube defects in newborns, which Jablonski and Chaplin favour, is plausible but the currently available evidence is rather conflicting and less than compelling. Disentangling the relative contribution of different selective forces historically responsible for skin coloration more than a million years ago is difficult and perhaps formally impossible. But it makes little sense to continue to exclude skin cancer from the mix.

References


