OPEN ACCESS

GAVIN PUBLISHERS

Advances in Biochemistry and Biotechnology

Review Article

Riley PA. Adv Biochem Biotechnol 3: 173.
DOI: 10.29011/2574-7258.000073

Radiative Heat Loss in Relation to Evolutionary Aspects of Melanin Pigmentation in Man

Patrick A. Riley*

Totteridge Institute for Advanced Studies, The Grange, London, UK

***Corresponding author:** Patrick A. Riley, Totteridge Institute for Advanced Studies, The Grange, Grange Avenue, London N20 8AB, UK. Tel: +442084455687; Email: p.riley@ucl.ac.uk

Citation: Riley PA (2018) Radiative Heat Loss in Relation to Evolutionary Aspects of Melanin Pigmentation in Man. Adv Biochem Biotechnol 3: 173. DOI: 10.29011/2574-7258.000073

Received Date: 10 August, 2018; **Accepted Date:** 20 August, 2018; **Published Date:** 28 August, 2018

Abstract

The degree of epidermal melanisation in humans is discussed in relation to the possible evolutionary benefits that may be associated with the known properties of the pigment. These include photo protection both by radiation absorption and free radical scavenging, genoprotective effects through the oxidative initiation of apoptosis, and heavy-metal binding and excretion. An aspect that has hitherto received little attention is the possibility that the degree of melanisation may influence the efficiency of heat loss and contribute to temperature homeostasis.

Keywords: Emissivity; Evolution; Infra-Red Radiation; Melanin

Introduction

Within the wide range of living organisms there are many examples of melanism and a number of diverse functions are subsumed by the dark pigment. In general, we may define melanin as a polymerisation product of orthoquinones. The biological significance of orthoquinones in microorganisms, plants, insects and cephalopods is well recognized [1] and the properties of melanin suggest several functions that may have evolutionary significance [2]. The general properties of melanins are well-established. Minor properties include the formation of charge-transfer complexes with certain drugs and photon-phonon absorption, but basically the major functions of melanins include: (a) absorbance of light over a fairly wide range of wavelengths; (b) facile redox properties associated with stabilization of free radical (semiquinone) states; and (c) powerful cation binding through negatively charged functional groups.

Absorbance of light is of great interest because of the photoreceptor shielding that is afforded by melanin which is used in primitive organisms to confer directionality on phototropism. In animals with eyes, light absorption by melanin is important in increasing visual acuity and adjusting exposure in light adaptation. Absorption of infra-red radiation by melanin is also of importance in temperature control in poikilotherms and animals in desert habitats. Melanin is also a major pigment visible in various external displays, either transient signalling or camouflage (as in flat fish and chameleons), or more permanent social recognition signals.

In relation to surface pigmentation in humans there are two evolutionary matters that merit examination: (i) What are the advantages of epidermal melanin that led to the development of black skin; and (ii) What factors were responsible for the geographic loss of skin pigment?

Development of Black Skin

Because of the evolution of relative hairlessness in man, and the recognition that the ultraviolet component of solar radiation is biologically harmful, it has generally been assumed that the pronounced increase in epidermal pigmentation in humans in comparison with other land-based mammals, including apes, evolved as a photo protective feature [3].

This photo protection may occur in two ways. Probably the most evident contribution of melanin is afforded by dissipative energy absorption and by scavenging free radical products generated in skin by incident radiation. Alternatively, the pigment may function as a genoprotective agent. An interesting possibility is that cellular calcium levels might be regulated by absorbed light since photo oxidation of melanin shifts the balance of catecholic to quinone functions which would release calcium ionically bound to catechols. We have shown that calcium bound to catechols is released on oxidation [4]. We speculate that, since the intracytoplasmic calcium level is a controlling signal in the initiation of apoptosis [5], cell death will be initiated by high levels of photo-oxidation giving rise to 'sunburn cells' in irradiated epidermis. Such a mechanism may contribute to genoprotection by ensuring that cells exposed to radiation of sufficiently high intensity to suffer mutations are eliminated by apoptosis.

An alternative explanation of the possible evolutionary role of epidermal melanin has been proposed in connection with the aquatic ape hypothesis [6,7] in which the metal chelating action of melanin would provide a method of excretion of potentially toxic metals from the littoral diet rich in molluscs which concentrate heavy metals [8]. According to this proposal human evolution, as a result of niche displacement from an overcrowded arborial habitat, took place at the water's edge. Such a habitat implies a diet rich in seafood which would include a high proportion of shellfish which are comparatively easily caught without much skill or sophistication. Shellfish are unusual in being able to concentrate metals, including potentially toxic heavy metals [9]. Thus, there would seem to be an evolutionary advantage accruing to those individuals with good metal-excreting pathways. Because of the metal-binding propensity of melanin it is possible that skin pigmentation represents a trans epidermal route of metal excretion. Melanin synthesized by melanocytes is transferred to epidermal keratocytes where it binds potentially toxic cations and is desquamated. Given the large surface area of the skin, with strong melanization and rapid epidermal turnover, this excretory route could be highly significant. It is conceivable, therefore, that the evolutionary origin of epidermal pigmentation was driven by diet rather than solar exposure [10].

Loss of Melanisation at Greater Latitude

According to this argument the loss of pigmentation in early human populations migrating to more Northerly latitudes would be secondary to changes in the staple diet. However, whilst the dietary argument may hold with regard to the acquisition of epidermal pigmentation, it is not a strong selective factor when the diet changes, and a more compelling explanation must be sought for the loss of epidermal melanisation.

Currently the most popular explanation regarding the evolutionary loss of skin pigmentation with increasing latitude is the Vitamin D hypothesis [11] which posits that the UV absorbance by epidermal melanin reduces the extent to which Vitamin D can be synthesized in the dermis. If the major source of Vitamin D were dermal photosynthesis, then heavily melanised individuals would be prone to rickets.

Radiative Heat Loss

However, another factor that hitherto has received scant attention is the potential radiative heat loss from pigmented skin. In a humid environment, where heat loss by sweating is not efficient, the thermostatic advantage of radiative (infra-red) heat loss from the skin surface might confer a significant advantage. It is estimated that about two thirds of the thermal losses in man are due to emissions in the 10 µm wavelength range.

Remarkably there have been few studies designed to measure the extent of infra-red emissions in relation to the degree of epidermal pigmentation. The most convincing data come from thermographic studies of zebras which demonstrate that the black stripes have higher infra-red emission than white stripes (see Figure 1). In a systematic survey of the geographical distribution of African zebras it was concluded that the most probable function of the black stripes was connected with temperature regulation [12]. This radiative mode of heat loss might historically have been advantageous to early humans in pursuing game and might account for the evolution of epidermal pigmentation.

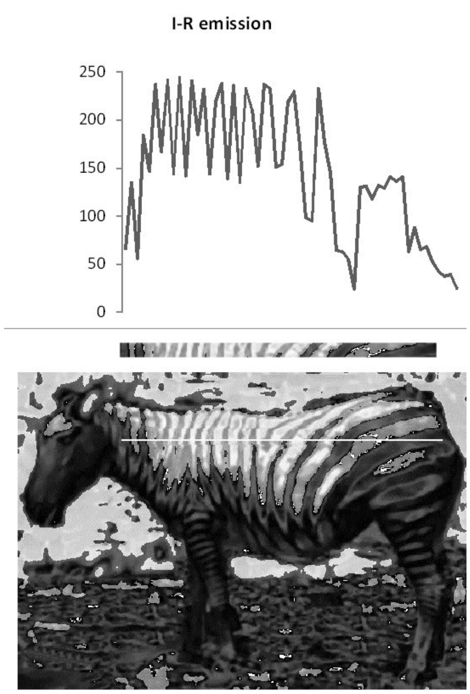

Figure 1: Zebra thermography showing emission peaks associated with the black stripes. The difference in infra-red emission along the line indicated is plotted in the upper panel.

Some evidence relevant to this aspect comes from the finding that cold weather injuries are more prevalent in pigmented individuals. Candler and Ivey [13] reported that African-American soldiers were significantly more susceptible to frostbite of the distal extremities than Caucasian soldiers with a relative risk score of 3.94 (95% confidence interval 2.77-5.59). A subsequent study of 2143 hospitalizations due to Cold Weather Injury (CWI) similarly showed that African-American men and women were approximately 4 and 2.2 times more likely to be affected than their Caucasian counterparts [14]. These findings suggest that epidermal melanin enhances peripheral heat loss, presumably by radiative means. Such an argument would be consistent with the notion that epidermal pigmentation would be disadvantageous in colder environments and could explain the evolutionary loss of pigmentation in populations migrating out of Africa.

Conclusion

Because melanin has several significant properties there are a number of ways in which the degree to which it is present in the human skin might have been influenced in evolutionary terms. The major possibilities favouring increased epidermal melanisation seem to embrace photoprotection, genoprotection, and heavy metal excretion. The geographical distribution of the reduction of melanisation is less easily explained as the negative selective pressure associated with these properties is not strong. The traditional view is that in regions with relatively little solar UV radiation the presence of epidermal melanin prevents adequate intradermal vitamin D synthesis with the resultant propensity for the development of rickets. As argued here, an alternative selective pressure favouring a reduction in pigmentation might derive from the effect of epidermal melanin on radiative heat loss.

Acknowledgements

I am grateful to my colleagues at the Totteridge Institute for Advanced Studies for discussions and helpful comments.

References

1. Riley PA, Ramsden CA, Land EJ (2011) Biological Chemistry of o-Quinones. In: Melanins & Melanosomes: Biosynthesis, Biogenesis, Physiological and Pathological Functions (eds. J. Borovansky & P.A. Riley). Wiley-VCH, Weinheim: 63-86.
2. Riley PA (1995) The evolution of melanogenesis. In "Melanin: Its Role in Human Photoprotection" (eds. L. Zeise, M.R. Chedekel, T.B. Fitzpatrick). Valdenmar Publishing Co, Overland Park, KS: 1-10.
3. Jablonski NG, Chaplin G (2010) Human skin pigmentation as an adaptation to UV radiation. Proc Natl Acad Sci USA 107: 8962-8968.
4. Riley PA, Stratford MRL (2015) Oxidative calcium release from catechol. Bioorganic & Medicinal Chemistry Letters 25: 1453-1454.
5. Mattson MP, Chan SL (2003) Calcium orchestrates apoptosis. Nature Cell Biol 5: 1041-1043.
6. Hardy A (1960) Was Man more Aquatic in the past? New Scientist 17: 642-645.
7. Morgan E (1972) The Descent of Woman. London: Souvenir Press.
8. Riley PA (1997) Epidermal melanin: sun screen or waste disposal? Biologist 44: 408-411.
9. Reinfelder JR, Wang W-X, Luoma SN, Fisher NS (1997) Assimilation efficiencies and turnover rate of trace elements in marine bivalves: a comparison of oysters, clams and mussels. Marine Biol 129: 443-452.
10. Riley PA (2010) The evolution of human pigmentation: some speculative comments. Nederlands Tjidschrift Derm. Vener 20: 277-278.
11. Loomis WF (1967) Skin-pigment regulation of vitamin D biosynthesis in man. Science 157: 501-504.
12. Larison B, Harrigan RJ, Thomassen HA, Rubenstein DI, Chan-Golston AM, et al. (2015) How the zebra got its stripes: a problem with too many solutions. R Soc Open Sci 2: 140452.
13. Candler WH, Ivey H (1997) Cold weather injuries among U.S. soldiers in Alaska: a five-year review. Mil Med 162: 788-791.
14. DeGroot DW, Castellani JW, Williams JO, Amoroso PJ (2003) Epidemiology of U.S. Army cold weather injuries, 1980-1999. Aviat Space Environ Med 74: 564-570.