PHILOSOPHICAL TRANSACTIONS B

rstb.royalsocietypublishing.org

Review

Cite this article: Jablonski NG, Chaplin G. 2017 The colours of humanity: the evolution of pigmentation in the human lineage. *Phil. Trans. R. Soc. B* **372**: 20160349.
http://dx.doi.org/10.1098/rstb.2016.0349

Accepted: 9 December 2016

One contribution of 19 to a theme issue 'Animal coloration: production, perception, function and application'.

Subject Areas:
evolution, genetics, health and disease and epidemiology

Keywords:
eumelanin, phaeomelanin, ultraviolet radiation, natural selection, sexual selection, genetic drift

Author for correspondence:
Nina G. Jablonski
e-mail: ngj2@psu.edu

THE ROYAL SOCIETY PUBLISHING

The colours of humanity: the evolution of pigmentation in the human lineage

Nina G. Jablonski and George Chaplin

Department of Anthropology, The Pennsylvania State University, 409 Carpenter Building, University Park, PA 16802, USA

NGJ, 0000-0001-7644-874X

Humans are a colourful species of primate, with human skin, hair and eye coloration having been influenced by a great variety of evolutionary forces throughout prehistory. Functionally naked skin has been the physical interface between the physical environment and the human body for most of the history of the genus *Homo*, and hence skin coloration has been under intense natural selection. From an original condition of protective, dark, eumelanin-enriched coloration in early tropical-dwelling *Homo* and *Homo sapiens*, loss of melanin pigmentation occurred under natural selection as *Homo sapiens* dispersed into non-tropical latitudes of Africa and Eurasia. Genes responsible for skin, hair and eye coloration appear to have been affected significantly by population bottlenecks in the course of *Homo sapiens* dispersals. Because specific skin colour phenotypes can be created by different combinations of skin colour–associated genetic markers, loss of genetic variability due to genetic drift appears to have had negligible effects on the highly redundant genetic 'palette' for the skin colour. This does not appear to have been the case for hair and eye coloration, however, and these traits appear to have been more strongly influenced by genetic drift and, possibly, sexual selection.

This article is part of the themed issue 'Animal coloration: production, perception, function and application'.

1. Introduction

Discussions of the cardinal features of the human lineage usually focus on bipedalism, relative brain size, language and technology, and ignore the remarkable distinctions of the integument and eyes that have figured importantly in human evolution. In this paper, we explore the diversity of skin, hair and eye coloration in the human lineage, and seek explanations for the evolution of pigmentation traits in relation to the history of human dispersals and population history. Our focus is primarily on the evolution of pigmentary traits in anatomically modern *Homo sapiens*, but brief consideration of integumentary coloration in non-human primates and during pre-*sapiens* phases of human evolution is necessary for understanding the starting point for *Homo sapiens*.

2. Human coloration in context

Like most mammals, primates have hair covering most of their bodies. Most non-human primates have coats consisting of brown or grey agouti hairs, but lavish variation exists. The trunk and limbs of most primates exhibit darker dorsal and lighter ventral pelage, probably for both concealment and thermoregulation [1–4]. Striking patterns of coat colours are found in some lineages [1,5–7], with the most conspicuous patterns found on both the bodies and faces of forest-dwelling diurnal primates such as marmosets (e.g. *Mico* species), guenons (*Cercopithecus*) [8], and doucs and snub-nosed monkeys (*Pygathrix* and *Rhinopithecus*). Nocturnal strepsirrhines such as lorises and galagos, and the only nocturnal haplorhine, the night monkey *Aotus*, are remarkable for cryptic body coloration combined with mask-like patterns of facial hair, which have almost certainly evolved primarily to facilitate species and mate recognition

under markedly reduced light conditions [1]. Compared with other catarrhines, the apes (gibbons, orangutans, chimpanzees and gorillas) lack agouti banding, and the large apes mostly lack regional patterning on their coats and colourful facial markings.

In some platyrrhines and all catarrhines, the skin of the face, and sometimes also the perineum, is not covered with hair and instead is glabrous and, sometimes, spectacularly coloured. The facial masks and perineal regions of the mandrill and golden snub-nosed monkey, to take two extreme examples, exhibit highly modified naked skin. Zones of red coloration are produced by a rich capillary bed perfused with haemoglobin-carrying red blood cells, while the zones of blue are structural colours produced by coherent scattering of light from oriented arrays of dermal collagen [9]. In some female catarrhines, the perineal skin turns pink or red near the time of ovulation as blood and interstitial fluid infuses the region as an advertisement of oestrus [10,11]; in some macaques, these changes are also accompanied by darkening of the facial skin, presumably also caused by perfusion of the region by blood [12]. In Old World monkeys and apes, the intensity of coloration of glabrous skin is under hormonal control and is considered a sign of fertility in females and competitive ability in males [12–14].

The genetic and developmental processes that produce the complex topographical arrangements of coloration on the face, trunk and limbs are not well understood [15], and are still mostly a matter of conjecture. The fact that many patterns follow presumed pathways of migration of the neural crest-derived melanocytes into the face and trunk during development warrants further study [5].

Humans are distinguished from their non-human relatives by an absence of most fur and an almost complete lack of regional patterning in hair coloration, except for male facial hair in some populations [16]. Most of the surface area of human skin is covered with almost invisible vellus hairs and is often referred to as 'functionally naked'. The remnants of the primate hair coat are the localized concentrations of non-agouti terminal hairs of similar colour that occur on the scalp, and of a different type in the axillae and pubic region. The evolution of hair loss in the human lineage has been discussed extensively in the literature and is reviewed elsewhere [17]. Because skin is mostly not preserved in the fossil record, arguments about the evolutionary causation of hair loss have relied on critical examination and integration of pertinent comparative anatomical, physiological, palaeoecological and climatological evidence. This evidence is consistent with an explanation based on natural selection for enhanced thermoregulation during high physical activity levels under conditions of high environmental heat load [18–20]. Under these conditions, most body hair was lost and a high density and broad somatic coverage of eccrine sweat glands was gained in order to increase the capacity for heat dissipation by evaporation from the surface of the skin [21–23]. Loss of body hair was accompanied by disadvantages, notably, loss of some protection against abrasion and ultraviolet radiation (UVR). Compensatory changes evolved quickly in hominin skin, as evidenced by genomic comparisons between humans and our closest relative, the chimpanzee. These have revealed that the human lineage is characterized by accelerated evolution of keratinization and epidermal differentiation genes that contribute to enhanced barrier functions of the epidermis [24–26].

While these changes were occurring, others affecting constitutive pigmentation were also taking place. Decades before genomic evidence became available, the timing of the origin of permanent darkly pigmented skin over the entire hominin body surface had been deduced from anatomical, physiological and climatological evidence [27]. Since genomic evidence has become available, comparative study of the human melanocortin 1 receptor (*MC1R*) locus demonstrated that the timing of evolution of permanent, dark, eumelanin-rich, skin pigmentation coincided with the evolution of functional hairlessness and increased density of eccrine sweat glands early in the history of the genus *Homo*, approximately 1.2 Ma or earlier [28]. Adaptive evolution for sun-resistant alleles of the *MC1R* locus appears to have occurred, therefore, when early members of the genus *Homo* became mostly hairless and highly physically active inhabitants of open savannah environments in Africa. For members of the *Homo* lineage evolving since then in sub-Saharan Africa—including the earliest modern people, *Homo sapiens*—the absence of functional polymorphism has been maintained by purifying selection [29,30]. Protective, eumelanin-rich constitutive pigmentation has persisted because of its contribution towards the survival and successful reproduction of populations living under high UVR at low latitudes.

3. Variation in human skin coloration is mostly a product of natural selection

Skin colour (as measured by skin reflectance) and levels of UVR are highly correlated. Biologically effective UVR is often described in units of minimal erythemal dose or MED, which expresses the amount of UVR radiation that will produce minimal erythema (sunburn or redness caused by engorgement of capillaries) in lightly pigmented human skin within a few hours following UVR exposure. Skin colour can be almost fully modelled as an effect of autumn UVMED alone ($r = 0.927$; $p < 0.0001$) [21,31]. If this relationship developed under natural selection, plausible causation must demonstrate real or probable enhanced reproductive success for specific skin pigmentation phenotypes under specific UVR conditions. Many adaptive explanations for the evolution of variation in human skin colour have been put forward in the last century, as reviewed elsewhere [17], and most have suffered from a lack of evidence for likely differences in survivorship and reproduction of different skin colour phenotypes under the same UVR conditions. Such was the fate of the 'skin cancer hypothesis', recently revived [32], that has invoked dark pigmentation as an evolutionary adaptation against sunburn, DNA damage and skin cancer. Skin cancers rarely cause death or adversely affect reproductive success during the peak reproductive years [33], so this explanation was dismissed long ago as being a primary cause of the evolution of dark skin pigmentation. The recent argument that ancestral hominins had pale, cancer-prone skin similar to that of individuals with OCA2 albinism and that evolution of dark skin spared the human lineage from skin cancer–related mortality lacks support [34]. Other explanations have insufficient explanatory power. These include the hypothesis that the eumelanin was most important in affording protection against tropical parasites and tropical skin diseases because of its potent antimicrobial properties [35–37], an idea that fails to explain the near-absence of eumelanin on the

primary environmental interfaces of the volar surfaces of the hands and feet and the lips. More recently, mooted is the hypothesis that the primary function of eumelanin was augmentation of epidermal barrier function by increasing the skin's resistance to desiccation under arid conditions, and that depigmentation did not occur under positive selection for vitamin D production [38–40]. This hypothesis has been disproved by multiple lines of evidence, including the fact that tanning occurs in the presence of UVR, not desiccation [41], and that positive selection for depigmented skin capable of producing vitamin D under low and highly seasonal UVB conditions is now well established [42].

The strongest hypothesis for the evolution of dark skin colour is that it afforded protection against photodegradation of cutaneous and systemic folate under high UVR conditions for early members of the genus *Homo*. The physiological effects of photodegradation of folate were explored long before the full extent of folate's roles in DNA biosynthesis, repair, DNA methylation, amino acid metabolism and melanin production were appreciated [43]. Because folate (in its main form of 5-methyltetrahydrofolate or 5-MTHF) is sensitive to photodegradation [44–47], protective eumelanin-rich pigmentation evolved in early *Homo* primarily to prevent reduction of fertility due to loss of folate in cutaneous blood vessels and the systemic circulation [21,48]. Folate deficiencies are associated with potentially fatal birth defects such as neural tube defects and male infertility [49–52]. Folate metabolism is regulated by genes and epigenomic factors, which have evolved to favour conservation of folate under conditions of longer day length and greater potential UVR-related folate loss [53,54]. Recent physiological evidence also indicates the importance of folate (in the form of 5-MTHF) in thermoregulation, via its effect on controlling nitric oxide-mediated cutaneous vasodilation [55–58]. Maintaining the integrity of folate metabolism is important with respect to evolution because it directly affects reproductive success and survival early in life [21,59]. Natural selection has, thus, affected varied genetic and physiological mechanisms in order to protect folate and 5-MTHF in the face of high UVR. The primary role of constitutive dark skin colour in hominin and modern human evolution is that of a natural sunscreen to conserve folate. Protection of epidermal DNA against strand breaks was the important secondary role played by dark skin colour.

The genus *Homo* and the species *Homo sapiens* emerged in equatorial Africa under conditions of intense and relatively invariant sunlight and UVR. Dispersal of hominins into non-equatorial Africa, Eurasia and the Americas involved movements into habitats with more seasonally variable patterns and differing wavelength mixtures of UVR [60]. In this review, we shall confine our discussion to the consequences for skin colour of dispersal of *Homo sapiens* into Eurasia and the Americas, a process which began around 55 000 years ago [61]. Dispersing populations were small and bottlenecks further reduced available genetic variation in populations crossing major geographic boundaries [62]. Note also that there is no evidence that dispersing archaic *Homo sapiens* used sewn clothing or other methods of full-coverage protection against the sun and elements. Non-sewn animal skins probably afforded some protection, but for the most part people were subjected to the full force of UVR apart from the times when they sought natural shelter. Skin was thus the primary interface with the environment for most of human evolution.

The UVR regimes faced by dispersing hominins were a major selective pressure affecting the evolution of skin pigmentation. Outside of the tropics, consideration of the pattern of UVB is relevant because only some wavelengths of UVB (between 270 and 300 nm, with peak synthesis between 295 and 297 nm) catalyse production of vitamin D in the skin. Within the tropics, average UVB is high and has two equinoctial peaks, but outside of the tropics, average UVB levels are lower and exhibit but a single peak at the Summer Solstice [60]. Average UVB in northern Eurasia and North America is extremely low and highly variable. Because eumelanin in skin is a highly effective sunscreen, the potential for cutaneous vitamin D production is reduced by dark skin [21,63–66]. Darkly pigmented hominins dispersing out of equatorial Africa thus faced conditions that significantly affected their vitamin D physiology. Penetration of UVR into the skin is related to the amount and distribution of melanin; larger and more superficial melanosomes and 'melanin dust' present in the stratum corneum are highly effective at reducing UVB transmission [67]. Pre-vitamin D production occurs in skin of all colours, but in eumelanin-rich skin, low doses of UVB do not raise 25(OH)D levels to physiologically adequate levels at which storage can take place; higher doses over longer periods of time are required for this, and these conditions are not met outside of equatorial latitudes. For people with dark skin living outside of the tropics, and especially north or south of 43°, there is insufficient UVB available in the sunlight outside of the time immediately around the summer solstice to satisfy the body's vitamin D requirement [21,41,60,68,69]. Long-term occupation of non-tropical latitudes, thus, would not have been possible without loss of some constitutive eumelanin pigmentation in order to prevent the serious sequelae of vitamin D deficiency [21]. At extreme high latitudes, year-round occupation is not possible without a diet that is centred on consumption and storage of vitamin-D rich foods such as oily fish, marine mammals, or caribou and reindeer, which concentrate vitamin D in their muscle meat and fat [21,70].

Most people living in the tropics and subtropics (at latitudes below 38°) gain eumelanin pigment in their skin as the result of seasonal high UVR exposure in a process referred to as the tanning response. Melanin produced by the tanning response is the outcome of UV-induced stress and is regulated by melanocortins in the skin [71]. Melanocortins in the skin reduce the production of reactive oxygen species, enhance repair of DNA damage caused by UVR and inaugurate eumelanin synthesis in individuals who carry specific, naturally expressed variants of *MC1R* [71]. Tanning is thus a response to damage caused by UVR. Contrary to popular belief, tanned skin affords little or no protection from damage from subsequent UVR exposure. Recent experimental studies have shown that a tan developed under suberythemal UVB exposure provides minimal photoprotection, but a tan developed under UVA-rich sunlamps such as those used in tanning salons provided no photoprotective benefit [72–74].

Depigmented skin evolved not once, but multiple times in human history, and was accomplished by different combinations of genetic mutations. For modern European populations, different signatures of selection on the *MC1R* and *SLC24A5* genes imply that both natural selection and genetic drift contributed to the evolution of depigmented skin. These genetic changes affected the amounts of melanin being produced in melanocytes and the size of the melanosomes in which the melanin was packaged [75]. For modern eastern

Asian populations, depigmentation was not achieved through mutations at the *SLC24A5* locus [75], but via a different set of genetic changes, which are still incompletely known [76,77]. The fact that depigmented skin evolved independently in the ancestors of modern Europeans and East Asians suggests that at least two (and probably more) distinct genetic mutation events occurred and that multiple loci underwent positive selection in these two regions receiving relatively low levels of UVB [78–80]. The most likely reason for this was that it was associated with a loss of skin pigment that favoured vitamin D production under conditions of low UVB [69,78,81]. Depigmented skin also evolved independently in *Homo neanderthalensis* [82] probably for the same reason. The 'palette' of skin and hair colour genes is extensive and, especially outside of Africa, there is evidence that multiple genes of small effect have contributed to subtle differences in integumentary coloration [83].

There has been a cause and effect relationship between UVR and skin pigmentation in human evolution, and skin colour phenotypes have been modified under the action of natural selection to maintain an optimum balance between photoprotection and photosynthesis over spatially varying conditions of UVR. Skin colour thus evolved as the product of two opposing clines, one emphasizing dark pigmentation and photoprotection against high loads of UVA and UVB near the equator, the other favouring depigmented skin to promote seasonal, UVB-induced photosynthesis of vitamin D_3 nearer the poles [60]. Intermediate latitudes with seasonally high loads of UVB favoured the evolution of people with intermediate colour capable of tanning [84,85]. The most important points to reinforce here are that the geographical gradient of human skin colour evolved under the influence of natural selection, and that very similar skin colour phenotypes (dark, light and intermediate) have evolved independently numerous times under similar UVR conditions. Diverse combinations of skin colour genes occurred during the course of prehistory as the combined result of natural selection, gene flow due to migration, and founder effect or genetic drift due to population bottlenecks occurring in the course of dispersal events [62,86]. Indigenous populations of the New World have generally lighter skin colours than those of the Old World, probably because they have not resided in their homelands for as long a time and because their adaptations to the environment have been more strongly cultural than biological, as exemplified by the wearing of sewn clothing and the making of shelters [21,22,87].

Sexual selection does not appear to have been a major influence on the evolution of human skin coloration, but it probably did increase the degree of sexual dimorphism in skin colour in some populations [21,87]. The unexposed skin of females is lighter than that of males in most populations [21,83], possibly because of the greater need of females to produce vitamin D in the skin to absorb and mobilize calcium during pregnancy and lactation. A persistent, directional preference for lighter-coloured females as marriage partners has been recognized during historic times in some east- and south-Asian cultures, and it is likely that this has contributed to the greater sexual dimorphism in coloration observed [21,87].

4. Hair and eye coloration are not under strong natural selection

Like skin, the coloration of human hair and eyes is determined primarily by the amount and type of melanin produced and stored in melanosomes [88,89]. Iris pigmentation is also influenced by structural features within the eye itself and by the degree of pupillary dilation [90–92]. Although skin coloration varies according to the intensity and seasonality of UVR on a global scale, no such regular geographical pattern is observed with hair and eye coloration [93]. In fact, little variation in hair and eye coloration occurs in indigenous populations outside of Europe (for hair) and outside of Europe, North Africa, the Middle East, Central Asia and South Asia for eyes [88,89,94]. The lack of variation in hair and eye coloration in Africa has been assumed to be caused by the importance of eumelanin in affording protection, as it does in the case of skin coloration, but this has not been empirically established. Hair and eye coloration appear to have not been under as strong natural selection as skin coloration, and loss of genetic variation at one or more population bottlenecks probably contributed to the patterns of phenotypic variation observed in the hair and irises of modern people [95].

Scalp hair in most non-European populations is very dark brown, with little phenotypic variation [96]. Many genes appear to contribute to the dark brown hair colour phenotype [88,93], and the relative importance of different loci is not yet known. The relatively high prevalence of blond hair in Northern Island Melanesia has been traced to the 93C allele of the *TYRP1* gene [97,98], which has been dispersed throughout the region in the course of human colonization of the South-west Pacific. In Europe, blond hair has been traced to establishment of variation in a regulatory enhancer of the *KITLG* gene, while red hair is produced by a specific range of variants of the *MC1R* locus [99–102]. Sexual selection is thought to have influenced the high prevalence of blond- and red-hair phenotypes in Europe [93], but this has not been established empirically.

The nature and coloration of human male facial hair—beards and moustaches—have been a matter of curiosity and speculation, but little formal research [16,103,104]. Male facial hair, manifested as moustaches, cheek hair (whiskers) and beards, occurs in some male primates and appears to represent secondary sexual characteristics that evolved as amplified visual signals of rank, dominance and attractiveness [8,105]. This supports claims that age-related changes in human beard and moustache coverage and colour on the male face serve as honest signals of age or social dominance [104], and that they evolved as products of contest competition between males [106]. Beards augment the effectiveness of human aggressive facial displays, but are rated as ambivalent or unattractive by females [107,108].

Iris coloration in modern people is mostly brown, but this label is misleading because colours described as 'brown' vary greatly from light to dark [89]. Until very recently, studies of human iris coloration focused primarily on European populations, where the greatest range and variety of eye colours—from dark brown to pale blue—are found. Iris colour phenotypes are determined by amounts of melanin and by the ratio of eumelanin to phaeomelanin in the iris, with brown eyes having a higher ratio than light eyes [88,89]. Other categorical eye colours, such as blue, green and hazel, are common in Europe and parts of the Middle East, and Central and South Asia, with Europeans having the lightest eye colours [88]. The sets of genetic markers associated with variation in iris coloration in Europe, South Asia and East Asia are distinct, and relatively little is known of the

combination of evolutionary forces—including natural and sexual selection—that influenced their distribution [89]. Dark iris coloration is associated with less scattering of intraocular light, a trait that may be protective under conditions of bright sunlight and high UVR. Blue eye coloration, on the other hand, is associated with greater intraocular light scattering and a higher level of melatonin suppression, traits that may have been adaptive under highly seasonal sunshine regimes in northwestern Eurasia [109]. Blue-eyed women have been found to be preferred by blue-eyed men, possibly as a manifestation of a male adaptation for the detection of extra-pair paternity based on eye colour, as a phenotypically based assurance of paternity [110]. Arguments for the action of natural and sexual selection on iris colour need to be examined with great care as more data on the genetic basis of the trait are revealed. A recent study showing that the genetic markers associated with iris coloration are also associated with skin and hair pigmentation traits suggests that iris coloration was a pleitropic effect associated with selection on pigmentation genes whose primary effect was skin or hair pigmentation, not iris coloration [89].

The genetic basis of human coloration is complex because some genetic variants affect all pigmentary systems—skin, hair and eyes—through pleiotropic effects, while others affect only one type [111] and because different genes and gene combinations can create similar coloration phenotypes. Despite the technical difficulties of such studies, the fascination of humans with their own coloration phenotypes will certainly continue to propel research forward quickly.

5. Conclusion

Skin, hair and eye coloration in humans is variable, and has been influenced by different combinations of evolutionary forces. Skin coloration has been strongly influenced by natural selection, globally and throughout human prehistory, because of the importance of melanin as a natural sunscreen on naked skin. The role of natural selection in the evolution of hair and eye coloration appears to have been negligible, but genetic bottlenecks followed by sexual selection may have played more significant roles in establishing the patterns of variation recognized outside of Africa.

In recent centuries, humans have migrated faster and over longer distances than during any time in prehistory. Many of these movements have brought people into regions with markedly different solar regimes than their homelands. Many people now live under levels of solar radiation that are much stronger, or much weaker and more seasonal, than those under which their ancestors evolved. These rapid changes in living circumstances have created significant health problems resulting from too much UVR exposure (skin cancer, accelerated ageing of the skin) and from too little UVR exposure (vitamin D deficiency and its many sequelae) that have greatly impacted individual well-being and public health. Mitigating these problems is now the focus of considerable attention in many health professions [112–115]. Rapid, long-distance migrations have also brought people together from disparate and widely separated places, creating unprecedented and novel opportunities for gene flow. The twenty-first century world contains a sepia rainbow of human skin colours, created from old and new combinations of skin colour–related genetic markers. The effects of these new genetic admixtures on health are not known. More significant to health and overall human well-being, however, are the problems of social segregation and behavioural bias that are rooted in cultural constructions of skin colour–based race categories [87]. Humans are visually oriented primates, and our varied colours are badges of our recently shared evolutionary history. Our skin colours unite us, not divide us.

Authors' contributions. The authors contributed equally to the conception and design of this paper and to the drafting and revising of the article for important intellectual content; they also gave the final approval of the version to be published.

Competing interests. We have no competing interests.

Funding. We received no funding for this study.

Acknowledgements. We are grateful to Tim Caro for extending the generous invitation to us to be part of the Animal Colouration Workshop at the *Wissenschaftkolleg zu Berlin* (WIKO) in May 2016. Tim and two anonymous reviewers provided excellent suggestions for improvement of this contribution. We thank Theresa Wilson of Penn State for providing comprehensive support and assistance to our research programme, including the maintenance of our reference library and preparation of this manuscript.

References

1. Caro T. 2013 The colours of extant mammals. *Semin. Cell Dev. Biol.* **24**, 542–552. (doi:10.1016/j.semcdb.2013.03.016)
2. Walsberg GE. 1988 Consequences of skin color and fur properties for solar heat gain and ultraviolet irradiance in two mammals. *J. Comp. Physiol. B* **158**, 213–221. (doi:10.1007/BF01075835)
3. Caro T. 2005 The adaptive significance of coloration in mammals. *BioScience* **55**, 125–136. (doi:10.1641/0006-3568(2005)0550125:tasoci]2.0.co;2)
4. Hetem RS, de Witt BA, Fick LG, Fuller A, Kerley GIH, Meyer LCR, Mitchell D, Maloney SK. 2009 Body temperature, thermoregulatory behaviour and pelt characteristics of three colour morphs of springbok (*Antidorcas marsupialis*). *Comp. Biochem. Physiol. A* **152**, 379–388. (doi:10.1016/j.cbpa.2008.11.011)
5. Chaplin G, Jablonski NG. 1998 The integument of the odd-nosed colobines. In *The natural history of the Doucs and snub-nosed monkeys* (ed. NG Jablonski), pp. 79–104. Singapore: World Scientific Publishing Co.
6. Bradley BJ, Mundy NI. 2008 The primate palette: the evolution of primate coloration. *Evol. Anthropol.* **17**, 97–111. (doi:10.1002/evan.20164)
7. Caro T. 2009 Contrasting coloration in terrestrial mammals. *Phil. Trans. R. Soc. B* **364**, 537–548. (doi:10.1098/rstb.2008.0221)
8. Kingdon JS. 1980 The role of visual signals and face patterns in African forest monkeys (guenons) of the genus *Cercopithecus*. *Trans. Zoolog. Soc. Lond.* **35**, 425–475. (doi:10.1111/j.1096-3642.1980.tb00062.x)
9. Prum RO, Torres RH. 2004 Structural colouration of mammalian skin: convergent evolution of coherently scattering dermal collagen arrays. *J. Exp. Biol.* **207**, 2157–2172. (doi:10.1242/jeb.00989)
10. Domb LG, Pagel MD. 2001 Sexual swellings advertise female quality in wild baboons. *Nature* **410**, 204–206. (doi:10.1038/35065597)
11. Nunn CL. 1999 The evolution of exaggerated sexual swellings in primates and the graded-signal hypothesis. *Anim. Behav.* **58**, 229–246. (doi:10.1006/anbe.1999.1159)
12. Higham JP, Hughes KD, Brent LJN, Dubuc C, Engelhardt A, Heistermann M, Maestriperi D, Santos

LR, Stevens M. 2011 Familiarity affects the assessment of female facial signals of fertility by free-ranging male rhesus macaques. *Proc. R. Soc. B* **278**, 3452–3458. (doi:10.1098/rspb.2011.0052)

13. Setchell JM, Smith T, Wickings EJ, Knapp LA. 2008 Social correlates of testosterone and ornamentation in male mandrills. *Horm. Behav.* **54**, 365–372. (doi:10.1016/j.yhbeh.2008.05.004)
14. Setchell JM. 2015 Color in competition contexts in non-human animals. In *Handbook of color psychology* (eds AJ Elliot, MD Fairchild, A Franklin), pp. 546–567. Cambridge, UK: Cambridge University Press.
15. Mundy N, Kelly J. 2006 Investigation of the role of the agouti signaling protein gene (ASIP) in coat color evolution in primates. *Mamm. Genome* **17**, 1205–1213. (doi:10.1007/s00335-006-0056-0)
16. Held LI. 2010 The evo-devo puzzle of human hair patterning. *Evol. Biol.* **37**, 113–122. (doi:10.1007/s11692-010-9085-4)
17. Jablonski NG. 2004 The evolution of human skin and skin color. *Ann. Rev. Anthropol.* **33**, 585–623. (doi:10.1146/annurev.anthro.33.070203.143955)
18. Wheeler PE. 1985 The loss of functional body hair in man: the influence of thermal environment, body form and bipedality. *J. Hum. Evol.* **14**, 23–28. (doi:10.1016/S0047-2484(85)80091-9)
19. Zihlman AL, Cohn BA. 1988 The adaptive response of human skin to the savanna. *Hum. Evol.* **3**, 397–409. (doi:10.1007/BF02447222)
20. Bramble DM, Lieberman DE. 2004 Endurance running and the evolution of *Homo*. *Nature* **432**, 345–352. (doi:10.1038/nature03052)
21. Jablonski NG, Chaplin G. 2000 The evolution of human skin coloration. *J. Hum. Evol.* **39**, 57–106. (doi:10.1006/jhev.2000.0403)
22. Jablonski NG. 2006 *Skin: a natural history*, 266. Berkeley, CA: University of California Press.
23. Lieberman DE. 2011 Human locomotion and heat loss: an evolutionary perspective. *Compr. Physiol.* **5**, 99–117. (doi:10.1002/cphy.c140011)
24. The Chimpanzee Sequencing and Analysis Consortium. 2005 Initial sequence of the chimpanzee genome and comparison with the human genome. *Nature* **437**, 69–87. (doi:10.1038/nature04072)
25. Toulza E *et al.* 2007 Large-scale identification of human genes implicated in epidermal barrier function. *Genome Biol.* **8**, R107. (doi:10.1186/gb-2007-8-6-r107)
26. Gautam P, Chaurasia A, Bhattacharya A, Grover R, Consortium IGV, Mukerji M, Natarajan VT. 2014 Population diversity and adaptive evolution in keratinization genes: impact of environment in shaping skin phenotypes. *Mol. Biol. Evol.* **32**, 555–573. (doi:10.1093/molbev/msu342)
27. Walter HV. 1971 Remarks on the environmental adaptation of man. *Humangenetik* **13**, 85–97. (doi:10.1007/BF00295790)
28. Rogers AR, Iltis D, Wooding S. 2004 Genetic variation at the MC1R locus and the time since loss of human body hair. *Curr. Anthropol.* **45**, 105–124. (doi:10.1086/381006)
29. Hudjashov G, Villems R, Kivisild T. 2013 Global patterns of diversity and selection in human tyrosinase gene. *PLoS ONE* **8**, e74307. (doi:10.1371/journal.pone.0074307)
30. Harding RM *et al.* 2000 Evidence for variable selective pressures at MC1R. *Am. J. Hum. Genet.* **66**, 1351–1361. (doi:10.1086/302863)
31. Chaplin G, Jablonski NG. 2002 Environmental correlates of human skin color, revisited. *Am. J. Phys. Anthropol.* **117**, 53. (doi:10.1002/ajpa.20010)
32. Greaves M. 2014 Was skin cancer a selective force for black pigmentation in early hominin evolution? *Proc. R. Soc. B* **281**, 20132955. (doi:10.1098/rspb.2013.2955)
33. Blum HF. 1961 Does the melanin pigment of human skin have adaptive value? *Q. Rev. Biol.* **36**, 50–63. (doi:10.1086/403275)
34. Jablonski NG, Chaplin G. 2014 Skin cancer was not a potent selective force in the evolution of protective pigmentation in early hominins. *Proc. R. Soc. B* **281**, 20140517. (doi:10.1098/rspb.2014.0517)
35. Mackintosh JA. 2001 The antimicrobial properties of melanocytes, melanosomes and melanin and the evolution of black skin. *J. Theor. Biol.* **211**, 101–113. (doi:10.1006/jtbi.2001.2331)
36. Wassermann HP. 1965 Human pigmentation and environmental adaptation. *Arch. Environ. Health* **11**, 691–694. (doi:10.1080/00039896.1965.10664280)
37. Wassermann HP. 1974 *Ethnic pigmentation: historical, physiological, and clinical aspects*, p284p. Amsterdam, The Netherlands: Excerpta Medica.
38. Elias PM, Menon G, Wetzel BK, Williams JW. 2009 Evidence that stress to the epidermal barrier influenced the development of pigmentation in humans. *Pigment Cell Melanoma. Res.* **22**, 420–434. (doi:10.1111/j.1755-148X.2009.00588.x)
39. Elias PM, Menon G, Wetzel BK, Williams JW. 2010 Barrier requirements as the evolutionary 'driver' of epidermal pigmentation in humans. *Am. J. Hum. Biol.* **22**, 526–537. (doi:10.1002/ajhb.21043)
40. Elias PM, Williams ML. 2016 Basis for the gain and subsequent dilution of epidermal pigmentation during human evolution: the barrier and metabolic conservation hypotheses revisited. *Am. J. Phys. Anthropol.* **161**, 189–207. (doi:10.1002/ajpa.23030)
41. Jablonski NG, Chaplin G. 2013 Epidermal pigmentation in the human lineage is an adaptation to ultraviolet radiation. *J. Hum. Evol.* **65**, 671–675. (doi:10.1016/j.jhevol.2013.06.004)
42. Grant WB. 2016 The UVB–vitamin D3–pigment hypothesis is alive and well. *Am. J. Phys. Anthropol.* **161**, 752–755. (doi:10.1002/ajpa.23076)
43. Branda RF, Eaton JW. 1978 Skin color and nutrient photolysis: an evolutionary hypothesis. *Science* **201**, 625–626. (doi:10.1126/science.675247)
44. Off MK, Steindal AE, Porojnicu AC, Juzeniene A, Vorobey A, Johnsson A, Moan J. 2005 Ultraviolet photodegradation of folic acid. *J. Photochem. Photobiol. B Biol.* **80**, 47–55. (doi:10.1016/j.jphotobiol.2005.03.001)
45. Steindal AH, Juzeniene A, Johnsson A, Moan J. 2006 Photodegradation of 5-methyltetrahydrofolate: biophysical aspects. *Photochem. Photobiol.* **82**, 1651–1655. (doi:10.1562/2006-06-09-RA-915)
46. Steindal AH, Tam TTT, Lu XY, Juzeniene A, Moan J. 2008 5-Methyltetrahydrofolate is photosensitive in the presence of riboflavin. *Photochem. Photobiol. Sci.* **7**, 814–818. (doi:10.1039/b718907a)
47. Tam TTT, Juzeniene A, Steindal AH, Iani V, Moan J. 2009 Photodegradation of 5-methyltetrahydrofolate in the presence of uroporphyrin. *J. Photochem. Photobiol. B Biol.* **94**, 201–204. (doi:10.1016/j.jphotobiol.2008.12.003)
48. Hasoun LZ, Bailey SW, Outlaw KK, Ayling JE. 2015 Rearrangement and depletion of folate in human skin by ultraviolet radiation. *J. Dermatol.* **173**, 1087–1090. (doi:10.1111/bjd.13885)
49. Fleming A, Copp AJ. 1998 Embryonic folate metabolism and mouse neural tube defects. *Science* **280**, 2107–2109. (doi:10.1126/science.280.5372.2107)
50. Lucock MD. 2000 Folic acid: nutritional biochemistry, molecular biology, and role in disease processes. *Mol. Genet. Metab.* **71**, 121–138. (doi:10.1006/mgme.2000.3027)
51. Tamura T, Picciano MF. 2006 Folate and human reproduction. *Am. J. Clin. Nutr.* **83**, 993–1016.
52. Wallock LM, Tamura T, Mayr CA, Johnston KE, Ames BN, Jacob RA. 2001 Low seminal plasma folate concentrations are associated with low sperm density and count in male smokers and nonsmokers. *Fertil. Steril.* **75**, 252–259. (doi:10.1016/S0015-0282(00)01697-6)
53. Lucock MD, Glanville T, Ovadia L, Yates Z, Walker J, Simpson N. 2010 Photoperiod at conception predicts C677T-MTHFR genotype: a novel gene-environment interaction. *Am. J. Hum. Biol.* **22**, 484–489. (doi:10.1002/ajhb.21022)
54. Lucock MD *et al.* 2014 Vitamin D, folate, and potential early lifecycle environmental origin of significant adult phenotypes. *Evol. Med. Public Health* **2014**, 69–91. (doi:10.1093/emph/eou013)
55. Stanhewicz AE, Bruning RS, Smith CJ, Kenney WL, Holowatz LA. 2012 Local tetrahydrobiopterin administration augments reflex cutaneous vasodilation through nitric oxide-dependent mechanisms in aged human skin. *J. Appl. Physiol.* **112**, 791–797. (doi:10.1152/japplphysiol.01257.2011)
56. Alexander LM, Kutz JL, Kenney WL. 2013 Tetrahydrobiopterin increases NO-dependent vasodilation in hypercholesterolemic human skin through eNOS-coupling mechanisms. *Am. J. Physiol. Regul. Integr. Comp. Physiol.* **304**, R164–R169. (doi:10.1152/ajpregu.00448.2012)
57. Smith CJ, Kenney WL, Alexander LM. 2013 Regional relation between skin blood flow and sweating to passive heating and local administration of acetylcholine in young, healthy humans. *Am. J. Physiol. Regul. Integr. Comp. Physiol.* **304**, R566–R573. (doi:10.1152/ajpregu.00514.2012)
58. Stanhewicz AE, Alexander LM, Kenney WL. 2015 Folic acid supplementation improves microvascular function in older adults through nitric oxide-dependent mechanisms. *Clin. Sci.* **129**, 159–167. (doi:10.1042/cs20140821)

59. Lucock M, Beckett E, Martin C, Jones P, Furst J, Yates Z, Jablonski NG, Chaplin G, Veysey M. 2017 UV-associated decline in systemic folate: implications for human nutrigenetics, health, and evolutionary processes. *Am. J. Hum. Biol.* **29**, e22929. (doi:10.1002/ajhb.22929)
60. Jablonski NG, Chaplin G. 2010 Human skin pigmentation as an adaptation to UV radiation. *Proc. Natl Acad. Sci. USA* **107**, 8962–8968. (doi:10.1073/pnas.0914628107)
61. Hershkovitz I *et al.* 2015 Levantine cranium from Manot Cave (Israel) foreshadows the first European modern humans. *Nature* **520**, 216–219. (doi:10.1038/nature14134)
62. Henn BM, Cavalli-Sforza LL, Feldman MW. 2012 The great human expansion. *Proc. Natl Acad. Sci. USA* **109**, 17 758–17 764. (doi:10.1073/pnas.1212380109)
63. Webb AR, Kline L, Holick MF. 1988 Influence of season and latitude on the cutaneous synthesis of vitamin D_3: exposure to winter sunlight in Boston and Edmonton will not promote vitamin D_3 synthesis in human skin. *J. Clin. Endocrinol. Metab.* **67**, 373–378. (doi:10.1210/jcem-67-2-373)
64. Webb AR. 2006 Who, what, where and when–influences on cutaneous vitamin D synthesis. *Prog. Biophys. Mol. Biol.* **92**, 17–25. (doi:10.1016/j.pbiomolbio.2006.02.004)
65. Clemens TL, Henderson SL, Adams JS, Holick MF. 1982 Increased skin pigment reduces the capacity of skin to synthesise vitamin D_3. *The Lancet* **319**, 74–76. (doi:10.1016/S0140-6736(82)90214-8)
66. Chen TC *et al.* 2007 Factors that influence the cutaneous synthesis and dietary sources of vitamin D. *Arch. Biochem. Biophys.* **460**, 213–217. (doi:10.1016/j.abb.2006.12.017)
67. Nielsen KP, Zhao L, Stamnes JJ, Stamnes K, Moan J. 2006 The importance of the depth distribution of melanin in skin for DNA protection and other photobiological processes. *J. Photochem. Photobiol. B Biol.* **82**, 194–198. (doi:10.1016/j.jphotobiol.2005.11.008)
68. Chaplin G, Jablonski NG. 2009 Vitamin D and the evolution of human depigmentation. *Am. J. Phys. Anthropol.* **139**, 451–461. (doi:10.1002/ajpa.21079)
69. Jablonski NG, Chaplin G. 2012 Human skin pigmentation, migration and disease susceptibility. *Phil. Trans. R. Soc. B* **367**, 785–792. (doi:10.1098/rstb.2011.0308)
70. Chaplin G, Jablonski NG. 2013 The human environment and the vitamin D compromise: Scotland as a case study in human biocultural adaptation and disease susceptibility. *Hum. Biol.* **85**, 529–552. (doi:10.3378/027.085.0402)
71. Abdel-Malek ZA, Knittel J, Kadekaro AL, Swope VB, Starner R. 2008 The Melanocortin 1 Receptor and the UV response of human melanocytes—a shift in paradigm. *Photochem. Photobiol.* **84**, 501–508. (doi:10.1111/j.1751-1097.2008.00294.x)
72. Coelho SG, Yin L, Smuda C, Mahns A, Kolbe L, Hearing VJ. 2015 Photobiological implications of melanin photoprotection after UVB-induced tanning of human skin but not UVA-induced tanning. *Pigment Cell Melanoma. Res.* **28**, 210–216. (doi:10.1111/pcmr.12331)
73. Wang F, Smith NR, Tran BAP, Kang S, Voorhees JJ, Fisher GJ. 2014 Dermal damage promoted by repeated low-level UV-A1 exposure despite tanning response in human skin. *JAMA Dermatol.* **150**, 401–406. (doi:10.1001/jamadermatol.2013.8417)
74. Miyamura Y *et al.* 2011 The deceptive nature of UVA tanning versus the modest protective effects of UVB tanning on human skin. *Pigment Cell Melanoma. Res.* **24**, 136–147. (doi:10.1111/j.1755-148X.2010.00764.x)
75. Lamason RL *et al.* 2005 SLC24A5, a putative cation exchanger, affects pigmentation in zebrafish and humans. *Science* **310**, 1782–1786. (doi:10.1126/science.1116238)
76. Maria de Gruijter J *et al.* 2011 Contrasting signals of positive selection in genes involved in human skin-color variation from tests based on SNP scans and resequencing. *Invest. Genet.* **2**, 1–12. (doi:10.1186/2041-2223-2-24)
77. Yin L, Coelho SG, Ebsen D, Smuda C, Mahns A, Miller SA, Beer JZ, Kolbe L, Hearing VJ. 2014 Epidermal gene expression and ethnic pigmentation variations among individuals of Asian, European and African ancestry. *Exp. Dermatol.* **23**, 731–735. (doi:10.1111/exd.12518)
78. Norton HL *et al.* 2007 Genetic evidence for the convergent evolution of light skin in Europeans and East Asians. *Mol. Biol. Evol.* **24**, 710–722. (doi:10.1093/molbev/msl203)
79. Wilde S *et al.* 2014 Direct evidence for positive selection of skin, hair, and eye pigmentation in Europeans during the last 5,000 y. *Proc. Natl Acad. Sci. USA* **111**, 4832–4837. (doi:10.1073/pnas.1316513111)
80. Allentoft ME *et al.* 2015 Population genomics of Bronze Age Eurasia. *Nature* **522**, 167–172. (doi:10.1038/nature14507)
81. Saternus R, Pilz S, Gräber S, Kleber M, Marz W, Vogt T, Reichrath J. 2015 A closer look at evolution: variants (SNPs) of genes involved in skin pigmentation, including EXOC2, TYR, TYRP1, and DCT, are associated with 25(OH)D serum concentration. *Endocrinology* **156**, 39–47. (doi:10.1210/en.2014-1238)
82. Lalueza-Fox C *et al.* 2007 A melanocortin 1 receptor allele suggests varying pigmentation among Neanderthals. *Science* **318**, 1453–1455. (doi:10.1126/science.1147417)
83. Candille SI *et al.* 2012 Genome-wide association studies of quantitatively measured skin, hair, and eye pigmentation in four European populations. *PLoS ONE* **7**, e48294. (doi:10.1371/journal.pone.0048294)
84. Quillen EE. 2015 The evolution of tanning needs its day in the sun. *Hum. Biol.* **87**, 352–360. (doi:10.13110/humanbiology.87.4.0352)
85. Nan H *et al.* 2009 Genome-wide association study of tanning phenotype in a population of European ancestry. *J. Invest. Dermatol.* **129**, 2250–2257. (doi:10.1038/jid.2009.62)
86. Quillen EE, Bauchet M, Bigham AW, Delgado-Burbano ME, Faust FX, Klimentidis YC, Mao X, Stoneking M, Shriver MD. 2012 OPRM1 and EGFR contribute to skin pigmentation differences between Indigenous Americans and Europeans. *Hum. Genet.* **131**, 1073–1080. (doi:10.1007/s00439-011-1135-1)
87. Jablonski NG. 2012 *Living color: the biological and social meaning of skin color*, 260. Berkeley, CA: University of California Press.
88. Norton HL, Edwards M, Krithika S, Johnson M, Werren EA, Parra EJ. 2016 Quantitative assessment of skin, hair, and iris variation in a diverse sample of individuals and associated genetic variation. *Am. J. Phys. Anthropol.* **160**, 570–581. (doi:10.1002/ajpa.22861)
89. Edwards M, Cha D, Krithika S, Johnson M, Cook G, Parra EJ. 2016 Iris pigmentation as a quantitative trait: variation in populations of European, East Asian and South Asian ancestry and association with candidate gene polymorphisms. *Pigment Cell Melanoma. Res.* **29**, 141–162. (doi:10.1111/pcmr.12435)
90. Sturm RA, Frudakis TN. 2004 Eye colour: portals into pigmentation genes and ancestry. *Trends Genet.* **20**, 327–332. (doi:10.1016/j.tig.2004.06.010)
91. Sturm RA, Larsson M. 2009 Genetics of human iris colour and patterns. *Pigment Cell Melanoma. Res.* **22**, 544–562. (doi:10.1111/j.1755-148X.2009.00606.x)
92. Mackey DA, Wilkinson CH, Kearns LS, Hewitt AW. 2011 Classification of iris colour: review and refinement of a classification schema. *Clin. Experiment. Ophthalmol.* **39**, 462–471. (doi:10.1111/j.1442-9071.2010.02487.x)
93. Liu F, Wen B, Kayser M. 2013 Colorful DNA polymorphisms in humans. *Semin. Cell Dev. Biol.* **24**, 562–575. (doi:10.1016/j.semcdb.2013.03.013)
94. Westgate GE, Botchkareva NV, Tobin DJ. 2013 The biology of hair diversity. *Int. J. Cosmet. Sci.* **35**, 329–336. (doi:10.1111/ics.12041)
95. Lazaridis I *et al.* 2014 Ancient human genomes suggest three ancestral populations for present-day Europeans. *Nature* **513**, 409–413. (doi:10.1038/nature13673)
96. Shriver MD, Parra EJ. 2000 Comparison of narrow-band reflectance spectroscopy and tristimulus colorimetry for measurements of skin and hair color in persons of different biological ancestry. *Am. J. Phys. Anthropol.* **112**, 17–27. (doi:10.1002/(SICI)1096-8644(200005)112:1<17::AID-AJPA3>3.0.CO;2-D)
97. Norton HL, Correa EA, Koki G, Friedlaender JS. 2014 Distribution of an allele associated with blond hair color across Northern island Melanesia. *Am. J. Phys. Anthropol.* **153**, 653–662. (doi:10.1002/ajpa.22466)
98. Kenny EE *et al.* 2012 Melanesian blond hair is caused by an amino acid change in TYRP1. *Science* **336**, 554. (doi:10.1126/science.1217849)
99. Valverde P, Healy E, Jackson IJ, Rees JL, Thody AJ. 1995 Variants of the melanocyte-stimulating hormone receptor gene are associated with red hair

and fair skin in humans. *Nat. Genet.* **11**, 328–330. (doi:10.1038/ng1195-328)

100. Rees JL. 2003 Genetics of hair and skin color. *Annu. Rev. Genet.* **37**, 67–90. (doi:10.1146/annurev.genet.37.110801.143233)
101. Beaumont KA, Shekar SN, Cook AL, Duffy DL, Sturm RA. 2008 Red hair is the null phenotype of MC1R. *Hum. Mutat*. **29**, E88–E94. (doi:10.1002/humu.20788)
102. Rees JL. 2010 Vexed by red-headed conundrums. *J. Invest. Dermatol.* **130**, 1771–1773. (doi:10.1038/jid.2010.132)
103. Weiss K. 2002 Good vibrations: the silent symphony of life. *Evol. Anthropol.***11**, 176–182. (doi:10.1002/evan.10034)
104. Kingdon J. 2004 *Lowly origin: where, when, and why our ancestors first stood up*, 416. Princeton, NJ: Princeton University Press.
105. Grueter CC, Isler K, Dixson BJ. 2015 Are badges of status adaptive in large complex primate groups? *Evol. Human Behav.* **36**, 398–406. (doi:10.1016/j.evolhumbehav.2015.03.003)
106. Puts DA, Bailey DH, Reno PL. 2015 Contest competition in men. In *The handbook of evolutionary psychology* (ed. DM Buss), pp. 385–402. New York, NY: John Wiley & Sons, Inc.
107. Saxton TK, Mackey LL, McCarty K, Neave N. 2016 A lover or a fighter? Opposing sexual selection pressures on men's vocal pitch and facial hair. *Behav. Ecol.* **27**, 512–519. (doi:10.1093/beheco/arv178)
108. Dixson BJ, Vasey PL. 2012 Beards augment perceptions of men's age, social status, and aggressiveness, but not attractiveness. *Behav. Ecol.* **23**, 481–490. (doi:10.1093/beheco/arr214)
109. Higuchi S, Motohashi Y, Ishibashi K, Maeda T. 2007 Influence of eye colors of Caucasians and Asians on suppression of melatonin secretion by light. *Am. J. Physiol.* **292**, R2352–R2356. (doi:10.1152/ajpregu.00355.2006)
110. Laeng B, Mathisen R, Johnsen J-A. 2007 Why do blue-eyed men prefer women with the same eye color? *Behav. Ecol. Sociobiology* **61**, 371–384. (doi:10.1007/s00265-006-0266-1)
111. Guenther CA, Tasic B, Luo L, Bedell MA, Kingsley DM. 2014 A molecular basis for classic blond hair color in Europeans. *Nat. Genet.* **46**, 748–752. (doi:10.1038/ng.2991)
112. Hossein-Nezhad A, Holick MF. 2013 Vitamin D for health: a global perspective. *Mayo Clin. Proc.* **88**, 720–755. (doi:10.1016/j.mayocp.2013.05.011)
113. Holick MF. 2014 Sunlight, ultraviolet radiation, vitamin D and skin cancer: How much sunlight do we need? In *Sunlight, vitamin D and skin cancer (ed*. J Reichrath), pp. 1–16. New York, NY: Springer Science+Business Media.
114. Palacios C, Gonzalez L. 2014 Is vitamin D deficiency a major global public health problem? *J. Steroid Biochem. Mol. Biol.* **144**, 138–145. (doi:10.1016/j.jsbmb.2013.11.003)
115. Bodekær M, Petersen B, Philipsen PA, Heydenreich J, Thieden E, Wulf HC. 2015 Sun exposure patterns of urban, suburban, and rural children: a dosimetry and diary study of 150 children. *Photochem. Photobiol. Sci.* **14**, 1282–1289. (doi:10.1039/C5PP00052A)