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Human pigmentation: its geographical and racial distribution and biological significance

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Synopsis

Skin pigmentation shows a regularity in its geographical distribution. There appears a strong clinal component to the variation in each of the four quadrants of longitude, apparently independent of a strong continental component. Quantitative studies, based on reflectance spectrophotometry, have resulted in a better understanding of the genetics and biological significance of skin colour variation. The genetic component of normal pigmentation variation within a population is polygenic. Heritability estimates derived from a Sikh sample centre around 60–80%. The **genetic** basis of differences between populations appears to reside in some four gene pairs for the difference between Europeans and Africans, and two gene pairs for the difference between Indians and Europeans.

Quantitation of the geographical distribution shows a remarkably close relationship of mean pigmentation with environmental variables, and in particular with latitude, the biological significance of which appears to reside in protection against **ultraviolet radiation**. It appears that skin colour is also involved in thermoregulation. New hypotheses envisage a role of pigmentation in, among others, disease protection and intracellular metabolism.

Introduction

Of all biological variation in man, skin pigmentation differences are the most conspicuous and, after sex, the most emotive. Recognized from early prehistoric times, anecdotes on the origin of variation in skin pigmentation occur in the mythology and folklore of the ancient civilizations. Thus the Babylonians, the dark people, are thought of as the descendants of Ham, the 'curse of darkness' being the consequence of Ham's disobedience to Noah (Genesis 9: 29). In Greek mythology differences in skin colour originated from the catastrophe that occurred when Phaeton took the reins of the sun chariot from his father Helios, but was unable to steer accurately so that at some places the sun passed too close to the earth burning the inhabitants black, while at others it rose too high resulting in the inhabitants turning pale.

Early attempts to classify races were based on skin colour, following Blumenbach who in 1775 distinguished five races, yellow (Mongolian), brown (Malaysian), red (American), black (Ethiopian), and white (Caucasian), and such attempts continued right through the nineteenth century and into the twentieth century until the dynamic nature of the race process and the fruitlessness of classification for classification's sake was realized.

In the earliest days, therefore, pigmentation was identified with continental group or race of man. But the awareness of colour differences that had become widespread among

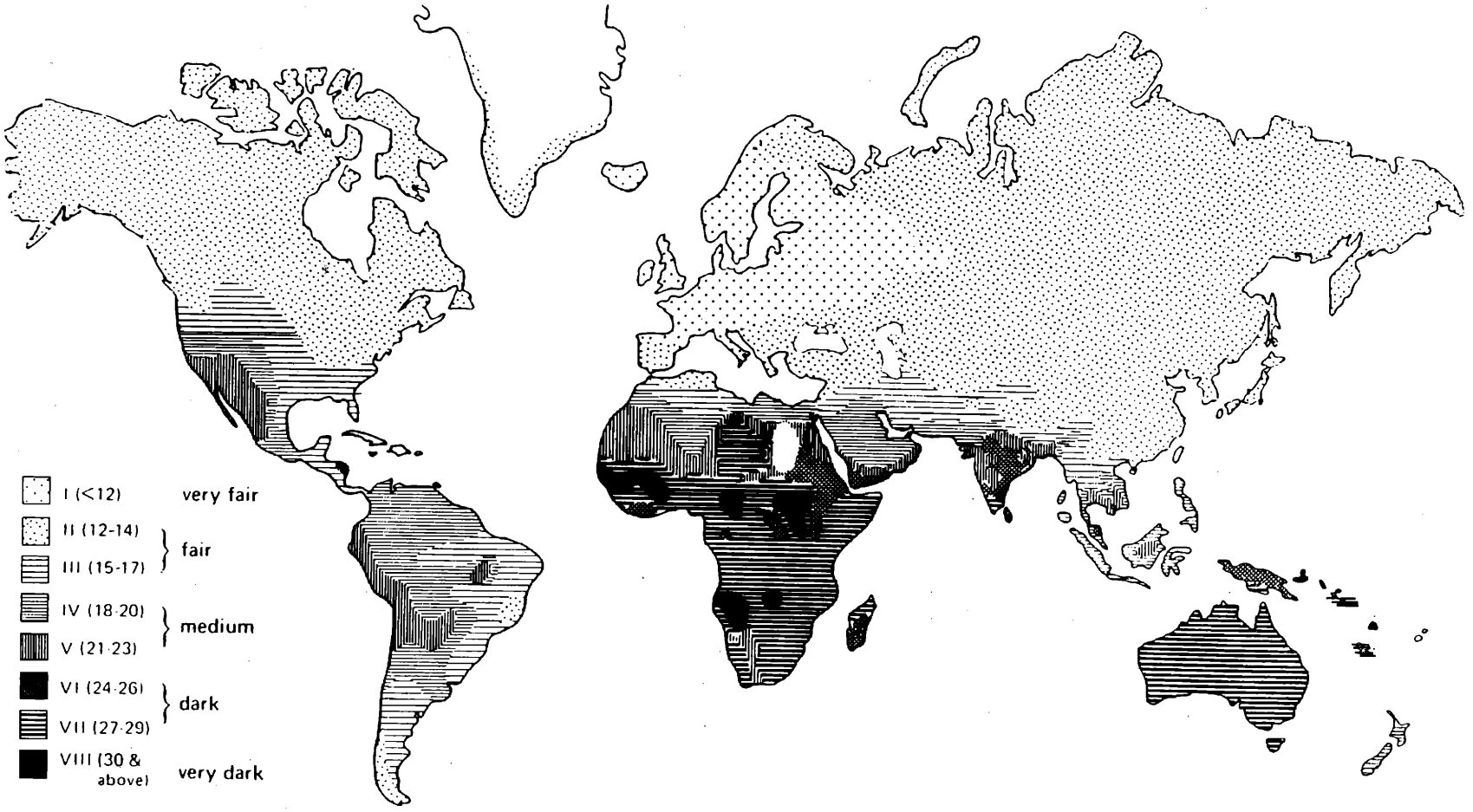

Figure 1. Geographical distribution of pigmentation (von Luschan's scale).

laymen and investigators alike led to the accumulation of descriptive observations of skin colour. These were of necessity highly subjective, but they were the foundations for the more systematic approach to colour measurement of later years. The earliest though crude approach to actual measurement of pigmentation was by arbitrary categorization of the whole range of colour into a number of shades, and subsequently colour scales were developed, such as those devised by Broca and von Luschan, respectively of paper and coloured porcelain tiles, comparison with which improved the accuracy of description. These continued to be developed until the postwar period, the latest being that of Gates (1). With all their disadvantages, nonetheless they provided the basis for a somewhat more objective comparison of human skin colours.

As a result it was possible to draw outline maps of the geographical distribution of pigmentation (von Luschan's scale) such as in *Fig. 1*. The general regularity of geographical distribution of pigmentation is clear even from these early studies. First there is a strong continental component to the variation, e.g. Europe is distinguished from Asia. Secondly there is a strong clinal component. There is no doubt about the tendency for darker skins to occur in tropical regions, and lighter in temperate, and this holds in all four quadrants of longitude; even in the Americas the generalization holds though the intensity of the gradient is rather less than in the Old World. But then on closer inspection there are further generalizations that appear to emerge. In Africa deepest pigmentation occurs not at the lowest latitudes, but appears to coincide with the great horseshoe of open savanah grasslands, while in north-west Europe pigmentation reaches a particularly low level for its latitude. Fleure's (2) discussion drew attention to obvious exceptions to the pigmentation gradient, but his attempts to explain them, in the absence of agreement on the selective function of pigmentation, essentially remain mere speculation.

In 1951, Weiner (3) described a portable reflectance spectrophotometer (EEL), suitable for field work. This made it possible to obtain, outside the confines of a laboratory, objective and accurate measures of skin colour on human populations in their native environments in the form of reflectance measures at standard wavelengths. For the first time it was possible to quantitate pigmentation. Thus it was possible to obtain information from family studies in order to investigate the inheritance of skin pigmentation, from experimental studies to investigate its physiology. The way was open to examine a variety of problems of its biology.

GENETICS

Though there is some phenotypic variation in skin colour due for example to suntanning or some disease states, there is no doubt of the fundamental genetic basis of skin colour differences, both between and within races. Take a negro child born in Africa and bring him up in Britain, or a white child born in London and raise him in Africa, and they remain respectively dark and light. There are some individual major genes, which segregate in Mendelian fashion, which affect skin colour, for example a child with phenylketonuria will show appreciably lightened pigmentation, as is dramatically illustrated in a Yemeni family (*Fig. 2*); phenylketonuria is a recessive condition, the child possessing two deleterious genes, one from his father and one from his mother, which cause deficiency of phenylalanine hydroxylase and inhibit the metabolism of phenylalanine, affecting all subsequent steps in the metabolic sequence. Such major genes are few, there is no indication in normal families of skin colour segregation such as would be explained by a major

gene effect, and it is generally thought that the genetic component of pigmentation variation is polygenic. But we have no good family studies made within a single population that allow this hypothesis to be tested, and this is a sad gap in our genetic knowledge, which my colleagues and I have endeavoured to fill.

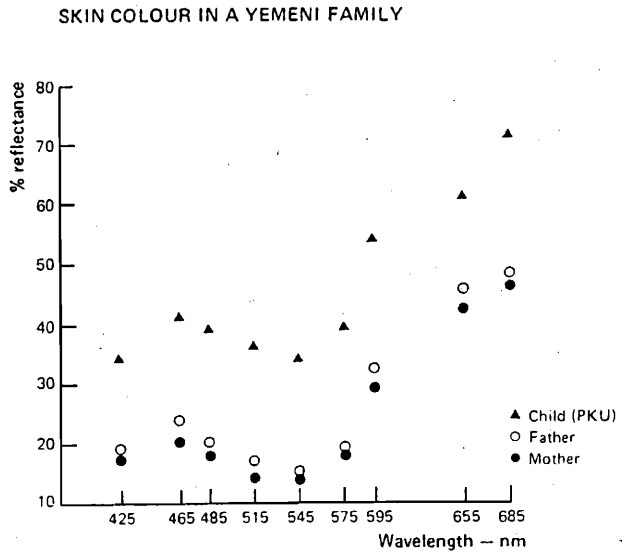

Figure 2. Effect on skin colour of phenylketonuria homozygosity.

We first looked at 141 husband and wife pairs of Sikhs (4) and showed that there was no assortative mating for skin colour at the upper inner arm site. Hence a straightforward analysis of intrafamilial correlation and regression allows estimates of heritability to be made (5). Reflectance data were collected from these 141 families, in which the number of children per family varied from one to five. We examined the regression of children on each parent at nine wavelengths, first counting each child separately, secondly using the mean of all children in a sibship, and thirdly making a weighted estimate according to the family size. The regression coefficient of each child on father ranged from 0·256 $\pm$ 0·053 to 0·399 $\pm$ 0·053 (wavelengths 545 and 685 nm). For child on mother the range was from 0·440 to 0·571 (the same wavelengths), while the regression coefficient on midparent ranged from 0·618 $\pm$ 0·068 to 0·807 $\pm$ 0·062. The regression coefficients using the sibship mean, and the weighted mean, were similar both in general level and in the curious discrepancy between the estimates on the father and on the mother. The sib/sib correlations ranged from 0·476 $\pm$ 0·055 to 0·591 $\pm$ 0·047. Breaking down the offspring by sex makes little difference. The general levels are compatible with polygenic inheritance, but there are clearly some problems of interpretation. The regression coefficients on midparent at the longer wavelengths are somewhat lower, and the sib/sib correlations at the longer wavelengths somewhat higher, than would be expected for a character competely under polygenic control, with no environmental contribution to the variance. The lower regressions on the fathers seem to imply some environmental involvement, while the fact that some sib/sib correlations are rather high again suggests the operation of some environmental factors. On account of these difficulties heritability estimates have to be accepted with critical caution.

Heritability using the data on fathers ranges from 54 to 84%, on mothers from 65 to 100%, and on midparent from 59 to 78%. I am inclined to regard the estimates from the mother as being the least reliable.

Within a population then, a polygenic hypothesis appears reasonable, and on this skin colour is of moderately high heritability. But the genetic basis of difference between populations is something quite different, and to elucidate this studies of hybrid populations are necessary. One method of analysis (6) is to compare observed distributions of pigmentation (by discrete classes) with the binomial distributions expected were a given number of genes involved. This approach assumes that the reflectance measurement is linearly related to the number of genes for pigmentation, that the genes are of equal effect, that they are additive, and that the population is in genetic equilibrium and in a state of random mating. One also needs to know the relative contributions of the parental populations to the hybrid. Stern, using United States negro data, concluded that relatively few genes are involved in the skin colour differences between Europeans and Africans, and his best estimate was four–five gene pairs. Harrison *et al.* (7) applied a similar method to Brazilian negroes and again found that relatively few gene pairs were involved, three to four giving the best fit.

A different method was used by Harrison & Owen, (8), in their investigation of the Afro-European hybrid population in Liverpool. They partitioned the quantitative variation into components and calculated the number of effective factors responsible for the interparental difference. Their calculations indicate the number of effective factors (= genes) again to be three to four.

The authors of these studies are fully aware of the difficulties attending the methods they use, and quite properly are cautious in the presentation of their results. But one cannot overlook the similarity that appears to be emerging in the results of these several studies, i.e. that the number of effective factors or genes or chromosome sections responsible for the pigmentation differences between Africans and Europeans are few in number, of the order of three or four.

A similar method of analysis has been applied to Indian/European hybrids (5). Our material relates only to first generation (F1) hybrids, so clearly one of the assumptions of the model, genetic equilibrium, is not fulfilled. Moreover, the two parental populations do not present the complete contrast in skin pigmentation that was the case in the African/European mixtures, so one cannot consider the genes for less and more pigmentation as being equal in proportion in the hybrids. In these circumstances the analysis is to be regarded only as exploratory. The best fit of the observed and the expected distributions in these hybrids seems to be accomplished at one to two pairs of factors or genes, and as the number of gene pairs increases there is a worsening of fit. While it seems entirely reasonable to expect the number of differences between European and Indian to be fewer than between European and African, these results must be regarded as tentative.

Interesting indirect supporting evidence as to the small number of genes involved in population differences comes from simulation studies by Livingstone (9, 10). He finds that with four loci the evolution of the range of human skin colour differences would take about 800 generations with no dominance, even with relatively slight differences in fitness (6% maximum) and migration between populations. In the subsequent model he also took into account the effects of population size, genetic drift and allele fixation. This work shows that observed differences are not incompatible with reasonable estimates of selection in man and duration of modern human evolution.

GEOGRAPHICAL DISTRIBUTION

Equally fascinating are the pronounced geographical gradients in skin colour, and these too can be further examined using the results of reflectance spectrophotometry. So far some 130 samples, approximately half of each sex, are available to represent the world's indigenous populations. Results are not all technically comparable, for though most were obtained with the EEL spectrophotometer, others were obtained with other models where the wavelengths used are not the same; for only a proportion of the samples are the data given at all nine wavelengths available on the EEL instrument (425, 465, 485, 515, 545, 575, 595, 655, 685 nm); the skin sites examined are not always the same; the samples vary in size, relate to individuals of different ages. The data are certainly too few for mapping. However, they can be used for an examination of those environmental variables with which pigmentation variation is most closely related.

The available data were scrutinized for comparability, and seventy-seven samples were regarded as sufficiently comparable for analysis. Each sample was located sufficiently precisely for values of the following environmental variables to be assigned to it: latitude, mean annual temperature, mean maximum temperature (average of the highest each year), mean minimum temperature (average of the lowest each year), maximum mid-day humidity (highest monthly mean), minimum midday humidity (lowest monthly mean), and altitude. These geographic and climatic variables are of course not independent of each other, as the zero order correlations show; there is for example an obvious influence of latitude on temperature, and a close correlation of mean temperature with maximum and minimum temperature. On account of these intercorrelations, our analysis (11) took the form of an examination of zero order correlation coefficients of reflectance readings with each environmental variable to show the general associations, and subsequent examination by stepwise regression in an endeavour to identify the order of importance in which these environmental variables contribute to the variation in skin pigmentation.

At the upper inner arm, most samples are available at wavelength 685, fewest at wavelengths 485 and 575. Association with latitude predominates (*Table 1*), accounting for between 88% (485 nm) and 70% (685 nm) of the total variance in mean reflectance. The second important variable appears to be mean temperature which, considered alone, would account for between 83% (485 nm) and 36% (655 nm) of the total variance. The order of the remaining variables differs from wavelength to wavelength, but in general the extreme temperature readings come next, then the humidity, while altitude shows the lowest correlations. Associations of reflectance with latitude and humidities are consistently positive, with temperatures and altitude negative. The association with geographical variables is clearly very strong, reflectance increasing with increasing latitude, decrease in temperature, and increasing humidity.

Taking into account the intercorrelation of the geographical variables in order of importance (i.e. proportion of the total variance accounted for) by stepwise regression, the order of the variables changes (*Table 2*). The contribution of latitude alone (since it predominates in the zero order analysis) remains unchanged, accounting for between 77 and 88% of the total variance for all wavelengths except 685 nm where it accounts for 70%. The addition as an independent second variable of maximum temperature at wavelengths 425, 465, 515 and 545 accounts for a further 10–21%, or the addition of mean temperature at wavelengths 485 and 575 for 11%. At the three longer wavelengths maximum humidity moves up into second place, accounting for some 2–13%.

Table I. Correlation coefficients (zero order) of environmental variables with mean reflectance at the upper inner arm
Male and female samples

Wavelength:	425 nm	465 nm	485 nm	515 nm	545 nm	575 nm	595 nm	655 nm	685 nm
Latitude	0·895	0·932	0·938	0·907	0·880	0·928	0·928	0·889	0·835
Mean temperature	−0·824	−0·795	−0·913	−0·797	−0·836	−0·898	−0·898	−0·598	−0·681
Minimum temperature	−0·533	−0·581	−0·616	−0·575	−0·596	−0·657	−0·658	−0·599	−0·590
Maximum temperature	−0·556	−0·481	−0·687	−0·511	−0·616	−0·680	−0·654	−0·101	−0·400
Minimum humidity	0·347	0·239	0·623	0·317	0·400	0·622	0·590	0·103	0·281
Maximum humidity	0·403	0·264	0·506	0·284	0·318	0·478	0·446	0·210	0·331
Altitude	−0·238	−0·285	−0·372	−0·255	−0·265	−0·371	−0·347	−0·405	−0·236
	$N = 61$	N = 45	$N = 32$	$N = 45$	$N = 55$	$N = 32$	$N = 36$	$N = 51$	$N = 77$

Table II. Percentage contribution to the variance of skin colour at the upper inner arm by environmental variables

	Contributions to variance (%)									
Wavelength 425 nm		Wavelength 465 nm		Wavelength 485 nm		Wavelength 515 nm		Wavelength 545 nm	
Latitude	80·1	Latitude	86·9	Latitude	88·0	Latitude	82·3	Latitude	77·4
Max. temp.	15·9	Max. temp.	9·9	Mean temp.	11·7	Max. temp.	12·3	Max. temp	21·4
Altitude	3·3	Altitude	2·7			Altitude	2·6	Min. humidity	0·8
Min. temp.	0·1	Min. temp.	0·3			Min humidity	0·9		
						Max. humidity	0·6		
						Min. temp.	0·5		
						Mean temp.	0·1		
	$N = 61$		$N = 45$		$N = 32$		$N = 45$		$N = 55$
Wavelength 575 nm		Wavelength 595 nm		Wavelength 655 nm		Wavelength 685 nm			
Latitude	86·0	Latitude	86·2	Latitude	79·0	Latitude	69·7		
Mean temp.	11·1	Max. humidity	13·3	Max. humidity	1·7	Max. humidity	6·6		
				Max. temp.	0·9	Max. temp.	4·0		
				Mean temp.	0·6	Min. temp.	1·6		
				Min. temp.	0·1	Mean temp.	1·2		
						Altitude	0·7		
						Min. humidity	0·2		
	$N = 32$		$N = 36$		$N = 51$		$N = 77$		

The predominance of latitude and the subordinate roles of the other variables is clearly shown by the first order partial correlation coefficients at the four wavelengths for which there are most samples. The correlation with latitude diminishes very slightly, if at all, after exclusion of the effects of maximum temperature or maximum humidity, diminishes rather more but still remains appreciable after the exclusion of mean temperature. Clearly the latitudinal correlation owes little to these three variables. Excluding the effects of latitude, the correlations with mean temperature diminish very markedly, those with maximum temperature or maximum humidity rather less.

The regression equations of reflectance on the relevant environmental variables at each wavelength (both sexes combined) show that these account for a remarkably high proportion of the total variance. Only for the two longest wavelengths does this drop below 97%, though exact comparison is precluded by the different number of samples available at each wavelength.

There is no doubt at all about the dominating influence of the latitudinal associations, nor of the appreciable independent contribution of maximum or mean temperature at wavelengths 425 to 475 nm. Humidity makes a negligible contribution to the variance at wavelengths below 595 nm, the effect being restricted to maximum humidity at the three longest wavelengths. The remaining environmental variables make negligible contributions. Particularly interesting is the contrast between the shorter and longer wavelengths in the importance of temperature and humidity.

These results indicate a remarkably close relationship of mean pigmentation with environmental variables, and in particular a dominating association with latitude. It is reasonable to argue therefore, that some factor associated with latitude has a strong biological influence. Of the environmental variables associated with latitude, the amount of ultraviolet radiation received at the earth's surface varies inversely with latitude, while temperature is also strongly associated; the effect of both at a given locality is, of course, modified by other variables such as cloudiness or altitude. Temperature is already taken into account in the present analysis, the latitude association of reflectance values is independent of it, so it seems most likely that ultraviolet radiation is the factor responsible for the latitudinal association of pigmentation.

BIOLOGICAL SIGNIFICANCE

Protection against ultraviolet radiation

Darwin (12) suggested selective advantage of the Negro's black skin through the protection it afforded against the harmful rays of the sun. The ultraviolet parts of the spectrum in particular, while at minimal exposures they have some beneficial effects, at greater doses promote injury in the form of sunburn and carcinogenesis. The carcinogenic wavelengths lie between 2537 and 3341Å, while erythema is induced increasingly rapidly at wavelengths of less than 3200, reaching a maximum at 2800Å. However, the relationship of pigmentation to solar radiation is not simple. Much of the radiation reaching the surface of the body does not reach the deeper melanin-containing layers. It is scattered, reflected, and absorbed in the outer horny layer of the skin (though in dark skins this layer too contains some melanin). However, some radiation reaches the deeper layers. When sheets of the stratum corneum from Africans and Europeans in Nigeria, shown to be similar in thickness to each other, were placed on a photographic plate and

exposed to ultraviolet irradiation, the European specimen showed pronounced penetration, whereas the degree of penetration in the darker African skin was much less (13). Comparing darker Europeans with fair, Mackie & McGovern (14) found that the fairer absorbed more radiant energy in the dermis, while the darker absorbed the greater part in the basal layer. Deeper penetration of ultraviolet radiation into the dermis in less melanized whites has also been demonstrated in several more recent studies (15, 16, 17). Moreover, the positive results of ultraviolet therapy in whites with rickets show that active rays do penetrate deeply enough to affect at least the superficial capillary circulation or to stimulate the nerve endings. Other experimental studies of differential penetration have shown that while transmission of invisible rays is reduced, wavelengths of 3650Å penetrate white human skin 2·2 mm thick. Pronounced penetration into the corium and subdermal layers requires rays of over 4000Å. A considerable proportion of ultraviolet rays at 2500Å reaches the corium, but rays at 2700–2800Å are practically all absorbed in the epidermis, and Negro skin absorbs particularly in the region from 2537 to 2800Å. While the photoprotective role of melanin is a complex phenomenon, involving attenuation of radiation by scattering, effective absorption, and dissipation of the damaging rays as heat, the barrier of melanin in the basal epidermal layers is obviously effective.

There are other lines of evidence besides the experimental. The fate of those dwellers in the tropics who have little or no melanin is instructive. Adult albinos in tropical areas develop a thickening (absent in young children) of the horny layers of the skin, a less efficient barrier than an abundance of melanin. Second, there is the evidence of cancer development. The rarity of cutaneous cancers in dark-skinned as compared with light-skinned subjects has often been noted. Those Europeans who are affected tend to be outdoor workers whose face and dorsum of hand (exposed areas) are the most frequent sites, while in whites in the United States there is a latitudinal north-to-south gradient in the increase of these tumours. The selective effect of protection against these tumours is likely to be small, for they are relatively infrequent and they occur mainly in the later years of life after reproduction has ceased. Another type of tumour is the melanoma, also rare but selectively more severe since it can occur in young adults; this is a pigmented tumour which in Europeans arises from pigmented spots in the epidermis, and in Negroes from relatively unpigmented regions of the body. While cases are rare in Europe, there were 395 melanoma cases in 20 000 white Australian patients, all of whom had lived a long time in Australia, and this high prevalence could well be related to excessive exposure to sunlight. Melanomas are said to be rarer in Negroes, yet malignant melanoma, next to squamous-cell cancer, is the commonest form of malignant disease seen in east and central Africa, and in Bengal it is much more common than in Europe. Most of the squamous-cell cancers are due to malignant changes in chronic tropical ulcers of the legs and the epitheliomata associated therewith, while the malignant melanomas occur mostly on the foot on its unprotected plantar surface. The extent to which radiation is involved, in that injury exposes the deeper tissues to it, is not clear. There is, however, considerable confirmatory evidence from animal experiments on the relation of pigmentation to cancer.

The protective function of melanin it seems is twofold. First in the short-term the melanized epidermis protects the deeper layers of the dermis against immediate damage, and secondly in the long-term it affords protection against cancer. Short-term damage can be prevented to some extent by secondary pigmentation in the form of sun tan, but the long-term effects seem affected more by primary pigmentation.

Thermoregulation

The results of the present analysis suggest also that thermoregulation may also be invoked as a selective mechanism for skin pigmentation, though in a subordinate but measurable role. Although melanin may bestow on deeply melanized skin the potential selective advantage of protection against the carcinogenic effects of ultraviolet rays, at the same time it imposes an additional heat load, because of the greater absorption and relatively less reflection of solar energy. Black skin absorbs 80% or more of incident visible light, while light skin absorbs only about 60% or less. Black and white skin reflectance curves differ less at the shorter infrared wavelengths (700–1000 μ), though again the black absorbs slightly more. About 95% of the infrared part of the radiation responsible for thermal effects is absorbed by a depth of 2 mm of skin surface, and 99% of it by a depth of 3 mm, thus suggesting that the differential response of heat load from solar energy resides in the pigmented parts of the epidermis (18). It is therefore to be expected that black skin would be heated more than white skin by the absorption of sunlight. The expected energy absorption has been calculated for white and for black skin exposed to radiant energy of similar spectral distribution to sunlight at the surface of the atmosphere; relatively untanned black skin absorbed 34% more energy than white skin. Since the passage of sunlight through the atmosphere filters out more energy in the infrared region than in the visible, the contrast in heat load from the visible spectrum may well be greater by the time sunlight has reached the earth's surface. In the American desert the heat absorption of unclothed white men exposed to sunlight amounted to 140 calories per hour on the average, a considerable heat load when it is remembered that the resting heat production is usually taken as 90 calories per hour. Under hot desert conditions, therefore, the additional heat absorption of a Negro due to skin colour may be 40 calories per hour or more, a potentially heavy strain on the heat-regulating mechanism under conditions of severe stress. Baker (19) reported a greater rise in rectal temperature accompanied by greater sweating in Negroes than in White soldiers while exercising in the nude, a difference which disappeared when exercising in clothing. Hence disadvantage of the pigmented skin in thermoregulation can be imagined in heat stress either where there is little chance of replenishing water lost in sweating, or in conditions of high humidity where evaporative heat loss is very difficult; indeed, in humid heat stress conditions, it was found that in nineteen matched pairs of negro and white males heat casualties claimed twenty-two victims and only one of them was Negro. With this the distribution of pigmentation in Africa appears to conform. But on the world scale the temperature association appears to be the wrong way round. Perhaps the deeper pigmentation triggers earlier activation of heat loss mechanisms such as sweating, or perhaps the heat load is insufficient for the disadvantages to operate. Another possibility comes from the earlier discussion. Dark skins are much less susceptible to sunburn damage than are light skins. Severe sunburn damage to the skin involves the sweat glands, disturbs their functioning and hence heat regulation. Under conditions in which efficient sweating is critical, the survival value of any feature such as pigmentation which may prevent sweat gland damage will be great. Though not understood fully, acclimatization to heat as a component of the adaptive role of melanin against solar radiation appears reasonable.

Another possibility is that pigmented skin may act as a more efficient dissipator of the solar heat load by radiation (20). Here the evidence is conflicting, and several studies (e.g. 21, 22) suggest small differences of about 2–3% in the emissivity coefficients of black

and white skins between 5000 nm and 8000 nm. But even a small physical effect of this magnitude may be significant from the point of view of adaptation or survival.

Vitamin D synthesis

The slight pigmentation of European populations poses a different problem. The role of melanin in vitamin D synthesis was suggested by Murray as long ago as 1934. Vitamin D is synthesized in the epidermis from a precursor 7-dehydrocholesterol, by photoactive reaction to UVR, medicated by melanin and keratin in the epidermis. Excessive or deficient synthesis can result respectively in hypervitaminosis or rickets in children. Fremon & Loomis (23) suggested that while the heavily melanized skin of the Negro screens out excessive UVR by absorption, and so prevents excessive amounts of vitamin D, the less melanized and keratinized skin of the northern Asians and north-west Europeans enables the available amounts of ultraviolet light to be fully utilized for synthesis of adequate quantities of vitamin D. On this hypothesis there would be dual selection for heavy pigmentation to prevent toxic overdoses of vitamin D where there is heavy ultraviolet radiation, and for slight pigmentation in regions of low ultraviolet. The first half of this hypothesis does not hold, for example Fremon (23) found no cases of hypervitaminosis amongst the Whites in the tropics. But selection for fair skin may well have occurred in north-west European conditions of coolness, heavy cloud cover, and relatively little radiation and indeed the suggestion from recent genetic studies of overall dominance of the genes from the white parent over those from the black parent carries the implication that selection for depigmentation may have been strong. In conditions of minimal sunshine where dietary vitamin D is low, increased access of these rays to the deeper skin layers could well be selectively advantageous in preventing the development of rickets. There is little direct evidence, but it is interesting that black children dwelling in the great cities in the northern part of America and Pakistani children in Glasgow are more susceptible to rickets than are white children. Certainly the occurrence of rickets amongst the more deeply pigmented inhabitants of some cities of the United Kingdom is today causing concern (24). This may indicate something more than a dietary deficiency. It may be due to the fact that, of the lessened amount of ultraviolet light reaching the skin in the temperate latitudes, again diminished by atmospheric pollution and indoor living, what is left after absorption by the melanin of black skin is insufficient to stimulate the production of vitamin D in the dermal layers.

Disease protection

An intriguing suggestion is that of Wasserman (25, 26), who argues that the main selective factor in the evolution of darker pigmentation in the tropics is disease and not climate. The primary need for survival in tropical peoples must have been protection against many infective and parasitic diseases. In their defence, the tropical dwellers evolved a mechanism of increased reticuloendothelial activity and elevated gammaglobulins. These features are related inversely to the size and acitvity of the adrenal cortex, so that through the decrease in adrenal cortical activity there would be increased MSH and ACTH levels which in turn result in increased pigmentation. Thus according to this hypothesis the dark skin pigmentation is only a byproduct of successful adaptation of a more efficient reticuloendothelial system. Such a hypothesis apart from ignoring other adaptive values of melanin, is certainly inadequate to explain the lighter pigmentation of whites.

New roles

Recently new concepts of the protective role of melanin have emerged. Melanin acts as an efficient photosensitizer in the presence of incident radiation. It has been suggested that melanin effectively eliminates those cells genetically damaged on exposure to ultraviolet. Ebling & Rook (27) propose a phototoxic role of melanin in addition to its photoprotective role, Riley (28) speculates on the phototoxic role of melanin as a substitute for photoprotection, suggesting that by their lighter skin the Caucasoids in the tropics suffer more from skin cancer more through failure to remove the genetically damaged cells than through the absence of the melanin buffer.

Also on account of its stable free radical nature, which gives a free electron receptor site on its molecule, melanin can combine with many other substances. It is able to form lipofuscin, a lipomelanin important in ageing (29). It forms complexes with chloropromazine, a drug which not only promotes melanin transport (30), but is also effective in treatments of some mental disorders and leaves symptoms of Parkinsonism and hyperpigmentation as side effects. Melanin moreover, is phagocytized by leucoytes, can therefore circulate in the body (31, 32), and indeed melanin granules have been reported in 75% of skin-draining lymph nodes of Bantu but in only 20% of Caucasoid lymph nodes. Wasserman (33) therefore suggests a possible role for melanin in intracellular metabolism. That some of the new concepts of the role of melanin may be true finds support from Ebling & Rook (27), who point out that it occurs in many organs besides the epidermis and this may have a variety of roles other than environmental adaptation.

Conclusion

Whatever the mechanisms, the close correlation with geographical factors suggests a very strong adaptive role. It seems that protection against ultraviolet radiation in areas where this is intense, and increased synthesis of vitamin D where there is minimal ultraviolet radiation, provide the two most important selective roles for heavy and light melanin pigmentation.

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