Developing a predictive model of human skin colouring

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ABSTRACT

A model of colour formation within human skin has been developed to aid the characterisation of pigmented skin lesions from their digitized colour images. The model is based on the Kublenka–Munk theory of scattering and absorption within inhomogeneous materials and the physics pertaining to their colour properties. By considering the skin to be a layered construction of such materials, the stratum corneum, epidermis, papillary dermis and reticular dermis, and by exploiting the physics related to the optical interface between these layers, the model generates all possible colours occurring within normal human skin. In particular, the model predicts that all skin colours have to lie on a simple curved surface patch within a three-dimensional colour space bounded by two physiologically meaningful axes, one corresponding to the amount of melanin within the epidermis and the other to the amount of blood within the dermis. These predictions were verified by comparing the CIE LMS coordinates of a representative, cross-racial sample of fifty skin images with the LMS coordinates predicted by the model. The results show that, within the predicted error bounds, the coordinates for normal skin colours do indeed lie on the curved surface generated by the model. Several possible applications of this representation are outlined, including images representing the melanin and blood components separately, as well as the possibility of measuring the Breslow thickness of melanocytic invasion within malignant melanoma.

Keywords: skin optics, skin colouring, melanin, melanoma, breslow thickness, computer modelling, color, colour

1 Introduction

This work forms a part of research at the University of Birmingham into developing theories and techniques which aim to aid clinicians in the early diagnosis of Malignant Melanoma. The prescence of variegated colouring is one of the major features in MacKie's[11] revised seven-point check-list and as such is an important indication of the malignancy of a lesion. It is also apparent, following talks with clinicians that they place a great deal of importance on this feature, often finding it difficult to provide a confident diagnosis when a black and white image of a lesion is presented. As such an understanding of the formation of skin colouration should provide interesting and useful information which may assist with the fulfillment this task.

During the process of this work it became apparent that, when projected into a three dimensional colour space, all the colours found within human skin seemed to lie on a curved surface. If this observation is true then there may exist an extremely useful transformation which will allow a dimensional reduction from a three dimensional colour space to a two

dimensional surface. Furthermore, if the processes leading to the formation of this surface are understood then it should be possible to provide a mapping from a certain position on the surface to histological measurements.

2 Optics of human skin

To understand the development of colouring within the skin, and hence the particular colouring within malignant skin lesions the optics of the skin needs to be studied. The skin is a complex structure which can essentially be thought of as two separate and structurally different layers, the epidermis and dermis which have very different scattering, refracting and absorption properties.

2.1 Interaction of light with the epidermis

The epidermis is a layer of epithelium cells which contain varying amounts of melanin with an outer layer, the stratum corneum, of keratinised epithelium cells. The stratum corneum varies considerably in thickness, being thickest in areas exposed to friction such as the soles of the feet. It is chiefly the thickness of the stratum corneum that accounts for differences in thickness between the epidermis of thick and thin skin.[14] As the surface of the stratum corneum is not smooth and planar, normal skin lacks specular reflectance but rather causes the incident radiation to become diffuse. This is not the case with "crusty" or "flaky" skin which is generally formed from stacked flakes of abnormal corneocytes which present multiple optical interfaces resulting in a greater regular reflectance and a white scaly appearance.

Within the body of the epidermis there are many absorption peaks, for instance nucleic acids with an absorption peak of 260nm and urocanic acid with a peak at 277nm. However, as discussed by Anderson et al.,[1] "in the visible portion of the spectrum, melanin is essentially the only pigment affecting the transmittance of normal human epidermis, giving rise to the wide range of discernible skin colours form 'black' to 'white' ". As the epidermis contains no blood vessels, blood vessels being confined to the dermis, it is also stated by Hertzman et al.[8] in their claim that "The 'color' of the skin is primarily due to two factors: melanin in the skin and the prescence of blood". Anderson et al.[1] go on further to point out that "Melanin is not a 'neutral density' filter of the skin; its absorption increases steadily toward shorter wavelengths over the broad spectrum of 250 to 1200nm" which results in blue light being more strongly absorbed than red light.

Other than regular reflectance, only about 5% of incident radiation in the range 350–3000nm is remitted by scattering within the epidermis, this is shown by Anderson et al.[1] to be due to either a small back scattering component within the epidermis or that the absorption factor relative to scattering is large. They go on further to state that whatever back scattering does occur "in the normal epidermis over this range it is for practical purposes weak, and that any strong scattering within the epidermis that does occur must be forward directed".

2.2 Interaction of light with the dermis

The dermis contrasts strongly in structure to that of the epidermis being highly vascular, containing many sensory receptors and being made largely from *collagen* fibres which range from $2\mu m$–$15\mu m$ in diameter[9] and provide the essential structure of the skin. Between the epidermis and dermis the junction presents an extremely uneven boundary with finger like dermal protrusions called *dermal papillae* projecting towards the skin surface. The dermis can be split into two further histologically distinct layers the *papillary dermis* and the *reticular dermis*[14] within which the structure of the collagen fibres differs significantly. The first of these layers is situated directly below the epidermis within which the collagen exists as "a fine network of fibres".[9] This contrasts with the reticular dermis where the collagen fibres are aggregated into thick bundles which are arranged nearly parallel to the skin surface.

The papillary reticular division is given further emphasis when the vascular supply of the skin is considered. For instance, as discussed by Rosen,[13] "Firstly medium sized arteries in the subcutaneous tissue branch just below the reticular dermis to form the *deep vascular plexus*. These then give rise to vertical branches whose caliber diminishes as they extend into the dermis. In the upper reticular dermis these vessels give rise to a plexus of horizontally oriented arteries the *superficial arteriolar plexus*. From this plexus, afferent branches arise, extend into the dermal papillae, loop and return to the upper reticular dermis, forming a *superficial venous plexus* that extends above and beneath the arteriolar plexus. Thereafter the venous circulation parallels that of the arterial". It is clear here that above the arteriolar plexus, and hence within the papillary dermis, the structure of the blood vessels is largely capillary in nature with the larger vessels existing within the reticular dermis. Furthermore, as the nature of the blood vessels becomes more capillary in nature as the epidermal–dermal interface is reached the ratio of blood vessel area to collagen fibre will also increase.

The dermis, being constructed from a densely fibrous collection of collagen fibres and blood vessels, has distinctly different optical properties to the epidermis. Firstly the absorption coefficient for bloodless dermis is far smaller than the scattering coefficient and as such scattering is of major importance. Indeed the results of Findlay's work,[1] in which he measured the remittance and transmittance of thin sections of bloodless dermis, indicate that practically none of incident light falling on the *in vitro* dermis is absorbed. Anderson and Parish point out that this scattering coefficient increases with decreasing wavelength of light with red light penetrating far deeper than blue. They then go on further to say that "Dermal scattering therefore plays a major role in determining the depth to which radiation of various wavelengths penetrate the dermis, and largely accounts for observations that, in general, longer wavelengths across the UV–visible-near infrared spectrum penetrate the dermis to a greater extent than do shorter wavelengths.".[1]

The appearance of blue skin nevi, a type of skin lesion with a black–bluish appearance, can be explained based upon this fact. In blue nevi melanin is deposited within the dermis. As the dermis exhibits far higher scattering for blue light than red, blue light will encounter far less of the melanin and as such suffers far less from absorption. Anderson and Parish point out that "Such scattering is the only way in which a pigment, such as melanin, which absorbs shorter wavelengths more strongly than long wavelengths can produce blue colours.",[1] this is likely to also account for the blue colourings commonly found in cases of melanoma where tumour cells have invaded the dermis.

When viewed *in vivo*, the blood borne pigments hemoglobin, oxy-hemoglobin, beta-carotene and bilirubin are the major absorbers. As the depth of penetration of light into the dermis is wavelength dependent only the superficial vessels contained within the papillary layer and consisting of capillaries will be exposed to significant blue light with the larger vessels in the reticular layer being exposed to light of a longer wavelength. This papilliary-reticular layer divide is further emphasised by the small amount of back scatter within the reticular layer which, as will be discussed is due to the bunching of collagen fibres into thick bundles.

Within the dermis and epidermis it is interesting to note that the chromophores responsible for scattering appear to be different from those that cause absorption.[1] Within the dermis these separate into collagen fibres which provide the scattering and blood born pigments which provide the absorption. This is convenient for modelling because, in normal skin, the scattering component is therefore fixed with the absorption depending on the amount and nature of the blood present.

2.3 Overall effect of epidermis and dermis on incident light

Firstly the light entering the epidermis is rendered diffuse by the non-planar surface of the stratum corneum. Within the epidermis scattering is negligible or at least forward directed and absorption, in the visible spectrum, is due solely to melanin. As melanin absorbs short wavelength more strongly than long wavelength the resulting light entering the dermis will have lost a blue component, the magnitude of this loss depending on the amount of melanin present.

Within the dermis this "brown" light is subject to scattering with the shorter wavelength light being scattered to a greater extent than the long wavelength. This largely determines the depth of penetration of the incoming light and thus controls the amount and nature of blood vessels various parts of the spectrum encounter. These blood vessels contain pigments which absorb blue light more strongly than red, hence giving the red colour of blood.

Most of the light incoming to the dermis, other than that absorbed, is remitted. Indeed as Anderson et al.[1] point out there is zero transmittance for wavelengths less than 600nm. This light then passes back through the epidermis where further melanin absorption takes place before being remitted through the stratum coneum. In normal skin this light is "brown" in colour due largely to melanin absorption with the red hues being due to absorption within the vascular dermis. However, as discussed blue hues can be produced when melanin invades the dermis.

It can be seen from this description of the skin that both the epidermal and dermal layers can be considered as translucent materials where the light is scattered and absorbed by various materials embedded within these layers. These materials include melanin, collagen fibres, blood vessels and lead to a description of the skin as a translucent inhomogeneous material.

3 Model of the optics of human skin within the visible spectrum

3.1 Model of the epidermis

To understand the absorption of radiation by melanin it is useful to consider *Bouguer's law,*[16] which describes the attenuation of light where scattering is negligible,

$$\theta(\lambda, d_m) = e^{-d_m m(\lambda)} \tag{1}$$

where $\theta(\lambda, d_m)$ is the ratio of transmitted radiation to incident radiation, d_m is the path length and $m(\lambda)$ is the spectral absorptivity of the material.

If $\theta(\lambda, d_m)$ is the absorptivity for a certain wavelength and melanin absorption pathlength then the light emitted from the epidermis, S_e, is

$$S_e = \theta(\lambda, d_m) S(\lambda) \tag{2}$$

where $S(\lambda)$ represents the incident light. The amount of each primary emitted from the epidermis in LMS space is then given by

$$L_m(d_m) = \int_0^\infty \theta(\lambda, d_m) S(\lambda) S_L(\lambda) \mathrm{d}\lambda \tag{3}$$

$$M_m(d_m) = \int_0^\infty \theta(\lambda, d_m) S(\lambda) S_M(\lambda) \mathrm{d}\lambda \tag{4}$$

$$S_m(d_m) = \int_0^\infty \theta(\lambda, d_m) S(\lambda) S_S(\lambda) \mathrm{d}\lambda \tag{5}$$

where $S_L(\lambda)$, $S_M(\lambda)$ and $S_S(\lambda)$ are the spectral response curves for the CIE L, M and S primaries[16] . This then allows a vector representation of the emitted light $\mathbf{P_1}$ where

$$\mathbf{P_1}(d_m) = \mathbf{i} L_m(d_m) + \mathbf{j} M_m(d_m) + \mathbf{k} S_m(d_m) \tag{6}$$

If the radiation was emitted from the skin at this point the spectral composition would lie on the locus of points described by the vector $\mathbf{P_1}$ over the range of melanin thicknesses present in the skin. In the special case where the melanin thickness is constant this spectral composition becomes single valued.

3.2 Model of the dermis

As discussed there is very little scattering within the epidermal layer with the small amount that occurs being forward directed. The result of this is that all light not absorbed by melanin can be considered to pass into the dermis. Within the

dermal layer, in contrast, the scattering coefficient is high and as such light is scattered back towards the surface: this high scattering coefficient then renders it unsuitable for modelling with Bouger's law.

To model the dermis it is therefore necessary to consider a more rigorous approach encompassing both scattering as well as absorption. Within an inhomogeneous material the nature of such scattering varies markedly with the size and structure of the inhomogeneties. If they are large compared with the wavelength of the incoming light then the scattering is highly forward-directed and known as *Mie scattering*.[7] If, however, they are small then the scattering can be modelled by considering *Rayleigh scattering*[12] in which the amount of scattering varies inversely with the fourth power of incident wavelength.

Within the papillary dermis the conditions for Rayleigh scattering are met with the collagen fibres having a diameter of an order of magnitude less than the incident visible light. Within the reticular dermis, however, this is not the case with the large bundles of collagen fibres resulting in scattering becoming Mie scattering and as such highly forward-directed with very little light being remitted.

In constructing a dermal model it is therefore convenient to consider the papillary and reticular layers as distinct with very different scattering properties. Indeed, to a first approximation, the remitted light due to the reticular layer can be considered negligible with the papillary layer being assumed to be an inhomogeneous material that combines absorbing particles with Rayleigh scattering. One such theory that models this is the Kubelka–Munk theory[7] in which it is assumed that the material contains particles small in comparison to the material thickness and that the incident radiation is diffuse. The first of these conditions is easily met for skin and as discussed in section 2.1 the stratum corneum causes the incident light to become diffuse meeting the second condition.

3.2.1 The Kubelka–Munk theory

Within the Kubelka–Munk theory the radiation passing through an inhomogeneous translucent material is divided into two opposing diffuse fluxes: the radiant flux in the direction of increasing sample depth is termed I with that returned (as a result of scattering) being termed J.

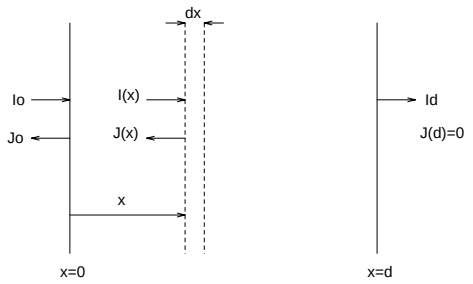

Figure 1: Diagram showing Kubelka–Munk two flux model

At a distance x from the material surface the change in flux over an infinitesimal distance $\mathrm{d}x$ for I and J is

$$\mathrm{d}I = -\alpha a_0 I \mathrm{d}x - \varsigma a_0 I \mathrm{d}x + \alpha b_0 J \mathrm{d}x \tag{7}$$

and

$$\mathrm{d}J = \alpha b_0 J \mathrm{d}x + \varsigma b_0 J \mathrm{d}x - \alpha a_0 I \mathrm{d}x \tag{8}$$

where α is the fraction of radiation absorbed per unit path length, ς is that fraction of radiation scattered per unit path length and a_0 and b_0 are constants that relate $\mathrm{d}x$ to the average path lengths for I and J.

If the radiation is directionally isotropic it can be shown that the average pathlength through an infinitesimal pathlength $\mathrm{d}x$ in any one direction is $2\mathrm{d}x$[1]: this condition is met if the radiation is diffuse, a condition already specified by the Kubelka–Munk model. For a diffuse medium the value of a_0 and b_0 is therefore 2 resulting in the differential equations 7 and 8 being written as

$$\mathrm{d}I = -(k+s)\,I\mathrm{d}x + sJ\mathrm{d}x \tag{9}$$

and

$$\mathrm{d}J = +(k+s)\,J\mathrm{d}x - sI\mathrm{d}x \tag{10}$$

where $k = 2\alpha$ and $s = 2\varsigma$.

The solution to these is an exponential and by application of the boundary conditions shown in figure 1, as shown by Egan and Hilgeman,[7] the expressions for the ratio of diffuse radiation $R(\beta, K, d)$ and transmission $T(\beta, K, d)$ to incident radiation are

$$R(\beta, K, d) = \frac{\left(1-\beta^2\right)\left(e^{Kd} - e^{-Kd}\right)}{(1+\beta)^2 e^{Kd} - (1-\beta)^2 e^{-Kd}} \tag{11}$$

and

$$T(\beta, K, d) = \frac{4\beta}{(1+\beta)^2 e^{Kd} - (1-\beta)^2 e^{-Kd}} \tag{12}$$

where

$$K = \sqrt{k\,(k+2s)} \tag{13}$$

and

$$\beta = \sqrt{\frac{k}{k+2s}}. \tag{14}$$

The remitted light from an inhomogeneous material thus depends on the value of α and ς for the material in question and d, the material thickness. For real materials it is likely that these are wavelength dependent and as such these coefficients become $\alpha(\lambda)$ and $\varsigma(\lambda)$ leading to k and s becoming $k(\lambda)$ and $s(\lambda)$ and R and T also becoming wavelength dependent.

3.2.2 Applying the Kubelka–Munk theory to the papillary dermis

Within the papillary dermis R and T are not solely dependent on wavelength and thickness but are also affected by the amount of blood born pigments present. As discussed in section 2.2 a useful simplification can be made due to the observation that the chromophores responsible for absorption are different from those responsible for scattering. Those responsible for scattering, namely the collagen fibres, then lead to values of ς which do not vary between *in vitro* and *in vivo* samples of the dermis: This results in a value for ς which is solely wavelength dependent and a value for α which varies with the amount of blood present. Hence they can be represented as $\varsigma(\lambda)$ and $\alpha(\lambda, \rho)$, where ρ represents the amount of blood born pigments, with R and T becoming $R(\beta(K(k(\alpha(\lambda,\rho)), s(\varsigma(\lambda)))), K(k(\alpha(\lambda,\rho)), s(\varsigma(\lambda))), d)$ and $T(\beta(K(k(\alpha(\lambda,\rho)), s(\varsigma(\lambda)))), K(k(\alpha(\lambda,\rho)), s(\varsigma(\lambda))), d)$.

To apply the Kubelka–Munk theory to the papillary dermis in such a way as to predict the spectral makeup of the remitted and transmitted light relies on a knowledge of the values of $\alpha(\rho, \lambda)$, $\varsigma(\lambda)$ and d_d (the papillary dermal thickness). As discussed above it is chiefly the stratum corneum that accounts for differences in skin thickness with the dermal layer having a constant thickness. Anderson et al.[1] assume this thickness to be 200 μm.

The values of $\varsigma(\lambda)$ and $\alpha(\lambda)$ for *in vitro* dermal tissue have been measured[1] as have the absorption coefficients for the blood born pigments. The value of $\alpha(\lambda, \rho)$ for *in vivo* dermis can then be found by considering the total absorption, θ_t, being the sum of θ_{iv} and θ_b, the absorption due to the blood born pigments and that measured for the *in vitro* dermis. Hence

$$\theta_t = \theta_{iv} + \theta_b \quad (15)$$
$$\theta_t = e^{-d\alpha_{iv}} * e^{-d\alpha_b} \quad (16)$$
$$\theta_t = e^{-d(\alpha_{iv}+\alpha_b)}. \quad (17)$$

Thus, it can be seen that α for the dermis is simply the product of α_{iv} and α_b. From the above equations it is possible to calculate the value of α and ς for any wavelength and concentration of blood leading to a calculation for R and T. For simplicity of notation it is helpful to consider R_1 and T_1 where $R_1(\lambda, \rho, d_d) = R(\beta(K(k(\alpha(\lambda, \rho)), s(\varsigma(\lambda)))), K(k(\alpha(\lambda, \rho)), s(\varsigma(\lambda))), d)$ and $T_1(\lambda, \rho, d_d) = T(\beta(K(k(\alpha(\lambda, \rho)), s(\varsigma(\lambda)))), K(k(\alpha(\lambda, \rho)), s(\varsigma(\lambda))), d)$

As with the epidermis the light remitted and transmitted from the papillary dermis can then be found by calculating the product of the incoming light with R and T respectively giving the following equations

$$S_{rd}(\lambda, \rho, d_d) = R_1(\lambda, \rho, d_d) S_d(\lambda) \quad (18)$$
$$S_{td}(\lambda, \rho, d_d) = T_1(\lambda, \rho, d_d) S_d(\lambda) \quad (19)$$

where S_d represents the light incident on the dermis and S_{rd} and S_{td} represent the remitted and transmitted light from the dermis.

3.2.3 Perturbing effect of the papillary dermis vascular structure

The above model assumes that the papillary dermis is constructed from a uniform distribution of blood vessels and collagen fibres. This, as discussed, is not the case with the blood vessels becoming more capillary in nature as they leave the *superficial arteriolar plexus* and extend towards the epidermis resulting in a larger surface area of blood born pigments in the upper papillary dermis. To accommodate this a two layer model of the papillary dermis is proposed where the amount of blood born pigments is larger in the top layer. The result of this is that the remitted light from the dermis is a combination of the remitting and transmitting properties within each layer leading to an overall model of the skin as shown in figure 2.

As can be seen from this the total light remitted from the papillary dermis, $R_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld})$ is,

$$\begin{aligned} R_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}) = \quad & R_{1ud}(\lambda, \rho_{ud}, d_{ud}) + T_{1ud}(\lambda, \rho_{ud}, d_{ud})^2 R_{1ld}(\lambda, \rho_{ld}, d_{ld}) \\ & + T_{1ud}(\lambda, \rho_{ud}, d_{ud})^2 R_{1ld}(\lambda, \rho_{ld}, d_{ld})^2 R_{1ud}(\lambda, \rho_{ud}, d_{ud}) \ldots \end{aligned} \quad (20)$$

where ρ_{ud} and ρ_{ld} are the amount of blood born pigments present in the upper and lower papillary dermis respectively and d_{ud} and d_{ld} are the thickness.

3.3 Model of the entire skin

As the light incident to the dermis is the result of epidermal absorption, the input to the dermal layer can be represented by equation 2. Thus the remitted and transmitted light from the dermis become

$$S_{rd}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m) = R_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}) \theta(\lambda, d_m) S(\lambda) \quad (21)$$
$$S_{td}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m) = T_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}) \theta(\lambda, d_m) S(\lambda). \quad (22)$$

The light remitted from the dermis passes through the epidermis again before being finally emitted from the skin resulting in the spectral composition of the final emitted light having the form

$$S_f(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m) = R_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}) \theta(\lambda, d_m)^2 S(\lambda) \quad (23)$$

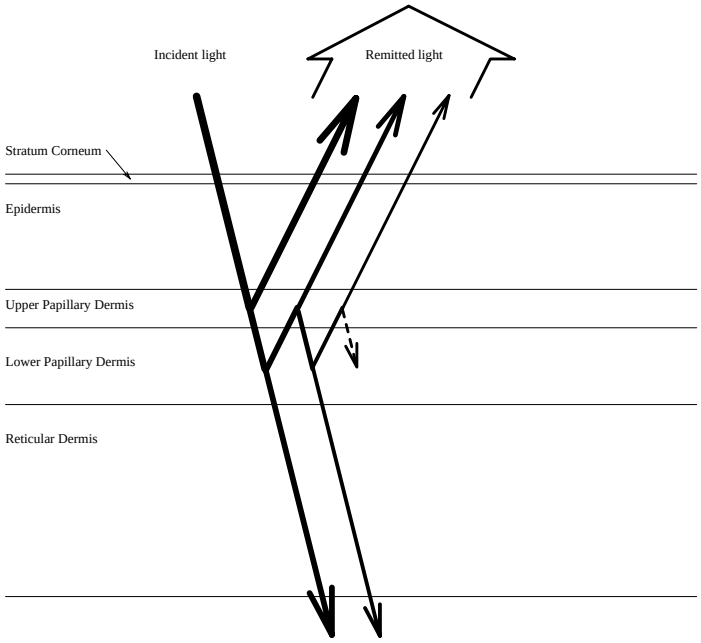

Figure 2: Model of the human skin

These then result in values for each primary being emitted given by

$$L_f(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m) = \int_0^\infty R_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld})\theta(\lambda, d_m)^2 S(\lambda) S_L(\lambda) \mathrm{d}\lambda \quad (24)$$

$$M_f(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m) = \int_0^\infty R_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld})\theta(\lambda, d_m)^2 S(\lambda) S_M(\lambda) \mathrm{d}\lambda \quad (25)$$

$$S_f(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m) = \int_0^\infty R_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld})\theta(\lambda, d_m)^2 S(\lambda) S_S(\lambda) \mathrm{d}\lambda \quad (26)$$

which can be represented in vector form as

$$\mathbf{P_2}(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m) = \mathbf{i}L_f(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m) + \mathbf{j}M_f(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m) + \mathbf{k}S_f(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m) \quad (27)$$

Just as the light transmitted from the epidermis lies on a characteristic locus of points, the light remitted from the dermis also lies on a characteristic trajectory. This becomes apparent when the limiting case of the epidermal absorption coefficient being zero is considered and equations 24, 25 and 26 then simplify to

$$L_f(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}) = \int_0^\infty R_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}) S(\lambda) S_L(\lambda) \mathrm{d}\lambda \quad (28)$$

$$M_f(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}) = \int_0^\infty R_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}) S(\lambda) S_M(\lambda) \mathrm{d}\lambda \quad (29)$$

$$S_f(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}) = \int_0^\infty R_{1total}(\lambda, \rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}) S(\lambda) S_S(\lambda) \mathrm{d}\lambda \quad (30)$$

As discussed in section 3.2.2 the dermal thickness can be considered to be constant and if it is assumed that the division between upper and lower papillary dermis is also constant L_f, M_f and S_f are left solely dependent on ρ_{ud} and ρ_{ld}, the amount of blood born pigments. If it is similarly assumed that the ratio of the amount of blood born pigments in the upper

papillary dermis to that in the lower papillary dermis does not vary the above equations thus define a single trajectory within LMS space. The assumption of an exact constant division between upper and lower papillary dermis for all skin is hard to justify, therefore what is important is that the effect of changes in this division are known. If the perturbations are small in comparison with the gross prediction then the proposition that remitted light from the dermis forms a single trajectory can still be considered true.

This affect of uncertainties is important for the formulation of the model as a whole since it depends heavily on measured histological and physical parameters. As such, the accuracy of the predicted results will depend on the accuracy to which these parameters are known and the manner in which these uncertainties propagate through the model. If the model is to be usable it is essential that the size and effect of these uncertainties are known. To this effect rigorous error analysis has been applied to the model[5] the result of which is to predict an uncertainty in the value of R_{1total} of around $\pm$2% to $\pm$4% which is arguably small enough to justify the above proposition. A standard deviation for the model as a whole is also predicted ranging from $\pm$5% to $\pm$10% over the range of remitted light for each primary.

The combined effect of the absorption processes within the dermis and epidermis result in the range of colours we see within human skin. This range of colours can be found theoretically by identifying the locus of points generated by the vector $\mathbf{P_2}(\rho_{ud}, \rho_{ld}, d_{ud}, d_{ld}, d_m)$ for all possible melanin absorption path lengths, dermal absorption path lengths and values of ρ. As discussed the light finally emitted from the skin is a product of that incident light transmitted by the epidermis and remitted by the dermis. As these both produce defined trajectories the product will define a fixed surface within LMS space.

To generate this locus of points it is necessary to calculate the absorption coefficients for melanin and blood and the values of α and ς for different wavelengths. These have been measured and have been published by Anderson et al.[1] By combining these with the CIE LMS spectral response curves shown in Wyszecki and Stiles[16] and integrating over all possible values of d_m and ρ it is then possible to obtain the surface within LMS space representing all skin colours. This surface is shown in figure 3.

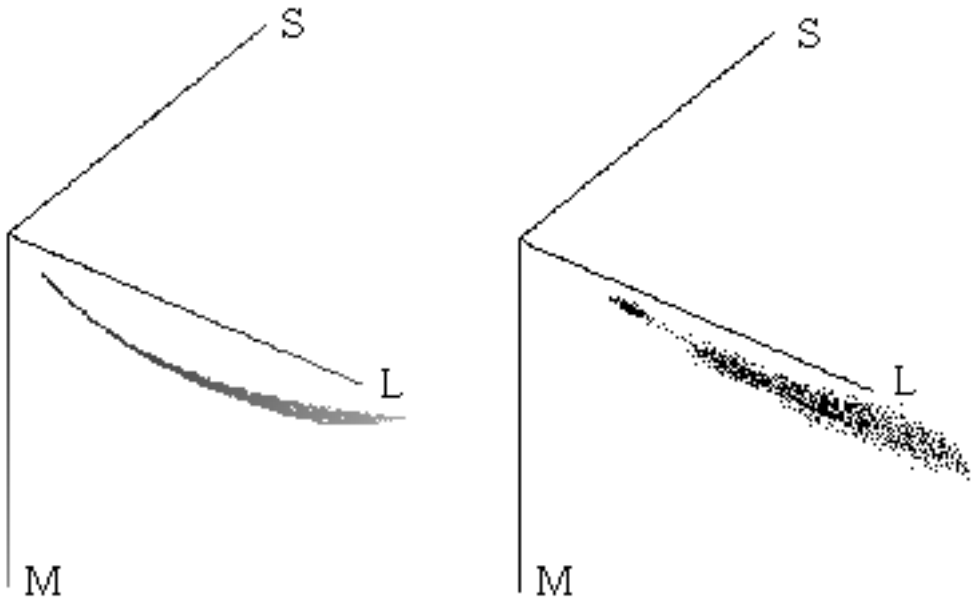

Figure 3: Left: Predicted surface of skin colours; Right: colours found in actual skin samples

4 Validation of the skin model

To validate the model a study was undertaken where images of skin from the interior and exterior forearm volunteers representing a racial cross section were obtained by the use of a colour CCD camera. These were then transformed into CIE 1964 LMS coordinates by means of a method described by Connonlly et al[3].[4] They were supplemented by the published LMS coordinates of ten skin samples by Weatherall and Coombs.[15] The surface formed by the combination of all measured skin samples is shown in figure 3 along with that predicted by the model in LMS space.

As previously discussed there is an inherent uncertainty in the model results due to the original uncertainties in parameter values. Further, it is a well known statistical result that approximately 66%[10] of measured results lie within one standard deviation of the predicted values. If therefore, approximately 66% of the measured deviations lie within this range then the model can considered valid. This can be tested by measuring the actual deviation to the closest point on the model and comparing it with the predicted standard deviation. This analysis was performed on the skin samples and the cumulative frequency of the ratio of actual deviation to predicted standard deviations are shown in figure 4.

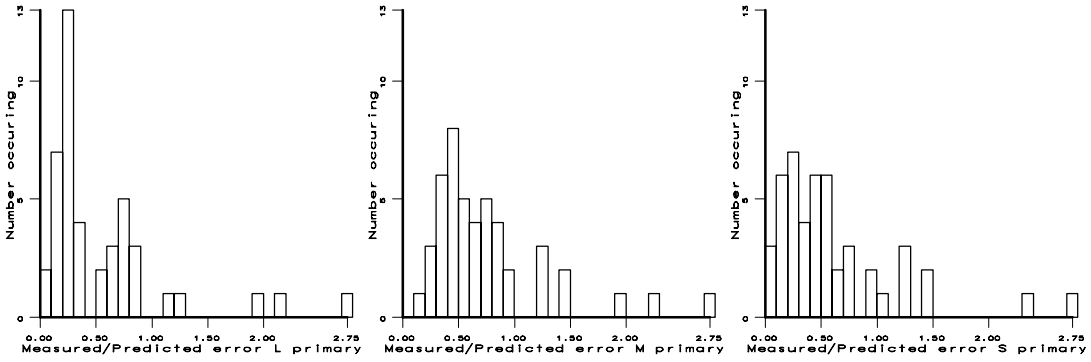

Figure 4: Ratio of measured deviation to predicted standard deviation for each plane

These results show that for the L primary 86% of measured LMS values lie within a single standard deviation of the model with 78% and 82% for the M and S primaries respectively. The model can therefore be considered correct within the bounds of experimental errors.

4.1 Discussion and conclusion

The model presented here predicts a single curved surface within the CIE LMS colour space on which should lie, except for the discussed perturbations, the entire spectrum of possible skin colours. The model has been proven by comparison with experimental data. An interesting property of the predicted surface is that it is formed from two physiologically derived axes, namely the amount of melanin within the epidermis and the amount of blood within the dermis. This therefore allows the transformation from a three dimensional colour space to a two dimensional feature space which is accessible for interpretation by a clinician and thus allows close clinical involvement with the design of any analysis based on this surface. As the surface is independent of particular skin samples it is possible to assign regions to psychologically determined colours, such as those interpreted by a clinician, which will allow classification of a skin lesion as red, brownish–blue etc.

For instance it is possible to transform every known skin colour within an image to the corresponding amount of blood and melanin. An example of this is the left image in figure 5 which shows a region of interior forearm with the feature space on the right representing the amount of blood present. Within this image the large blood vessels leading to the hand can be clearly seen. Similarly the model can be used to produce an image representing the amount of melanin present in the skin. In the case of the malignant melanoma shown on the left of figure 6 the extra melanin produced within the bounds of the legion can be clearly seen, in the right image, against the normal surrounding skin. It is interesting to note that the areas of

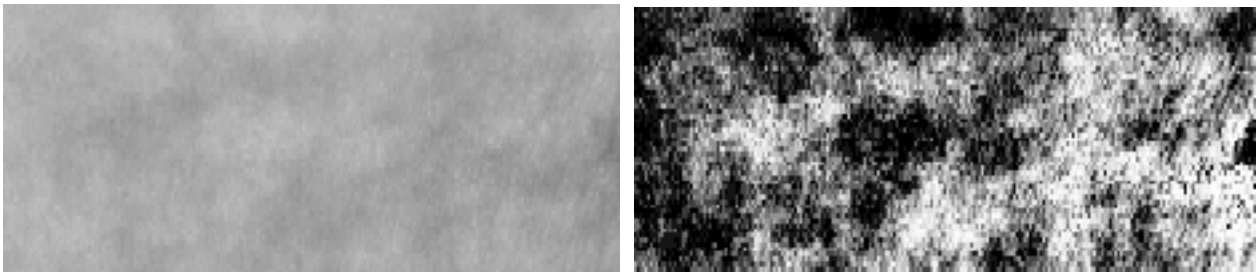

Figure 5: Enlarged section of forearm transformed into blood space showing major blood vessels leading to hand

regression within the lesion, where the amount of melanin has receded again, can be clearly defined. The lack of melanin detected within part of the center nodule is due to specular reflection from the smooth nodule surface.

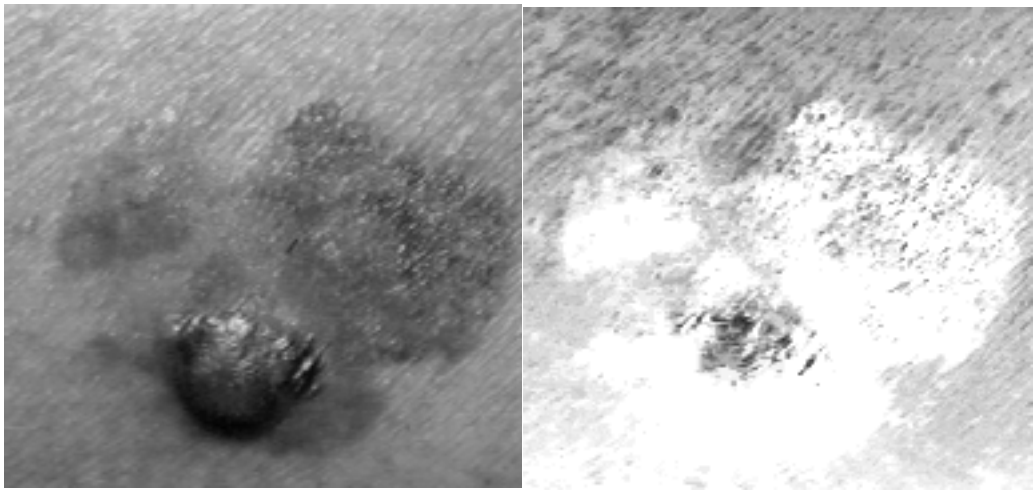

Figure 6: Malignant melanoma transformed into melanin space

4.1.1 Further work: Effect of melanocytes penetrating into the dermis

As discussed in section 2.2 melanocytes can sometimes penetrate into the dermis producing the characteristic blue hues of blue nevi and melanoma. This melanocytic ascent has been quantified by Clark et al.[2] into five levels of tumour invasion known as the Breslow thickness and in the case of melanoma Neville[6] states that “there is a strong relation between this level of invasion and prognosis”.

The effect of dermal invasion perturbs the model by moving points off the surface of predicted skin colours in the direction of the S primary. This deviation from the expected plane is easy to detect and work is underway to develop the model to predict this deviation and hence allow an accurate measurement of the Breslow thickness. Preliminary results are promising with areas of high Breslow thickness, and hence likelihood to be malignant, being detectable.

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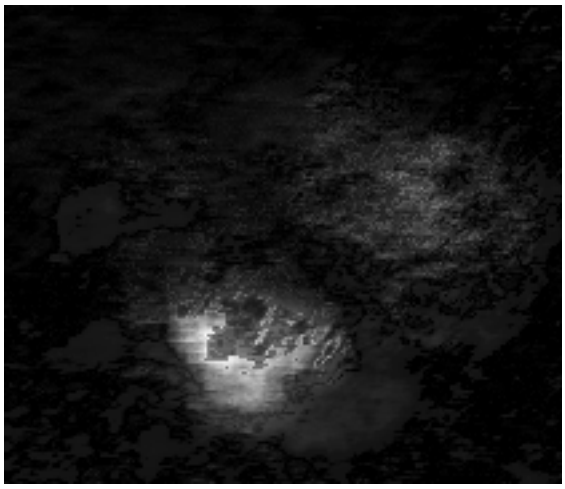

Figure 7: Suspect malignant regions within malignant melanoma

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