International Journal of Cosmetic Science, 2006, **28**, 359–370

Natural skin surface pH is on average below 5, which is beneficial for its resident flora

H. Lambers, S. Piessens, A. Bloem, H. Pronk and P. Finkel
Sara Lee Household and Body Care Research, Fruitweg 25, 2525 KG, The Hague, The Netherlands

Received 10 January 2005, Accepted 3 July 2006

Keywords: microflora, skin moisture, skin pH, skin scaling, TEWL

Synopsis

Variable skin pH values are being reported in literature, all in the acidic range but with a broad range from pH 4.0 to 7.0. In a multicentre study ($N = 330$), we have assessed the skin surface pH of the volar forearm before and after refraining from showering and cosmetic product application for 24 h. The average pH dropped from 5.12 ± 0.56 to 4.93 ± 0.45. On the basis of this pH drop, it is estimated that the 'natural' skin surface pH is on average 4.7, i.e. below 5. This is in line with existing literature, where a relatively large number of reports (*c.* 50%) actually describes pH values *below 5.0*; this is in contrast to the general assumption, that skin surface pH is on average between 5.0 and 6.0. Not only prior use of cosmetic products, especially soaps, have profound influence on skin surface pH, but the use of plain tap water, in Europe with a pH value generally around 8.0, will increase skin pH up to 6 h after application before returning to its 'natural' value of on average below 5.0. It is demonstrated that skin with pH values below 5.0 is in a better condition than skin with pH values above 5.0, as shown by measuring the biophysical parameters of barrier function, moisturization and scaling. The effect of pH on adhesion of resident skin microflora was also assessed; an acid skin pH (4–4.5) keeps the resident bacterial flora attached to the skin, whereas an alkaline pH (8–9) promotes the dispersal from the skin.

Correspondence: H. Lambers, Sara Lee Household and Body Care Research, Fruitweg 25, 2525 KG The Hague, The Netherlands. Tel.: + 31 70 330 3796; fax: + 31 70 330 0932; e-mail: hlambers@hbcr.nl

Résumé

La littérature publie plusieurs valeurs du pH de la peau, toutes dans la gamme acide mais avec un éventail s'étalant de pH 4.0 à 7.0. Dans une étude multi-centres ($N = 330$) nous avons mesuré le pH de la surface de la peau de l'avant-bras, avant et après l'abstention de douche et d'application de produits cosmétiques pendant 24 heures. La moyenne du pH a chuté de 5.12 ± 0.56 à 4.93 ± 0.45. Basé sur cette chute de pH, on estime que le pH 'naturel' de la surface de la peau est en moyenne 4.7, i.e. en dessous de 5. Ceci est en ligne avec la littérature existante, dans laquelle un nombre de rapports relativement important (ca. 50%) décrit réellement les valeurs du pH en dessous de 5.0; Ceci est en contraste avec l'assomption générale que le pH de la surface de la peau est en moyenne entre 5.0 et 6.0. Non seulement l'usage préalable de produits cosmétiques, spécialement les savons, a une influence profonde sur le pH de la surface de la peau, même le simple usage d'eau du robinet, en Europe avec un pH généralement autour de 8.0, va accroître le pH de la peau jusqu'à 6 heures après l'application avant de retomber à sa valeur 'naturelle' d'une moyenne en dessous de 5.0. Il est démontré qu'une peau au pH en dessous de 5.0 est en meilleure condition qu'une peau au pH au dessus de 5.0, comme on peut l'illustrer par la mesure de paramètres biophysiques de la fonction barrière, de l'hydratation et de l'écaillement. L'effet du pH sur l'adhésion de la microflore résidant sur la peau a été également mesuré; un pH

de peau acide (4–4.5) garde la flore résidant sur la peau, alors qu'un pH alcalin (8–9) favorise le détachement de la flore de la surface de la peau.

Introduction

The skin surface has an acidic pH, called the 'acid mantle' [1]. This acidic surface pH as well as its concomitant pH gradient over the stratum corneum (SC) are important for a good skin condition, controlling the presence of resident skin microflora as well as supporting important physiological processes like the formation of an optimal structure of the lipid barrier and SC homeostasis [2– 5].

The exact origin of this low surface pH and accompanying pH gradient is not yet completely elucidated. Passive, no energy-requiring mechanisms as well as active, energy-requiring mechanisms have been proposed for the origin of the acidity of the SC [2, 3, 6]. One of the major components produced by the passive mechanism is lactic acid, naturally occurring in eccrine sweat and also derived from epidermal metabolic processes; the sweat-derived lactic acid will diffuse back into the skin and may thus acidify mainly the superficial layers [7]. Other important components of the passive mechanism are free fatty acids (FFAs), cholesterol sulphate, urocanic acid (from histidin by histidase) and pyrrolidone carboxylic acid (PCA from glutamic acid, probably by a cyclase). Active proton pumps mainly form the active, energy-requiring mechanism: recently, the sodium/hydrogen anion exchanger proteins (NHE1) have been described, present in the membranes of the lamellar bodies. They are responsible for acidification of the extracellular space in the lower layers of the SC [8].

The generation of all these acid compounds creates a desired pH gradient over the SC. Since the viable epidermis has a pH of around 7, a steep pH gradient exists going from the stratum granulosum (SG), just below the SC, to the skin surface [9]; The proton concentration at the surface may thus be a factor 100–1000 higher when compared with the SG, only 10–20 μm lower, resulting in a surface pH, which is generally assumed to be on average between 5 and 6 [10].

The acidic surface pH is also an important determinant for the growth conditions of both resident microflora, i.e. normally found on the skin, as well as transient microflora, i.e. opportunistic, potentially pathogenic. *Staphylococcus epidermidis* (*S. aureus*) is a most typical example of the resident cocci, which normally represent more than 80% of the total microflora of 'dry' body areas like arms, legs and lower torso [11]; *S. aureus*, on the other hand, is a typical example of the potentially pathogenic transient flora [12].

Skin has a mutualistic symbiotic relationship with its microflora: the human skin provides the right biotope for the resident flora, while the resident flora in turn strengthens human's defence by prevention of the colonization of harmful bacteria as well as playing a role in the acidification of the skin [12, 13].

The aims of the present study were to determine the average natural skin pH value of volar forearm after refraining from washing for at least 24 h and to investigate the relation between skin pH and skin condition. Since pH is an important determinator for skin's microflora, we also investigated the influence of pH on bacterial growth and the consequences of skin surface pH on dispersal (e.g. detachment) of resident flora.

Material and methods

Multicentre study

The pH of the volar forearm of 330 subjects (The Netherlands: 167, Germany: 87, The Philippines: 40, Spain: 36) was measured in the morning, between 10.00 a.m. and 12.00 a.m. and 24 h later. The subjects were asked to refrain from any contact with water or cosmetic product during the period between the first and the last assessment.

pH measurement

For pH measurements the pH Meter PH900 (Courage + Khazaka, Cologne, Germany) was used with a flat glass electrode (Mettler-Toledeo, Greifensee, Switzerland) according to EEMCO Guidelines [4]. No significant differences were found in pH value, whether carefully standardized water volumes between the skin and electrode of 50 μl, 100 μl, or when water adhering to the glass electrode, were used. Pressure of the electrode on the skin was also not a critical parameter but was controlled as much as possible [4].

TEWL measurement

Trans-epidermal water loss (TEWL) was measured using the Tewameter TM210 (Courage + Khaz-

aka) [14]. Before measurement, volunteers were acclimatized in a conditioned room at 22 ± 1°C and 50 ± 2% Relative Humidity.

Skin moisturization

Skin moisturization was measured with a Corneometer® CM825, which measures capacitance at low frequency (40–75 Hz) and is sensitive to water having a high dielectric constant, according to EEMCO guidelines [15]. Before measurement, volunteers were acclimatized in a conditioned room at 22 ± 1°C and 50 ± 2% Relative Humidity.

Scaling

The amount of scaling was determined by visual scoring (scale 0–4) of skin image measurements with the Visioscan VC98 (Courage + Khazaka) [16].

SLS challenge

A 24 h patch test was performed with 50 μL 0.15% sodium lauryl sulphate (SLS), followed by TEWL-assessment two hours after removal of the patch.

Bacteriological tests

In-vitro growth test
Test medium used was buffered pepton water (BPW, Merck: 7228, 50% as prescribed) to which 0.5% glucose was added. Growth was measured in the presence of 0.75% lactate at a pH 4.7 and 30°C after incubation for 72 h at $OD_{700\ nm}$ on a BioRad Spectrophotometer, Bench Mark Plus (Hercules, CA, U.S.A.). Reference was medium without lactate at pH 7.0.

In-vivo dispersal test
Volar forearms were first treated by applying 5 mL water containing 1% lactic acid (pH 3.0); after air-drying, contact dishes (Oxoid, CM145 + SR70) were pressed onto the skin. Thereafter, the same volar forearms were treated by applying 1% sodium carbonate decahydrate (pH 11.0); again, after air-drying, contact dishes were pressed onto the skin. The contact dishes were incubated for 2 days at 30°C, after which the number of colony forming units (cfus) per cm^2 were counted.

Results

Natural skin surface pH is on average below 5

Initial values of skin surface pH of the volar forearm have an average value of 5.12 ± 0.56; importantly, after 24 h without any product application or contact with water, the pH value decreases to an average value of 4.93 ± 0.45 (Fig. 1). Figure 2 demonstrates that subjects with a skin pH close to 4.7 show on average no change in pH after 24 h; however, the more out of balance, the larger the correction is. From these observations it can be estimated that the

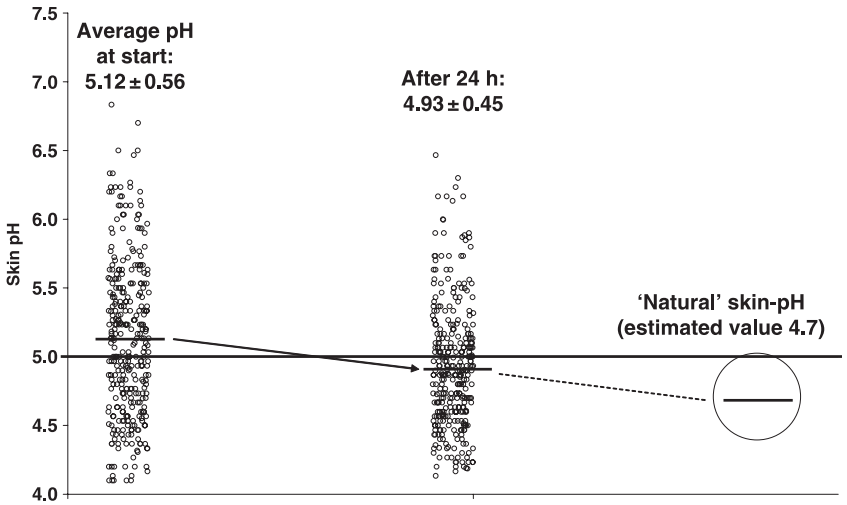

Figure 1 Skin surface pH shift assessed on volar forearm during 24 h without contact with water or cosmetic products. Total number of volunteers, $n = 330$.

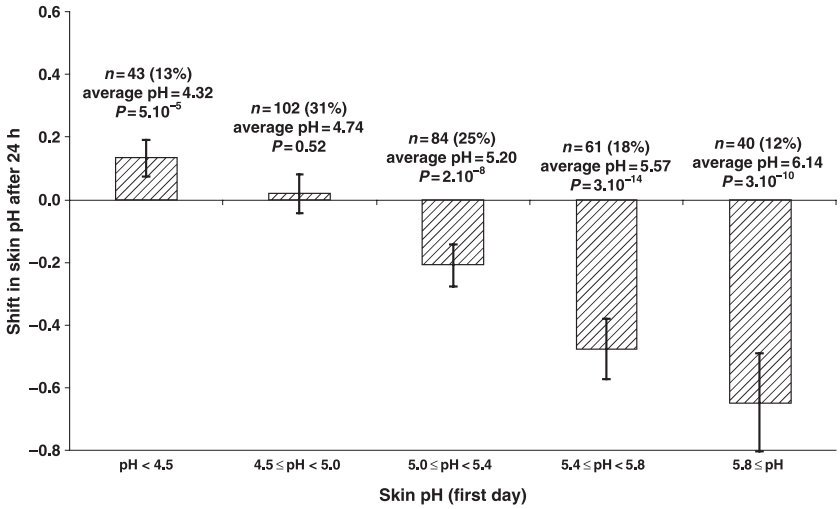

Figure 2 Shifts within various pH ranges on volar forearm during 24 h without contact water or cosmetic products. Error bars specify 95% confidence interval; *P*, the probability that the shift is caused merely by coincidence.

surface pH evolves to a 'natural' average value of 4.7.

Effect of tap water, soap and shower gel on skin surface pH

When the volar forearm of volunteers was treated with soap, a clear increase in skin pH can be measured, which after 6 h has not yet returned to its starting value (Fig. 3). Moreover, a commercial shower gel with a pH of 6.0, causes a considerable initial increase, which returns to the starting value in about 4 h. The same was found for washing with tap water. Interestingly, the skin pH drops below the initial value after 6 h, strongly suggesting that the skin pH was not in balance at the start of the experiment.

Skin pH and skin condition

Lower skin surface pH values correlate with a better resistance against SLS-induced irritant dermatitis (Fig. 4). Furthermore, subjects with skin pH < 5.0 show statistically significant less scaling and higher hydration levels than subjects with skin pH > 5.0 (Fig. 5). On the basis of these

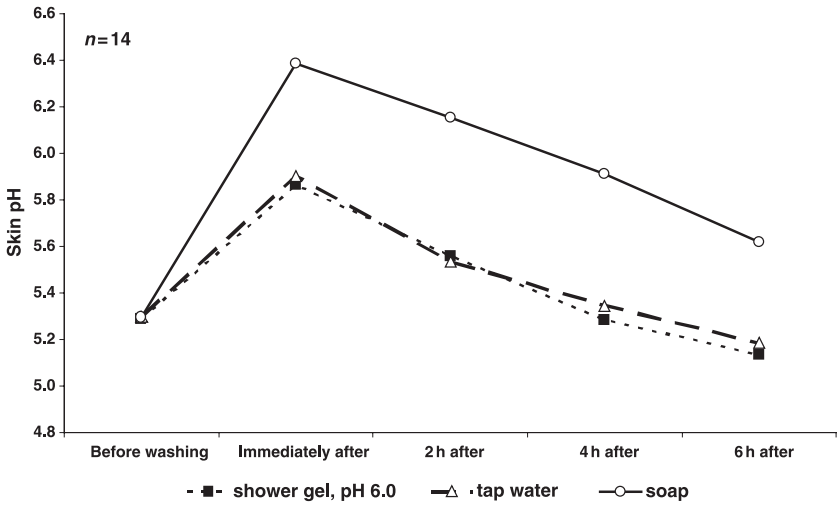

Figure 3 Effect of various single treatments of volar forearm on skin pH.

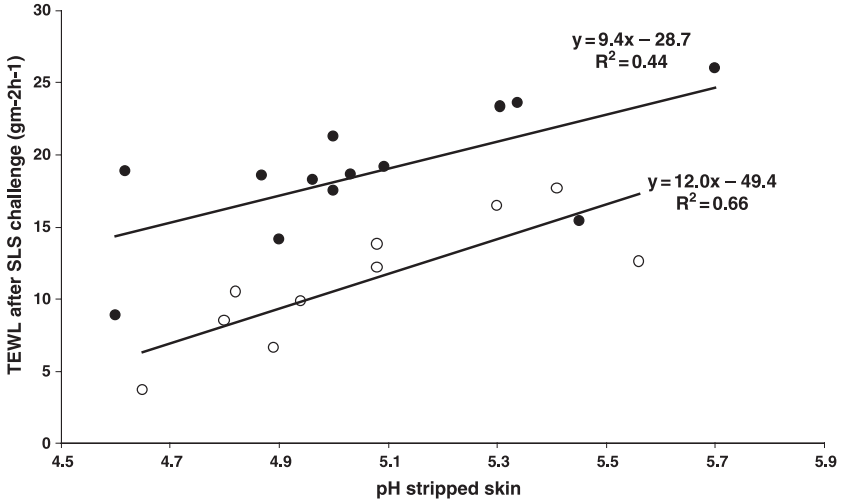

Figure 4 Relation between pH and vulnerability of the skin as measured by trans-epidermal water loss (TEWL) after sodium lauryl sulphate (SLS) patch testing. Closed circles: present study; Open circles: Data from Wilhelm [45].

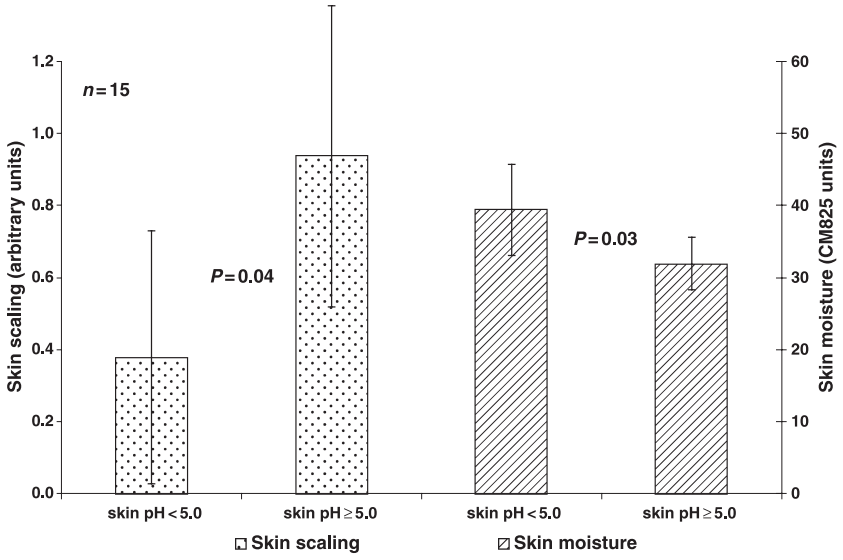

Figure 5 Skin scaling and moisture of volunteers with pH < 5.0 compared with volunteers with pH > 5.0.

biophysical parameters, it can be concluded that skin with pH < 5.0 has a better condition than skin with pH > 5.0.

pH and *in-vitro* growth of microflora

S. epidermidis shows growth at pH 4.7 in the presence of lactate buffer. This growth is enhanced compared with growth at neutral pH (pH 7). On the contrary, growth of *S. aureus* is strongly suppressed (Fig. 6). This demonstrates that in its natural habitat *S. epidermidis* may have an advantage over *S. aureus* in being able to grow at acidic pH in the presence of lactic acid; apparently, the presence of lactate, as one of the main acidifiers at the surface of the skin, influences metabolism and can act as a prebiotic for *S. epidermidis*.

pH and *in-vivo* dispersal of resident skin flora

Imprints, taken from volar forearms, which were acidified to *c.* pH 4.0 by pretreatment with lactic

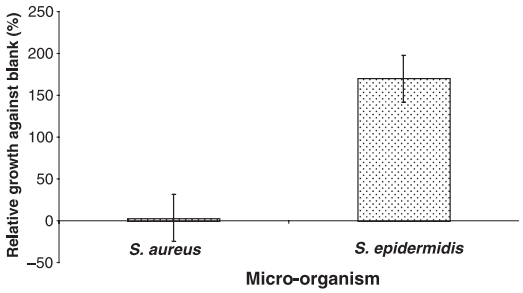

Figure 6 Growth of *S. areus* and *S. epidermidis* at pH 4.7 in the presence of 0.75 lactic acid, relative to growth at pH 7.0 without lactic acid.

acid, show low counts of the resident flora (mainly staphylococci and micrococci); on the contrary, imprints, taken from exactly the same sites after the acid treatment but now brought to *c.* pH 9.0 by pretreatment with carbonate, reveal much higher counts of resident flora. The overall results ($n = 20$) show low dispersal of 24 cfu cm^{-2} after an acid treatment, whereas 428 cfu cm^{-2} were found when the same site was treated with an alkaline solution. Apparently, under acidic conditions resident bacteria do not easily detach from skin. This was further confirmed by putting one hand in acid and the other in alkaline water, resulting in much higher (*c.* 10×) counts of residents in the water under alkaline conditions.

Discussion

Skin surface pH

This study, performed on 330 volunteers, demonstrates that the surface pH of human volar forearm, when left alone without any product application or contact with water, evolves to a 'natural' average value of around 4.7 (Figs 1 and 2). The initial measurements showed an average pH value of 5.12 and after 24 h the average pH value had dropped to 4.93; this was accompanied by a decrease of the standard deviation going from 0.56 to 0.45 with about 70% of the pH values lying within the 4.3–5.1 range. From the 24 h pH shift (Fig. 2), it can be estimated that eventually the skin surface pH will evolve to a value of on average 4.7.

The average skin pH 4.7 reported here, is lower than the skin pH range between 5.4 and 5.9, which is currently generally accepted as being the 'neutral' skin pH range and used on many cosmetic product labels [10].

We also performed a literature survey, collecting skin surface pH data with focus on human forearm, but also including data from cheek and forehead. Although this overview is not claimed to be complete, it is believed to give a fair reflection of the general state of the art. The following 'distribution' of pH values is found (Table 1): about 30% of the publications report average skin surface pH values exclusively below pH 5.0, about 40% report skin surface pH values in a broader range between 4.0 and 6.0 and again about 30% exclusively above pH 5.0. The survey demonstrates that indeed a large number of publications report aver-

Table 1 References of human skin pH values found in literature

Reference	pH range
Dikstein *et al.* [22]	4.0–5.5
	4.3–5.9
Zlotogorski [26]	4.0–5.5
	4.2–5.9
Treffel *et al.* [79]	4.1–4.2
Krien *et al.* [80]	4.3–>6.0
Korting *et al.* [23]	4.3–4.4
Aly *et al.* [81]	4.4
Blank [24]	4.4–5.1
Öhman *et al.* [7]	4.5–4.6
Gottfreund and Meyer [82]	4.5–4.8
Fluhr and Elias [6]	4.5–5.3
Rieger [9]	4.5–5.3
Öhman *et al.* [30]	4.5–5.3
Kubota *et al.* [83]	4.6–4.9
Wilhelm [29]	4.6–5.3
Schmid [84]	4.7
Matousek [85]	4.8
Surber *et al.* [86]	4.8–4.9
Berardesca [33]	4.8–5.0
Seidenari *et al.* [25]	4.8–5.5
Chikakane and Takahashi [72]	4.9–5.2
Thune *et al.* [87]	4.9–6.3
Wilhelm and Maibach [45]	5.0–5.5
Yosipovitch [35]	5.0–5.4
Fluhr [20]	5.0–5.5
Eberlein-Konig *et al.* [38]	5.1–5.3
Warrier *et al.* [88]	5.1–5.5
Barel *et al.* [21]	5.3–5.5
Braun-Falco *et al.* [89]	5.4–5.9
Rippke *et al.* [3]	5.4–5.9
Murahata *et al.* [90]	5.5
Ehlers *et al.* [32]	5.5–5.8
Locher *et al.* [91]	5.5–5.8
Bock *et al.* [92]	5.7

age pH values below 5 or in a more acid range than the skin 'neutral' 5.2–5.9 pH range (Table 1 and references), which is in line with our findings.

The relatively high skin pH values reported in literature may partly be explained by assuming that in various studies the history of product-use before the measurements was not taken into account. In many cases, pH values were measured in the morning without specified 'wash-out' period and possibly only a short-time after volunteers had taken a shower or had applied cosmetic products. For instance, a pH shift of the skin into the more alkaline region has also been described by Grunewald *et al.* after washing the skin with SLS-containing solutions [17, 18].

In this respect, it is interesting to mention that tap water in many (European) countries has neutral to alkaline pH values [19]. In our study, the local tap water has a pH value of around 8, containing enough bicarbonate buffering capacity to increase the pH of the (non-pre-equilibrated) volar forearm for >4 h after showering with plain tap water (Fig. 3). It can be rationalized that products with buffered pH levels around pH 4.7 or lower will minimize disturbance of the 'natural' skin surface pH.

Next to differences in washing habits, other factors may partly explain the differences in pH values found in the literature. There are several parameters, ranging from physiological, pathological, extrinsic to methodological, which may affect the measured pH value and which may partly explain the broad range of pH data found in literature.

Device and protocol

The use of different measuring devices and protocols. It should be noted that until today there is no standardized method to measure pH. The pH Meter PH 900 (Courage + Khazaka) seems to be the most used pH meter. Some general guidelines for pH measurements are given by Parra [4] and these were also followed in this study.

Body site

Most authors report similar pH values at different 'regular' body sites like forearm, neck, forehead, cheek [20– 24]; however, significant differences have been described by others [25, 26]. 'Special', more occluded, body sites like axilla and intertriginous areas, have deviating, normally higher pH values; they are often referred to as 'holes' in the acid mantle. Values reported in Table 1 are mainly measured on the forearm.

Age

At low and high ages skin pH values are higher. Newborns start with near neutral pH after which the pH rapidly declines to acid values within the first month [27, 28]. At higher age again, usually around 70–80 years, increased surface pH values have been documented [22, 26, 29].

Sex

Conflicting data exist concerning differences in pH values between male and female. Men have been claimed to have more acidic surface pH [24, 30, 31]. In the study presented here, we also found significantly lower skin pH values on male when compared with women [results not shown]. Others, however, have reported exactly the opposite [32]. Then again, Zlotogorski [26] and Wilhelm [29] have found no gender differences.

Race

Skin of black people was found to be slightly more acidic than white skin [29, 33]; in contrast, early publications find the opposite [34].

Biorhythm

Differences in pH values have been reported during the day (circadian rhythm with high pH values of *c.* 5.3 in the afternoon vs. low pH values of *c.* 4.9 at night) [35] and also during the seasons (pH in winter slightly higher than in summer) [36].

Disease

There is ample evidence that various pathological skin conditions are associated with higher skin pH values. Examples are atopic dermatitis [25, 37, 38], irritant contact dermatitis [39, 40], ichthyosis [7] and acne [41, 42].

In our study, we have tried to keep the variables to a minimum, i.e. we used one defined body site (volar forearm, left and right), middle-aged volunteers (males as well as females) with healthy skin and we performed measurements in the morning between 10.00 a.m. and 12.00 a.m. under controlled washing conditions. It is proposed that in the future the above-mentioned variables are taken into account as much as possible while measuring skin pH values.

The importance of the acidic surface pH and pH gradient are implicated in the control of various

important physiological processes like the formation and the repair of a competent lipid barrier [2, 3, 6, 43, 44]. Indeed, we have found that skin with lower skin pH values correlate with a better resistance against SLS induced irritant dermatitis (Fig. 4); this confirms the results from Wilhelm [45, 46], who found similar enhanced resistance at lower surface pH (Fig. 4). The formation of the lipid barrier occurs after secretion of precursor-lipids from the lamellar bodies, and subsequent enzymatic conversion of these polar precursors into the apolar ceramides, FFAs and cholesterol, thus constituting a continuous bilayer system, the lipid impermeability barrier. Proper enzymatic extracellular lipid processing is driven by acidic pH. Indeed, enzymes involved, e.g. glucocerebrosidases [47] and phospholipases (at least a specific phospholipase A2) have acidic pH optima [6, 47, 48]. It is interesting to mention in this respect that competent lipid barrier formation in neonatal skin [2] and barrier repair of damaged skin are delayed at neutral pH conditions [49]. Furthermore, regeneration of barrier function after damage with acetone or extensive tape stripping proceeds significantly faster when the skin is exposed to acidic pH (5.5) than neutral pH (*c.* 7.2), indicating that barrier formation and restoration (both in mice and in humans) is a process stimulated by low pH as well as a steep pH gradient [2, 43, 44, 49]. It is also suggested here, that, next to water [50–52], the proper acidic pH could equally well play an important role in activation of filaggrin degrading enzymes and consequently in enhanced natural moisturizing factor (NMF) formation in the skin. The only indirect evidence for such a mechanism is the reported breakdown of filaggrin by both cathepsin-B and cathepsin-L-like proteinases, which are only active in the pH range of 4.0–5.5 and not at pH > 6.0 [53, 54]. This is in line with the finding reported here that skin with pH values below 5 have higher hydration levels than skin with pH values above 5 (Fig. 5).

Skin surface pH and microflora

Skin and skin flora may be considered as an example of a symbiotic relationship [55, 56]. More specifically, it may be called a mutualistic symbiosis, rather than a commensalistic one, where both benefit from the relationship: human skin provides sebum (lipids), sweat (minerals) and dead skin cells (proteins) to the resident flora; in turn, the resident flora strengthens skin's first defence line, the so-called 'acid mantle' by e.g. the enzymatic production of free fatty acids, by the production of anti-bacterials and by competing and preventing the colonization of harmful bacteria. Acid skin pH is clearly associated with regulation of the skin flora [57, 58].

In this study, we have shown that the average natural pH value of the skin is 4.7, lower than currently assumed; this 'acid mantle', next to factors like hydration and presence of minerals as nutrients [56], creates an environment where resident flora (mainly members of staphylococci, micrococci, coryneforms and propionibacteria) can grow, while growth of transient flora (e.g. Gram negative bacteria like *Escherichia* and *Pseudomonas* specs or Gram positive, coagulase positive *Staphylococcus aureus* or *Candida albicans*) is inhibited (see also Fig. 6). Growth and presence of the resident flora effectively contributes to preventing these potentially pathogenic microorganisms to colonize the skin.

This study further shows that the use of normal tap water increases the natural skin surface pH for prolonged time (Fig. 3) and this in turn will undoubted have its effect on the 'quality' of skin's microflora, which on the long-term may even lead to various skin problems and disorders; this hypothesis is supported by the observation that children living in an area with low water hardness, i.e. lower buffer capacity of the tap water resulting in less impact on skin pH, had significantly lower occurrence of eczema than in an area with higher water hardness [59]. In general, it is found that eczematous dermatitis is associated not only with higher, more alkaline skin pH than normal, healthy skin [60, 61], but also with the occurrence of *S. aureus* [62, 63], whose secreted enterotoxins are able to induce eczema even on intact skin [64].

Next to the acid conditions as an effective antibacterial defence system *per se* [65], skin also contains specific substances which add to its antibacterial activity and which is part of the innate immune defence system; e.g. epidermal lipids like free sphingoid bases and fatty acids [66] and epidermal proteins like cathelicidins and defensins [67, 68] and the recently described dermcidin occurring in sweat [69]. A little-investigated but important factor in the ecology of the skin bacteria is the fact that skin flora itself also produces bacteriocins, that control survival in this competitive

environment. These bacteriocins are chemically very heterogeneous and can be either proteinaceous or lipidic in nature [70]. An interesting example is Pep 5 by *S. epidermidis*; this anti-bacterial peptide is especially active against other Staphylococci, specifically *S. aureus* [71].

In this context, it is interesting to mention that the activity of these antibacterial lipids and peptides are boosted at an acidic pH [66, 67, 71, 72], possibly because uncharged lipids and cationic peptides will have a better and more efficient interaction with the bacterial membranes.

Besides controlling bacterial growth on the skin, both adhesion and the prevention of adhesion are important factors which determine the composition and relative numbers of microbes on the skin. It is becoming increasingly clear that a combination of (a) specific interactions like lectin or sugar binding; (b) hydrophobic interactions; and (c) electrostatic interactions, play a crucial role in the binding capacity of both residents and transients to the skin [73, 74].

Here, we have demonstrated the importance of the electrostatic interaction by showing that the pH of the skin surface has important consequences for the binding of resident bacteria on the skin, i.e. under acidic conditions the dispersal rates of endogenous bacteria are much lower than under alkaline conditions. This phenomenon was already described in 1942 by Arnold [75], who used relatively strong acidic and alkaline solutions; here, we have used more realistic conditions, in line with the more acidic and/or alkaline cleansing products available on the personal care market.

The exact mechanism, which may explain these differences in dispersal rates of resident flora, is not known. It is suggested, that under alkaline conditions both the keratins, which constitute the corneocyte, and the bacterial surfaces are negatively charged resulting in repulsion. The role of the lipid-cornified envelope in adhesion of bacteria and the binding to sugar-containing receptors, would be less important under these conditions. Another factor, which may explain the enhanced dissociation of skin bacteria, is the high swelling of the skin under alkaline conditions due to the high netto negative charge of the keratins; this may open up the 'sponge'-like corneocytes, allowing the bacteria to diffuse to the surface. This explains not only the higher numbers of bacteria detaching from the skin at alkaline conditions, but also the fact that repeated washings hardly show diminished numbers of bacteria; apparently, the resident skin bacteria are located even at relatively deep layers in the SC of the skin [76]. It is well known that washing the hands with conventional alkaline soap will liberate large amounts of skin bacteria; this can easily be visualized by pressing the fingers on agar-plates after washing and subsequently count the colonies after breeding. Repeated washings (up to 10 times) fail to reduce these numbers of bacteria. This is why in hospitals the intensive washing of the hands before operations has been questioned [77, 78].

Conclusions

Natural healthy human skin surface pH is on average 4.7, lower than currently assumed (pH 5.4–5.9). Skin with pH < 5.0 is in better condition than skin with pH > 5.0. Growth of *S. epidermidis*, under *in-vitro* acidic pH conditions (pH 4.7) and in the presence of lactate, is enhanced when compared with neutral pH, whereas growth of *S. aureus* is strongly inhibited under these acidic conditions. An acid pH seems to preserve the resident bacterial flora, whereas an alkaline pH causes dispersal from the skin.

References

1. Schade, H. and Marchionini, A. Der Sauremantel der Haut. *Klin. Wochenschr.* **7**, 12–14 (1928).
2. Fluhr, J.W. and Elias, P.M. Stratum corneum pH: formation and function of the 'acid mantle'. *Exog. Dermatol.* **1**, 163–175 (2002).
3. Rippke, F., Schreiner, V. and Schwanitz, H-J. The acidic milieu of the horny layer. *Am. J. Clin. Dermatol.* **3**, 261–272 (2002).
4. Parra, J.L. and Paye, M. EEMCO Guidance for the in vivo assessment of skin surface pH. *Skin Pharmacol. Appl. Skin Physiol.* **16**, 188 (2003).
5. Elias, P.M. The epidermal permeability barrier: from the early days at Harvard to emerging concepts. *Soc. Invest. Dermatol.* **122**, xxxvi–xxxix (2004).
6. Fluhr, J.W., Kao, J., Jain, M., Ahn, S.K., Feingold, K.R. and Elias, P.M. Generation of free fatty acids from phospholipids regulates SC acidification and integrity. *J. Invest. Dermatol.* **117**, 44–51 (2001).
7. Öhman, H. and Vahlquist, A. The pH gradient over the SC differs in X-linked recessive and autosomal dominant Ichthyosis: a clue to the molecular origin of the Acid Skin Mantle? *J. Invest. Dermatol.* **111**, 674–677 (1998).
8. Behne, M., Och, Y., Murata, S., Holleran, W.M. and Mauro, T.M. Functional role of sodium-hydrogen

antiporter NHE1. *J. Invest. Dermatol.* **114**, 797 (2000).
9. Rieger, M.M. The pH of the SC: an update. *Cosm. Toil.* **115**, 43–45 (2000).
10. Müller, R. Datenblätter zur Bewertung der Wirksamheit von Wirkstoffen in Kosmetischer Mitteln: Angabe zum pH-Wert von Kosmetische Mitteln. GDCh, Arbeitsgruppe Kosmetische Mitteln, Frankfurt, Germany (2002).
11. Leyden, J.J., McGinley, K.J., Nordstrom, K.M. and Webster, G.F. Skin microflora. *J. Invest. Dermatol.* **88**, 65–72 (1987).
12. Noble, W.C. Staphylococci on the skin. In: The Skin Microflora and Microbial Skin Disease (Noble, W.C., ed.), pp. 135–152. Cambridge University Press, Cambridge (1993).
13. Holland, K. Cosmetics, what is their influence on skin microflora?. *Am. J. Clin. Dermatol.* **3**, 455–459 (2002).
14. Rogiers, V. EEMCO guidance for the assessment of transepidermal water loss in cosmetic sciences. *Skin Pharmacol. Appl. Skin Physiol.* **14**, 117–128 (2001).
15. Berardesca, E. EEMCO guidance for the assessment of stratum corneum hydration: electrical methods. *Skin Res. Technol.* **3**, 126–132 (1997).
16. Lambers, H. and Pronk, H. Biophysical methods for stratum corneum characterization. In: Cosmetic Lipids and Skin Barrier Föster (Förster T., ed.), 185–227. Marcel Dekker, New York (2001).
17. Grunewald, A.M., Gloor, M. and Kleesz, P. Barrier recompensation mechanisms. In: Prevention of Contact Dermatitis, Volume 25 (Elsner, E.P., Lachapelle, J.M., Wahlberg, J.E. and Maibach, H.I., eds), 206–213. Karger, Basel (1996).
18. Gehring, W. and Gloor, M. Die Bedeutung des pH-Wertes bei der Hautreinigung. *Parfümerie und Kosmetik* **71**, 254–256 (1990).
19. Council-Directive 98/83/EC. Quality of water intended for human consumption. *Official J. Eur. Commun.* 44–45. 5/12/98 (1998).
20. Fluhr, J.W. Impact of anatomical location on barrier recovery, surface pH and SC hydration. *Br. J. Dermatol.* **146**, 770–776 (2002).
21. Barel, A.O., Lambrecht, R., Clarys, P., Morrison, B.M. and Paye, M. A comparative study of the effects on the skin of a classical bar soap and syndet cleansing bar in normal use conditions and in the soap chamber test. *Skin Res. Technol.* **7**, 98–104 (2001).
22. Dikstein, S. and Zlotogorski, A. Measurement of skin pH. *Acta Derm. Venereol.* **185**, 18–20 (1994).
23. Korting, H.C., Huebner, K., Greiner, K., Hamm, G. and Braun-Falco, O. Differences in the skin surface pH and bacterial microflora due to long term application of synthetic detergent preparations of pH 5.5 and pH 7.0. *Acta Derm. Venereol.* **70**, 429–431 (1990).
24. Blank, I.H. Measurement of pH of the skin surface; I and II. *J. Invest. Dermatol.* **2**, 67–79 (1939).
25. Seidenari, S. and Giust, G. Objective assessment of the skin of children affected by atopic dermatitis: a study of pH, capacitance and TEWL. *Acta Derm. Venereol.* **75**, 429–433 (1995).
26. Zlotogorski, A. Distribution of skin surface pH on the forehead and cheek of adults. *Arch. Dermatol. Res.* **279**, 398–401 (1987).
27. Yosipovitch, G. Skin barrier properties in different body areas in neonates. *Pediatrics* **106**, 105–108 (2000).
28. Fluhr, J.W., Mao-Qiang, M., Brown, B.E. et al. Functional consequences of a neutral pH in neonatal rat stratum corneum. *J. Invest. Dermatol.* **123**, 140–151 (2004).
29. Wilhelm, K-P. Skin aging: effect on TEWL, hydration, pH and sebum. *Arch. Dermatol.* **127**, 1806–1809 (1991).
30. Öhman, H. and Vahlquist, A. In vivo studies concerning a pH gradient in human SC and upper epidermis. *Acta Derm. Venereol.* **74**, 375–379 (1994).
31. Yosipovitch, G., Tur, E., Morduchowitcz, G. and Boner, G. Skin surface pH, moisture and pruritis. *Neprol. Dial. Transplant* **8**, 1129–1132 (1993).
32. Ehlers, C., Ivens, U.I., Moeller, M.L., Senderovitz, T. and Serup, J. Females have lower skin surface pH than men. *Skin Res. Technol.* **7**, 90–94 (2001).
33. Berardesca, E. Differences in SC pH gradient when comparing white causasian and black skin. *British J. Dermatol.* **139**, 8955–857 (1998).
34. Draize, J.H. The determination of the pH of skin of man and common laboratory animals. *J. Invest. Dermatol.* **5**, 77–85 (1942).
35. Yosipovitch, G. Circadian rhythms of the skin. *Cosm. Toil.* **114**, 45–47 (1999).
36. LeFur, M. Seasonal Changes in Skin Biophysical Properties in Healthy Caucasian Women. Stratum Corneum III, Basel, poster 12 (2001).
37. Sparavigna, A., Setaro, M. and Gualandry, V. Cutaneous pH in children affected by atopic dermatitis and in healthy children: a multicenter study. *Skin Res. Technol.* **5**, 221–227 (1999).
38. Eberlein-Konig, B., Schaefer, T., Huss-Marp, J. et al. Skin surface pH, SC hydration and TEWL. *Acta Derm. Venereol.* **80**, 188–191 (2000).
39. Berg, R.W., Milligan, M.C. and Sarbaugh, F.C. Association of skin wetness and pH with diaper dermatitis. *Ped. Dermatol.* **11**, 18–20 (1994).
40. Francomano, M.A., Mantovani, L. and Pepe, P. Baseline biophysical parameters in subjects with sensitive skin. *Skin Res. Technol.* **2**, 225 (1996).
41. Korting, H.C., Bau, A. and Baldauf, P. pH-abhängigkeit des Wachstums-verhaltens von *Staph. aureus* and *Propion acnes*. *Ärztliche Kosmet.* **17**, 41–53 (1987).
42. Korting, H.C. and Braun-Falco, O. The effect of detergents on skin pH and its consequences. *Clin. Dermatol.* **14**, 23–27 (1996).

43. Mauro, T.H., Holleran, W.H., Grayson, S., Behne, M., Feingold, K.R. and Elias, P.M. Barrier recovery is impeded at neutral pH, independent of ionic effects. *Arch. Dermatol. Res.* **290**, 215–222 (1998).
44. Schreiner, V., Maerker, U. and Hoppe, U. Dependence of barrier repair in human skin on intra- and extracellular pH. *J. Invest. Dermatol.* **106**, 119 (1996).
45. Wilhelm, K-P and Maibach, H. Susceptibility to irritant dermatitis induced by SLS. *J. Am. Acad. Dermatol.* **23**, 122–124 (1990).
46. Wilhelm, K-P and Maibach, H. Factors predisposing to cutaneous irritation. *Dermatol. Clin.* **8**, 17–22 (1990).
47. Takagi, Y., Kriehuber, E., Imokawa, G., Elias, P.M. and Holleran, W.M. Beta-glucocerebrosidase activity in mammalian stratum corneum. *J. Lip. Res.* **40**, 861–869 (1999).
48. Freinkel, R.K. and Traczyk, T.N. The phospholipases A of epidermis. *J. Invest. Dermatol.* **74**, 169–173 (1980).
49. Hachem, J-P., Crumrine, D., Fluhr, J., Brown, B., Feingold, K.R. and Elias, P.M. pH directly regulates epidermal permeability barrier homeostasis, and stratum corneum cohesion/integrity. *J. Invest. Dermatol.* **121**, 345–353 (2003).
50. Rawlings, A.V. and Harding, C.R. Moisturization and skin barrier function. *Dermatol. Ther.* **17**, 43–48 (2004).
51. Rawlings, A.V. Trends in stratum corneum research and the management of dry skin conditions. *Int. J. Cosm. Sci.* **25**, 63–95 (2003).
52. Harding, C.R. Dry skin, moisturization and corneodesmolysis. *Int. J. Cosm. Sci.* **22**, 21–52 (2000).
53. Kawada, A., Hara, K., Morimoto, K., Hiruma, M. and Ishibashi, A. Rat epidermal cathepsin B: purification and characterization of proteolytic properties towards filaggrin and synthetic substrates. *Int. J. Biochem. Cell Biol.* **21**, 175–183 (1995).
54. Kawada, A., Hara, K., Hiruma, M. Noguchi, H. and Ishibashi, A. Rat epidermal cathepsin-L like proteinase: purification and some hydrolytic properties toward filaggrin and synthetic substrates. *J. Biochem.* **118**, 332–337 (1995).
55. Katsuyama, M., Ogawa, S., Sayama, K., Kobayashi, Y. and Ichikawa, H. A novel way to attack the balance of skin microflora. Part 1. *J. Dermatol. Sci.* **38**, 197–205 (2005).
56. Holland, K. Cosmetics, what is their influence on skin microflora? *Am. J. Clin. Dermatol.* **3**, 455–459 (2002).
57. Chikakane, K. and Takahashi, H. Measurement of skin pH and its significance in cutaneous diseases. *Clin. Dermatol.* **13**, 299–306 (1995).
58. Noble, W.C. Physical factors affecting skin flora and disease. In: The Skin Microflora and Microbial Skin Disease (Noble, W.C., ed.), pp. 78–81. Cambridge University Press, Cambridge (1993).
59. Mc Nelly, N., Williams, H.C., Philips, D.R., Smallman-Raynor, M., Lewis, S. and Britton, J. Atopic eczema and domestic water hardness. *The Lancet* **352**, 527–531 (1998).
60. Eberlein-König, B., Schäfer, T. and Huss-Marp, J. Skin surface pH, stratum corneum hydration, transepidermal water loss and skin roughness related to atopic eczema and skin dryness in a population of primary school children. *Acta Derm. Venereol.* **80**, 180–191 (2000).
61. Sparavigna, A., Setaro, M. and Gualandri, V. Cutaneous pH in children affected by atopic dermatitis and in healthy children: a multicenter study. *Skin Res. Technol.* **5**, 221–227 (1999).
62. Tada, J. Characteristics of patient with atopic dermatitis associated with severe facial lesions. *Jpn. J. Dermatol.* **103**, 1429–1435 (1993).
63. Rippke, F., Schreiner, V., Doering, T. and Maibach, H.I. Stratum corneum pH in atopic dermatitis: impact on skin barrier function and colonization with staphylococcus aureus. *Am. J. Clin. Dermatol.* **5**, 217–223 (2004).
64. Strange, P. Staphylococcal enterotoxin B applied on intact normal and intact atopic skin induces dermatitis. *Arch. Dermatol.* **132**, 28–33 (1996).
65. Gijsen, R.M.R. Lactates as natural active ingredients. *Eurocosmetics*, June, 8–9 (1998).
66. Arikawa, J., Ishibashi, M., Kawashima, M., Takagi, Y., Ichikawa, Y. and Imokawa, G. Decreased levels of sphingosine, a natural antimicrobial agent, may be associated with vulnerability of the stratum corneum form patients with atopic dermatitis to colonization by Staphylococcus aureus. *J. Invest. Dermatol.* **119**, 433–439 (2002).
67. Braff, M.H., Bardan, A., Nizet, V. and Gallo, R.L. Cutaneous defense mechanisms by antimicrobial peptides. *J. Invest. Dermatol.* **125**, 9–13 (2005).
68. Ali, R.S., Falconer, A., Ikram, M., Bisset, C.R., Derioa, R. and Auinn, A.G. Expression of the peptide antibiotic human beta defensin-1 and defensin-2 in normal human skin. *J. Invest. Dermatol.* **117**, 106–111 (2001).
69. Schittek, A. Dermcidin: a novel human antibiotic peptide secreted by sweat glands. *Nat. Immunol.* **2**, 1133–1137 (2001).
70. Woodruffe, R.C. Natural control and ecology of microbial populations on skin and hair. *Soc. Appl. Bacteriol. Symp Ser.* **3**, 13–34 (1974).
71. Sahl, H.G. and Brandis, H. Production, purification and chemical properties of an anti-staphylococcal agent produced by S. epidermidis. *J. Gen. Microbiol.* **127**, 377–384 (1981).
72. Chikakane, K. and Takahashi, H. Measurement of skin pH and its significance in cutaneous diseases. *Clin. Dermatol.* **13**, 299–306 (1995).

73. Costerton, J.W., Geesey, G.G. and Cheng, K.J. How bacteria stick. *Sci. Am.* **238**, 86–95 (1978).
74. Ofek, I. and Beachy, E.H. General concepts and principles of bacteria adherence in animals and man. In: Bacterial Adherence (Beachy, E.H., ed.), 3–29. Chapman and Hall, London (1980).
75. Arnold, A. Relationship between certain physicochemical changes in the cornified layer and the endogenous bacterial flora of the skin. *J. Invest. Dermatol.* **5**, 207–223 (1942).
76. Beetz, H.M. Depth distribution of skin bacteria in the stratum corneum. *Arch. Dermatol. Forsch.* **244**, 76–80 (1972).
77. Ojajarvi, J. Effectiveness of hand washing and desinfection methods in removing transient bacteria after patient nursing. *J. Hyg.* **85**, 193–203 (1980).
78. Ojajarvi, J. The importance of soap selection for routine hand hygiene in hospital. *J. Hyg.* **86**, 275–283 (1981).
79. Treffel, R., Paniset, F., Faivre, B. and Agache, P. Hydration, TEWL, pH and skin surface parameters: correlations and variations between dominant and non-dominant forearms. *Br. J. Dermatol.* **130**, 325–328 (1994).
80. Krien, P.M. and Kermici, M. Evidence for the existence of a self-regulated enzymatic process within human SC. *J. Invest. Dermatol.* **420**, 414–420 (2000).
81. Aly, R., Shirley, C., Cunico, B. and Maibach, H.I. Effect of prolonged occlusion on the microbial flora, pH, CO_2 and TEWL. *J. Invest. Dermatol.* **71**, 378–381 (1978).
82. Gottfreund, J. and Meyer, T. Die Bedeutung des pH-Wertes 5.5 in Emulsionen. *Kosmetische Medizin.* **19**, 146–151 (1998).
83. Kubota, K., Machida, I., Tamura, K., Akiba, T. and Tamura, J. Treatment of refractory cases of atopic dermatitis with acid hot spring bathing. *Acta Derm. Venereol.* **77**, 452–454 (1997).
84. Schmid, M.H. The concept of the acid mantle of the skin: its relevance to the choice of skin cleansers. *Dermatology* **191**, 276–280 (1995).
85. Matousek, J.L. and Campbell, K.L. A comparative review of cutaneous pH. *Veterin. Dermatol.* **13**, 293–300, (2002).
86. Surber, C., Itin, P. and Rufli, TH. Skin surface pH after short exposure to model solutions. In: The Environmental Threat to the Skin (Marks, R. and Plewig, G., eds), pp. 277–281. Martin Dunitz Publishers, London (1992).
87. Thune, P., Nilsen, T., Hanstad, I.K., Gustavsen, T. and Dahl, H.L. The water barrier function of the skin in relation to the water content of SC, pH and skin lipids. *Acta Derm. Venereol.* **68**, 277–283 (1988).
88. Warrier, A.G., Kligman, A.M., Harper, R.A., Bowman, J. and Wickett, R.R. A comparison of black and white skin using noninvasive methods. *J. Soc. Cosmet. Chem.* **47**, 229–240 (1996).
89. Braun-Falco, O. and Korting, H.C. Der normale pH-wert der menschlichen Haut. *Hautartz* **37**, 126–129 (1986).
90. Murahata, R.I., Toton-Quinn, R. and Finkey, M.B. Effect of pH on the production of irritation in a chamber irritation test. *J. Am. Acad. Dermatol.* **18**, 62–66 (1988).
91. Locher, G. Permeabilitätsprüfung der Haut. *Dermatologica* **124**, 159–182 (1962).
92. Bock, M. and Schwanitz, H.J. Prävention irritativer Kontaktekzeme bei Friseuren durch topische Anwendung von CO_2-imprägniertem Wasser. *Occup. Environ.* **47**, 53–57 (1999).