A systematic review of the evidence for ‘myths and misconceptions’ in acne management: diet, face-washing and sunlight

Parker Magina, Dimity Ponda, Wayne Smithb and Alan Watsonc


Background. Lay perceptions that diet, hygiene and sunlight exposure are strongly associated with acne causation and exacerbation are common but at variance with the consensus of current dermatological opinion.

Objectives. The objective of this study was to carry out a review of the literature to assess the evidence for diet, face-washing and sunlight exposure in acne management.

Methods. Original studies were identified by searches of the Medline, EMBASE, AMED (Allied and Complementary Medicine), CINAHL, Cochrane, and DARE databases. Methodological information was extracted from identified articles but, given the paucity of high quality studies found, no studies were excluded from the review on methodological grounds.

Results. Given the prevalence of lay perceptions, and the confidence of dermatological opinion in rebutting these perceptions as myths and misconceptions, surprisingly little evidence exists for the efficacy or lack of efficacy of dietary factors, face-washing and sunlight exposure in the management of acne. Much of the available evidence has methodological limitations.

Conclusions. Based on the present state of evidence, clinicians cannot be didactic in their recommendations regarding diet, hygiene and face-washing, and sunlight to patients with acne. Advice should be individualized, and both clinician and patient cognizant of its limitations.

Keywords. Acne, diet, hygiene, sunlight.

Introduction

This article reviews the evidence base for common lay and medical beliefs regarding acne. A 2001 article advocated ‘debunking myths about acne’ and, among ‘myths’ nominated for debunking, were those related to diet (chocolate and fatty foods), hygiene, face cleansing and sun-exposure.

These perceived myths and misconceptions about acne causation, exacerbating factors and treatment efficacies are common, and not just among patients. In an analysis of examination answers of final year medical students at Melbourne University, Green and Sinclair found that 67% of students identified stress, 10% identified smoking and alcohol, and 25% identified poor facial hygiene as exacerbating factors in acne. Dietary factors (especially chocolate, oily or fatty foods and high sugar-content foods) were nominated by 41% as exacerbating acne. The corollary of these beliefs about aetiology were some of the treatment recommendations of the final year students—cleaners and washes, antiseptics and medicated soaps, and improved facial hygiene and diet.

In these matters the students’ responses reflect popular attitudes and opinions, but they are at variance with a consensus of dermatological opinion that diet, stress and uncleanliness are unrelated to acne pathogenesis and that skin cleansing and dietary manipulation are ineffective in acne treatment.

Similarly, in a 2001 Australian study of obstetric ward doctors and nurses, 12% of doctors and 20% of nurses believed sun exposure to be therapeutic for acne. There has been a lay and traditional dermatological opinion that sunlight exposure was beneficial in acne and that this caused acne to improve in summer and worsen in winter, though this is now generally thought to be more likely not to be the case.
Methods

A literature search was performed in July 2003 using the databases Medline, EMBASE, AMED (Allied and Complementary Medicine), CINAHL, Cochrane, and DARE. Search terms used were acne vulgaris with combinations of myths, misconceptions, diet, chocolate, sugar, hygiene, wash, cleanse, sun, light, and ultraviolet. The search was confined to English language articles. Reference lists of identified articles were examined for further relevant studies. There were no pre-specified quality criteria for study inclusion. Methodological information was extracted to assist in interpretation of results. Many studies had methodological shortcomings—for example, small sample sizes with no power considerations, lack of control subjects, lack of blinding, or unclear or unstated statistical methods. Given the paucity of high quality studies found and the seemingly considerable effect some studies of limited methodological quality have had on current opinion and practice, no studies were excluded from the review on methodological grounds. The methodological limitations of studies, and the resultant implications for interpretation of findings, are noted.

Results

Medline, EMBASE, CINAHL and AMED searches identified 221, 171, 28 and 1 references, respectively. Examination of these citations and abstracts produced eleven trials of dietary, washing or ultraviolet-light exposure treatment modalities. Hand-searching of references from papers obtained in the search located a further 14 studies.

The role of dietary factors (Table 1)

There are, perhaps surprisingly, few studies that examine the role of diet in acne. Three studies have specifically examined the role of chocolate.

Grant and Anderson and Anderson performed trials of chocolate, milk and roasted nuts in university students and found no effect on acne, but the trials were small, uncontrolled, had very short follow-up, and inadequate statistical analysis. Fulton et al. in a single-blind placebo-controlled cross-over trial in American hospital acne clinic attendees and male prisoners found no effect of chocolate on acne (on sebum production or composition). A small study of patients with acne (16 subjects and 13 matched controls) found no difference in sugar consumption between the two groups—though patients with seborrhoeic dermatitis had higher levels of sugar consumption.

A study of 2720 British soldiers found no difference in weight between soldiers, aged 15–19, with or without acne. Soldiers aged 20–40 with acne however were significantly heavier (5.6 kg) than soldiers aged 20–40 without acne. This study has been quoted in the context of a possible link between diet and acne, but the evidence of this study for such a link would appear tenuous.

Recently, a reappraisal of the current thinking regarding diet and acne has been proposed following a cross-sectional study of acne in native, non-westernised New Guinean and Paraguayan populations. This study showed no cases of acne in either of these populations, and this was contrasted with prevalence of acne in western populations. It is proposed that western diets, with characteristically high glycaemic indexes, lead to hyperinsulinaemia and a resulting cascade of endocrine consequences (increased androgens, increased insulin-like growth-factor 1, altered retinoid signaling pathways) which mediate acne pathogenesis.

A further recent study has demonstrated a correlation of worsening ‘diet quality’ (during a pre-examination period in university students) and exacerbation of acne. The main factor examined in this study was the effect of examination stress on acne severity, and the dietary variable measured was self-assessed diet quality (on a scale 1–4) rather than a validated objective measure of dietary components.

The role of dirt, hygiene, cleanliness and washing (Table 2)

Soap has been advocated in the treatment of acne since the 19th century and Solomon and Shalita, in a 1996 review of the use of detergents, soaps, cleansers, foaming solutions, moisturisers and washes in acne, make detailed recommendations regarding skin cleansing in acne. But they cite very little evidence for their recommendations.

Improvements in acne have been noted in a small (ten subjects) uncontrolled study of a medicated face wash, a further uncontrolled, and incompletely reported, study of face washing, a study in which an abrasive was used in addition to a medicated wash in some subjects but had no non-wash controls, an open uncontrolled and incompletely reported study of a cleansing bar and the ‘Buf-puf’ abrasive device, and in studies in which medicated soap or acidic syndet bar was compared with unmedicated soap but in which, again, no non-wash comparison was studied. The large placebo effect in placebo-controlled acne therapy trials—19–56% in a sample of seven placebo controlled trials of tetracyclines in acne and in one study, 5% benzoyl peroxide and, in another two studies, the vehicle employed in the chlorhexidine preparation. There was no significant difference in acne lesion counts at 8 and 12 weeks in the chlorhexidine/benzoyl peroxide study. The combined data of the two chlorhexidine/vehicle studies showed significantly less...
A povidone-iodine cleanser was reported to improve acne in randomized controlled trials, but statistical reporting was deficient. Swinyer et al. studied the effect on acne of three treatment regimens. Each treatment regimen included a different cleansing/washing modality. However each regimen also included a different

<table>
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<tr>
<th>Study</th>
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<th>Brief description of intervention</th>
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<th>Summary of results and comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grant²⁹ 1965</td>
<td>SBA</td>
<td>Chocolate (9&amp;3/4 ounces) on two successive days</td>
<td>University students with mild or moderate acne.</td>
<td>8</td>
<td>4 of the 8 subjects developed up to 5 new papules or pustules. Considered not significant change—but no statistical evaluation, very small sample size, uncontrolled, follow-up may not be long enough to detect changes.</td>
</tr>
<tr>
<td>Anderson³⁰ 1971</td>
<td>SBA</td>
<td>Chocolate, milk or peanuts (daily for one week)</td>
<td>University students with acne who identified dietary triggers.</td>
<td>Not specified</td>
<td>Reporting limited. No apparent control group. Numbers and statistical analysis not reported. Follow-up not clear—possibly 3–7 days following dietary intervention. Treatment duration and follow-up may not be long enough to detect changes.</td>
</tr>
<tr>
<td>Fulton³¹ 1969</td>
<td>Single blind</td>
<td>Chocolate bar vs placebo bar with similar caloric and fat composition</td>
<td>Acne clinic patients and male prisoners. Mild-moderate acne.</td>
<td>65</td>
<td>No difference in acne severity during chocolate and control bar study periods. High fat content of control bar could be acneigenic. 4 week duration of treatment in cross-over design may be too short, given natural history of acne lesions.</td>
</tr>
<tr>
<td>Bett³² 1967</td>
<td>Cross-sectional</td>
<td>N/A</td>
<td>Specialist outpatient clinic with seborrheic dermatitis or acne. Controls: patients with warts or no skin disease.</td>
<td>16 dermatitis and 16 acne. 32 controls</td>
<td>No difference in sugar consumption between controls and acne patients. Significantly greater consumption of sugar by patients with seborrheic dermatitis than by controls.</td>
</tr>
<tr>
<td>Bourne³³ 1956</td>
<td>Cross-sectional</td>
<td>N/A</td>
<td>Soldiers aged 15–40.</td>
<td>2720</td>
<td>No correlation of acne presence/severity in ages 15–19. Aged 20–40, subjects with acne were significantly heavier than those without acne.</td>
</tr>
<tr>
<td>Cordain³⁴ 2002</td>
<td>Cross-sectional</td>
<td>N/A</td>
<td>Paraguayan and New-Guinean tribal societies.</td>
<td>1315 (315 aged 15–25)</td>
<td>No acne lesions observed in any subject.</td>
</tr>
<tr>
<td>Chiu³⁶ 2003</td>
<td>Cohort</td>
<td>N/A</td>
<td>University students with acne during exam period.</td>
<td>22</td>
<td>An increase in stress (on the Perceived Stress Scale) was significantly correlated with increases in acne severity. Self-assessed dietary quality (graded 1–4) was also significantly correlated with acne severity (r = −0.48).</td>
</tr>
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</table>

acne lesions at 8 and 12 weeks in the chlorhexidine-using subjects than in the unmedicated vehicle-using controls.
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<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Cunliffe(^{39}) 1972</td>
<td>SBA</td>
<td>Facial washing with medicated wash</td>
<td>Mild or moderate acne</td>
<td>10</td>
<td>Significant improvement in acne severity after 3 months. Uncontrolled. Method of severity assessment not reported.</td>
</tr>
<tr>
<td>Hulme(^{40}) 1986</td>
<td>SBA</td>
<td>Facial washing with a detergent-containing product</td>
<td>Mild-moderate acne</td>
<td>55</td>
<td>Significant decrease in inflammatory lesions, but not comedones.</td>
</tr>
<tr>
<td>Fulghum(^{41}) 1982</td>
<td>Paired design (right vs left side of face)</td>
<td>Cleanser vs cleanser + polyethylene granules</td>
<td>Mild-moderate acne in specialists’ practices</td>
<td>44</td>
<td>No difference in acne severity or adverse effects between the two products. No non-cleanser control.</td>
</tr>
<tr>
<td>MacKenzie(^{42}) 1977</td>
<td>SBA</td>
<td>Abrasive polyester cleansing pad</td>
<td>Comedonal, papular/pustular, or cystic acne</td>
<td>97</td>
<td>Reported to be effective, but uncontrolled, subjective assessment of efficacy and no statistical analysis.</td>
</tr>
<tr>
<td>Bettley(^{43}) 1972</td>
<td>Cross-over trial</td>
<td>Medicated wash + clearasil cream vs unmedicated wash + clearasil cream</td>
<td>Not specified</td>
<td>41</td>
<td>Medicated wash significantly greater improvement than unmedicated wash. Means of assessment of severity and of randomization of treatment order not clear. Periods of use (1 month) physiologically unlikely to be long enough to achieve therapeutic effect. Statistical analysis not specified.</td>
</tr>
<tr>
<td>Korting(^{44}) 1995</td>
<td>RCT</td>
<td>Acidic Syndet Bar vs Alkaline Soap Bar</td>
<td>Mild acne (‘pre-acne’)</td>
<td>120</td>
<td>Significantly less inflammatory lesions with acidic syndet: evident at 4 weeks and effect still present at 12 weeks. No blinding. No non-wash controls.</td>
</tr>
<tr>
<td>Stoughton(^{47}) 1987</td>
<td>RCT</td>
<td>Chlorhexidine gluconate skin cleanser vs 5% benzoyl peroxide</td>
<td>Acne with at least 10 papules or pustules</td>
<td>50</td>
<td>No difference in acne lesion counts at 8 and 12 weeks between 2 treatments.</td>
</tr>
<tr>
<td>Stoughton(^{47}) 1987</td>
<td>RCT</td>
<td>Chlorhexidine gluconate skin cleanser vs placebo (inactive vehicle)</td>
<td>Acne with at least 10 papules or pustules</td>
<td>110</td>
<td>Significantly less acne lesions at 8 and 12 weeks in chlorhexidine treated group. Combined data of two studies reported.</td>
</tr>
<tr>
<td>Millikan(^{48}) 1976</td>
<td>RCT</td>
<td>Povidone-iodine cleanser vs control cleanser</td>
<td>Mild acne</td>
<td>17</td>
<td>Reported superiority of povidone-iodine. But result seemingly non-scientific.</td>
</tr>
<tr>
<td>Millikan(^{48}) 1976</td>
<td>RCT</td>
<td>Povidone-iodine cleanser + tetracycline vs control cleanser + tetracycline</td>
<td>Moderate to severe acne</td>
<td>27</td>
<td>No difference.</td>
</tr>
<tr>
<td>Swinyer(^{49}) 1980</td>
<td>RCT</td>
<td>Soap + scrub + tetracycline vs tretinoin + soap + tretinoin vs cleanser + water avoidance + tretinoin + benzoyl peroxide</td>
<td>Hospital out-patients with mild–moderate acne</td>
<td>118</td>
<td>Tretinoin + benzoyl peroxide + cleanser as effective as tetracycline + tretinoin + soap. And more effective than tetracycline + abradant scrub. Unable to identify efficacy of individual components of regimens.</td>
</tr>
</tbody>
</table>
systemic or topical antibiotic combination, so the role of cleansing or washing cannot be adequately assessed in this study.40

The role of sunlight (Table 3)
The authors of a Saudi Arabian study which found that new cases of acne at a hospital dermatology clinic increased during the winter months concluded that this was due to the favourable effect of UV-light on acne during the warmer months.28 But they do not present any data on referral procedures and patterns, waiting times or other factors which might confound this observation. Elsewhere, acne has been found to improve in one third of subjects in summer, worsen in another third, and remain the same in the other third—though these results were obtained in a retrospective study26 and are thus prone to recall bias as well as not having controlled for potential confounders such as comedogenicity of sunscreens.

It might be thought that studies of the effect of natural sunlight on acne might be inherently problematic methodologically. In this circumstance, inferences drawn from trials of artificial light-sources may be of relevance. Trials of artificial UV-B, UV-A and combined light sources,27 and phototherapy with artificial blue-red, blue, violet, green and full-spectrum light sources50–53 have been found to improve acne, but methodological concerns—statistical significance or lack of controls or lack of blinding—limit interpretation of these results to varying degrees. While these studies may suggest a possible ameliorating effect of UV-light on acne, a trial

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</thead>
<tbody>
<tr>
<td>Al-Ameer28 2002</td>
<td>Cross-sectional</td>
<td>N/A</td>
<td>Hospital outpatients</td>
<td>220</td>
<td>More patients were seen during the colder months. No consideration of potential confounders.</td>
</tr>
<tr>
<td>Gfesser26 1996</td>
<td>Cross-sectional</td>
<td>N/A</td>
<td>All grades of severity. Source not specified</td>
<td>139</td>
<td>One third had summer exacerbation, one third had winter exacerbation, and one third didn’t vary seasonally. Exacerbation measured retrospectively by patient recall. No consideration of confounders.</td>
</tr>
<tr>
<td>Mills27 1978</td>
<td>Controlled trial. Means of assignment to groups not reported</td>
<td>UV-A vs UV-B vs UV-A + UV-B vs photo-chemotherapy vs 2 photo-sensitization regimens</td>
<td>Specialist clinic patients with moderately severe acne</td>
<td>126</td>
<td>No reduction in comedones. UV-B and UV-B + UV-A reported to reduce overall lesion counts. But statistical significance not tested. No non-photoraphy controls. Insufficient reporting of methodology.</td>
</tr>
<tr>
<td>Papageorgiou50 2000</td>
<td>RCT</td>
<td>Artificial blue light vs blue + red light vs white light vs benzoyl peroxide</td>
<td>Hospital out-patients with mild-moderate acne</td>
<td>107</td>
<td>Significant improvement in inflammatory acne with blue-red light compared to benzoyl peroxide or white light. Significant improvement in comedonal acne with blue-red light compared to white light. Unblinded.</td>
</tr>
<tr>
<td>Ammad51 2002</td>
<td>SBA</td>
<td>Artificial blue light</td>
<td>Mild-moderate acne</td>
<td>21</td>
<td>Improvement in acne severity (significant for inflammatory lesions, non-significant for comedones) and patients’ quality of life. Uncontrolled.</td>
</tr>
<tr>
<td>Kawada52 2002</td>
<td>SBA</td>
<td>Artificial blue light</td>
<td>Mild-moderate acne</td>
<td>30</td>
<td>Improvement in acne lesion counts and physician rating of improvement (greater for inflammatory than comedonal acne). No controls. Statistical significance of results not stated.</td>
</tr>
<tr>
<td>Sigurdsson53 1997</td>
<td>RCT</td>
<td>Green vs violet vs full-spectrum light-source. No non-light controls</td>
<td>Mild-moderate acne</td>
<td>30</td>
<td>No differences in acne lesion counts for three light sources. All three light sources improved acne counts significantly (but no non-light-source control group).</td>
</tr>
</tbody>
</table>
of UV-radiation has also found it to enhance the comedogenicity of sebum in the ear skin of rabbits—the best available animal model of human facial skin.54

**Discussion**

**The context of the debate: lay beliefs regarding acne causation.**

The question of whether common perceptions and beliefs regarding diet, cleanliness and sunshine prove to be fact or misconception is of considerable importance due to the practical implications of these beliefs for acne management, adverse effects, expense and potential psychological sequelae, and due to their prevalence and consistency across different western cultures. Studies from the United States,55,57 New Zealand,56 Britain,58 Germany,59 Nigeria,60 Saudi Arabia61 and Sweden62 report high prevalence of belief in the causal or therapeutic roles of diet, cleanliness and sunlight in acne.

**Diet.** Dietary modifications are commonly practised by patients with acne. Though not usually having the potential for adverse nutritional sequelae of dietary restrictions employed in some other conditions, they can nevertheless be burdensome for patients. The evidence for their efficacy or otherwise is not strong. Convincing trials are lacking.

The studies of acne and chocolate of Grant and Anderson29 and Anderson30 have considerable methodological shortcomings. The subjective self-assessed measure of global dietary quality in Chiu et al.'s study76 renders these findings of limited relevance to this review. The trial of Fulton et al.31 was methodologically stronger. But the findings of this study are worthy of closer inspection. Most importantly, the placebo bar was of a similar fat and sugar composition to the study (chocolate) bar. Therefore, while this study might suggest no role for the cocoa content of chocolate bars in acnegenesis, the role of the complete product remains open to question. Furthermore the treatment period for both chocolate and placebo bars was just four weeks. Consideration of the pathogenesis of acne lesions may be relevant. It has been hypothesized that chocolate may exacerbate acne by production of more comedogenic sebum—by increasing blood lipid levels5 or by producing 'less fluid sebum'63—and thence greater obstruction of pilosebaceous follicles, setting the stage for follicle rupture and secondary inflammatory changes. A further possible mechanism by which acne could be exacerbated is via hyperinsulinaemia and changes in the HPA axis34,35 (see below). Neither of these mechanisms might be expected to produce changes in a short time-frame. Certainly, all efficacious medical treatments of acne take two months or more to produce clinically significant changes.64,65 Given the four week treatment periods and three week wash-out period in the cross-over design, it may be that there was insufficient study duration to observe the relevant changes.

The New Guinea/Paraguay study cited is consistent with the often quoted observations of Schaefer that acne “used to be unknown among Eskimos, but one can see it readily amongst [eskimo] teenagers . . . many eskimos themselves blame their pimples on ‘pop, chocolate and candies’.66 Furthermore, a physiological mechanism of causation (initiated by hyperinsulinaemia with subsequent androgen increases) has been proposed. Indirect support for this proposition is to be found in the high rates of acne in the condition polycystic ovary syndrome which is characterised by hyperinsulinaemia and high androgen levels,67 and in studies which have found an association between acne in females and higher levels of androgens.68,69

But comparing the findings of studies in New Guinean and Paraguayan hunter-gathers with rates of acne in Western populations, even when supported by biologically plausible hypotheses of mechanism of causation, does not provide proof of a causal relationship—it is inherently prone to “ecological fallacy”70: the individual diet of the subjects who develop acne is not known and confounders cannot be assessed. As has been commented on,35,71 the obvious alternative explanation of the low prevalence of acne in these non-westernized populations is that of genetic susceptibility to acne.72 But, perhaps, of more immediate practical significance, is the fact that the therapeutic institution of such a non-western diet is unlikely to be acceptable to adolescents with acne. The dietary restrictions which Western adolescents with acne employ in managing their condition are trivial compared to the differences between their diets and those of hunter-gatherers.

Thus, in 2003 there is not yet compelling evidence on which GPs or other clinicians can base advice regarding nutrition in relation to acne.

**Facial hygiene and face cleansing.** Not only are the facial cleansing regimens of patients with acne often burdensome, they can be expensive. The evidence for the role of a lack of facial hygiene in acne pathogenesis and for face cleansing in its management is mostly of poor quality. Furthermore, face-washing has been proposed as being traumatising, and so exacerbating acne73 and as increasing the skin irritation adverse effects of topical tretinoin and isotretinoin (though not other topical therapies) in acne treatment.49,74,75

Additionally, commonly used soaps and shampoos have been found to be comedogenic when applied to the rabbit ear.76 Even the author of one of the above cited studies concedes face washing for acne “continues to be empirical therapy”.40

**Sunlight.** Convincing direct evidence for a positive effect of sunlight exposure on acne is lacking. Recent findings suggesting various spectrums of artificial light to
be efficacious may not be directly generalizable to natural sunlight. Another consideration is that the long-term sequelae of sun exposure for acne therapy may well be an increased risk of melanoma and non-melanoma skin malignancy— notwithstanding the intriguing association of acne with decreased melanoma risk.\textsuperscript{77} Additionally, photosensitivity is an issue with commonly used, efficacious medical treatments of acne—tetracyclines\textsuperscript{78–80} and isotretinoin.\textsuperscript{81,82}

**Evidence from twin studies**

Indirect evidence of a lack of a major role for environmental factors including diet and skin hygiene in acne pathogenesis comes from studies of the genetic determinants of acne. Research in this area has found strong evidence, especially from twin studies, for the role of heredity in acne.\textsuperscript{83} A large twin study of 458 pairs of monozygotic and 1099 pairs of dizygotic female twins in the UK\textsuperscript{84} was performed to assess the relative contribution of genetic and environmental factors on the liability to acne. The study found that 81\% of the variance of the disease was attributable to genetic factors and only 19\% to environmental factors. These results contradict the results of an earlier study which found similar rates of acne concordance in monozygotic and dizygotic twins— but this was a much smaller study (only 20 pairs of twins in each group). A further British case–control study of hospital dermatology adult patients with persistent acne\textsuperscript{85} found a significantly greater risk of adult acne in relatives of patients than in relatives of controls (odds ratio 3.93), though this may be explained by shared environmental exposures among families.

Thus, the potential for intervention in environmental areas may be limited. The devotion of considerable time, effort and expense on the part of patients in efforts to address purported environmental factors in their condition might be reassessed and be better directed towards optimising medical therapy—management strategies for which strong evidence of efficacy exists.\textsuperscript{9}

**Psychological implications**

The psychological dimension of this debate should also be considered. A 1976 *British Medical Journal* editorial, noting the potential for excessive or obsessional face-washing to exacerbate acne, suggested that obsessional washing may be related to the perception that acne was caused by dirt and that it is regarded as “an outward sign of moral defilement”.\textsuperscript{73} An academic dermatologist has observed “most of the dietary manipulations advocated by non-professional advisors seem more calculated to punish rather than to cure, and none are backed up by experimental evidence".\textsuperscript{13} And Green and Sinclair\textsuperscript{2} suggest a worrying implication of the linkage of facial hygiene to acne aetiology and treatment—the implication that acne is a consequence of being dirty and that sufferers are unhygienic. A reasonable hypothesis following from this proposition is that such misconceptions may exacerbate the recognized psychological sequelae of acne—especially in the areas of self-esteem, shame and embarrassment.\textsuperscript{56,58,87–91}

**Conclusion**

The evidence base for current recommendations regarding dietary, face-washing and UV-exposure behavioural modifications in acne management is incomplete at best. Studies have often been of small sample size, uncontrolled, or unblinded.

There are also, perhaps, a number of other factors that may influence recommendations to patients. The potential for sun-exposure to increase risk of skin malignancy must be considered. The anecdotal evidence of patients that certain foodstuffs exacerbate their acne cannot be dismissed out of hand. The cost of medicated washes for acne can be considerable. Methodologically rigorous research is clearly required to address the effect of these exposures on acne.

The inescapable conclusion is that, given our present state of knowledge, clinicians cannot be didactic in their recommendations. Advice should be individualized, and both clinician and patient cognizant of its limitations.

**Declaration**

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Acne ‘myths and misconceptions’: a review


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