



An introduction to the factors that influence acne skin conditions



What is acne?

There are various forms of acne which include acne vulgaris, acne conglobata and post-adolescent female acne amongst others. To the layman, however, the term “acne” generally refers to a condition known as acne vulgaris.

Acne vulgaris is a chronic inflammatory disorder of the pilosebaceous unit. This condition presents itself by a variety of lesions such as comedones, papules, pustules and in severe cases, nodules and cysts. Depending on the severity of the condition and the level of inflammation present, erythema and oedema are evident.

Severe acne can cause extreme disfiguration which, in young individuals, can impact personality development. It is a common skin disease that affects the quality of life and is associated with a relatively high prevalence of depression and suicide.

Cells and structures involved in acne

> Keratinocytes

> Connective tissue

- fibroblasts
- macrophages
- mast cells
- lymphocytes
- collagen fibres
- elastin

> Sebaceous gland (SG)

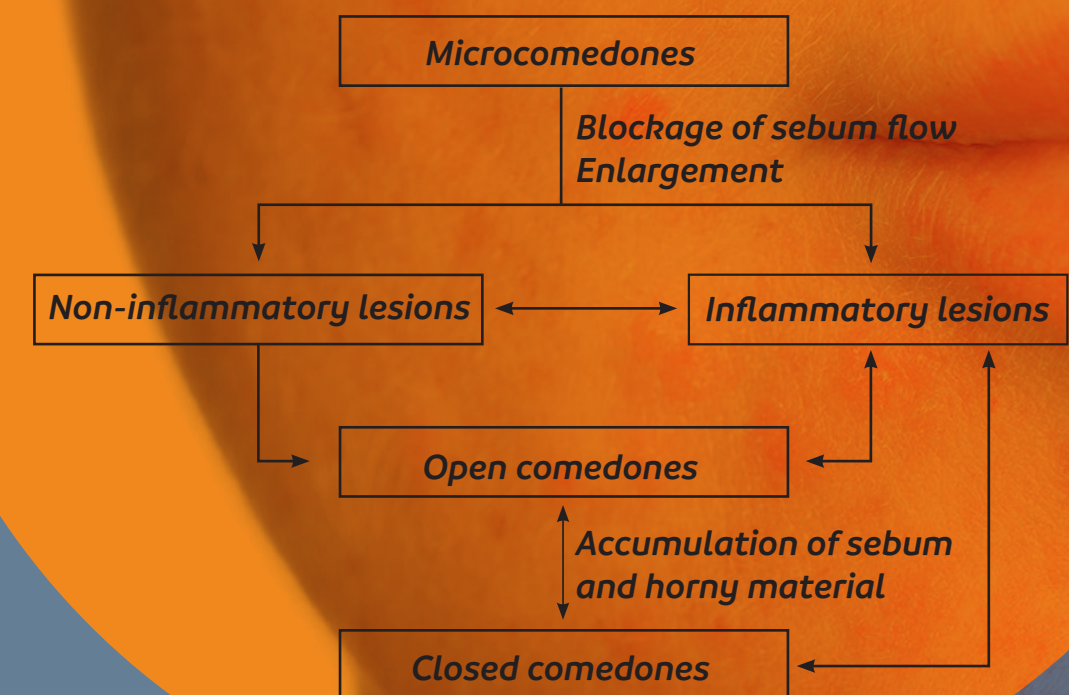
- Sebocytes
- Sebum

> Hair follicles

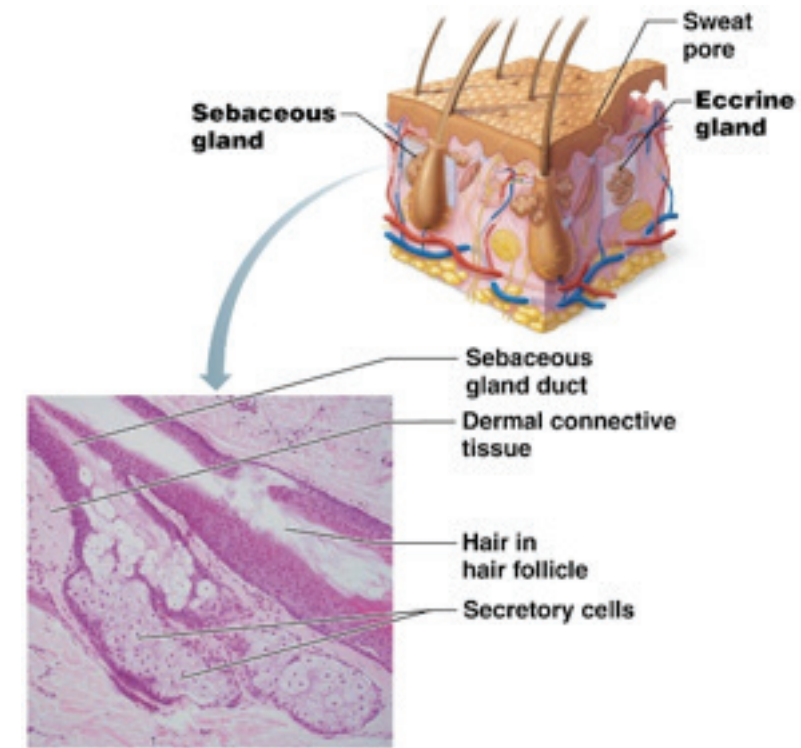
Together, the sebaceous gland and the hair follicle form the pilosebaceous unit.

Pathogenesis of acne

The pathogenesis of acne is a multidisciplinary process not yet well understood. It includes four major events: hyperkeratosis, microbial invasion, hormonal involvement and the presence of chronic inflammation. We will have a look at each of these major events:



Hyperkeratosis and comedogenesis



(a) Photomicrograph of a sectioned sebaceous gland (100×)

Figure 1: Photomicrograph of a sectioned sebaceous gland (Marieb, 2015:120)

In normal skin, the keratinocytes within the hair follicle are loosely layered and because regular desquamation can occur, carry desquamated cells to the surface of the skin via the flow of sebum. This means that there will be a balance between new cells and desquamated ones. In skin affected by acne, there is an increased proliferation of keratinocytes, disrupting that fine balance.

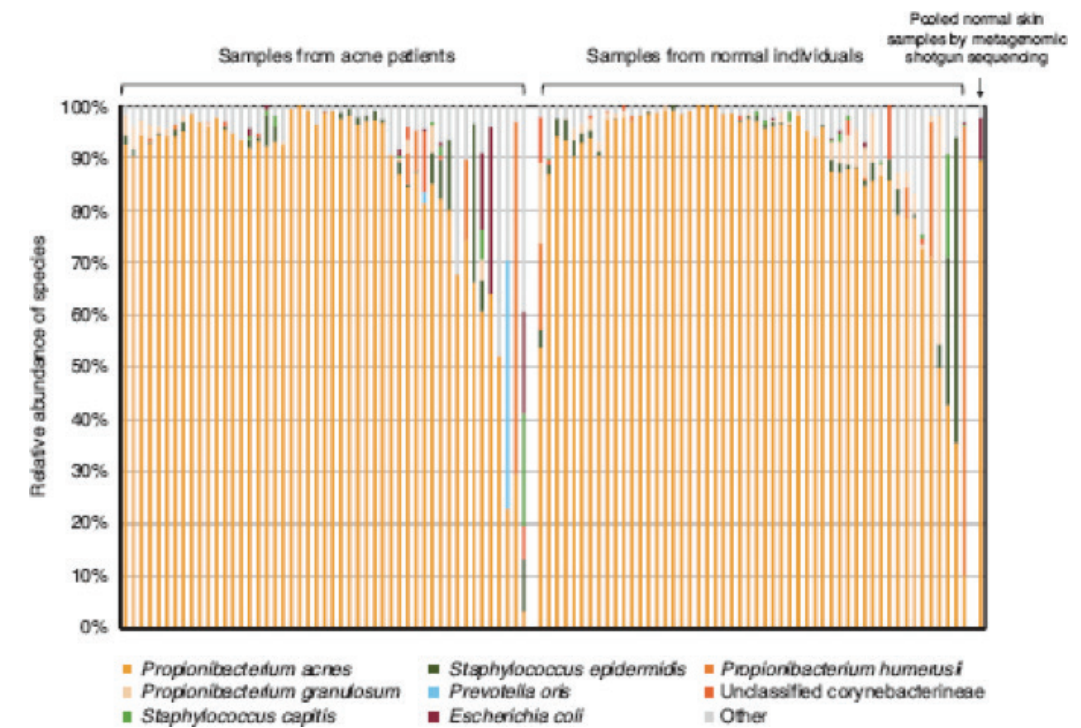
The conventional view is that the pilosebaceous unit becomes plugged with hyperkeratinised cells. It creates an ideal breeding ground for the multiplication of anaerobic microbes and since sebum is now trapped within the follicle, hydrolysis of the triglycerides into FFA's continues, resulting in irritation and inflammation which gives rise to various acne lesions.



— Microbial involvement in acne: ●

It is increasingly believed that the interaction between skin microbes and host immunity plays an important role in this disease as microbiome disruptions are observed in acne skin. —

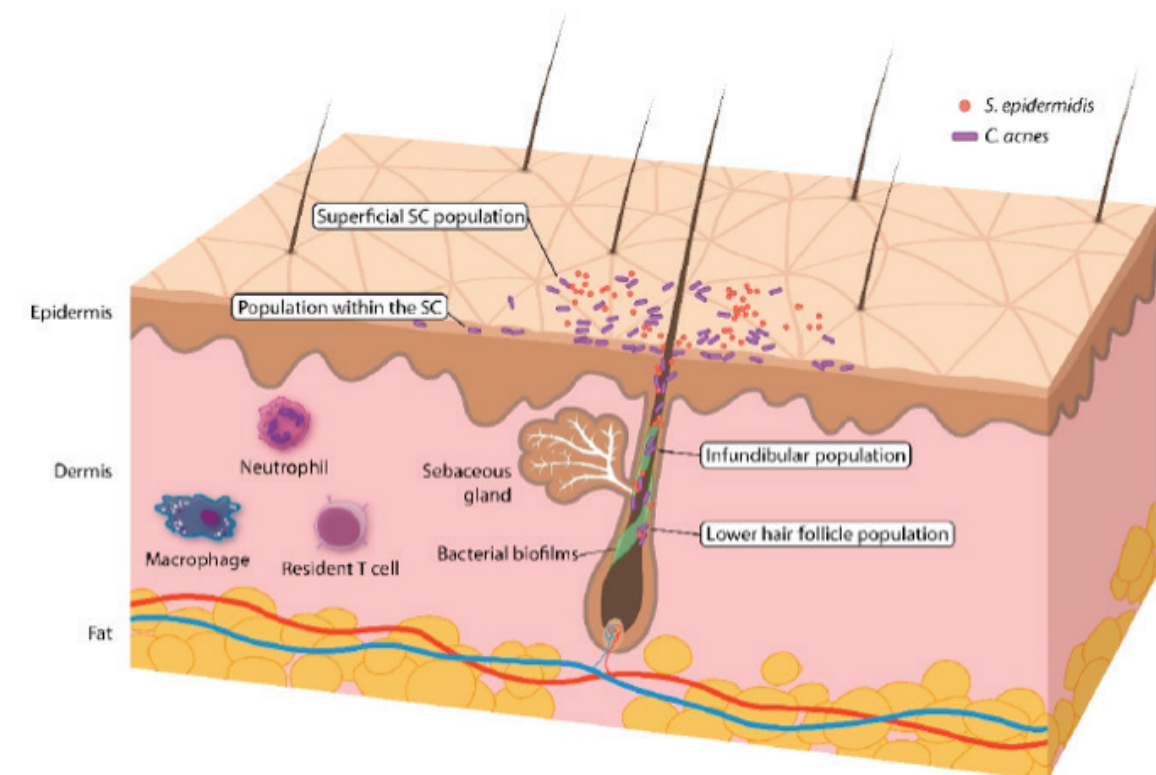
Microbial imbalances and acne

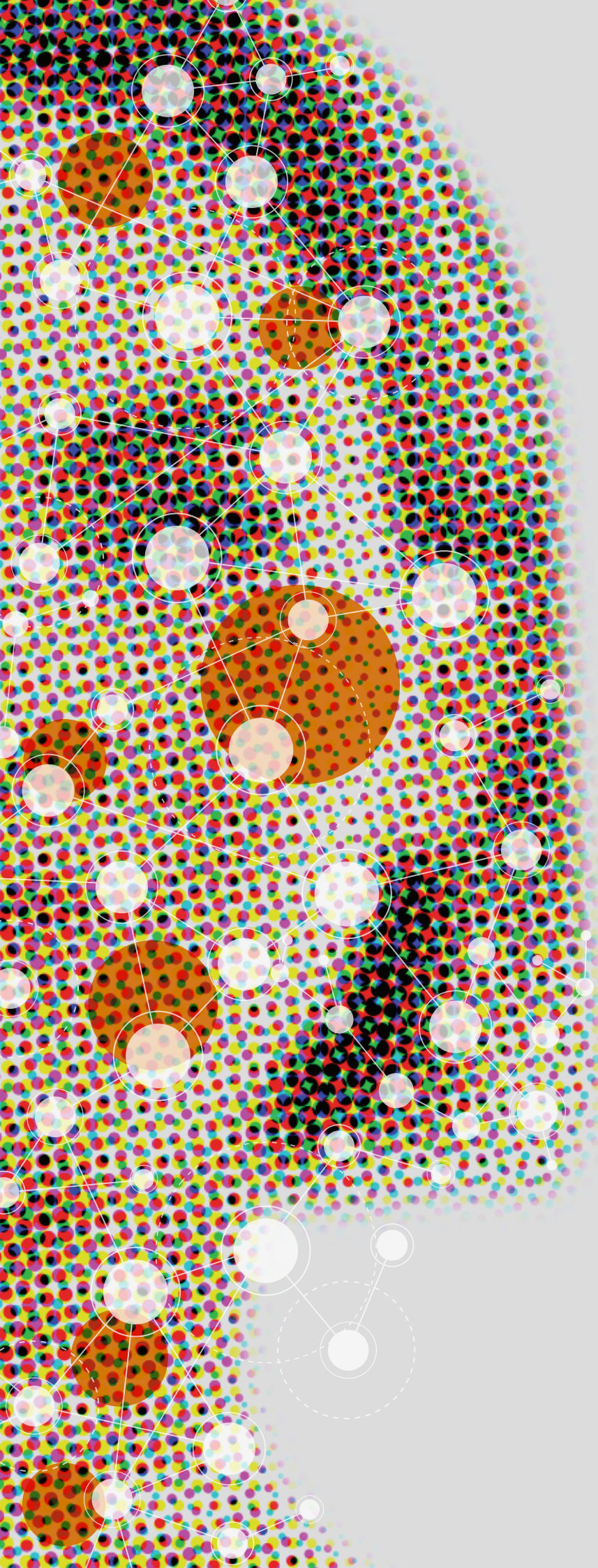


Even though commensal, some resident microbes have been associated with inflammatory skin diseases, such as *P. acnes* (acne), *Malassezia furfur* (seborrheic dermatitis) and *Demodex* (rosacea) when they over proliferate.

Other microbes are known pathogens, like *Staphylococcus aureus* and *Streptococcus pyogenes*.

P. acnes has been considered the likeliest cause of acne. Although the role of *P. acnes* in the development of acne remains uncertain, it is important to note that there is no real difference in the abundance of *P. acnes* in normal vs acne skin. *P. acnes* accounts for 87% of all the microbiota found in the pilosebaceous glands and is primarily known as a beneficial commensal.



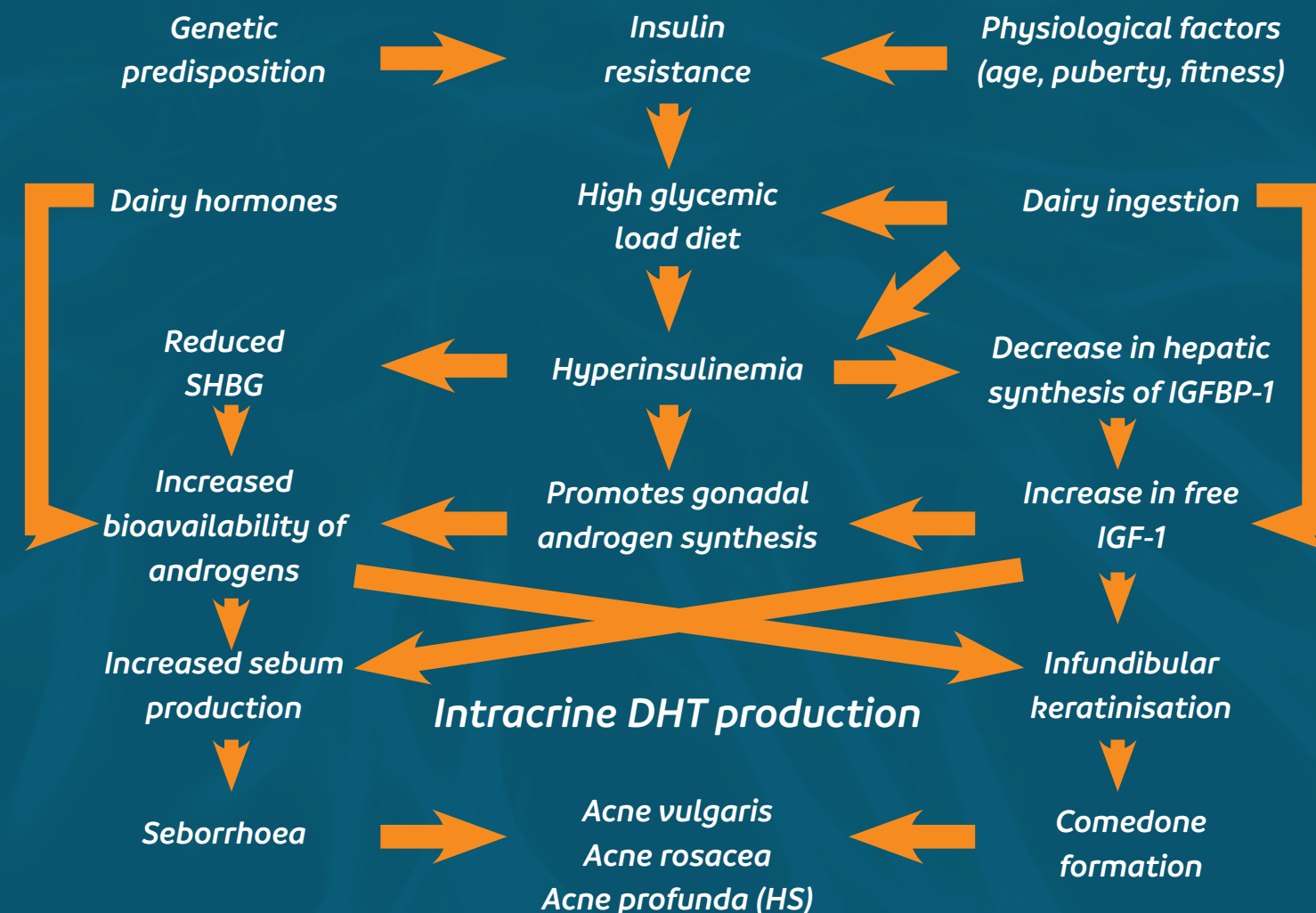


P. acnes is thought to contribute to acne pathogenesis through several different mechanisms including interaction with innate cutaneous immunity and keratinocyte and sebocyte function, leading to amplification of the three key pathologic processes implicated in acne development: inflammation, keratinisation, and sebogenesis.

Although *P. acnes* is best-known for its connection with acne, it is speculated that other bacteria might also (indirectly) contribute to the inflammatory process such as *Malassezia*, *S. epidermidis* and *S. aureus*.

Hormonal involvement

Hormonal involvement in acne is intricate and contributes to the development of acne due to involvement in sebocyte proliferation, differentiation and lipogenesis, increase in the sebaceous secretion, hyperkeratinisation and comedone formation.



Inflammation and acne

Even at subclinical stages, acne is a chronic inflammatory and immune-mediated disease. Some strains of *P. acnes* can be responsible for the local inflammatory response of acne. *P. acnes* colonises sebaceous follicles and releases lipase and proinflammatory cytokines. An increase in neutrophils and lymphocytes can be observed in acne, regardless of severity. It is the accumulation of dead white blood cells in the pilosebaceous cavity that constitutes the pus in an acne lesion.

Inflammation is caused by the activation of the skin's innate immune system, including keratinocytes, macrophages and sebocytes, that produce pro-inflammatory lipids and cytokines.

Barrier function and acne

It is imperative to understand that barrier function in the pilosebaceous unit is governed by tight junctions (T-junctions). Acne can most simply be explained by a breakdown in barrier function in the PSU that activates an inflammatory response that results in the redness and swelling associated with a lesion. The accumulation of dead white blood cells in the PSU adds to the building pressure.

Microbiome and acne

The skin can be considered as a complex ecosystem, being home to a diverse group of micro-organisms including bacteria, viruses, archaea and fungi. The skin is constantly challenged to maintain homeostasis with resident microbes, and microbial fluctuations can contribute to modifications and diseases in this ecosystem.

All though microbes are primarily commensal, under certain circumstances, they can become opportunistic pathogens, causing skin disorders such as acne.

Aggressive treatment approaches can also alter the skin microbiome and can create environments for infection and inflammation.



Lifestyle and acne

Stress, dietary changes, lack of sleep and exercise and other lifestyle changes can all be contributing factors to the development of acne. Modern lifestyle has been linked to the increase in adult and menopausal acne. There is constant pressure from society to balance a career, work and family life. Interruption in sleep cycles can lead to a rise in stress hormones that have been directly linked to the development of acne. Emotional stress, depression and anxiety have been thought to aggravate acne by altering the gut microbiota and increasing intestinal permeability, potentially contributing to skin inflammation. Getting enough sleep, reducing stress, exercising, maintaining a healthy diet with a low glycemic load, quitting smoking, avoiding bovine milk and maintaining a healthy body weight can all help reduce acne.

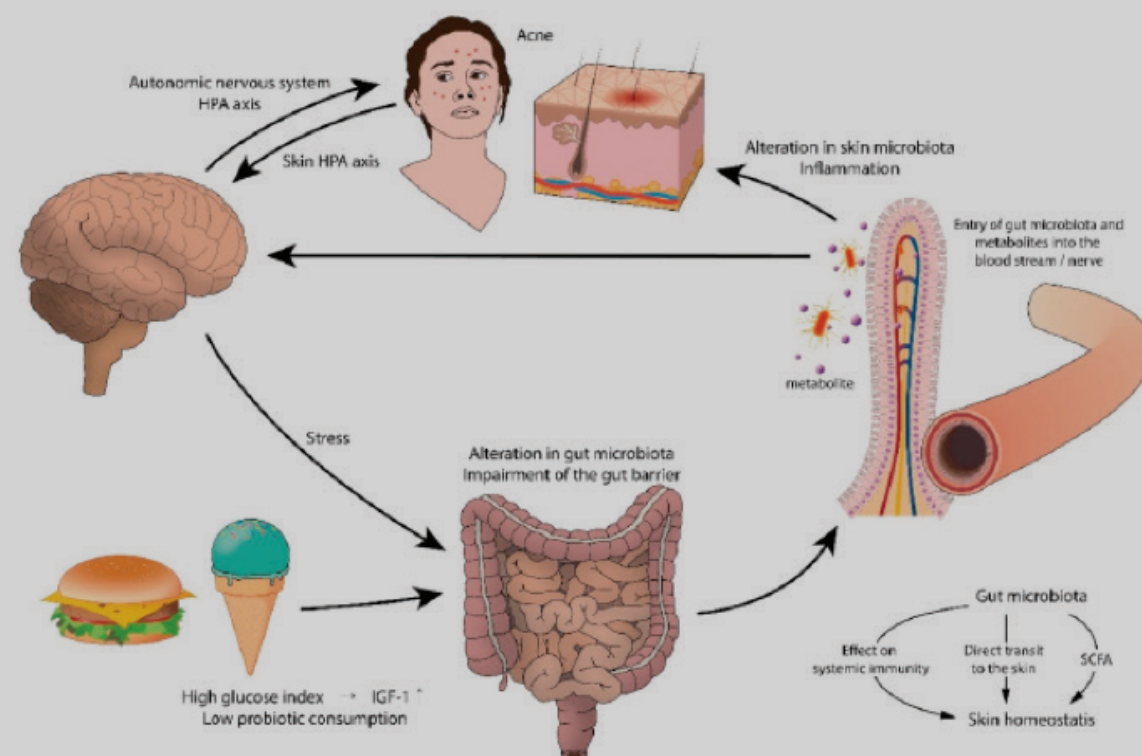


Figure 4: Sourced from <https://www.mdpi.com/2077-0383/8/7/987/htm>

Conclusion

There is a complex relationship between the sebaceous gland, the integrity of the skin barrier and the microbiome in the development of acne. Often, conventional acne treatments cause damage to the skin barrier and microbiome with prolonged use, which further affects barrier function and the skin's ability to defend itself.

One of the main treatment approaches to acne should be the rebalancing of the natural equilibrium of the microbiome, which will allow the repair and restoration of the natural skin barrier function.

The development of acne is a complex relationship between hyper keratinisation, sebaceous gland activity, microbial involvement, hormonal and lifestyle influences and inflammation. Exactly which step in this development cascade first initiates the development of this disease remains unclear. What we can do is to manage the things we have control over and supplement the skin where need be with the probiotics, oil and care it deserves.

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