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Human sebocytes: the new leptin connection?

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Recent discoveries of the past two decades have shown that the sebaceous glands are not 'atavistic fossils of human development', which have no other roles besides causing trouble for patients with acne, but should be considered as living, highly active organs of the skin.[1,2] However, the exact cellular mechanisms that regulate the neuroimmunoendocrine functions of the sebaceous glands and the sebocytes are still poorly understood, leaving a gap in our knowledge of how sebaceous glands could be integrated into both the physiology and pathophysiology of the skin. Therefore, identification of novel sebaceous modulators and their related signal transduction pathways[3,4] is unquestionably one of the most interesting fields in dermatological research, giving renaissance to this forgotten organ.

Of further importance, their complex and intense lipid metabolism and the production of inflammatory mediators[4,5] put sebocytes in a unique position that suggests also approaching them from a seemingly different field such as adipocyte biology, where lipid metabolism and inflammation are linked at a cellular level.

In an elegant paper published in the current issue of *BJD*, Törőcsik *et al.*[6] challenged sebocytes from this interesting aspect by using a wide repertoire of methods on the human immortalized SZ95 sebocyte model.[7] They have provided evidence that administration of leptin, the key adipokine in adipocyte biology, most probably via activation of the identified leptin receptor and key 'leptin signalling pathways' (signal transducer and activator of transcription-3 and nuclear factor-κB), led to an enlargement of intracellular lipid bodies, induced proinflammatory changes in the sebaceous lipid profile, and increased the expression of certain inflammatory enzymes and cytokines.

Addressing the pathophysiological and clinical relevance of these intriguing results is undeniably an interesting and challenging issue. By showing that leptin is a novel sebaceous player in inducing inflammation and altering the lipid profile in sebocytes, the authors also implicate another piece of the puzzle in the 'everlasting' issue of the role of nutritional factors in the development of acne vulgaris.[8,9] Namely, keeping the limits of their *in vitro* data in mind, one may speculate how these findings could be translated into the pathogenesis of acne: is leptin a possible link between food intake and the development of the disease? Is it more the quantity than the quality that plays a key role in inducing inflammation of the sebaceous glands via increased levels of serum leptin? Is it indeed systemic (serum) leptin that should be addressed, or could other cells of the skin (e.g. keratinocytes) also serve as a possible local source? And finally, going even beyond acne, could leptin be a 'universal' stimulus for the sebaceous glands, with a possible pathognomonic role in other skin diseases where previously sebocytes were never thought to have a role? These are only a few of the many ideas that this elegant work delivers, warranting further basic as well as clinical research.

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