Should we use Body Mass Index to Predict Disease Onset and Severity in Psoriasis?

In this issue of *Acta Dermato-Venereology* (p. 492–497), Katarina Wolk and co-workers, present data from a population-based case-control study showing a statistically significant positive association between body mass index (BMI) and onset of plaque psoriasis. In the study, a higher BMI was also associated with higher psoriasis activity, as judged by considering the Psoriasis Area Severity Index (*PASI*) at onset. Finally, in the study, smoking but not alcohol consumption, was significantly associated with increased risk of developing plaque psoriasis without a clear dose-response relation. These data largely confirm observations from case-control and cohort studies conducted in several other countries, and add indications, for the first time, that increased BMI may affect the activity of psoriasis at onset. The results have several important implications for research, public health, and clinical practice.

BMI, or Quetelet index, from the Belgian mathematician Adolphe Quetelet, is calculated as the individual's body weight divided by the square of his or her height. In spite of some limitations, such as lack of account for muscularity, BMI has been used as the standard for recording obesity statistics since the early 1980s. Being closely correlated with body fat in the abdominal cavity (central visceral fat) and with increased cardiovascular risk, the measurement of waist circumference may represent a useful adjunct to BMI. Interestingly, in addition to BMI, increased waist circumference has also been linked with increased risk of developing psoriasis (1).

The relation between overweight, obesity and psoriasis is intriguing. As discussed by the authors, adipose tissue has been shown to produce pro-inflammatory cytokines, e.g. tumour necrosis factor-alpha (TNF-α). Adipokines, such as leptin and resistin, may affect cytokine and chemokines expression in psoriasis, and serum resistin correlates with disease severity (2). Both genetic and environmental factors influence body weight, with heritability estimates ranging from 55% to 85%. Exploration of genetic influences on obesity may also throw some light on psoriasis.

There is a mismatch between the modern obesogenic environment and biological mechanisms of weight regulation. These mechanisms evolved and became efficient in preventing weight loss, but are relatively ineffective in preventing excessive weight gain. If similar genetic environmental interactions influence psoriasis then secular trends of increased incidence and severity of psoriasis can be expected. Although a variety of factors may be involved, a study has documented that the annual incidence of psoriasis almost doubled between the 1970s and 2000 in Rochester, Minnesota, USA (3).

Patients with severe psoriasis experience increased mortality rates compared with the general population (4). Population-based studies have documented that excess body weight during midlife, including overweight, is associated with an increased risk of death in all racial or ethnic groups, and at all ages. Overweight in adolescents also predicts future adult cardiovascular disease (5). Interestingly, recent data suggest that the risk of psoriasis increases with increased BMI not only in adults but also in children (6). The role dermatologists should play in working to reduce the epidemic is obvious. Not only higher BMI may be associated with higher psoriasis activity, but it may also influence clinical response to systemic treatment (7). All in all, the documented correlation between BMI and psoriasis highlights the need to adopt a more integrated research approach in disease causation, and a more holistic strategy in psoriasis management.

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Luigi Naldi, MD
Director, Centro Studi GISED,
Fondazione per la Ricerca Ospedale Maggiore,
Unità Complessa di Dermatologia,
Ospedali Riuniti, Bergamo (Italy)

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