cohort who survive to an older age and the presence of comorbidities in the older general population.

The study also found an increasing prevalence of psoriasis as the level of social deprivation became worse. Some deprived areas of the U.K. were likely to have not been included in the study.

A limitation of the study is that, because prevalence and incidence rates were determined by general practice electronic health records using the U.K. Clinical Practice Research Datalink, some cases of psoriasis may have been missed if patients did not see a primary care physician about their symptoms.[1] In addition, the database includes only a fraction of practices in the U.K., and so may not be geographically and demographically representative of the whole population of patients with psoriasis.

The study's finding that the incidence of psoriasis in the U.K. is not increasing is somewhat surprising, given the increasing incidence of insulin resistance in the population, a factor associated with psoriasis. However, according to the U.K. Census bureau,[9] the proportion of 'skin of colour' residents in the U.K. has risen over the past 20 years, and this may have confounded the observed incidence trends because psoriasis is uncommon in people of non-European ethnicity. It would be interesting to see whether the incidence trends change after accounting for the underlying proportion of whites in the population.

Like other chronic diseases, the fact that people are living longer with psoriasis has important implications for healthcare service delivery and resource allocation. The significant morbidity and higher mortality rates at younger ages associated with this disease highlights the sustained need for investigating early for comorbidities known to be associated with psoriasis, such as insulin resistance and hypercholesterolaemia, and managing these early to prevent secondary complications such as obesity, hypertension and cardiac complications. Furthermore, treatments that have less burden on fatty liver disease and renal impairment, often a consequence of severe psoriasis, may be reflected in the future by reduced mortality rates in these patients.

Conflicts of interest

None to declare.

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Could maternal stress increase the risk of developing psoriasis in the offspring?

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Linked Article: Liu *et al. Br J Dermatol* 2017; **176**:659–666.

Very few data exist on the role of prenatal stress as a risk factor for psoriasis. As reported in this issue of the *BJD*, the impressive findings of the study by Xiaoqin Liu *et al.* from Aarhus University in Denmark show that prenatal exposure to severe maternal stress increases the risk of developing psoriasis in the offspring.[1]

The authors performed a registry-based cohort study including 1 811 917 live singletons born during 1978/2008 in Denmark. If mothers lost a child, partner/spouse, parent or sibling during pregnancy or up to 12 months before pregnancy, the children were assigned to the bereaved group. The authors found that 7956 children were hospitalized or prescribed medications for psoriasis during 28 million person-years of follow-up. By age 30 years, 1·54% and 1·34% of children from the bereaved and nonbereaved groups, respectively, were diagnosed with psoriasis. In general, prenatal exposure to maternal bereavement was not associated with the risk of psoriasis. However, prenatal stress following maternal loss of an elder child or partner/spouse during pregnancy, which are the most stressful life events, significantly increased the risk of psoriasis development in the offspring (hazard ratio 1·33, 95% confidence interval 1·02–1·73).

The death of a child or partner/spouse are recognized as relevant sources of severe stress.[2] Stress is a well-known risk factor for psoriasis.[3,4] Prospective studies have shown that periods of high stress moderate the course of psoriasis, with more daily stressors predicting increased disease severity.[5] One potential mediator between stress and psoriasis is the hypothalamic–pituitary–adrenal axis, which was found to be dysregulated in patients with psoriasis. Experimentally induced stress tests have shown that cortisol levels in patients with psoriasis exposed to a public speaking task are significantly more acutely elevated than in healthy controls.[6] It could be speculated that children exposed to severe maternal stress *in utero* may be impaired in hypothalamic–pituitary–adrenal axis function. The altered adrenal activity has also been observed in other chronic inflammatory diseases such as atopic dermatitis and rheumatoid arthritis, and could potentially contribute to the inflammatory state.[7,8]

Stress relief interventions can lead to changes in the cortisol responses of highly distressed individuals. In particular, patients with rheumatoid arthritis who participated in stress management training had a significant reduction of interleukin-8 serum cytokine levels at the 2-month follow-up compared with controls.[9] Whether this may be applicable also in psoriasis is not yet known, although several stress reduction interventions have been proposed.[10] Moreover, psoriasis itself can generate psychological distress, because it can be a significantly disabling disease, interfering with activities of daily life and increasing levels of social stigmatization.

In conclusion, the implications of this work for clinical practice are thus to acknowledge that intense stress, including prenatally, may be a risk factor for development and/or worsening of psoriasis, and interventions for stress management may have an adjuvant role in the complex management of patients with psoriasis.

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Conflicts of interest

P.G. has been a consultant and/or speaker for Abbott, Celgene, Janssen, LEO Pharma, Lilly, Merck Sharp & Dohme, Novartis and Pfizer.

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Indoleamine 2,3-dioxygenase in psoriasis: a defective mechanism

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Linked Article: Llamas-Velasco *et al. Br J Dermatol* 2017; **176**:695–704.

Indoleamine 2,3-dioxygenase (IDO or IDO1) is an immunosuppressive enzyme that catalyses the metabolism of tryptophan along the kynurenine pathway.[1] Tryptophan degradation and kynurenine accumulation result in the inhibition of T-cell proliferation, activation and survival, and in the expansion of regulatory T cells.[2] IDO1 has a tolerogenic/immunosuppressive role in different biological settings, such as maternal tolerance towards the allogeneic fetus, suppression of allograft rejection, tumour immune escape and autoimmune diseases.[3] In the context of the role of IDO1 in autoimmune diseases, the paper in this issue of the *BJD* by Llamas-Velasco *et al.* shows that even if IDO1 is upregulated in patients with