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Whither scabies?

Scabies occurs in epidemics, and, though it is still seen fairly often in Britain, there is reason to hope that the condition may soon become much less common. At the start of the second world war the mode of transmission of scabies was not fully understood, and in 1941 there were, according to Mellanby,[1] over one million people in Britain with scabies. Mellanby's war-time experimental work on scabies transmission in volunteer conscientious objectors showed that scabies is not strictly a venereal disease and that a brief sexual encounter is less likely to transmit the infection than a more prolonged platonic sharing of a bed. Furthermore, his work showed that not only did regular bathing have little protective value but also that infrequent laundering of clothing and bedding did not increase the rate of transmission.

These facts suggested that the epidemic of scabies had less to do with wartime living conditions than had previously been believed. The disease had admittedly also been a scourge during the first world war, but a study of the children treated each year at a London cleansing centre had shown that the incidence of scabies had remained unchanged until 1920, when it had declined rapidly.[2] Scabies was uncommon during the next 10 years, but it began to be seen more frequently in 1930, and the incidence rose precipitously again in 1937, well before the second world war started. The epidemic continued for the next 12 years, but the disease declined sharply in 1949[3] and maintained a relatively low profile until 1964, when its incidence began to rise once more.[4]

Shrank and Alexander[4] argued in 1967 that increased sexual promiscuity was unlikely to explain that latest increase, since there had been no concomitant increase in gonorrhoea or syphilis during the period of their study. Increased resistance of the mite to treatment was also excluded as a possibility on the grounds that no cases failed to respond to correct treatment. They also pointed out that each of the recent epidemics has lasted 10-15 years, with a similar quiescent period between epidemics. The highest incidence of scabies occurs in the age range from 15 to 30 years, so the facts are consistent with the theory that the "herd immunity" of the population gradually increases during an epidemic, and that afterwards about 15 years have to elapse for the partially immune population to be replaced by non-immune individuals.

Indeed, the concept of partial immunity to scabies was introduced by Mellanby,[5] who showed that it is more difficult to induce a second attack of scabies and that during an established second attack fewer mites are found. The importance of immune responses in scabies is also suggested by the observation that the disease can be reactivated by methotrexate.[6] A particularly severe variant called Norwegian scabies has been reported in patients with a specific anergy[7] and in patients receiving immunosuppressive drugs.[8] Some supportive laboratory evidence has come from a recent study showing that patients with scabies tend to have a low serum IgA concentration; if the serum values correlated with low IgA concentrations in the skin secretions, this might predispose to scabies infection.[9] The possibility that the low IgA concentration was the result rather than the cause of the infection was not excluded, but the decrease was noted in early as well as late cases, and skin diseases are more usually associated with an increase in serum IgA.[10] The relation between the serum IgA levels and the herd immunity to scabies remains to be elucidated.

If scabies runs true to form the present mild epidemic, which started in 1964, should be over by 1979. Unfortunately, data from other countries show that the 30-year cycle is by no means constant,[11] and it may well be that scabies has not been common enough in Britain in recent years for herd immunity to have developed.

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Vaginal candidosis

Symptoms resulting from infections of the vagina with yeasts are a growing problem among patients attending general practitioners, gynaecologists, genitourinary physicians, and venereologists. The usual cause is the fungus *Candida albicans*, but in a minority of cases symptoms may be due to *Torulopsis glabrata*.[1 2] Candida is now the most common infectious agent found in women attending clinics for sexually transmitted diseases.[1 3] For example,[4] in 1973 in England and Wales 27 545 cases were diagnosed in women at the clinics, an increase of 25% over 1971; the corresponding figure for men was 5058 cases, an increase of 68%.

Whether or not clinical disease with symptoms results from infection depends on host factors, but little is known about the virulence of different strains of fungi. Factors such as glycosuria, diabetes, obesity, pregnancy, and the recent ingestion of antibiotics or steroids and other immunosuppressant agents have been known for some time to favour the growth of fungi in the vagina. Even so, the role of oral contraceptive pills is more controversial, particularly since the introduction of those with a lower oestrogen content.

The diagnosis can be established easily by identification of the mycelium and yeast spores on Gram-stained slides of specimens from the vaginal wall and cultures using a maltose-peptone agar such as Sabouraud's medium. One of the more difficult problems is to assess the meaning of asymptomatic candidosis in women, for the condition is quite frequently