Letters to the Editor

Troop-related multiple sclerosis outbreak in the Orkneys?

SIR—The multiple sclerosis (MS) epidemic in the Faroe Islands associated with a British troop presence during the second world war[1] has led to a search for similar MS outbreaks, as has since been reported for Iceland.[2] Did a similar epidemic occur in the Orkney Islands? As identified in four surveys, MS cases apparently rose sharply in the Orkneys and Shetlands in 1940–44 due largely to increases in the Orkneys (fig 1). The apparent increase in Orkney MS incidence dates from 1940. Only three cases were found in each of the two five-year periods before 1940, yet of the 13 new Orkney cases in the 1940–44 period, four had onsets in 1940 alone.[3] Further, the apparently high incidence of MS in the Orkneys during and after the war, and subsequent gradual decline, has since been confirmed.[4] This suggests a log-normal Orkney MS case distribution like that seen in the Faroes epidemic.[1] The Orkney-Shetland military build-up in 1939–40 near the Home Fleet base at Scapa Flow (fig 2)[5] is a point of further analogy. By early 1941, troop levels (not including naval and air forces) in the Orkneys were three times those in the Shetlands, in keeping with a greater apparent rise in MS incidence in the Orkneys than in the Shetlands and suggesting a possible dose-response relation between troop exposure and disease.

If this view of Orkney MS is correct, the Faroe Islands common source outbreak is the paradigm of a more general phenomenon in MS epidemiology. Further, postulation of a prolonged incubation interval between exposure to an exogenous agent or important risk factor and the appearance of clinical MS, required by some hypotheses [reviewed in ref 6], is not required in every case, as increased numbers of cases began to appear in the Orklands within 18 months of the arrival of troops.

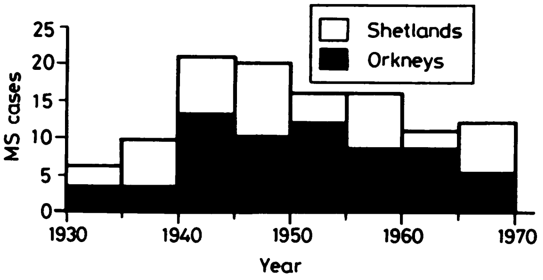

Fig 1 *MS cases in the Orkney Islands, 1930–70 (after Poskanzer et al,* ref 3).

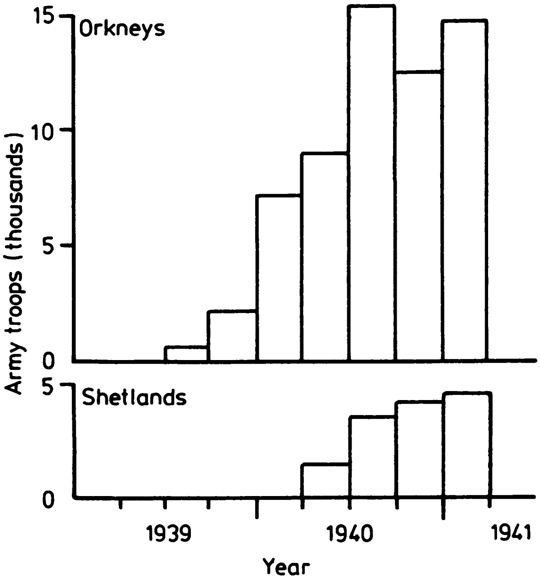

Fig 2 *Army troops in the Orkney and Shetland Islands, 1939–41. No recorded troops on the islands before 1939 (3rd quarter). For 1940 (4th quarter) and 1941 (1st quarter), not included are 1122 and 119 men, respectively, stationed at sites not found in either island group on prewar ordnance survey maps. From 1941 (2nd quarter) onward, regimental strength is not organised chiefly by station site, and later is very limited.*

The alternative proposal, that case ascertainment in prewar years was incomplete,[3] could explain the apparent rise in Orkney-Shetland MS incidence (15 cases, 1930–39; 42 cases, 1940–49). But the assumption that no significant ($p<0{\cdot}05$) increase occurred requires that at least half (15 or more) of the Orkney-Shetland MS cases with onsets in 1930–39 escaped detection. Further study, including comparison of the quality of diagnosis and record keeping before and after the war, will be needed to determine which hypothesis is correct.

References

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JOHN R MARTIN
Laboratory of Experimental Neuropathology
National Institute of Neurological
and Communicative Disorders and Stroke,
National Institutes of Health,
Bethesda, Maryland, 20892, USA

Maternal blood lead

In their recent paper, McMichael *et al* describe the results of a comprehensive study of reproductive outcomes among women environmentally exposed to lead (JECH 1986 **40:** 18). The authors report a positive association between maternal blood lead concentration and preterm deliveries but no association between blood lead and spontaneous abortion. We would like to suggest that the authors' data are inadequate for making such a conclusion about spontaneous abortion.

The abortions included in their study are only a small subset of the abortions experienced by the study population. In addition, they are heavily weighted toward second trimester abortions. Sixty-two percent of exposed women in the study were at least 14 weeks' pregnant at enrollment. Yet prospective and life-table studies have shown that nearly 90% of all spontaneous abortions occur before this gestational age (ie, during the first trimester).[1 2] This suggests that the large majority of abortions experienced by McMichael's population occurred before they could be enrolled. This is supported by the fact that only 3·4% of exposed pregnancies ended in abortion—far less than the 12–15% usually reported in unexposed populations.[3] Therefore, there is reasonable doubt that the small group of late abortions observed in this study is representative of spontaneous abortions as a whole.

The authors conclude that "the lack of any clear positive association of spontaneous abortion with maternal PbB within this study population suggests that small, non-occupational increases in lead exposure are insufficient to disrupt the early stages of pregnancy". We suggest that there are not enough data from the early stages of pregnancy to offer reassurance on this point.

ANDREW ROWLAND
ALLEN WILCOX
National Institutes of Health
National Institute of
Environmental Health Sciences
P.O. Box 12233
Research Triangle Park,
N.C. 27709, USA

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[3] Leridon H. *Human fertility: The basic compenents.* Chicago: University of Chicago Press 1977; 44–81.

The author replies as follows:

Your correspondents correctly point out that the spontaneous abortions observed within our cohort of lead-exposed pregnant women in Port Pirie were almost certainly a minority of all spontaneous abortions. Our conclusion, which they quote in their third paragraph, should rather have ended: ". . . non-occupational increases in lead exposure are insufficient to affect the risk of second trimester spontaneous abortion".

While the sample of spontaneous abortions observed was censored with respect to gestational age—and may therefore have been biased with respect to any overall association of spontaneous abortion with lead exposure—we do not know of any reason why the sample might have been biased with respect to any association of second trimester events with lead exposure.

A J MCMICHAEL
Department of Community Medicine
University of Adelaide
Royal Adelaide Hospital
Adelaide
South Australia 5001