matters arising

T-mycoplasmas and infertility

SIR,—IN the first report about the association of T-mycoplasmas with human infertility the authors considered that the high incidence of isolation (89%) from the cervix of infertile women compared with the low incidence of isolation (23%) from pregnant women suggested that the T-mycoplasmas were playing some rôle in the infertility. This was based on the isolation discrepancy and was principally a result of the low incidence in pregnant women. Others have, however, been more successful in isolating T-mycoplasmas from pregnant women. Mårdh and Weström² isolated T-mycoplasmas more frequently from pregnant (68%), than from non-pregnant women (46%), and thought that the former rate might have been due to changes in the genital tract at pregnancy conducive to the growth of T-mycoplasmas. Gnarpe and Friberg tried to substantiate the possible association of T-mycoplasmas with infertility by treating infertile couples with tetracycline and 29% of the women they treated subsequently became pregnant. In the absence of untreated controls, however, this finding is meaningless. Mr G. T. Smedley and I, in an unpublished study, found that of 32 infertile women, 22 (69%) carried T-mycoplasmas. Within a year, nine of these women became pregnant without treatment and 5 (56%) of them carried T-mycoplasmas. Gnarpe and Friberg's second report³ concerns the attachment of T-mycoplasmas to human spermatozoa. I find it difficult to regard the 'blobs' on the spermatozoa that they saw by electron microscopy as evidence for attached T-mycoplasmas, as these structures could also be *Mycoplasma hominis*, chlamydiae or some other agent. I do agree, however, that adherence of human T-mycoplasmas occurs in the same way as those of bovine origin adhere to bovine spermatozoa⁴. It is interesting that practically all bovine semen samples that we examined contained T-mycoplasmas, yet they all had a high fertilising capacity⁵. It seems likely that abnormal appearance, low motility and viability of spermatozoa in man are not due to the presence of T-mycoplasmas in semen, because the organisms are isolated from such samples no more frequently than from samples which contain apparently normal spermatozoa. Spermatozoa of low fertilising potential might also be

adversely affected by mycoplasmas present in the vagina and cervix. This comes back to the original point about the incidence of T-mycoplasma isolation from these sites. Whether the incidence of isolation is greater in infertile women is a moot point, but it would also be interesting to know whether they harbour more organisms in the genital tract than do fertile women. I feel that the evidence put forward to incriminate T-mycoplasmas as a cause of infertility is far from convincing and that they are being placed on a pedestal that they do not, so far, deserve.

Yours faithfully,

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¹ Gnarpe, H., and Friberg, J., *Nature*, **242**, 120 (1973).

² Acta Path. Microbiol. Scand., **78**, 367 (1970).

³ Gnarpe, H., and Friberg, J., *Nature*, **245**, 97 (1973).

⁴ Taylor-Robinson, D., and Manchee, R. J., *Mycoplasma Diseases of Man*, Veb Gustav Fischer Verlag, Jena, p. 113 (1969).

⁵ Taylor-Robinson, D., Thomas, M., and Dawson, P. L. J. *Med. Microbiol.*, **2**, 527 (1969).

DRS GNARPE AND FRIBERG REPLY: we have read with interest the above letter and although Dr Taylor-Robinson's comments are valuable we have by no means expressed the opinion that T-mycoplasmas might be one of the major causes of infertility.

There are, however, a few points we wish to make clear. In our first investigation¹ we found a high incidence of T-mycoplasmas in infertile men and women compared with a lower incidence in men and women with proven fertility. All infertile patients had an unexplained infertility of 5 yr or more. None of the spermatozoa specimens investigated had abnormal appearance under light microscopy, low motility or reduced viability. We still consider that the incidence of pregnancy after tetracycline treatment is higher than can be expected to appear by chance among patients with this long-standing infertility².

We agree with Dr Taylor-Robinson that the 'blobs' on human spermatozoa discovered in scanning electron microscopy might be other agents such as *M. hominis* or Chlamydiae. In all specimens where cultures were made of

thoroughly washed spermatozoa³, however, we found T-mycoplasma colonies and not other agents growing. Whether Chlamydiae were present or not was, of course, impossible to establish with the technique used. We also found that some T-mycoplasma isolates produced substances cross-reacting with neuraminidase antibodies and suggested that these substances might render conception more difficult³.

These findings make it possible that some T-mycoplasmas might be of importance for some cases of unexplained infertility.

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¹ Gnarpe, H., and Friberg, J., *Nature*, **242**, 120 (1973).

² Friberg, J., and Gnarpe, H., *Am. J. Obstet Gynec.*, **116**, 23 (1973).

³ Gnarpe, H., and Friberg, J., *Nature*, **245**, 97 (1973).

African swells, magmatism and plate tectonics

SIR,—Some of the 'flaws'¹ in my lateral compression model² seem to be matters of faith, for example, that the model is "at odds with modern global tectonics", whilst many (including the "petrological observations") are arguable matters of interpretation. One "flaw", apparently supported by data, concerns the faster rate of uplift of post-glacial regions compared with the doming of East Africa. It is argued that since there is no evidence of deep partial melting in the post-glacial regions, the probability of such melting by warping of the African craton is insignificant. But the two mechanisms are quite different. The post-glacial regions are rising in response to a uniform isostatic pressure at the base of the plate. Below the post-glacial uplift there is no localised decompression, as might be the case below an 'upwarp'. As was pointed out in 1964 (ref. 2), in the case of the African swells "lighter material would have to be added at depth to compensate each succeeding uplift". So far as I am aware, this deduction has not been challenged, nor an alternative offered.

In any case, since 1964 my original hypothesis² has been considerably modified and refined³, and Gill's critique is