Flatfeet are known to occur in conditions with hyperlaxity, such as the rare Ehlers-Danlos syndrome [1]. In such conditions, flatfeet are known to deteriorate during pregnancy [2]. There are several reasons why flatfeet may occur in pregnancy: significant weight gain and increased ligamentous laxity due to the hormonal changes will both play a part. Relaxin is a peptide hormone with a structure not dissimilar to insulin. There is evidence that it is produced by the corpus luteum in pregnant women and has important hormonal functions, including a direct effect on collagen [3, 4]. It could be postulated, in view of the history of recurrent miscarriages in our cases, that this hormonal effect is cumulative. A positive family history in the first case is interesting, but there was no evidence of generalized joint laxity. Perhaps pregnant women should be given advice regarding supportive footwear and avoid going barefoot in the hope that this could prevent the development of flatfeet. In the future, it may be appropriate to carry out a prospective study to gain further information on the incidence and severity of foot problems during pregnancy.

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CD69 on Synovial T Cells in Rheumatoid Arthritis Correlates with Disease Activity

Sir—A number of activation markers have been described on the surface of synovial fluid T cells which have different functions. CD69 is a very early T-cell activation marker and up to 70% of synovial T cells express it [1]. We have recently presented evidence to show that its upregulation occurs at the time that T cells enter the inflammatory focus [1]. The particular importance of CD69 is that it is involved in the direct stimulation of macrophages by activated T cells to secrete inflammatory mediators such as interleukin 1 [2]. We reasoned that the number of CD69 T cells in the synovial fluids of patients with rheumatoid arthritis (RA) should have some relationship to the degree of joint inflammation.

The peripheral blood and paired synovial fluid T cells from 10 patients (five males, five females; age 54.5 ± 12.7 (s.d.) yr), fulfilling the ARA criteria for RA, were examined by cytofluorographic analysis using the FACScan (Becton-Dickinson). As expected, the percentage of CD69-positive T cells was low in the peripheral blood (3.2 ± 2.0 s.d.), while there was a significant increase in the synovial fluid (65.4 ± 19.0 s.d.; P < 0.001). Similar results were obtained for the surface expression of CD69 (mean fluorescence intensity: for blood, 1.7 ± 1.0 s.d.; for synovial fluid, 32.1 ± 13.0 s.d.; P < 0.001). Of interest was the finding that both the percentage of CD69 and the mean fluorescence intensity correlated with the number of inflamed joints (Fig. 1; r = 0.76, P = 0.04 and r = 0.81, P = 0.03, respectively).

From these results and the in vitro data of Dayer and colleagues [2], it may be concluded that inhibition of the actions of CD69 could suppress disease activity in RA.

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