Origin of the diphtheroid bacteria, mycoplasmas, etc., reported in association with autoimmune conditions

SIR, Those of your readers who saw my previous note (Bisset, 1977) on the isolation of a bacterium from the erythrocytes of normal and arthritic subjects may be interested to know how these very slow and lengthy cultural procedures progressed. The final figures gave 30% positive isolations from 200 persons, who were either healthy or suffering from conditions other than arthritis (Bisset and Bartlett, 1978). Early cultures (which in this case means those only a few weeks old) usually consisted of diphtheroid bacilli or the spheroplasts that are frequently described as mycoplasmas; the latter were often acid-fast or grew as small, acid-fast rods on subculture. All these forms have proved to be phases in the L-cycle of the sporing bacillus now referred to as BLE (*Bacillus licheniformis* var. *endoparasiticus* Benedek). Although the rate of reversion to the fully sporogenous, parent form is very low indeed, in primary cultures or subcultures on ordinary medium, further work in this laboratory shows that it can be greatly increased by the use of known L-form reversion stimulants, which serves to substantiate the relationship of the various phases one to another.

These results seem clearly to indicate that most if not all the bacterial forms claimed to be associated with arthritis are forms of a hitherto almost unsuspected cryptoparasite or symbiont of the blood. This includes so-called mycoplasmas, and supporting evidence has recently been provided by Walker and RajBhandary (1978), who have shown by analysis of tRNA sequences, that classical mycoplasmas are related to bacteria of precisely this type. Mutually corroborative evidence from 2 such different sources is much more impressive than either one by itself.

Such a conclusion does not by any means imply that these bacteria are disqualified from playing a role in arthritis or other, mainly autoimmune, conditions. They may act as an immunological irritant, and initiate the sort of interaction, proposed with great clarity of detail by Pease (1965), which seems to be in excellent accordance with the known facts. It is worth keeping a record that Dr Pease was also the first to suggest the true identity of the well-known diptheroid bacilli as they occur in arthritis (Pease, 1974).

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T cells in ankylosing spondylitis

Sir,

In a recent article in the *Annals* P. T. Fan *et al.* (1977) found that the percentage of T cells in peripheral blood of patients with ankylosing spondylitis (AS) was decreased. An increase in null cells was proposed by the authors as an explanation for the lymphocyte abnormality. It is known that a common feature in ankylosing spondylitis is elevated C-reactive protein (CRP).

Morstensten *et al.* (1975) found that binding of CRP can result in the modulation of certain of the T cell functional characteristics in vitro. According to these authors CRP binds selectively to T lymphocytes and inhibits their ability to form spontaneous rosettes with sheep erythrocytes. By contrast CRP does not bind to B lymphocytes and does not alter such B cell functions as binding to activated complement component or to the Fc portion of immunoglobulin. In view of this the 'decrease' of T cells in peripheral blood of patients with AS may be related to CRP.

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Sir,

Dr Sotnik's suggestion that the presence of C-reactive protein (CRP) in the peripheral blood of patients with AS may be responisible for our finding of a decreased T cell percentage in these patients is indeed intriguing and plausible. CRP had been found in 8 of 9 patients with active ankylosing spondylitis ("rheumatoid spondylitis" (Shetlar, *et al.*, 1956) In patients with acute rheumatic fever elevation of CRP is correlated with an increase in the proportions and absolute numbers of CRP-binding lymphocytes (Williams *et al.*, 1978). The CRP binding persisted even after overnight incubation of the lymphocytes in medium at 37°C and was found to reside mainly in that population of T cells bearing receptors for Fc IgG (Williams *et al.*, 1978).

Mortensen *et al.* (1975) have found that CRP inhibited