Sir - Bach et al (March 9, p 644) [1] report the benefits of a coordinated rheumatic fever control programme. They included control of streptococcal skin disease, while reiterating the dogma that pyoderma does not lead to rheumatic fever. We have recently described the highest published rates of rheumatic fever in the world in the Aboriginal population of northern Australia. [2] Among this population, streptococcal pyoderma is endemic, with up to 70 percent of children in some communities being affected, much of it secondary to scabies infestation. In the same population, throat carriage rates of group A streptococci (GAS) are extremely low (usually 1 percent to 5 percent) and symptomatic GAS pharyngitis is uncommon.

Early claims that pyoderma could not precipitate rheumatic fever came largely from epidemiological studies conducted in military populations and in rheumatic fever hospitals. Few studies have looked at this issue in populations with endemic streptococcal skin infections, and those that have all provided support for the prominent role of pharyngitis in rheumatic fever pathogenesis, but did not exclude completely a role for pyoderma. Recent work from New Zealand [3] has identified many rheumatic fever-associated isolates of GAS as coming from serotypes traditionally associated with pyoderma. In one of the Trinidad studies referenced by Bach et al [4] half of all rheumatic fever episodes were not preceded by symptomatic pharyngitis, 38 percent of rheumatic fever patients had recent skin infection, and one of the two most common rheumatic fever-associated strains of GAS came from a "pyoderma" serotype. We have shown that individuals with multiple skin sores may be infected with multiple strains of GAS, and individual sores may also be co-infected with multiple strains. [5] This would provide the ideal milieu for horizontal transfer of genetic material between strains of GAS. In regions with endemic GAS infections, the reservoir of GAS strains present on skin may allow constant genetic reassortment, with development of new epitopes (to allow evasion of host defences) and sharing of pathogenic factors (including putative rheumatogenic factors).

The pathogenetic basis of rheumatic fever awaits elucidation, together with a better understanding of the molecular epidemiology of skin and throat streptococci. Even if rheumatic fever results only from ultimate infection of the throat, we believe that it is important in endemic areas to include major primary prevention efforts where the majority of GAS disease exists - the skin - with community-based scabies and pyoderma control programmes.

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REFERENCES


