## AMYLOIDOSIS—A POSSIBLE MINOR FACTOR CONTRIBUTING TO THE DISAPPEARANCE OF LEPROSY FROM NORTHERN EUROPE

Sir,

The disappearance of leprosy from Northern Europe even before the advent of antileprosy drugs is an epidemiological mystery. Peter Richards[1] has traced the rise and fall of the disease in Europe in his book and enumerated the various theories regarding this topic. The various factors thought to be responsible for the fall of the disease include the isolation of the patients; plague epidemics which contributed by killing the patients and by reducing the total population; increase in the prevalence of tuberculosis which conferred immunity against leprosy and, lastly, improved socioeconomic conditions which improved housing and nutrition. However, none of the theories can explain the historical facts. Richards concludes that many circumstances have contributed to differing extents at different times.[1]

One more factor that may have contributed is reactive systemic amyloidosis. It is known that Europeans are more likely to contract the lepromatous form of the disease than are Indians or Africans.[2] Also, recurrent erythema nodosum leprosum (ENL) reactions are associated with development of amyloidosis.[3] The prevalence of amyloidosis in leprosy patients is variable.[4] Renal amyloidosis is reported in 5% leprosy patients in tropical regions but a high prevalence of more than 30% is reported from the United States and Argentina.[4] Cochrane had noted that amyloidosis is rare, except in Caucasian races and to some extent in Mongolian people as a terminal complication of leprosy.[5] There are at least 3 different phenotypes of the serum amyloid A protein (SAA),[6] an acute phase protein whose levels increase during ENL reactions.[3] The amyloid fibrils are derived from the SAA. It is possible, though not yet proved, that the susceptibility to develop amyloidosis may depend on the SAA phenotype. It is likely that the leprosy patients in Europe had two

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peculiarities—high prevalence of the lepromatous form of the disease and increased susceptibility of these lepromatous patients to develop reactive systemic amyloidosis as a complication of ENL reactions which were untreated or poorly controlled. Death of lepromatous patients from amyloidosis may have reduced the infectious pool in the community thus reducing the chances of infection. Thus amyloidosis may have contributed to the disappearance of leprosy from Northern Europe along with the other factors that have been mentioned.

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## References

[1] Richards P. *The medieval leper*. Cambridge: D. S. Brewer, 1977. pp. 83–97.
[2] Spickett SG. Genetic mechanisms in leprosy. In: *Leprosy in theory and practice*, 2nd ed. Cockrane RG, Davey TF (eds), Bristol: John Wright and Sons, 1964, pp. 98–124.
[3] McAdam KPWJ, Anders RF, Smith SR, Russel DA, Price MA. Association of amyloidosis with erythema nodosum leprosum reactions and recurrent neutrophil leucocytosis in leprosy. *Lancet*, 1975; **ii:** 572–6.
[4] Date A. The immunological basis of glomerular disease in leprosy—a brief review. *Int J Lepr*, 1982; **50:** 351–4.
[5] Cochrane RG. Complicating conditions due to leprosy. In: *Leprosy in theory and practice*, 2nd ed. Cochrane RG, Davey TF (eds), Bristol: John Wright and Sons, 1964, pp. 331–43.
[6] Pepys MB. Amyloidosis. In: *Oxford textbook of medicine*, Vol. 1, 2nd ed. Weatherall DJ, Ledingham JGG, Warvell DA (eds), Oxford: Oxford University Press, 1987, pp. 9.145–9.157.