We thank Hanck and colleagues for their interest in our article.1 We agree with their main observations. For historical reasons, the term “Tropical Pancreatitis” came into usage because this type of chronic pancreatitis was first described from the tropics, almost all the first few reports of this disease emanated from tropical countries, and it had characteristic differences from alcoholic pancreatitis. Earlier workers did not feel the need for further laying down very rigid criteria to diagnose this disease.2 The label, “tropical pancreatitis”, based on geographic prevalence, came into vogue to describe the disease, and few workers looked beyond at the possible etiological factors that led to this disease. Cassava consumption was assumed to be a major etiological factor because tropical calcific pancreatitis (TCP) was usually observed among populations that consumed cassava as a staple diet.3 Theoretically the cyanogenic glycosides in many varieties of cassava could cause tissue injury, including the pancreas.4 Pancreatologists were possibly complacent, when they could identify one major risk factor to explain the genesis of pancreatitis - alcohol in the case of alcoholic pancreatitis (AP) and cassava in the case of TCP. In the case of alcohol, it is only recently that the importance of co-factors such as smoking, genetic mutations, viral infections or diet that contribute to the genesis of the disease or its progression, has been investigated.5 In TCP too, other factors, in view of changes in socio-economic conditions and life styles in developing countries, need to be considered.6 A change in the pattern of clinical picture has been reported in the past few years in southern India, particularly Kerala, as well as from areas which are outside the tropics, such as northern India, Bangladesh and China, underlining the need for strictly defining TCP.7,8

The other problem in categorizing chronic pancreatitis into different groups arises from the recent spurt in alcoholism and smoking observed in India and particularly in Kerala state during the past two decades. The per capita alcohol intake in Kerala is highest in the country and about three times the national average.9 There has been corresponding increase in ACP in the state, and nearly 33% of chronic pancreatitis are now associated with alcoholism; another 10% to 12% of our chronic pancreatitis patients are social drinkers.10 Small quantities of alcohol can contribute to pancreatic injury, and this is abetted by smoking,11 also, there is no lower threshold for ACP.12 Hence one can assume that even in the “non alcoholic” group of TCP patients, alcohol and smoking in smaller degrees may contribute to the development of pancreatitis. We agree with Hanck and colleagues that social drinkers and occasional smokers should be excluded in future scientific studies on TCP.

As yet we do not know the etiology or distinguishing features of tropical pancreatitis. The changing pattern of TCP and its possible relationship to socioeconomic and lifestyle changes are fascinating subjects for further research.

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