Letters to the Editor

VIGOROUS EXERCISE IN LEISURE-TIME AND CORONARY HEART-DISEASE

SIR,—In their fascinating report (Feb. 17, p. 333) Professor Morris and his colleagues discuss the possible causal pathways whereby vigorous leisure exercise might protect against clinical coronary heart-disease (C.H.D.) and indicate that it is not associated with a lower prevalence of hypercholesterolæmia. While we agree that the effects of exercise on plasma-cholesterol concentrations are unconvincing, it may be of interest to point out that lower fasting plasma-triglycerides have been fairly consistently observed in people taking regular exercise. This is relevant because in the Stockholm prospective study fasting plasma-triglycerides as well as cholesterol were found to be risk factors for C.H.D., independent of each other.

The absence of any association between vigorous exercise and cigarette-smoking was another interesting observation in Professor Morris's study. In a smaller number of subjects we were able to demonstrate a significant inverse correlation between degree of smoking and degree of physical activity. However, this may have been due to the fact that our subjects were younger (20–55 years) and some of them engaged in competitive sports.

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SIR,—The article by Professor Morris and his colleagues on the relationship between vigorous exercise and cardiovascular health reflects only the deplorable state of fitness in a sedentary, over-indulgent, carbohydrate-consuming society. What is not generally realised is that the human body demands only a modicum of athletic activity to maintain an adequate standard of fitness, both cardiovascular and musculoskeletal. Recent studies on 15 untrained men (aged 25-33) and all 5 kg. (± 1.5 kg.) above their standard weight have shown that as little as 17-20 minutes' vigorous exercise on three days per week could produce a significant increase in athletic performance (3-5 minutes faster over 4000 metres) (P < 0.001), with a reduction in body-weight of approximately 1-2 kg. per week to standard weight, increased muscle tone, and lower resting pulse-rate (average decrease 15 per min.). Such short, sharp bursts of activity (10 kcal. per min.) utilise muscle glycogen, free fatty acids, and stored triglycerides 7; while the basal metabolic-rate is stimulated for several days as muscle protein and glycogen are resynthesised.8 Thus, the benefits of exercise in producing weight-loss extend beyond the immediate energy expenditure, which may be quite small (200 kcal.). However, it is

an undisputed fact that even such modest athletic pursuits seem beyond the desire and capabilities of the majority of the middle-age population.

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STREPTOKINASE IN MYOCARDIAL INFARCTION

SIR,—The Australian multicentre trial of streptokinase in myocardial infarction (Jan. 13, p. 57) has, like previous trials, failed to reach a conclusion regarding its effectiveness. Could it be that systemic infusion of the lytic agent is less effective than a more local delivery might be? Kordenat and Kezdi 1 achieved lysis of an induced thrombus in dogs when streptokinase was infused directly into the coronary artery within 24 hours of occlusion. We appear to achieve partial lysis of an induced thrombus in pig coronary arteries by instilling streptokinase into the left ventricle, but we are worried by the high incidence of ventricular fibrillation that results. Sommers and Jennings ² also noticed this effect after sudden release of a mechanical obstruction in the coronary vessels, and we feel that partial lysis may lead to distal embolisation of the thrombus, leading to the arrhythmias observed.

Similarly, in a recent patient with an acute myocardial infarction from coronary-artery thrombosis, streptokinase instilled directly into the affected vessel led to an alarming myocardial hæmorrhage as demonstrated by coronary angiography. This effect has been observed in dogs.³

These facts, taken in conjunction with the problem of the antigenic potential of streptokinase when used for a recurring event (as is coronary-artery thrombosis), lead us to be cautious in the use of this agent in coronary disease. We feel that more experimental work is required to determine the best site of delivery and the optimum dosage before further clinical trials are undertaken.

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COFFEE DRINKING AND ACUTE MYOCARDIAL INFARCTION

SIR,—The Boston Collaborative Drug Surveillance Program detected an apparent relationship between heavy coffee consumption and myocardial infarction (Dec. 16, p. 1278). This relationship is further substantiated by comparing coffee consumption to death-rates from ischæmic heart-disease for different countries (see accompanying figure). A strong correlation (r = 0.6114, P < 0.01) is shown between annual per-caput coffee consumption in kilogrammes ⁴ and fatal ischæmic heart-disease ⁵ in 24 countries.

Association does not prove causality, however, and objections can be raised to the use of international comparisons for studying the pathogenesis of arteriosclerotic disease. Nevertheless, a large prospective clinical trial

Holloszy, J. O., Skinner, J. S., Toro, G., Curteton, T. K. Am. J. Cardiol. 1964, 14, 753.

² Hoffman, A. A., Nelson, W. R., Goss, F. A. ibid. 1967, 20, 516.

^{3.} Carlson, L. A., Linstedt, S. Acta med. scand. 1969, suppl. 493.

^{4.} Truswell, A. S., Mann, J. I. Atherosclerosis, 1972, 16, 15.

Hurter, R., Swale, J., Peyman, M. A., Barnett, C. W. H. Lancet, 1972, ii, 671.

^{6.} Carlson, L. A., Bottiger, L. E. ibid. 1972, i, 865.

^{7.} Johnson, R. H., Walton, J. L. ibid. 1971, i, 566.

^{8.} Bergstrom, J., Hultman, E. Scand. J. clin. Invest. 1967, 19, 218.

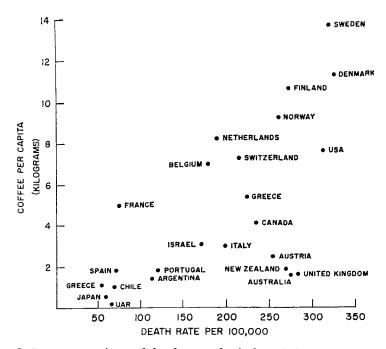
^{1.} Kordenat, R. K., Kezdi, P. Am. J. Cardiol. 1972, 30, 640.

^{2.} Sommers, A. N., Jennings, R. B. Fedn Proc. 1964, 23, 444.

^{3.} Moschos, C. B., et al. Cardiovasc. Res. 1970, 4, 228.

^{4.} United Nations Statistical Yearbook 1969; p. 484. New York, 1970.

^{5.} United Nations Demographic Yearbook 1969; p. 622 New York.



Coffee consumption and death-rates for ischæmic heart-disease.

designed to study coffee consumption as a risk factor in the pathogenesis of atherosclerosis is needed.

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THE WINDMILLS OF SHINGLES

SIR,—It is a little unkind of Dr Slack and Dr Taylor-Robinson (Feb. 17, p. 369) from their Mount Olympus to try so dogmatically to exorcise the clinico-epidemiological description. Must those beyond the portals of immunology think-tanks be ever barred from tilting? Was Pickles 1 then so wrong? Perhaps Dr Smith's idea (Feb. 3, p. 267) is at variance with "presently accepted theories", but if only theories, must these remain immutable? May not his observations carry some germ of truth? Few of us are practising nudists and if not hands directly why not clothes and some air turbulence surrounding even the act of handshaking. It may be "erroneous to presume that zoster could be caught". Does this mean that reinfection with varicella-zoster virus can never occur? Immune mechanisms may be more than half the battle, but the virus spark must come in somewhere. There are difficulties in the assay of varicella-zoster virus and calculation of its rate of destruction, so that it is bold indeed to deny its ability to survive on occasion.

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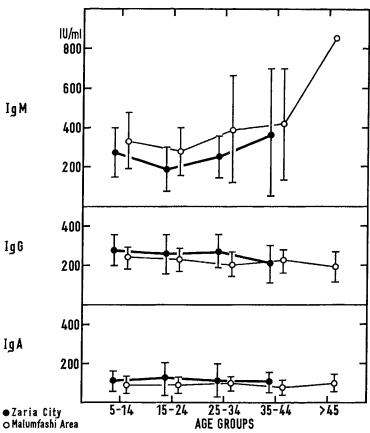
NORMAL IMMUNOGLOBULINS IN THE TROPICS

SIR,-Dr Rowe 2 has shown that the mean immunoglobulin levels of healthy subjects may differ widely between different countries. Wide regional variations in environment occur within many tropical countries and these may have an important influence on immunoglobulin levels. Patients come to Ahmadu Bello University Teaching Hospital, Zaria, Nigeria, from both the old walled city of Zaria and from the surrounding rural areas. We thought that it would be of interest to compare the immunoglobulin levels of these two communities.

Sera were obtained from 109 healthy subjects living in Zaria city and from 89 subjects living in villages around Malumfashi, 60 miles to the north of Zaria. 101 subjects were female and 97 were male. There were approximately equal numbers of subjects within each age decade. Immunoglobulin levels were measured by radial immunodiffusion and the mean results (+1 s.d.) in international units per ml. were (see also accompanying figure):

		Zaria City	Malumfashi	
		N. 109	N. 89	
IgA	 	 120 ± 70	94 ± 40	P < 0.001
IgG	 	 259 ± 92	225 ± 57	P < 0.01
IgM	 	 260 ± 166	346 ± 200	P < 0.001

Significant differences were found in the mean levels of each immunoglobulin between the two populations.



Mean immunoglobulin levels in Zaria City and Malumfashi by age.

The reason for these differences is not obvious. population of both communities is predominantly Hausa and the samples were all collected at the same time of the year. However, there are differences in the pattern of parasitic infection seen in the two communities, and this may be important.

Immunoglobulin levels are widely used in hospital studies of patients with a variety of diseases. Our findings indicate that in the tropics very careful selection of a control population may be required.

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RESIDUAL T CELLS IN CHRONIC LYMPHOCYTIC LEUKÆMIA

SIR,—We were very interested in the paper by Dr Wybran and his colleagues (Jan. 20, p. 126) confirming that T cells remain in chronic lymphocytic leukæmia

^{1.} Pickles, W. Epidemiology in Country Practice. Bristol, 1939. 2. Rowe, D. S. Lancet, 1972, ii, 1232.