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Exercise, health, and medicine

As ever more runners and cyclists appear on city streets the publication of two reviews of the effects of exercise on health is timely. The Norwegian Confederation of Sport and its medical council prepared its assessment in response to a parliamentary and governmental initiative[1]; the other review is based on an international symposium held in Finland.[2] Between them they provide a comprehensive account of present knowledge and growth areas, with many pointers to the research that is needed. They also outline the actions already indicated in both clinical medicine and public health.

Most of the physiological effects of physical activity and exercise described in these reviews are familiar—but particularly welcome are the discussions of the effects of exercise on psychological states and mental health.[1] The need for more research is plain—as it is in assessing the specific and non-specific contributions of exercise in the treatment and rehabilitation of patients with obesity and diabetes, with asthma and chronic obstructive lung disease, with hypertension, coronary and peripheral vascular disease, with muscle diseases and rheumatic disorders. Benefit is established in a few instances, but this is a range of problems that cries out often for controlled trials.

For despite universal agreement that exercise and physical fitness are cardinal aspects of health promotion and a widespread hope that they help prevent the chronic diseases of Western industrialised countries, much remains to be learnt on both benefits and costs.

One example is exercise for the elderly. Evidence is growing —though sadly little is yet done about it—that exercise can be beneficial for the elderly in easing the activities of daily living and avoiding immobility, in maintaining nutrition, in temperature regulation, and in social wellbeing. Exercise programmes may also be valuable for mental functioning both at home and in institutional settings.[1] The possibility that exercise may delay aging processes is wide open for prospective study. Even the ancient question of exercise and longevity is still unsettled, and in general the field is clogged with confused thinking, mythology, and mystery. One tantalising possibility is that physical activity may have some value in preventing osteoporosis—and so its complications.

Both volumes have much to say on exercise and coronary heart disease. The inconsistencies in associations with physical activity at work[2] are an increasingly academic issue in the West; it is the exercise of leisure time that matters.[3,4] The three latest and very disparate reports[5–7] give further encouragement, strengthening the association of exercise with a reduction in the incidence of coronary heart disease and its mortality. The association seems to be largely independent of the classical risk factors—high serum cholesterol concentration, raised blood pressure, and cigarette smoking. Among the possible mechanisms through which exercise might protect against coronary heart disease[8] are lipoprotein patterns and the increase in high density lipoprotein cholesterol, though the amount of exercise required to achieve this effect as described in a recent investigation[9] is disconcerting. Further data are wanted, too, on the relations with cardiovascular training. The most relevant of the haemostatic mechanisms seems to be activation of fibrinolytic activity; the increased production of prostacyclin PGI_2 observed with vigorous exercise[10] is a new mechanism to explore.

In this generally underdeveloped research area one of the glaring deficiencies is the lack of collaboration between physiologists and epidemiologists over cardiovascular problems. A contrast may be drawn with lung function, where the external stimulus of industrial pulmonary disease encouraged cooperation. In practical terms, our present methods of assessing physical activity and cardiovascular fitness leave much to be desired. How worth while such collaboration can be is evident in the fitness survey carried out with characteristic imagination in Canada during 1981 on a large representative sample of the population aged 7 to 70. Results are beginning to appear, with standardised measurements of fitness, muscular strength and endurance, flexibility, body composition, and many data on exercise—behaviour and attitudes.[11,12] Such a national fitness survey is now being considered for Britain; the interest and enthusiasm for sport and exercise it could be expected to generate, the new information it would yield on the people's health, and its baseline for monitoring progress could well add a new dimension to the national effort in health promotion and health education.

A former president of the American Heart Association is quoted[2] as saying, rather severely, that "the use of exercise in prevention, treatment, and rehabilitation is in a state of transition from unfounded faddism to scientific legitimacy." Sponsored by the Sports Council and its fitness and health advisory group, the Health Education Council, and the Medical Research Society, a scientific symposium on exercise, health, and medicine was held earlier this month at the Sports Council's National Centre in Lilleshall, Shropshire, to speed that transition in Britain (see p 1637). Meanwhile as the 1983

marathon season opens, with its expected further huge increase in participants, the public may be said—literally—to be voting with its feet.

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Hypocholesterolaemia and cancer?

The serum cholesterol concentration of a population can be reduced and this is accompanied by a decrease in the incidence of coronary heart disease.[1] This observation raises some important questions: can the serum cholesterol concentration be reduced to an excessively low level by diet alone; is a low serum cholesterol concentration harmful; and is there an ideal range for the concentration of serum cholesterol?

Lewis and coworkers reported a decrease of 29% in mean serum cholesterol concentration in 12 Trappist monks after taking a modified fat diet supplemented with fibre for five weeks[2]—a result obtained with optimal compliance over a short period. The Oslo Study Group admitted 1232 healthy normotensive men with serum cholesterol concentrations between 7·5 and 9·8 mmol/l to a randomised controlled trial to determine the effects of reducing smoking and reducing serum cholesterol concentrations by modifying the diet.[1] Mean serum cholesterol concentrations were about 12% lower after five years in the treatment group, who experienced significantly fewer coronary events. This suggests that with appropriate dietary advice the mean serum cholesterol concentration in a population may be reduced by 10-15%—a reduction which can hardly be regarded as excessive.

A highly significant positive correlation was obtained when death rates from different countries for coronary heart disease were compared with those from cancer of the colon.[3] Rose and coworkers investigated the possibility that a raised serum cholesterol concentration might be a factor common to both diseases. Surprisingly, cancer of the colon was shown to be associated with low concentrations of serum cholesterol. The Framingham group studied 5209 people prospectively over 24 years,[4] finding that in men there was a significant inverse association between serum cholesterol concentrations and cancer at several sites, of which cancer of the colon achieved the greatest significance. There are several explanations for this association. Firstly, it may be the effect of competing lethal risks: people with high serum cholesterol concentrations may have died prematurely from ischaemic heart disease while those with low concentrations survived to develop cancer of the colon. Secondly, possibly a low serum cholesterol concentration is a consequence of cancer of the colon rather than a predisposing factor, though if this were true the Framingham patients would have had low cholesterol concentrations for at least 10 years before their cancers were detected. The absorption of cholesterol from the diet is variable but low, decreasing as cholesterol intake increases. The remainder is excreted in the faeces as neutral sterol.[5] A diet with a high content of animal fat will therefore increase the amount of cholesterol entering the colon. A third suggestion, therefore, is that cholesterol acts as a cocarcinogen within the colon.[6] Finally, a high fat diet stimulates the secretion of bile and bile salts may be converted by bacteria to cocarcinogens within the colon.[7 8] This effect may be enhanced if transit time through the colon is slow and the exposure of the colonic mucosa to carcinogens is prolonged. Thus individuals who absorb cholesterol poorly, and ingest a high cholesterol-low fibre diet, might be especially at risk of developing cancer of the colon.[9-11] In support of this hypothesis, recent work has shown a highly significant positive correlation between cholesterol intake and cancer of the colon.[12]

Possibly a low serum cholesterol concentration may be associated with other aetiological factors contributing to cancer. A diet low in cholesterol may contain synthetic trans fatty acids which could increase the permeability of the colonic mucosa to carcinogenic substances.[13] Another possibility is that low serum cholesterol concentrations may be associated with low concentrations of plasma retinol. The concentration of plasma retinol and the dietary intake of β-carotene are inversely related to mortality from cancer.[14 15] Marenah and coworkers (p 1603) show a direct relation between low density lipoprotein concentrations and plasma concentrations of retinol and β-carotene. If hypocholesterolaemia is carcinogenic, what is the mechanism? Oliver suggests that it might be via a reduction in the amount of cholesterol within the membrane of the cell, which alters its fluidity.[16] Marenah *et al* examined the cholesterol content and fluidity of monocytes and fibroblasts over a wide range of cholesterol concentrations and conclude "that changes in cell membranes are unlikely to occur at serum cholesterol concentrations attainable by the dietary or drug treatment of hyperlipidaemia."

So what practical conclusions may we draw? The relationship between low serum cholesterol concentration and cancer is present only in men, whereas that between increased serum cholesterol and heart disease is present in both sexes. Mortality from ischaemic heart disease exceeds that of cancer of the colon at all concentrations of serum cholesterol in men.[16] Mortality from cancer of the colon is virtually on a plateau between serum cholesterol concentrations of 7·8 and 4·9 mmol/l, but below this rises quite steeply. Mortality from ischaemic heart disease is on a gently rising curve below a serum cholesterol concentration of about 7·0 mmol/l, but then increases markedly.[16] Within a population widespread mild hypercholesterolaemia will make a greater contribution to coronary artery disease mortality than a few individuals with severe hypercholesterolaemia. Thus, in the light of