Life expectancy among males at birth in European republics of former Soviet Union (sources World Health Organisation, Unicef, and V Shkolnikov (personal communication)). Data for 1983-4 not available for some republics.

vested interests, not least in the Ministry of Finance, that opposed it.²

Although alcohol consumption began to increase again in the late 1980s, especially as a result of illicit production and diversion from state trading organisations, alcohol is unlikely to explain all the recent deterioration in life expectancy.³ As Ryan notes, much high quality epidemiological research is now being undertaken in the former Soviet Union, although there is little evidence that it is being acted on by policymakers either there or in the West. What is needed is a mechanism for bringing this work to a wider audience in both regions.

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Improving the detection of glaucoma

Intraocular pressure of greater than 21 mm Hg is an indication to refer

EDITOR,—Ronald Pitts Crick and Maurice W Tuck argue that "to improve the present system of detecting glaucoma the trend to more comprehensive testing by optometrists needs to be encouraged."¹ They suggest that patients should be referred if they have an intraocular pressure of ≥ 26 mm Hg or an intraocular pressure of 22-25 mm Hg plus optic discs or visual fields with features characteristic of glaucoma. Under present regulations, however, a patient should be referred "if there appears to be signs of injury, disease or abnormality in the eye or elsewhere which may require medical treatment."^{2,3} The upper limit of normal intraocular pressure is generally taken to be 21 mm Hg. Thus an intraocular pressure of 22-25 mm Hg, even in the absence of other features of glaucoma, is a sign of disease or abnormality in the eye. An optometrist who did not refer such a patient could be in breach of current regulations.

This needs to be clarified before optometrists can take a more active role in the screening of glaucoma.

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- 1 Crick RP, Tuck M. How can we improve the detection of glaucoma? *BMJ* 1995;310:546-7. (4 March.)
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Link between glaucoma and diabetes is disputed

EDITOR,—Ronald Pitts Crick and Maurice W Tuck highlight the difficulties of screening for glaucoma and twice mentioned diabetes as an important risk factor.¹ Tielsch *et al* have recently cast doubt on this tenet in ophthalmology.² Using data from the Baltimore eye survey, they concluded that there is no evidence to support an association between diabetes and primary open angle glaucoma.

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Lipid lowering dietary advice

EDITOR,—Scientists and practitioners agree that dietary modification should be the first step of any cholesterol lowering regimen, but H A W Neil and colleagues report that the reduction in concentrations of total cholesterol and low density lipoprotein cholesterol after dietary advice given in general practice to hypercholesterolaemic patients by a dietitian, practice nurses, or in a diet leaflet alone was small.¹ The poor response seems to be related mainly to the low dietary compliance in poorly motivated patients. This agrees with our objective evaluations of the compliance to hypocholesterolaemic dietary prescriptions: on the basis of changes in the plasma fatty acid pattern, after two months on the diet only 40% of patients could be defined as good adherers, while 22% had a poor compliance and the remaining a variable adherence over time.² Neil and colleagues conclude that a more intensive intervention than is normally available in primary care is probably necessary and a mass approach to dietary changes is needed.

Instead, better results have been obtained in 65 patients attending a lipid clinic³ and in 45, 9 year old schoolchildren enrolled in a community project for control of chronic diseases.⁴ In the first study, dietary intervention was performed by a doctor and included the evaluation of a seven day food record, practical dietary recommendations, and distribution of a booklet suggesting the adoption of the Mediterranean diet. After six weeks, total cholesterol decreased by 11.3% and low density lipoprotein cholesterol by 15.3% (from 7.12 (SD 0.5) mmol/l to 6.31 (0.7) mmol/l, $P < 0.001$, and from 5.04 (0.5) mmol/l to 4.27 (0.6) mmol/l, $P < 0.001$, respectively) in 25 patients with moderate hypercholesterolaemia and by 9.1% and 9.9% in 40 patients with severe hypercholesterolaemia (from 9.49 (1.4) mmol/l to 8.76 (1.7) mmol/l, $P < 0.001$, and from 7.43 (1.4) mmol/l to 6.70 (1.8) mmol/l, $P < 0.001$).

In the second study simple dietary advice was given to parents when blood was taken. This included a list of foods to be avoided and a list of suggested foods to be consumed at home and at school. As an example to start with, a standard isocaloric, moderate fat, moderate cholesterol diet was provided, together with practical information on food preparation. After six weeks, total cholesterol dropped by 14.1% (from 4.58 (0.8) mmol/l to 3.93 (0.6) mmol/l, $P < 0.001$) and low density lipoprotein cholesterol by 16.9% (from 2.78 (0.7) mmol/l to 2.31 (0.5) mmol/l, $P < 0.001$). Both total cholesterol and low density lipoprotein cholesterol remained almost unchanged after 12 months (3.94 (0.6) mmol/l and 2.27 (0.5) mmol/l, respectively).

The above data are similar to those obtained in the long term treatment with some cholesterol lowering drugs, suggesting the high efficacy of simple and low cost dietary advice and the need for a careful evaluation of the compliance to hypocholesterolaemic dietary prescriptions before starting drug treatment. "True dietary resistance" due to genetic factors should also be borne in mind.⁵

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Magnesium in acute myocardial infarction

EDITOR,—Salim Yusuf and Marcus Flather¹ offer no satisfactory explanation for the statistical incompatibility ($P > 0.0001$ for heterogeneity) between the inefficacy of magnesium in the fourth international study of infarct survival (ISIS-4)² and the clearly positive pooled results of the 10 other trials of intravenous magnesium in acute myocardial infarction. Magnesium has been reported to protect contractile function in a dozen experimental models of myocardial ischaemia and reperfusion and is now known to act during the first few minutes of reperfusion.^{3,4} ISIS-4 was unique in that most patients underwent thrombolysis before being randomised to magnesium or control (to accommodate the factorial design). The interval was not recorded but most typically have been three or more hours after randomisation took place at a mean of seven hours from the onset of symptoms. The 30% of patients in ISIS-4 who were not given thrombolytic treatment were randomised at a mean of 12 hours after the onset of symptoms, when spontaneous reperfusion of viable myocardium is unlikely to occur.

ISIS-4 was not designed to test for an anti-reperfusion effect of magnesium. Any subgroup analysis put forward to disprove such an effect