The Association of British Insurers has placed on record its statement of practice regarding HIV testing. This confirms the non-prejudicial stance taken by insurers when medically assessing applicants for insurance. The association is actively redressing misleading and emotive statements with specific leaflets and other appropriate measures.

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Impaired glucose tolerance and height

SIR,—P M McKeigue is correct in stating[1] that our findings of an association between short stature and impaired glucose tolerance[2] may be a chance phenomenon or may result from an unusual characteristic of our study population. Since submitting that report, however, we have published further data confirming this association in a different (and larger) population—those taking part in the Isle of Ely diabetes study. In that analysis there was also a negative relation between height and the plasma glucose concentration two hours after a glucose load, used as a continuous measure of glucose tolerance.[3] There has been an independent report of a higher risk of ischaemic heart disease (which is associated with impaired glucose tolerance) in shorter men, thus confirming several similar, previous findings (J W G Yarnell *et al*, personal communication).

Furthermore, retrospective cohort studies of birth weight and weight at 1 year have shown striking relations with measures of the risk of ischaemic heart disease and glucose tolerance in adulthood.[4] Clearly, many factors affect adult height, such as inheritance, nutrition and infection during childhood, puberty, and, as we have observed,[3] osteoporosis in later life. Depending on the communities studied, the influences of these other factors will vary and, on occasions, obscure the small but significant differences that we have found. Their biological importance still has to be determined.

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Hypertension and non-insulin dependent diabetes

SIR,—In his editorial John S Yudkin focuses on the possibility that hypertension or its treatment may worsen insulin resistance and precipitate glucose intolerance,[1] but in addition to the results of a recent Finnish study[2] there is other evidence to support the converse of this "chicken and egg" association—namely, that insulin resistance or hyperinsulinaemia, or both, might have a primary pathophysiological role in the regulation of blood pressure.

Resistance to insulin stimulated glucose disposal, as well as being a feature of obesity, type II diabetes, and essential hypertension, occurs in old age and subjects with glucocorticoid excess and acromegaly, and it is relevant that these conditions too are associated with the development of increased blood pressure and impaired glucose tolerance. A similar pattern of metabolic abnormalities has been identified in spontaneous rodent models of hypertension, and, moreover, insulin resistance is present from an early stage of development when hypertension is barely detectable.[3] Animals with renovascular hypertension, however, do not develop insulin resistance as a consequence of the increase in blood pressure.[4]

Perhaps the most direct evidence for the role that insulin may have in the regulation of blood pressure comes, however, from the established evidence that weight loss and physical training—interventions that enhance insulin sensitivity and lower plasma insulin concentrations—are effective in reducing blood pressure in obese and diabetic patients and in essential hypertension. Furthermore, physical training may lower blood pressure even if body weight remains unchanged, particularly in those people who are hyperinsulinaemic before training starts.[5]

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Lp(a) concentrations

SIR,—Helge Kapelrud and colleagues report increased serum concentrations of Lp(a) lipoprotein in diabetic patients with microalbuminuria and suggest that this may explain their excess mortality. We performed a similar study but cannot confirm these observations.

We assigned patients from our diabetic renal unit to groups on the basis of their albumin excretion rate, estimated from overnight urine collections over six months. Twenty one patients had normoalbuminuria (albumin excretion rate <20 μg/min) and 15 had albuminuria (albumin excretion rate >20 μg/min). The groups were well matched for age, sex, duration of diabetes, and glycated haemoglobin concentration, and there were no significant differences in plasma cholesterol or triglyceride concentrations (table). Lp(a) concentrations, measured by electroimmunoassay, were heavily skewed, with only two values greater than 300 mg/l (both in patients with albuminuria). Median values, however, were similar (89·0 mg/l in patients with normoalbuminuria, 97·5 mg/l in patients with albuminuria; NS). Regression analysis showed a correlation between Lp(a) concentration and albumin excretion rate ($r=0·41$, $p<0·05$), but this was heavily influenced by the two outlying values.

Our patients' Lp(a) concentrations were similar to those in Kapelrud and colleagues' study but there was no difference between the groups. Raised Lp(a) concentrations have been reported with albuminuric renal disease.[2] The two patients in our study who had higher than normal Lp(a) concentrations had macroproteinuria. Lp(a) concentrations may also rise with impaired renal function.[3] None of our patients, however, had a serum creatinine concentration of >200 μg/min, but Kapelrud and colleagues do not give any data on renal function in their patients.

Lp(a) concentrations are influenced by glycaemic control[4] but not by age or sex. Glycated haemoglobin concentrations were similar in our groups and comparable with those in Kapelrud and colleagues' patients. Neither our patients' greater age nor differences in the sex ratio can explain the difference between our results. We think that further work is required before the excess cardiovascular mortality in diabetic patients with proteinuria is adequately explained.

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SIR,—James Scott's editorial[1] and the studies of Kapelrud and colleagues[2] and Farish and colleagues[3] show that high serum Lp(a) lipoprotein concentrations may be related to coronary heart disease in populations at risk such as diabetic patients with microalbuminuria and postmenopausal women taking norethisterone.

We measured serum concentrations of Lp(a), fibrinogen, and cholesterol and body mass index in 150 obese subjects (60 male, 90 female). We found much higher concentrations of Lp(a) in

Data on diabetic patients with normoalbuminuria and albuminuria. (All values expressed as means(SD) except where otherwise stated)

	Normoalbuminuria (n=21)	Albuminuria (n=15)	p
Albumin excretion rate (μg/min)	86 (18)	102 (31)	<0·001
M:F	9:12	6:9	
Age (years)	35·7 (15·3)	33·4 (14·8)	NS
Duration of diabetes (years)	17·3 (11·6)	15·8 (9·2)	NS
Glycated haemoglobin (%)	10·4 (2·5)	8·1 (2·7)	NS
Creatinine (μmol/l)	86 (18)	102 (31)	NS
Cholesterol (mmol/l)	5·0 (1·6)	5·9 (1·8)	NS
Triglyceride (mmol/l)	1·4 (1·0)	2·2 (2·5)	NS
Median (range) Lp(a) (mg/l)	89·0 (30-900)	97·5 (10-168)	NS