impact is small. More to the point, they have not been allocated between the different alcohol groups and so cannot be included in a comparison of the pattern of taxation.

The most relevant point in Wilson's letter is that sales taxes on wine have been raised since the original study was conducted. He claims, without supporting evidence or reference, that the retail tax component of wine is now 16.4%. Our figures, based upon Commonwealth data, show something smaller. However even his figure is several orders of magnitude less than the 42% and 91% mark-up on the cost of beer and spirits respectively which result from the excise on these products. Alcohol taxes in Australia remain arbitrary.

Wilson's data on the cost of producing a litre of alcohol also differ somewhat from the authoritative figures produced by the Commonwealth. However they are also largely irrelevant. If the object of public policy is to minimise the impact of taxes upon consumption benefits then the relevant data are the price elasticity of demand and the overall cost of production (of the product, not of the alcohol). If the policy objective is the minimisation of alcohol related ill health the relevant data are the price elasticity of demand and the alcohol content of the beverage. Current and past policies in Australia have not reflected either of these objectives.

Jeff Richardson
Professor, National Centre for Health Program Evaluation, Department of Economics, Monash University, Clayton, VIC 3168

Reducing agents and AIDS — why are we waiting?

To the Editor: In Australia we read or hear very little about the possible therapeutic benefits of the administration of reducing agents to individuals infected with the human immunodeficiency virus (HIV) and patients with acquired immunodeficiency syndrome (AIDS). In spite of the rapid escalation of biological knowledge much of medicine is still dependent on the (sometimes serendipitous) assembly of empirical observations. Perhaps this fact has been overshadowed by the masterful technology of the AIDS era.

There is now abundant evidence that HIV-positive individuals as well as AIDS patients have an altered redox state and that this may be an important factor in their disease process. For example, in February 1989 Eck and his colleagues showed that plasma levels of acid-soluble thiols (cysteine) and glutathione levels in peripheral blood mononuclear cells and monocytes are significantly decreased in the various AIDS groups.[1] Their in-vitro studies also showed a strong dependence of intracellular glutathione concentration on extracellular cysteine with an accompanying strong correlation between the glutathione concentration and the viability and functional activity of T cells.

In December 1989, Buhl et al. described systemic and lung epithelial lining fluid glutathione deficiency in symptom-free HIV-positive individuals.[2] The levels reported were respectively 30% and 60% of the levels in healthy controls. These authors also pointed out that, although unexplained, the glutathione deficiency might be a direct causative factor in the reduced immune function observed in patients with HIV infection. Glutathione is the major transport system in plasma for the sulphydryl-containing amino acid cysteine which itself is a major antioxidant. Oxidants cause breaks in the DNA strands of lymphocytes and damage many of their innate functions. Sulphydryl compounds also augment a number of lymphocyte functions *in vitro*, including mitogenic T cell proliferation and T and B cell differentiation. The fact that glutathione deficiency is clearly demonstrated in the lungs of HIV-positive patients may be of great importance in understanding the genesis of opportunistic pulmonary infection that characterises AIDS.

It is reasonable to argue that HIV infection is accompanied by a period of oxidative stress resulting in a lowering of glutathione levels which in itself may explain some of the phenomena of the infection.

As AIDS has a 100% fatality rate and 60% of HIV-positive individuals are said to develop AIDS in five years, would it not be reasonable to give urgent consideration to trials of therapy and prevention with reducing agents?

In these difficult times even the aetiological significance of HIV itself in Kaposi's sarcoma has been put in some doubt. An Australian graduate, Valerie Beral, and her colleagues at the Centers for Disease Control in Atlanta recently argued that on epidemiological grounds there is reason to believe that Kaposi's sarcoma, one of the principal AIDS diseases, "may be caused by an as yet unidentified infectious agent".[3] Therefore should we not be searching for other possible mechanisms that could point to other treatment options?

There are at least two reducing substances that are cheap, readily available and virtually devoid of any serious side effects. These are glutathione and *N*-acetylcysteine. The latter is familiar to clinicians as an agent originally used in the treatment of chronic bronchitis and probably more recently used as an antidote for paracetamol poisoning. Herzenberg from Stanford University has recently documented reversal of low systemic glutathione levels in HIV-positive individuals by use of *N*- acetylcysteine; experiments *in vitro* indicate that *N*-acetylcysteine can produce highly desirable effects on HIV replication including a reduction in the appearance of p24 antigen.[4] An Italian company, Zambon, will be assigned a patent on this drug for the purposes of treating AIDS when the United States Food and Drug Authority approves the first clinical trials.

Oxidative stress as an important mechanism in AIDS and its possible reversal by reducing agents was hypothesised as long ago as 1985 by another Australian researcher[5,6]; surely it is time someone carried out trials of therapy with reducing agents?

V F Turner, FRACS
40 Ventnor Avenue, West Perth, WA 6005

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Iron status of Australian children

To the Editor: In the paper by English and Bennett on the iron status of 1696 Australian children, iron deficiency was diagnosed when the levels of plasma transferrin and ferritin were both below specified cutoff points.[1] Citing the paper by Cook and Finch,[2] the authors suggest that there was "an increasing sensitivity in the ability to detect true iron deficiency when abnormal values for two or three iron status indicators were used rather than one".

A review of the original published data by Cook, Finch and Smith,[3] reveals a number of salient points. Firstly, the authors do not calculate and present the sensitivities and specificities of their diagnostic tests. Secondly, the authors themselves state that "an arbitrary decision was made to accept individuals as iron deficient who showed two or more abnormal parameters". Finally, sensitivities and specificities can only be calculated with regards to iron deficiency anaemia, since a "gold standard" for iron deficiency is not presented.

Analysis of the above data with respect to the sensitivity and specificity of the parameters of iron metabolism (serum ferritin level, transferrin saturation, red cell protoporphyrin level) to detect iron-deficiency anaemia (haemoglobin level < 130 g/L for adult males, and < 120 g/L for all others taken as the "gold standard") gives the results shown in the Table.

TABLE: Sensitivity and specificity of tests for iron deficiency anaemia

	Sensitivity	Specificity
One parameter abnormal	26%	81%
Any two parameters abnormal	17%	96%
All three parameters abnormal	33%	98%
Any two or more parameters abnormal	50%	94%

Parameters: serum ferritin level, transferrin saturation, red cell protoporphyrin level.

Although the sensitivity does increase when tests are combined, it is obvious that the parameters of iron metabolism as diagnostic or screening tests have an unacceptably low sensitivity.

It is important when establishing prevalences of disease states, that the sensitivity and specificity of the diagnostic tests be considered.

Since there isn't a practical "gold standard" for the diagnosis of iron deficiency without anaemia, it is impossible to assess the reliability of the various diagnostic tests, and hence the true prevalences.

B B Jalaludin, MRCP
Medical Research Officer, Department of Community Medicine, Westmead Hospital, Westmead NSW 2145

1 English RM, Bennett SA. Iron status of Australian children. *Med J Aust* 1990; 152: 582-586.
2 Cook JD, Finch DA. Assessing iron status of a population. *Am J Clin Nutr* 1979; 32: 2115-2119.
3 Cook JD, Finch DA, Smith NJ. Evaluation of the iron status of a population. *Blood* 1976; 3: 449-455.

In reply: Dr Jalaludin raises some important issues in regard to criteria for estimating the prevalence of iron deficiency and iron-deficiency anaemia in a population. As noted in our paper[1] there is general agreement that it is difficult to assess impaired iron status from single indicators. This statement also applies to the haemoglobin level as a measure of iron-deficiency anaemia, which has been described by Cook, Finch and Smith as a measure not only lacking specificity but also relatively insensitive because of the wide scatter of values in normal subjects.[2]

The "gold standard" for detecting iron deficiency is acknowledged to be the response or lack of response to orally administered iron therapy. Cook et al. note that in one study applying this standard, 20% of both anaemic and normal women were classified incorrectly on the basis of the initial haemoglobin concentration. Therefore, it would not have been appropriate to have calculated sensitivities and specificities for the data of Cook et al.

The estimate of the prevalence of iron deficiency in our paper is consistent with the criteria adopted by Cook et al. to report the prevalence of iron deficiency in a population.[1] It is also consistent with the criteria applied by an expert scientific group of the Federation of American Societies for Experimental Biology in its report on the iron status of the American population. Using two models, the group found that two or more abnormal values for iron metabolism indicators could be considered indicative of impaired iron status.[3] Concerning haemoglobin levels and iron status, the group was of the opinion that the number of iron-deficient individuals hidden among those with the normal range of haemoglobin concentration is probably as great as the number of iron-deficient individuals who can be recognised as anaemic.

A recent report on nutrition monitoring in the United States, which uses the expert scientific group's criteria for determining iron status, has suggested that there