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The white paper on public health

Is promising, but has some blind spots, which must be tackled

The white paper *Choosing Health: making healthy choices easier* lays out the government's approach to tackling a broad range of public health challenges from smoking, obesity, and drinking to mental and sexual health.[1] Positive aspects, such as signposting foods to indicate their fat, salt, and sugar contents have quite rightly been welcomed. Limitations, including the ironic coupling of the emphasis on individual choice with a failure to tackle secondhand smoke, are being highlighted by the relevant expert groups. The public are developing an awareness of the relevance of these public health issues in their lives, thanks in part to the substantial media coverage of the report's contents and of stakeholders' responses. At least these problems are beginning to get a thorough public airing, which must be a step in the right direction for further policy change. However, to maximise the benefits of such a substantial switch towards prevention, as urged by Wanless,[2] three fundamental blind spots need to be considered.

The first exemplifies a historical failure to match rhetoric with action. The report promises to help local health services deal with inequalities: "We are giving primary care trusts the means to tackle health inequalities and improve health through funding to give greater priority to areas of high health need . . ." This implies recognition of the need to provide greater resources to those primary care trusts in most need in order to move disadvantaged people up to the level of advantaged people.[3] However, the promise rings hollow for many primary care trusts serving deprived communities. The government has set inequalities targets for these primary care trusts requiring them to improve the health of their populations faster than the average for the United Kingdom.[4] Many of these are already struggling because they have not even received the funding they are due according to the government's own calculations of their requirements.[5] In northeast London, for example, all four of the primary care trusts defined as being in the government's "spearhead group" (which means they have the worst health and deprivation indices), are currently underfunded to a total of over £80m ($149m; €114m) against their weighted capitation target.

The second blind spot is an inability to work through the consequences of worrying about being labelled as a nanny state. As a result, the recommendations on banning smoking are inconsistent and may actually increase health inequalities due to differential uptake by people across the socioeconomic divide.[6] Encouraging smoking cessation on an individual level is one thing, but allowing smoking in pubs that do not serve prepared food undermines this effort. It is precisely people who visit pubs where food is not served who are in most need of protection from the effects of secondhand smoke. In this way the white paper fails to address the inequalities that purport to be the driving force behind it.

The third blind spot is the government's reluctance to take its own medicine. That decisions should be informed by robust research evidence is becoming embedded across the health service, and the white paper does pay some attention to the need to use evidence based interventions. A pity then that such emphasis is placed on individualistic interventions such as the provision of educational materials, when it is well established that information alone does not entice people to change behaviour. Nor will the provision of fruit for your lunch box alter eating habits so long as healthy eating is perceived to be posh.[7] That the government persists in believing that it is as simple as that is disappointing. Improving public health is about changing behaviour. We need an in-depth understanding of the personal values, beliefs, preferences, and aspirations that drive behaviours in different social groups. Only then can we begin to design interventions to modify deep seated cultural norms and to challenge ingrained ambivalence. Changing behaviour will require the implementation of comprehensive structural, environmental, and economic interventions. For example, the report has nothing about using the taxation system to increase the minimum weekly income for healthy living,[8] or for increasing taxes on tobacco, which are known to have the greatest effects on smoking levels of young people.[9]

The white paper does signal a seminal moment in terms of attention to public health and could have a profound impact. But the government must consider its blind spots and show a commitment to tackle complex environmental and personal barriers to behaviour change if it is to fulfil its pledge to make healthy choices easier.

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Angiotensin receptor blockers and myocardial infarction

These drugs may increase myocardial infarction—and patients may need to be told

The interpretation of large scale clinical trials is being increasingly scrutinised by leading journals,[1] with great emphasis being placed on the importance of sharing all potential side effects, no matter how trivial, with patients. The *Lancet* recently published the results of the valsartan antihypertensive long term use evaluation (VALUE) trial, a study of the effects of reducing blood pressure in patients at high risk.[2] The angiotensin receptor blocker valsartan produced a statistically significant 19% relative increase in the prespecified secondary end point of myocardial infarction (fatal and non-fatal) compared with amlodipine. A doctor who is a patient of one of the authors (SV) commented that if the incidence of myocardial infarction increased with valsartan it would be an essential component of informed consent to share this information when prescribing valsartan for high risk patients with high blood pressure. These peculiar results led us to examine carefully the evidence surrounding angiotensin receptor blocker and myocardial infarction.

Could the unexpected increase in the incidence of myocardial infarction in the VALUE trial represent a statistical aberration? Although the modest, yet significant differential in blood pressure in favour of amlodipine (1.8 mm Hg systolic and 1.5 mm Hg diastolic *v* amlodipine) may explain the 13% increase in the incidence of stroke in patients taking valsartan (P = 0.08), it is unlikely, according to some experts, to account for the 19% increase in the incidence of myocardial infarction.[3]

Unfortunately careful evaluation of the current evidence shows that angiotensin receptor blockers, unlike angiotensin converting enzyme inhibitors, are either neutral or increase the rates of myocardial infarction despite their beneficial effects on reducing blood pressure.

For example, the CHARM-alternative trial showed a significant 36% increase in myocardial infarction with candesartan (versus placebo) despite a reduction in blood pressure (4.4 mm Hg systolic and 3.9 mm Hg diastolic *v* placebo treatment).[4] Likewise, in the CHARM-preserved study, candesartan reduced admissions for chronic heart failure by 13% but did not prevent death despite a mortality of 11.3% and a reduction in blood pressure of 7 mm Hg systolic and 3 mm Hg diastolic compared with placebo.[5] In the study on cognition and prognosis in the elderly (SCOPE), candesartan was associated with a non-significant 10% increase in fatal plus non-fatal myocardial infarction despite lower blood pressure (3.2 mm Hg systolic and 1.6 mm Hg diastolic for candesartan *v* placebo).[6] Furthermore, the angiotensin receptor blocker losartan in the LIFE study did not reduce rates of myocardial infarction despite a 1.7 mm Hg lower pulse pressure compared with atenolol.[7] In the RENAAL trial, a study performed in diabetic patients with nephropathy, the angiotensin receptor blocker losartan offered nephroprotection, but no reduction in cardiovascular mortality, although about 30% of patients died of a cardiovascular event.[8] In a similar population the angiotensin receptor blocker irbesartan showed nephroprotection[9] but seemed to have no impact on the 24% incidence of cardiovascular events (a secondary composite end point). Although irbesartan lowered blood pressure (4 mm Hg systolic and 3 mm Hg diastolic *v* placebo), no reduction occurred in myocardial infarction, stroke, or cardiovascular death. Compared with amlodipine, irbesartan was associated with a 36% increase in non-fatal myocardial infarction (P = 0.06), a 48% non-significant increase in stroke, and a 29% non-significant increase in death despite similar blood pressure reduction (see advisory briefing of the Food and Drug Administration, NDA 20-757 (S-021), www.fda.gov).

These peculiar effects of angiotensin receptor blockers on myocardial infarction stand in contrast to those of angiotensin converting enzyme inhibitors, which consistently produce a 20% or greater reduction in myocardial infarction in patients with diabetes, hypertension, renal insufficiency, and atherosclerosis.

How could two pharmacological agents, considered by many to be interchangeable and equivalent, have such divergent effects on coronary vascular outcomes despite similar effects on blood pressure? Medicine contains several examples of similar pharmacological conundrums. For example, metformin and phenformin, agents of the same class that have similar effects on insulin sensitivity and glycaemic control, have different side effects, and phenformin is associated with a higher rate of lactic acidosis. Troglitazone, rosiglitazone, and pioglitazone are all thiazolidinedione insulin sensitisers, yet troglitazone was removed from the market because of increased rates of hepatocellular necrosis. Different

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