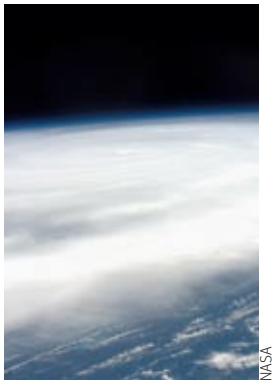
The economics of tackling climate change

Don't leave health benefits out of the equation



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Ian Roberts professor of epidemiology and public health, London School of Hygiene and Tropical Medicine, London, UK
Ian.Roberts@lshtm.ac.uk

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Tony Blair welcomed the publication of the Stern review on the economics of climate change calling it a wake up call to the world.[1] Its message was clear. The costs of taking action to stabilise the climate will be high but much less than the costs of inaction. Delay would be dangerous. Action is needed now. The review also exposed the economic cause of climate change. Climate change is market failure on the greatest scale the world has ever seen.[1]

Markets fail to provide the right quantity of goods and services when important costs are left out of our private economic decision making. In general, the extent to which people engage in activities that result in the emission of greenhouse gases depends on the cost of those activities. For example, when deciding whether to travel by car we might consider the cost of the time, petrol, parking, and wear and tear on the vehicle. Londoners might include the congestion charge, but few people would take into account the costs to the world and future generations of the emissions that their journey will produce. Costs on others that do not (without public action) enter into the private arithmetic of how we allocate resources are called externalities and are the economic basis of one of the greatest threats humanity has ever faced.

While economists have been grappling with the resource implications of policies to tackle climate change, the public health implications of these policies have also come under scrutiny.[2] The message this time is that they present unrivalled opportunities for improving public health. Policies that reduce greenhouse gas emissions could also substantially reduce obesity, diabetes, heart disease, cancer, road deaths and injuries, and air pollution.[2]

Take food production for instance, which results in substantial greenhouse emissions, similar in magnitude to those from transport or industry.[1] Livestock rearing for meat and dairy produce is a major source of emissions, including methane from enteric fermentation and carbon dioxide as a result of land clearance for cattle farming.[3] Polices that internalise the environmental costs of livestock production would reduce the consumption of animal products. This would help stabilise the climate, but would also—by reducing the amount of saturated fat and meat in the diet—reduce the incidence of cardiovascular disease and bowel cancer.[3] Similar policies on other foods might decrease the consumption of the carbon intensive fats and refined sugars that are helping to fuel the obesity pandemic.[4]

The transition to a low carbon transport system that involved more walking and cycling would substantially benefit health.[5] Road traffic crashes account for 1.2 million deaths worldwide each year and 10 times as many serious injuries. Their incidence is a function of the use of fossil fuels by the transport sector.[6] After all, the kinetic energy that breaks bones and tears soft tissues comes from the chemical energy stored in the fuel tank, the burning of which emits carbon dioxide. Death rates for pedestrians and cyclists exhibit steep social gradients, and reducing traffic volumes and speeds would have important equity implications.[7] Urban air pollution—much of which is related to transport—causes a further 800 000 premature deaths each year.[5] Walking, cycling, or using public transport instead of travelling by car would reduce the use of energy from fossil fuels; it would also reduce traffic injuries and air pollution. By increasing physical activity it would tackle the output side of the personal energy balance equation, again with implications for obesity.

Improvements in the efficiency of home energy will reduce mortality and morbidity from the extremes of heat and cold and reduce the vulnerability of the poor to fluctuations in the price of energy.[8] Greater use of renewable energy sources will also reduce urban air pollution. In generating electricity, the energy sources with the highest carbon dioxide emission profiles also have the greatest effect on air pollution and the incidence of occupational injuries.[9]

Providing access to clean energy for the 2.4 billion people who use biomass fuels for cooking would reduce the burden of mortality and morbidity from indoor air pollution, and universal access to clean electricity could initiate a tidal wave of human creativity and accelerate the pace of human development.[2 10]

Like Tony Blair, Gordon Brown also welcomed the Stern review calling it "the most comprehensive analysis yet" and commenting that "above all environmental policy is economic policy." Stern acknowledged that any positive effects of tackling climate change over and above those detailed in his review would strengthen the economic argument for stabilising the climate. The full extent of health benefits is only now coming to light.

Climate change, a globally important externality, is at least partly the result of leaving environmental impact out of economic decision making. To leave out health when considering the benefits of policies that tackle climate change would be a second serious omission. Environmental policy is economic policy. It is also health policy. Economists and health professionals must work together to ensure that the effects of environmental

policies on health and development are quantified and included in the economic models that inform our collective response to the threat from climate change.

It would not be the first time that environmental policy had substantial benefits for health. Two hundred years ago the streets of London were awash with sewage. In 1858 the smell from the Thames was so strong that MPs declared the House of Commons "unusable."[11] Infectious disease was a deadly scourge, but it was the "great stink" of 1858 that helped secure the funds needed to sort out London's sewage. Policy on sewage did more to improve the health of Londoners than any health policy that century. Indeed, in a recent *BMJ* poll, sanitation was voted the greatest medical advance in the past 166 years.[12] Could tackling climate be the next great medical advance?

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Aspirin resistance in cardiovascular disease

Carries a worse prognosis, but may indicate pre-existing higher risk

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Giuseppe Biondi-Zoccai assistant professor in cardiology, Division of Cardiology, University of Turin, 10126 Turin, Italy
gbiondizoccai@gmail.com
Marzia Lotrionte consultant cardiologist, Institute of Cardiology, Catholic University, 00136 Rome, Italy

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Aspirin has clear benefits in cardiovascular disease. It reduces total mortality, cardiovascular mortality, and cardiovascular morbidity in people with cardiovascular disease or those at high risk of the disease; it is also cheap, relatively safe, and easy to use.[1] So why does aspirin fail to work in some people who take it as prescribed? Research on this clinical "resistance" to aspirin has tried to assess whether the effect on the in vitro activation of platelets depends only on the dose and type of antithrombotic agent given, or whether some people respond poorly ("resist") to a specific drug, as some people do to specific antibiotics. In the accompanying systematic review, Krasopoulos and colleagues assess whether resistance to aspirin is related to cardiovascular outcomes in people with cardiovascular disease.[2]

To date, most research has focused on whether aspirin resistance really exists, whether antiplatelet resistance is specific to certain agents or classes of drug,[3] and whether resistance carries a worse prognosis. Firstly, no accepted gold standard test to define aspirin resistance is available.[4] This and other epidemiological considerations have led some authors to argue that what has been dubbed resistance is just part of normal (Gaussian) variability in pharmacokinetics and pharmacodynamics. Others admit that aspirin resistance exists but are doubtful about its prognostic importance.[5] They think that a lack of response to aspirin could just be a proxy marker for more advanced pre-existing disease or less controlled traditional risk factors.

Krasopoulos and colleagues review analysed data from 20 studies and 2930 patients and found resistance to aspirin in 28% of people. Resistance was significantly more common in women and those with renal failure and was associated with a statistically and clinically significant increase in the risk of death or adverse cardiovascular events, at least in univariate analysis. However, they found no association between the dose of aspirin or concomitant use of other antiplatelet agents and adverse events. The review has some limitations including use of fixed effect methods and lack of pooled multivariable adjusted estimates. A similar review recently came to the same conclusions, however, confirming the external validity of Krasopoulos and colleagues' review.[6]

Despite this work several questions remain. We don't know whether aspirin resistance is a true abnormal response or whether it reflects normal variability in drug activity. We also aren't clear whether aspirin resistance has a negative prognostic effect independent of more traditional risk factors, such as diabetes or obesity.[5] If aspirin resistance is an abnormal response that results in worse prognosis then what can clinicians do? We suggest that when aspirin resistance is suspected patients should be screened using available tests. Management of patients with aspirin resistance should include a comprehensive appraisal of thrombotic and bleeding risks, the likelihood of non-adherence to treatment, and access to other antiplatelet agents. On the basis of this assessment, several strategies can then be proposed. These include adding another antiplatelet agent (for people at high thrombotic risk and low bleeding risk), substituting aspirin with the more effective clopidogrel (for people at intermediate thrombotic risk and low bleeding risk), increasing the dose of aspirin (for example, to 325 mg/day in people at mildly increased thrombotic risk and low to intermediate bleeding risk), or continuing with the same antiplatelet regimen (for everyone at high bleeding risk).

The problem in finding truly scientific answers to the effectiveness of these strategies lies in the lack of randomised controlled clinical trials. This will