

How will we know if the London 2012 Olympics and Paralympics benefit health?

By measuring directly attributable effects in addition to opportunity costs

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The London 2012 Olympic and Paralympic Games will cost £9.3bn (€10.7bn; \$13.3bn), £150 for every man, woman, and child in the United Kingdom. For this investment, we have been promised legacy outcomes¹ for sport and physical activity, regeneration, culture, sustainability, the economy, and disability. The last of these legacy outcomes was added only recently,² after considerable criticism.³ Each of these areas has implications for health or relates to socioeconomic determinants of health.⁴

In the linked systematic review, McCartney and colleagues found little evidence that major multi-sports events deliver health or socioeconomic benefits.⁵ This suggests that £150 a head towards staging London 2012 is a poor investment made by the treasury on our behalf. However, the review shows that past research comprises a small number of poor quality studies, with large gaps in the outcomes evaluated. Furthermore, studies have evaluated incidental outcomes; London 2012 is the first Olympic and Paralympic Games that will explicitly try to develop socioeconomic legacies for which success indicators are identified—the highest profile of which is to get two million more people more active by 2012.¹ London 2012 therefore seems to fulfil one of McCartney and colleagues' recommendations to include longer term outcomes as legacy goals,⁵ and, in the case of increased physical activity, the goal is explicitly linked to improved public health.^{6,7}

However, if the chosen measure for this legacy outcome, the active people survey,⁸ shows that two million more people are more active by 2012, it will not be an indication that London 2012 has increased physical activity levels. This is because the survey cannot demonstrate attribution, which put simply means that it cannot provide evidence that London 2012 intervention programmes are the cause of two million more people becoming more active. Neither can the survey provide evidence of "additionality," which means it cannot show that London 2012 programmes have increased physical activity to levels greater than could have been achieved by investment in alternative interventions. McCartney and colleagues' review highlights these difficulties, with no included studies able to attribute changes to events, and few containing contemporary comparisons against which opportunity costs can be considered.



So, how will we know if our investment of £150 a person has generated additional health and socioeconomic outcomes clearly attributable to London 2012? Evaluation must focus on London 2012 intervention programmes rather than generic national surveys and consider net outputs not gross positive indicators. Free swimming for under 16 year olds and over 60s (a London 2012 intervention programme) may show high take-up by the public.⁹ However, this

may be confounded by several factors such as people who already swim simply doing so free of charge or increasing their frequency of swimming. In this case, free swimming would not have had additional benefit because it would not have resulted in more people being more active, and evaluations must take care to remove such behaviours from impact calculations.¹⁰

Opportunity costs must be considered if outcomes are to be attributed to London 2012. Because each major multi-sports event is unique, the contemporary comparisons that McCartney and colleagues call for cannot be derived from control groups or reference cases.¹¹ Detailed alternative scenarios, termed "counterfactuals," outlining what would have been most likely to happen in the absence of London 2012, must be modelled for comparison purposes.^{10,11} Unfortunately, the London 2012 legacy evaluation framework develops examples of only the most basic counterfactuals.¹² In fact, in two of four examples given, the framework outlines alternative scenarios in which there would be no alternative activity, indicating that there would be no opportunity cost. This suggests a limited understanding of how the principles of attribution and of additionality must be applied in practice.

Evidence provided in a systematic review for the Department of Health leads to the conclusion that detailed modelling of counterfactuals requires alternatives to be outlined in at least three broad areas.⁹ Firstly, a consideration of economic alternatives must model what would have happened to funding for intervention programmes without London 2012—would some or all of it remain within the relevant sector? Secondly, a consideration of alternative themes must outline what promotional messages would have been used as hooks to engage people in place of Olympic or Paralympic themes. Finally, alternative scenarios for support and enthusiasm must consider what political, practitioner, private sector, or third sector

(or combinations thereof) support and enthusiasm would exist for intervention programmes without London 2012. It is unlikely that free swimming would exist without London 2012, so the question is whether the funding would have been lost to the sector, or whether it would have been invested in other physical activity programmes, and if so for whom, with what emphasis, and with what levels of support and enthusiasm. Once these questions have been answered and an alternative scenario to free swimming established, its likely effects can be modelled from previous research. This provides a comparator case against which the effectiveness and cost effectiveness of free swimming can be measured, along with its true attributable and additional contribution to the physical activity legacy of 2012.

McCartney and colleagues conclude that how the costs of major multi-sports events can be justified in terms of health and socioeconomic benefits is unclear.⁵ Given that the legacy evaluation framework provides only a basic outline of how to apply in practice the concepts of attribution and of additionality to the opportunity costs,¹² the risk for the UK population is not that we will not get the benefits we want for our £150 a head investment in London 2012, but that there will be no robust evidence of what we have paid for.

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Improving the accuracy of predicting cardiovascular risk

QRISK2 supersedes Framingham as the risk prediction score of first choice

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Risk prediction tools are intended to help clinicians identify people at high risk of future disease events in whom more systematic use of preventive interventions is warranted. In formulating clinical recommendations and planning health services, professional groups and health authorities need to be aware of newly developed tools that more accurately stratify risk in the general population. In the linked study, Collins and Altman assess the performance of the QRISK2 score for predicting 10 year cardiovascular disease in an independent cohort of patients from general practice in the United Kingdom.¹ The authors also compare its performance with the version of the Framingham score previously recommended by the National Institute for Health and Clinical Excellence (NICE) and QRISK1.

Unfortunately, the use of risk prediction tools in routine clinical practice has a chequered history for several reasons. Firstly, the tools themselves are often poorly developed and inaccurate² or have not been subject to proper external validation.³ Secondly, they may be cumbersome to use or not readily accessible in busy practice settings.⁴ Thirdly, whether they alter clinical decision making and improve patient outcomes has rarely been evaluated,⁵ which leaves clinicians asking why they should bother using them if they do not add value to clinical management.

However, cardiovascular disease is a major cause of death and disability across the world, so better ways of identifying asymptomatic people at high risk who could

be targeted for aggressive prevention could yield big dividends in improved population health and productivity. Efforts to devise such methods are warranted given that clinicians' estimates of risk are often inaccurate, with a bias towards underestimating risk.⁶

To date, the Framingham equation (with some adjustments) has been the mainstay for predicting cardiovascular risk,⁷ but its limitations—having been derived from a middle class population in the United States in the context of what were considered to be risk factors and appropriate medical care more than 30 years ago—can no longer be ignored.

In response, in 2008 Hippisley-Cox and colleagues reported a new risk score, QRISK2. This score incorporates the traditional Framingham risk factors (age, sex, systolic blood pressure, smoking status, and lipid values) and, on the basis of new knowledge, adds in body mass index; family history of cardiovascular disease; social deprivation (Townsend score according to postcode); self assigned ethnicity; and conditions associated with cardiovascular risk, including type 2 diabetes, treated hypertension, rheumatoid arthritis, renal disease, and atrial fibrillation.⁸ These well defined variables, which are readily ascertainable in routine clinical practice, are entered into web based software (<http://www.qrisk.org>) that calculates the 10 year risk of cardiovascular disease. Collins and Altman have used data from more than 1.6 million people aged 35-74 years attending 382 general



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practices in the UK to subject this score to independent and external validation.¹

Compared with the adjusted Framingham risk score previously recommended by NICE, QRISK2 is better at discriminating between people who go on to have an event and those who do not (more so for women), explains a greater proportion of the variation in event rates, and more accurately estimates individual risks. The most important finding about QRISK2 is that it reclassifies between 43.0% of women and 45.4% of men previously deemed high risk ($\geq 20\%$ at 10 years) by NICE Framingham into a low risk category ($< 20\%$). Almost one in two people assessed as high risk using NICE Framingham (predicted mean risk 24-25%) were downgraded to low risk with QRISK2 (predicted mean risk 15%), with the observed 10 year risk being 13-14%. The correlation of predicted risk with observed risk across all levels of risk was much stronger for QRISK2, especially for men, than it was for NICE Framingham. This has major implications for clinical decision making. The validation study has some limitations—it excluded patients who were already using statins, and fewer than 20% of all included patients had complete data for all risk factors (with data on cholesterol levels being the most common omission). However, these limitations do not detract from its findings of greater accuracy of QRISK2 compared with NICE Framingham.

How might the use of QRISK2 affect clinical practice? A recent systematic review of the effects of providing information on the risk of coronary artery disease to asymptomatic adults found that people given this information perceived their risk more accurately and were more intent on taking preventive drugs if indicated.⁹ In particular, repeatedly providing patients with risk information and counselling was associated with modest reductions in

predicted risk (absolute risk reductions of 0.2-2%). Similarly, there is some evidence that prescription of anti-hypertensive and lipid lowering drugs, control of blood pressure, and uptake of physical exercise in patients at high risk can be enhanced (absolute increase 1.1-1.3%) by the use of risk scores in clinician-patient encounters, although an impact on event rates and patient outcomes has yet to be shown.¹⁰ It is hoped that the availability of more accurate clinically relevant tools such as QRISK2 may encourage their wider use and evaluation in proper impact studies. In the meantime NHS authorities should now consider QRISK2 as the risk prediction tool of choice for cardiovascular disease in UK populations.

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Balancing the intended and unintended effects of statins

When used according to guidelines, the benefits outweigh the risks

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In the past quarter of a century, statins—inhibitors of 3-hydroxy-3-methylglutaryl coenzyme A reductase—have become one of the most studied and prescribed classes of drugs in modern history. The efficacy of these drugs in reducing cardiovascular morbidity and mortality across a wide spectrum of risk is supported by an extensive dataset of randomised controlled trials,¹ which have been largely reassuring about the safety of currently available types of statin. However, safety data from trials are inherently incomplete, given the relatively short follow-up periods of clinical trials and their limited external validity. In the linked paper, Hippisley-Cox and Coupland used routinely collected data on more than two million men and women from 368 general practices in England and Wales to estimate the association of type, dose, and duration of statin use with the occurrence of several end points.² By using a large database they overcome the problem of the low incidence of statin associated adverse events, and they provide information on adverse effects that clinical tri-

als are not adequately powered to estimate. The analysis yielded several key observations.

Reassuringly, they found no significant association between statin use and the risk of Parkinson's disease, rheumatoid arthritis, venous thromboembolism, dementia, or osteoporotic fracture. The risk of a dose dependent rise in liver enzymes was increased for all statin users, which is consistent with reported clinical experience, although subgroup analysis showed a greater effect for specific statins. The rise in liver enzymes tended to occur within the first year of treatment. Statin users were more likely than controls to experience muscle related adverse events, such as myopathy, rhabdomyolysis, and raised concentrations of creatine phosphokinase. The risk was highest in the first year after starting treatment, was probably dose dependent, was consistent for all statins, and it seemed to persist for more than three years after stopping treatment. The authors also found an increased risk of cataract and acute renal failure among statin users,

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which was apparent within a year of starting treatment and returned to baseline within a year (for cataract) or one to three years (for renal failure) of discontinuing the drug. The only statistically significant association between statin use and cancer was for a lower risk of oesophageal cancer in statin users.

Hippisley-Cox and Coupland's study is an important addition to the existing literature on statins because it is the best available "real world" estimate of the risk of adverse events with statin treatment. The authors attempt to use their findings to estimate the risk-benefit ratio of statin treatment by calculating the number of "extra" statin related adverse events in their database compared with the number of cardiovascular events prevented by statins, estimated from trial evidence. When considering these risk-benefit ratios, however, it is important to recognise that the trial based efficacy estimate is a more accurate approximation of true effect size than the estimate of adverse event rates based on these observational data. Biases inherent in observational data may be impossible to adjust for. For example, ascertainment bias could contribute to the higher rates of adverse events noted in statin users, who would be more likely to have had laboratory tests during follow-up and to be older, with more comorbidity. Other potential confounding factors include clinical characteristics that were not measured or not adjusted for that could have explained some of the observed associations. For example, discouraging alcohol use in statin users to reduce anticipated liver toxicity may explain the lower risk of oesophageal cancer, and the use of anti-inflammatory drugs in older users of statins may have contributed to an increased risk of renal failure. Statin users typically take multiple drugs that may them-

selves cause adverse events, either directly or through interaction with statins.

For healthcare providers who prescribe statins, and their patients, the present findings are reassuring. Statin use is not associated with cancer, severe muscle toxicity is rare, and liver abnormalities seem to be reversible, which is consistent with analyses of trial data.^{1,3,4} Associations with renal failure and cataract are subject to the biases discussed above, and, if a causal association with statins is assumed and treatment is stopped, these side effects are reversible and may be considered an acceptable risk if the aim is to prevent an irreversible myocardial infarction or stroke. It would be wise to interpret the present observations in the context of the confirmed cardioprotective effects of statins and remind ourselves and our patients that these drugs, although considered safe, are, like any intervention in medicine, not entirely free of adverse events. We should neither overstate the size of the benefit of statins, nor exaggerate their side effects. Our understanding of the intended and unintended effects of statins is still incomplete and will continue to evolve. When used according to current guidelines, the benefits of statins outweigh their risks.

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Falling research in the NHS

A clear national strategy is needed to overcome local barriers to research



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The recently published Department of Health leaflet entitled *A Junior Doctor's Guide to the NHS* includes a statement that the director general of research and development is responsible for establishing the NHS as an international centre for research excellence.¹ Although medical research in the United Kingdom is clearly excellent and world leading, the NHS has underperformed as a leader in clinical research. In 2003 a government sponsored report described how the NHS would be transformed to produce high quality global clinical trials, giving the UK a competitive edge in clinical research.² Unfortunately, the UK's participation in global clinical trials dropped from 6% in 2002 to 2% by 2009.³ Other initiatives to support clinical research have also failed to stimulate research by NHS clinicians, including the establishment of the UK Clinical Research Collaboration in 2004, and the inauguration of the NHS research and development strategy under the auspices of the National Institute for Health Research in 2006.⁴

Despite concern that the introduction of the European Clinical Trials Directive might negatively influence

research,^{5,6} the directive has had a positive, albeit marginal, effect on the number of clinical trials conducted in the European Union, except in the UK, where the number is falling.⁷ So this directive cannot be blamed for the underperformance of the NHS in research.

Clinical trials are an indicator of the health of the nation's research, but they are not the only aspect of clinical research that has been affected. The National Research Ethics Service (NRES) confirms that the number of research applications fell from 9670 to 6321 between 2004 and 2010.⁸ So, clinical research into devices and techniques and any projects involving human subjects have also decreased. But why is this happening?

In part, the answer lies in the system of research governance in the UK, which despite attempts to harmonise and facilitate research has created barriers by allowing local trusts and other health organisations to create their own rules. Researchers may have to apply for multiple honorary contracts to carry out multicentre research. This may necessitate multiple CRB (Criminal Record Bureau) checks

Responses on bmj.com
“There is a ‘research passport’ scheme that streamlines procedures associated with issuing honorary research contracts or letters of access to researchers who have no contractual arrangements with NHS organisations.”

Kerina H Jones and Philip A Jones, Swansea University, United Kingdom

“...medical research in the United Kingdom was... world leading before local NHS research governance was introduced... Why not just abolish the recently created research governance infrastructure?”

Steve W Goodacre, University of Sheffield, United Kingdom

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and even multiple medical examinations; they may also have to follow local procedures, which often differ at each site, and all of this requires multiple submissions of the same research proposal.⁹

Another factor is the failure to develop a research culture and management targets. In 2002 the Strategic Learning and Research Advisory Group (established between the Department of Health and the Department for Education and Skills) attempted to stimulate research training and capacity, but this initiative failed.¹⁰ Consequently, doctors and other clinical staff have little support or time to conduct research.

Local NHS research and development governance imposes an additional hurdle to overcome once approval has been given by a research ethics committee and clinical trial authorisation has been obtained from the competent licensing authority. This additional hurdle has resulted in fewer new research projects being carried out and existing ones taking longer to complete, with the consequence that the competitiveness of UK researchers has been reduced and costs are rising in comparison with other countries.

The solution might be to standardise and streamline research governance in the same way as the NRES and the Medicines and Healthcare products Regulatory Agency (MHRA) have done. Currently under NRES, research ethics committees have to follow standard operating procedures, which include a requirement to complete their deliberations within 60 days of submission of any research project application; in parallel the MHRA has clear procedures and time lines.

A common application form, produced as part of the Integrated Research Application System (IRAS) is now used for a simultaneous application to a research ethics committee and to the MHRA, but NHS research and development governance is excluded from this procedure. The NHS research and development strategy under the auspices of the National Institute for Health Research, mentioned above, has failed to grasp the problem in governance that has arisen from the fragmentation of the NHS. This has meant that research ethics committees,

guided by the NRES, are under the administrative care of strategic health authorities, but individual NHS trusts are responsible for research and development governance. This has resulted in no single organisation being able to tell NHS trusts exactly what to do with an application to conduct a research project. Without proper guidance, the interpretation and implementation of clinical governance arrangements in research have put delivery of clinical targets above the delivery of research. Doctors have become demotivated about research, and major drug companies have started to withdraw clinical research from the UK.¹¹

There are signs that the National Institute for Health Research is taking action, by introducing systems designed to reduce bureaucracy and streamline governance.¹² However, effective implementation will depend on reducing local barriers to research by removing the ability of trusts to create their own local rules in addition to national systems.

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Health of indigenous peoples

Health systems must recognise culture and protect rights as well as needs

The health of the world's 350 million indigenous peoples continues to show that Western orthodoxy about health cannot be generalised. Although the health of many of these peoples has improved alongside advances in medicine, even in developed countries the health of indigenous peoples still falls short of the standards of other citizens. With the exception of rare and sometimes familial diseases, no systematic biological, physiological, or genetic causes for these persistent disadvantages seem to exist.¹

In spite of its 240 page length, the recently published United Nations' report on the state of the world's indigenous peoples provides a brief, contemporary, and cogent

overview of indigenous peoples today.² Ironically, it does not use any of the 7000 indigenous languages, but it does speak in terms once considered radical and now considered rational.

The state of indigenous peoples' health continues to cause concern. Poverty, conflict, dislocation, and powerlessness affect many of the world's indigenous peoples, manifesting themselves in high rates of maternal and child mortality, infectious diseases (tuberculosis, malaria, and HIV), and mental health illness. Indigenous peoples in developed countries may be better off in fundamental standards of living and terms of security—albeit at the

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Poor access to healthcare exacerbates the health risks for indigenous peoples

poorer end of the spectrum—yet cardiovascular illnesses, diabetes, mental health conditions, and premature mortality still prevail (in the United States, Canada, Australia, New Zealand, and the Pacific).

Epidemiology shows that health disparities exist between indigenous and non-indigenous populations in the incidence of almost all illnesses. Many of the widespread causes of mortality for children are preventable; it is the “methodology of access”—the design, philosophy, and implementation of health service access strategies—that needs attention. Of particular interest are areas where incidence is the same for both populations but morbidity and mortality are worse for indigenous people.^{3,4} Inappropriate clinical ethnic (or indigenous) profiling, where stereotypical assumptions may be wrongly made, and the frequent presence of comorbidities in indigenous peoples may explain inequalities in access to services.^{5,6}

Indigenous cultural views of health and wellbeing are remarkably consistent, although nuanced by language, practice over generations, and environmental influences. As the report states, “The indigenous concept of health articulates physical, mental, spiritual and emotional elements, from both individual and communal perspectives. It is shaped by indigenous peoples’ historical experiences and worldviews, and is expressed in the rules and norms that are applied in the [indigenous] community.”

Although lifestyle and profound choices, such as those exercised by the world’s nomadic tribes and by those who live in voluntary isolation, may expose indigenous peoples to poorer health outcomes (in Western terms), poor access to largely state run health systems exacerbates health risks for these people. These systems are culturally incongruent, dominated by Western philoso-

phy, and they force indigenous peoples to compromise or rationalise their cultural beliefs to access the best care. Such systems simply reproduce poor health outcomes by adopting a deficit approach—where underlying socioeconomic disparities are used to justify health disparities—and they often also result in a loss of cultural integrity for indigenous peoples.

Multicultural (the acknowledgment of different cultures), pluricultural (where specific traits of one culture are shared by other cultures), or bicultural (the existence of two main cultures, as in New Zealand) systems welcome and promote the presence of different cultures in society.⁷ Yet, according to the report, increasing recognition of diversity is not enough, because it fails to deliver equality.

Interculturalism goes beyond recognising different cultures to seeking exchange and reciprocity in a mutual relationship.⁸ Intercultural health initiatives combine traditional and Western medical practices within healthcare systems, and are preferably led by indigenous organisations.⁹ In New Zealand, for example, Maori health initiatives offer both Maori and mainstream (Western) health services.

Intercultural health systems not only improve the quality of health services for marginalised indigenous populations, but they also promote respect and solidarity within the context of national society. However, even this approach will fail if equitable resourcing does not follow identified needs.

The second UN Decade for Indigenous People began on 1 January 2005. This current UN report suggests that progress has slowed. The challenge for medical professionals and decision makers in health systems is to recognise that health is linked to rights as well as needs, and for indigenous peoples these rights extend to lands, natural resources, and the desire to conserve and practise traditional knowledge. Efforts within society that organise, protect, and advance these rights—in true partnership with indigenous peoples—will also have a positive effect on health. Furthermore, we should each inspect the “methodology of access” to the best standards of care for indigenous children, women, and people in general.

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