nourishment not only with attention to the particular micronutrient needs of patients on antiretroviral treatment, but also with special attention to local tastes and knowledge of food. An example is the use of guava to mitigate intestinal side-effects of antiretrovirals, thus having a positive effect on adherence.

Health information for all by 2015 is an admirable goal, as long as we realise that the health-knowledge space is not static, just as health needs are always evolving and vary by local conditions and behaviours. Establishing just what is essential health information for particular communities and making sure that information is not only evidence-based but usable, useful, and applied would be a great accomplishment. And this can only be done by breaking free of the linear relation between the developed world and the developing world, and recognising that knowledge systems must be true networks, locally owned and driven, with each point of equal value.

Ultimately in public health, we are moving from issues of access to knowledge, to the management and use of knowledge. This change may prove to have as positive an impact on peoples' health as evidence-based medicine had after its introduction 100 years ago.[8]

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Modelling the effect of screening for cervical cancer on the population

Public-health surveillance is a widely used method to assess the effect of interventions. In their book, *Monitoring the health of populations*,[1] Stroup and colleagues note that public-health policy is often based on analysis of disease trends within a population. Surveillance studies are important because the effectiveness of an intervention in the population can be quite different from its effect in a controlled setting, such as in a clinical trial. Moreover, when the results of clinical trials are unavailable to inform about an intervention that has been adopted, public-health surveillance can be an important approach for assessing its likely effect. Clinical trials in screening for prostate cancer by assay of prostate-specific antigen, for example, are ongoing. In the interim, inferences about the likely efficacy of such screening are based on mortality trends,[2–4] although difficulties in interpreting these trends have led to varied conclusions.

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Julian Peto and colleagues' study in this issue of *The Lancet* represents an application of public-health surveillance to assess the effectiveness of screening for cervical cancer. Although there is a broad consensus that screening prevents death caused by cervical cancer, these investigators are interested specifically in ascertaining the present and future effect of the UK national call-recall system—a screening programme set up in 1988.

Most surveillance analyses of cancer-control interventions face the challenge that the population under study has already adopted the intervention of interest. Peto and colleagues' analysis is no exception. Accordingly, identification of the effect of the intervention depends on projection of the outcomes that would have been observed in the absence of the intervention—ie, a projection of what disease-specific mortality would have been in the absence of the call-recall screening programme—and its subsequent comparison with observed mortality rates.

To project the mortality rate from cervical cancer in the absence of screening, Peto and colleagues adopt a widely used approach known as age-cohort modelling.[5,6] The researchers applied an age-cohort model to age-specific cervical-cancer mortality rates observed through 1987. Others have used this method to study mortality from cervical cancer,[7,8] although over different time intervals and with slightly different assumptions. The age-cohort model accounts for the increase in mortality in women born after 1932, caused by changes in presumed sexual behaviour and the spread of the human papillomavirus (HPV) during the 1960s and 1970s. Peto and colleagues then inflate the resulting projections to account for the fact that their data cover a calendar period during which opportunistic screening was taking place. The inflation factor is based on the assumed decline in mortality from cervical cancer due to screening before 1988.

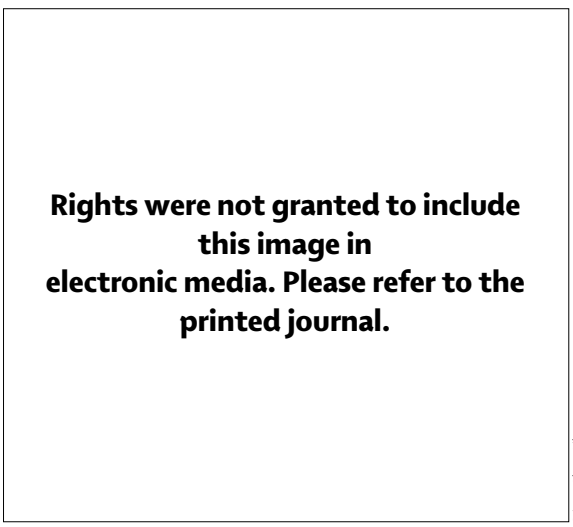
The validity of their model rests on three underlying assumptions—namely, that women born after 1952 have about the same lifetime risk as those born around 1952; that in the absence of screening, the age-specific risk of death from cervical cancer follows similar patterns in women born before and after 1952; and that by 1988, 40% of deaths from cervical cancer in young women were being prevented by screening. Based on these assumptions, Peto and colleagues conclude that the call-recall programme has had and will continue to have a great effect in the prevention of death from cervical cancer in the UK.

How does Peto and colleagues' model fit into the taxonomy of surveillance models proposed by Feuer et al,[9] in which a model's fit to surveillance data is classified as either shallow (empirically-based) or deep (biologically-based)? Shallow models generally focus on observable factors—eg, incidence and mortality—whereas deep models go beyond observable quantities, often modelling latent disease processes, such as onset and progression. Since the model described this week is based on mortality trends, it is a shallow model. By contrast, the model of cervical-cancer incidence and mortality described by Goldie et al[10] is a deep one. Their model consists of a series of transitions, from a healthy state to acquisition of HPV infection, to cervical disease and invasive cancer. By directly modelling the link between HPV infection and cervical cancer, Goldie et al project disease incidence and mortality in the absence of screening on the basis of information about the likelihood of infection with HPV.

What are the relative advantages and disadvantages of shallow and deep models? With respect to model structure, shallow models are generally much simpler and depend on fewer inputs than deep models. Peto and colleagues' model needs only data on historical disease-mortality trends and an estimate of the inflation factor to account for use of screening before 1988. By contrast, Goldie et al's model[10] depends on the input of data on many variables, including incidence of HPV infection, transition rates between various disease states, and disease-specific survival by stage at diagnosis. However, the modular nature of the inputs to the Goldie model makes them easier to interpret and, in some instances, to estimate than the inputs to the Peto model. For example, Peto and colleagues' inflation factor is itself a complex function of many factors, including the frequency of opportunistic screening before 1988 and rates of disease progression. Even if 40% of cervical cancers in women younger than age 40 were being prevented by 1978, translating this information into associated declines in mortality would depend on knowledge of disease progression and survival rates in the absence of screening. Whether, by 1983–87, this decline in incidence would induce a corresponding decline in mortality, as Peto and colleagues assume, is unknown. An indepth look at the process by which screening affects mortality is needed to confirm this assumption—in short, a deep model.

Empirically based shallow models are, hence, often simple in structure but tend to have complex inputs that can be difficult to quantify. Uncertainty in these inputs can be considerable and can lead to uncertainty about the results of the model. As the investigators note, the uncertainty of the estimated effect of screening for cervical cancer on the basis of the Peto model is difficult to quantify; the model needs to be validated. A biologically based model—a population version of the Goldie model,[10] for example—could be developed and the results compared with those of the Peto model. Similar results from both models would lend support to the findings of Peto and colleagues.

Funds for disease-control programmes are often reduced or withdrawn in response to success. However, just because a disease becomes rare does not mean that control of it should stop. It might rare because of the success of the programme being assessed. Modelling allows us to not only project the effect of an ongoing programme, but also to project what would happen to disease rates if that programme were terminated. If, as Peto and colleagues conclude, the population effect of the national screening programme is indeed great, then screening for cervical cancer should continue—even though it is no longer a major public-health issue in the UK—until a vaccine against HPV or some other means of primary prevention becomes available. However, if the screening programme is less successful, or the costs outweigh the benefits, funds for control of the disease might be more usefully directed elsewhere.



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Coloured transmission electron micrograph of human papillomaviruses on fractal background

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Programming obesity in childhood

Since the introduction of the first children's programmes in the 1950s, television has become the dominant pastime of youth throughout the industrialised world. A typical child in the USA watches television for 2·5 h each day,[1] exceeding time spent in vigorous physical activity by more than a factor of 10.[2] The fundamental transformation of children's lifestyle caused by television during the past half-century has long raised concern among mental-health experts, particularly with regard to violence.[3] More recently, the adverse effect of television viewing on physical wellbeing has been much discussed.

The first national study linking television viewing to childhood obesity was published in 1985.[4] There are now over 50 published articles on the topic in the medical literature. Cross-sectional epidemiological analyses have generally, although not always, shown direct independent associations between television viewing and measures of obesity;[5] sometimes, these associations were stronger than for any other obesity risk factor. Regional and nationally representative longitudinal studies suggest a causal relation.[4,6–8] Further evidence for causality comes from intervention studies; arguably, the only successful school-based obesity interventions have incorporated a focus on reducing television viewing.[9,10]

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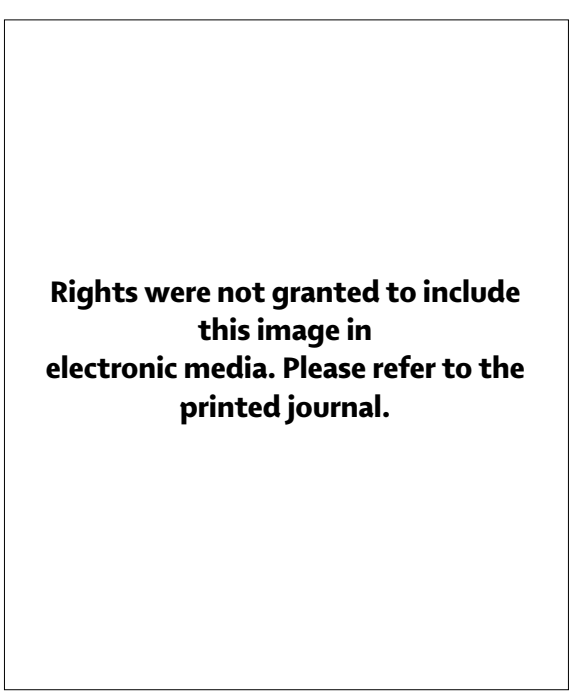
Television viewing has been proposed to cause weight gain through three main effects: by displacing physical activity, by depressing metabolic rate while watching television, or by adverse effects on diet quality, either while watching television or at other times, primarily due to food advertisements. Of these possibilities, the strongest evidence relates to diet. Each year, the food industry spends enormous sums of money advertising high-calorie poor-quality foods to children. Cartoons and other shows targeted at children broadcast food advertisements once every 5 min.[11] The behavioural effects of these advertisements have been extensively studied. As recently reviewed,[1,11] exposure of children to food advertisements increases the likelihood that they will choose the advertised products, that they will request those products from their parents, and that their parents will purchase those products. Exposure to food advertisements increases children's total energy intake significantly. Food advertisements have also been linked to overconsumption of fast food, sugar-sweetened soft drinks, and sweet and salty snacks, and to underconsumption of fruits and vegetables.



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One question that has received little attention is whether television viewing in childhood affects adult health. A study by Robert Hancox and colleagues in this week's *Lancet* aims to address this issue. The investigators prospectively examined about 1000 individuals in New Zealand from birth until age 26 years. They found that television viewing during childhood was independently associated with increased body-mass index, higher serum cholesterol, lower cardiopulmonary fitness, and cigarette smoking. Interestingly, child and adolescent television viewing remained a significant predictor of these adult endpoints even after adjustment for television viewing at age 21 years. A likely explanation for these findings is that dietary[12] and other lifestyle habits learned in childhood and influenced by television continue into adulthood.[13] Hancox and colleagues recognise two methodological issues: the possibility of residual confounding cannot be excluded and the validity of television measurements over time may differ. These limitations notwithstanding, the data indicate that television viewing in childhood has serious long-term consequences.

Ultimately, parents must reclaim from television the responsibility for educating and entertaining their young children. Admittedly, a parent working long hours, with inadequate daycare, and living in an unsafe neighbourhood may find it difficult to limit television time or deny requests for advertised junk foods. Fundamental socioeconomic changes may be needed before television viewing can be effectively reduced on a population basis.

In the meantime, the data presented by Hancox and colleagues strengthen the case for a ban on food advertisements