while leaving the unhealthy ones behind, or the result of some other factors.[14] One possibility is that it shows a lifecycle issue, with migrants benefiting from the positive health effects of migration (eg, access to better health care, reduced exposure to some types of communicable diseases), while the negative effects of migration (lifestyle diseases, some forms of workplace diseases) are lagging because they affect migrants much later in their lives.

If it is indeed a matter of lifecycles, the focus of research on migrant health in China should shift to trying to understand the entire lifecycle of rural migrants, including their changing exposures to various health risks, changes of cultural habits and of physical, social, and psychological environments, adaptations to their new conditions (both effective and suboptimum), and changes in their health knowledge, perceptions of risk, and expectations of health. To prevent an explosion of obesity and metabolic diseases in migration-receiving cities and countries in the near future, it is clear that any action must focus on the agency of the migrants themselves. Cultural habits that were positive or neutral in a rural context might become problematic after migration. It is easy to forget that in most traditional rural societies, lean and physically active did not connote health and attractiveness, but instead poverty, low status, and dependence on low-prestige manual labour—all undesirable social traits.[15] Similarly, weight loss was an alarming sign of deteriorating health, not a marker of fitness. Changing these perceptions to adapt to the health ideology of the cities and to a more modern conception of health risk will take time and understanding. Changing perceptions and habits is as difficult among migrants as it is for the rest of the population, but action must focus on understanding the origin of these unhealthy behaviours, their causes, and the reasons for their persistence or emergence.

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Preventing new-onset diabetes in thiazide-treated patients

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Although thiazide diuretics reduce blood pressure as effectively as other drugs,[1] one of the recognised disadvantages of drugs of this class is that they adversely affect lipid and glucose metabolism.[2] They cause a slight increase in serum LDL cholesterol and triglycerides, a decrease in serum HDL cholesterol, and an increase in blood glucose concentrations both in the fasting state and after a glucose load. The effects on blood glucose have raised special concern because data from clinical trials have shown that, when prolonged for years, thiazide-based treatment is associated with an increased incidence of type 2 diabetes compared with placebo or with drugs such as blockers of the renin–angiotensin system or calcium-channel blockers.[3,4]

Although in some studies drug-induced diabetes has not been associated with substantial modification of cardiovascular risk,[3] in others an increased risk of cardiac and cerebrovascular morbid or fatal events has been noted,[5] leading to the hypothesis that thiazides favour, perhaps in predisposed patients, the development of a so-called true diabetes—ie, diabetes with all the adverse clinical consequences. Some guidelines have therefore downgraded thiazides from their earlier position as first choice antihypertensives;[6] others have issued recommendations that discourage the use of thiazides in some categories of patients—eg, those with metabolic syndrome, in whom the risk of new-onset diabetes is substantially higher.[2,7]

Several possibilities have been investigated to determine whether and to what extent the adverse effects of thiazides on glucose metabolism can be attenuated. It is long established that the adverse effects of thiazides on blood glucose and other metabolic variables increase with increasing doses,[8] which is why the high diuretic doses used previously have been abandoned. Additionally, glucose metabolism might be better preserved if thiazides are given in combination with blockers of the renin–angiotensin system—perhaps because the protective effect of reducing the amount of (or blocking the effect of) angiotensin II offsets at least partly the adverse thiazide-dependent metabolic consequences.[8]

Another option to reduce the adverse effects of thiazides is to give potassium supplements, a strategy that stems from the observation that the hyperglycaemic effect of thiazide diuretics is enhanced by potassium depletion and inversely related to the serum potassium concentrations.[9] By extension, it is possible that the effect of thiazides on glucose metabolism would also be attenuated by giving them in combination with a potassium-sparing diuretic.

In an Article[10] by Morris J Brown and colleagues in *The Lancet Diabetes & Endocrinology*, the combination of a thiazide and potassium-sparing diuretic is shown to be therapeutically valid. Brown and colleagues randomly assigned several hundred patients with hypertension to 24 weeks double-blind treatment with hydrochlorothiazide (25 mg daily), amiloride (10 mg daily), or the combination of the two drugs at half doses (12·5 mg and 5 mg daily); all doses were doubled after 12 weeks. The investigators did 2 h oral glucose tolerance tests at baseline and at 12 and 24 weeks. Compared with baseline, the 2 h blood glucose concentrations increased in patients in the hydrochlorothiazide group and decreased in those taking amiloride, even when amiloride was taken together with hydroclorothiazide (difference between hydrochlorothiazide and combination groups: –0·42 mmol/L [95% CI –0·84 to –0·004]; 0·048).

Compared with thiazide monotherapy, blood pressure reduction (as shown by home blood pressure measurements) was not significantly different in the amiloride group and was slightly greater in the combination group. The number of patients with serious side-effects was small in all groups and the mean increase in serum potassium attributable to the potassium-sparing diuretic was negligible.

On the basis of these results, Brown and colleagues suggest inclusion of the combination of a low-dose thiazide diuretic with amiloride among the first-choice treatment strategies recommended for hypertension.[10] This suggestion is legitimate, because the world is facing an epidemic of diabetes, and use of diuretic-based treatments that do not have dysmetabolic effects could have far-reaching beneficial consequences. However, before this suggestion is incorporated into guidelines, further evidence is needed. A study with longer treatment duration than 24 weeks would be useful to ensure that the favourable metabolic effects of amiloride continue to prevail. Although Brown and colleagues argue that data about 2 h glucose concentrations can be extrapolated to accurately predict the risk of new-onset diabetes,[11] direct quantification of diabetes development in patients given a thiazide diuretic compared with those given a thiazide–amiloride combination is needed. Finally, and more generally, long-term studies are needed to clarify previous discrepancies and provide conclusive evidence that the diabetes induced by antihypertensive drugs (thiazides and β blockers)[3,4] has the same deleterious effects on macrovascular and microvascular disease as spontaneous diabetes to be sure that the effect of the hydrochlorothiazide–amiloride combination translates from a trial setting into real-life benefit for cardiovascular prevention for patients.

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Taking action on sugar

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On Oct 22, 2015, Public Health England (PHE) published its report[1] setting out the evidence for effective policies to reduce sugar intake on a population basis. The report followed those from the UK Department of Health's Scientific Advisory Committee on Nutrition (SACN)[2] and WHO,[3] both of which concluded that sugar has important effects on weight gain and dental caries, with the authors of the SACN report also noting that consumption of sugary soft drinks increase the risk of type 2 diabetes independently of the effect of sugar on weight gain. Both proposed limiting sugar to 5% of total dietary energy intake, either for the population (SACN[2]) or individually (WHO[3]), with SACN specifying that consumption of sugary drinks should be greatly reduced. The role of sugar in non-alcoholic fatty liver disease and other metabolic effects of fructose[4] were not considered.

In some people, especially those who are young and socially disadvantaged, sugar intake can be five times greater that the proposed 5% limit, partly accounting for the huge global burden of obesity[5] and dental caries[6] and the escalating incidence of diabetes and other non-communicable diseases in almost all countries.[7] The international consulting company McKinsey estimates that the economic cost of obesity worldwide is about US$2 trillion a year, equivalent to the cost of all warfare and terrorism.[8] There is also a huge oversupply of world sugar;[9] major plans for expansion of sugar production; falling world sugar prices; and transnational feed, food, advertising, and supermarket companies geared to selling this ever cheaper commodity in foods, snacks, and drinks for ever greater profits. Furthermore big free market developments threaten to make the situation worse. The European Union (EU) sugar price arrangements will end in 2017, allowing sugar to become even cheaper in Europe, and if the proposed EU–US international trade agreement goes ahead, fructose corn syrup could flood the EU market. In the Middle East and north Africa, countries have been subsidising rather than taxing sugar (and vegetable—usually palm—oil) for years,[10] thereby helping to promote their consumption and to induce the epidemics of obesity, diabetes, and other non-communicable diseases. In view of the expected global major escalation in obesity and diabetes, how can public health authorities tackle this seemingly overwhelming commercial drive?

In their report,[1] PHE concludes that although clinicians should advise patients on specific dietary changes, the pervasive industrial effects on consumer choices also need to be severely constrained. All forms of marketing influence food preferences, choices, and purchases for adults and children. Food retail price promotions are more widespread in Britain than elsewhere in Europe. Foods on promotion account for around 40% of all expenditure on food and drinks consumed at home. Higher sugar products are promoted more than other foods. Such promotions can increase purchases by a fifth, especially of sugary products, in all societal groups.[1] PHE therefore recommends a drastic reduction in sugary food promotions, as well as severe restrictions on marketing and sponsorship, but does not specify such measures