stimulation testing, and low concentrations of insulin-like growth factor (IGF)-1.[6] However, 81% of children with low GH concentrations in childhood were found to have normal values when retested after completion of puberty,[2] and no study has shown a difference in response to GH therapy between children with normal and those with subnormal GH concentrations.

As shown in the panel, treatment in the two studies that assessed final height was started at a much later age than in the studies that assessed height after 6 years. In the latter studies, most of the increase in height SDS occurred within 2 years, and the most impressive increment was seen in the continuously treated higher dose group. During the 6-year study period, skeletal maturity (bone age) advanced by between 7·1 to 7·6 years, and 46% of the children, now aged around 11 years, were in established puberty—not early for the normal population but arguably early for a cohort of short children. Thus, while it is to be hoped that these children will attain a more favourable adult height than their later-treated counterparts, it is possible that they will finish growing relatively early, thus "catching down" in terms of final height SDS. Long-term follow-up is required to settle this question, and the final height data of de Zegher and colleagues will be of the utmost interest.

To conclude, children of short stature who were small at birth are not a homogeneous group and include individuals who are naturally small because of genetic and constitutional factors. GH concentrations in these children are often subnormal but this feature does not seem to influence the response to treatment. GH therapy, when given from 5 years of age, results in an unequivocal improvement in height status mainly within 2 years but does cause some advance in bone age and may accelerate puberty. GH therapy started at age 10 does not increase the final height—at least when conventional doses are used.

In practical terms what treatment should be recommended? 2 years of GH treatment, at a dose of about 20 U/m^2 per day in selected children aged 4–7 years with marked short stature associated with smallness at birth is justified if the shortness is causing distress. There is, as yet, no evidence that a long course of GH therapy improves final height, and the results of current studies should be awaited before this approach can be recommended.

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1 Zucchini S, Cacciari E, Balsamo A, et al. Final height of short subjects of low birth weight with and without growth hormone treatment. *Arch Dis Child* 2001; **84**: 340–43.
2 Coutant R, Carel J-C, Letrait M, et al. Short stature associated with intrauterine growth retardation: final height of untreated and growth hormone-treated children. *J Clin Endocrinol Metab* 1998; **83**: 1070–74.
3 de Zegher F, Albertsson-Wikland K, Wollman HA, et al. Growth hormone treatment of short children born small for gestational age: growth responses with continuous and discontinuous regimens over 6 years. *J Clin Endocrinol Metab* 2000; **85**: 2816–21.
4 Karlberg J, Albertsson-Wikland K. Growth in full-term small-for-gestational-age infants: from birth to final height. *Pediatr Res* 1995; **38**: 733–39.
5 de Zegher F, Kimpen J, Raus J, Vanderschueren-Lodeweyckx M. Hypersomatotropism in the dysmature infant at term and preterm birth. *Biol Neonate* 1990; **58**: 188–91.
6 de Waal WJ, Hokken-Koelega AC, Stijnen T, de Muinck Keizer-Schrama SM, Drop SL. Endogenous and stimulated GH secretion, urinary GH excretion, and plasma IGF-I and IGF-II levels in prepubertal children with short stature after intrauterine growth retardation. The Dutch Working Group on Growth Hormone. *Clin Endocrinol (Oxf)* 1994; **41**: 621–30.

Hot foods—unexpected help with energy balance?

Urbanisation has often been blamed for the epidemic of obesity (and abdominal obesity), yet in some highly urbanised societies, such as Japan, Korea, and Hong Kong, the prevalence of obesity (BMI >30 kg/m^2) is as low as 3%, whereas in most western countries it is well over 10%.[1] Why? Although physical inactivity and the consumption of foods of high energy density are major contributors to this difference, other factors must be involved. Could certain food cultures provide protection against obesity?

Most individuals maintain a constant bodyweight and body composition by balancing energy intake and energy expenditure. The principal components of energy expenditure are the basal metabolic rate and physical activity, but food ingestion also has a thermic (energy-expending) effect, which may alter the efficiency of energy expenditure. Some foods have been shown or are thought to have an effect, by different mechanisms, on energy intake or energy expenditure. Food factors could alter energy intake by suppressing or stimulating appetite or by increasing or decreasing energy expenditure. Thus a change in quality as well as quantity of food may be of benefit in achieving energy balance.

The provision of a wide range of foods can stimulate appetite without leading to an increase in energy intake, and it can be associated with lower body fatness, provided that the foods have low energy density.[2] Food intake can be affected by changes in the organoleptic properties of food (ie, the taste, smell, and texture of foods) and even by the sound of chewing. Deficiencies of micronutrients, especially thiamine and zinc, can impair appetite and contribute to anorexia, an effect that suggests involvement of the central nervous system at cortical or subcortical levels, notably at the hypothalamus and pituitary. Some foods have anorectic properties that have been attributed to certain ingredients—for example, xanthines in tea and coffee, theobromine in cocoa, small peptides with opioid activity derived from gluten, β-casein, and molecules produced by roasting or brewing coffee.[3] And dietary supplementation with garlic (*Allium sativum*) for 12 weeks was shown to reduce fat and carbohydrate intakes in patients with mild or moderate hypercholesterolaemia.[4]

The choice of food may also affect energy expenditure. Capsaicin found in chilli (*Capsicum frutescens*) or red pepper (*Capsicum annuum*) may increase thermogenesis, mainly through effects on the vasculature.[5,6] Caffeine, which stimulates the central nervous system and heart, also results in an increase in energy expenditure. The combined effect of these two substances on energy balance has been investigated by Mayumi Yoshioka and colleagues.[7] Participants in their study, who were given free access to food, took both red pepper (Saemaul Kongjang) and caffeine, or neither (control diet), with some meals. Red pepper and caffeine were used in amounts that were within usual patterns of consumption. The researchers found that consumption of red pepper and caffeine was associated with a 4000 kJ per day reduction in energy balance; and, surprisingly, most of the difference was due to a reduction in energy intake rather than to expenditure. Precaution should, however, be taken with the ingestion of large doses of caffeine present in certain foods and diet pills, since this compound may induce cardiac arrhythmia.[8]

There is now enough evidence to justify definitive intervention studies that test whether specific foods, or food patterns, in countries with low prevalence of obesity

can favourably affect energy balance. It may well be that minor food components have synergistic and cumulative benefits in the maintenance of a healthy degree of body fatness.

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1 World Health Organization. Obesity. Preventing and managing the global epidemic. Report of a WHO Consultation on obesity. WHO: Geneva, 1998.

2 McCrory MA, Fuss PJ, McCallum JE, et al. Dietary variety within food groups: association with energy intake and body fatness in men and women. *Am J Clin Nutr* 1999; **69:** 440–47.

3 Wahlqvist ML. Functional foods in the control of obesity. In: Goldberg I, ed. Functional foods: designer foods, pharmafoods, nutraceuticals. New York: Chapman & Hall, 1994: 71–86.

4 Kannar D, Wattanapenpaiboon N, Savige GS, Wahlqvist ML. Hypocholesterolemic effect of an enteric-coated garlic supplement. *J Am Coll Nutr* 2001; **20:** 225–31.

5 Henry CJ, Emery B. Effect of spiced food on metabolic rate. *Hum Nutr Clin Nutr* 1986; **40:** 165–68.

6 Colquhoun EQ, Eldershaw TP, Bennett KL, Hall JL, Dora KA, Clark MG. Functional and metabolic evidence for two different vanilloid (VN1 and VN2) receptors in perfused rat hindlimb. *Life Sci* 1995; **57:** 91–102.

7 Yoshioka M, Doucet E, Drapeau V, Dionne I, Tremblay A. Combined effects of red pepper and caffeine consumption on 24 h energy balance in subjects given free access to foods. *Br J Nutr* 2001; **85:** 203–11.

8 Cannon ME, Cooke CT, McCarthy JS. Caffeine-induced cardiac arrhythmia: an unrecognised danger of healthfood products. *Med J Aust* 2001; **174:** 520–21.

Detention and mandatory treatment for tuberculosis patients in Russia

> "The only purpose for which power can be rightfully exercised over any member of a civilised community, against his will, is to prevent harm to others. His own good, either physical or moral, is not a sufficient warrant."—John Stuart Mill, *On Liberty*, 1859.

Over the past decade rates of tuberculosis in Russia have more than doubled,[1] and although this country accounts for 17% of the population of WHO's European region, it has a third of Europe's cases of tuberculosis. Since some Russian oblasts have among the highest rates of multidrug-resistant tuberculosis in the world, in all likelihood Russia's contribution to the prevalence of such types of tuberculosis is considerably more than a third.[2]

Tuberculosis affects the most marginalised in society. Rates of tuberculosis in prisoners in Russia, for example, are approximately a hundred times those of the general population.[3] Indeed, prisons have been graphically termed the epidemiological pump[4] of the Russian tuberculosis epidemic. The three important factors contributing to this pump are the conditions under which prisoners (and those in pre-trial detention centres) are held, the lack of resources for effective therapy, and the poor coordination between the vertical prison and civilian and other (such as military) health-service systems. Amnesties, such as the release of nearly 350 000 prisoners in March, 2001, put stress on efforts to coordinate health services, and the convergence of the HIV epidemic on these marginalised populations threatens to further challenge efforts to control tuberculosis.

Because of its historical, cultural, and geopolitical position, tuberculosis-control policies in Russia may influence strategic approaches to control of the disease elsewhere. On the June 6, 2001, the Russian Federal Council adopted a law, supported by WHO and the Council of Europe, on preventing dissemination of tuberculosis. It provides a legal framework for public policy in the prevention, diagnosis, and treatment of the disease and the rehabilitation of patients who have or have had tuberculosis. It also provides a structure for statistical monitoring, financing services, and occupational and social support, as well as details of individual and state responsibilities. The law makes clear that care will be free, and it highlights the fact that tuberculosis control is a government priority. There is little in the law, however, to encourage a formal shift from inpatient sanatoria-based care to ambulatory care based on WHO's DOTS strategy. Nor is there much to enhance the coordination of services for those moving between systems (eg, prison and civilian) or to promote the development of integrated health-care and social-support structures.

In response to the potential public-health threat posed by those with tuberculosis, the law provides the state with the authority to detain for up to 6 months in sanatoria individuals who do not comply with screening, diagnostic, or therapeutic regimens. Whether such authority covers surgical treatment, which is quite widely practised, is unclear. The law also stipulates, in its article on tuberculosis patients' rights, provision of legal advice.

The article relating to detention and mandatory treatment raises several important issues. First, in view of the impact on the epidemiology of tuberculosis that high incarceration rates and conditions within prisons and lack of linkages between the vertical health care structures seem to have, the attention being placed on "downstream" remedies may be premature. There is a concern, therefore, that this article in the law may provide a smokescreen for poor coordination of services and deficiencies in the criminal justice system.[5]

Second, in the years since WHO called tuberculosis a global epidemic, the use of coercive public-health practices to constrain those perceived to be posing a public-health threat may be increasing. The 1990s saw Norway adopt legislation similar to the new Russian one;[6] detention of individuals with tuberculosis in England rose during the past decade;[7] laws were adopted and applied across the USA to detain non-compliant, non-infectious individuals since the early 1990s;[8–11] and prisons rather than hospitals have been used as detention centres for individuals with tuberculosis in countries such as the USA[9] and Israel (unpublished) in recent years. Despite loud calls this decade for an evidence-based approach to health-care policy, the evidence to support sanctions such as detention and mandatory treatment is scant if not non-existent.

The third issue is that, perhaps in their haste to support reforms to tuberculosis control in Russia, WHO and the Council of Europe, both of which advocate observance of human rights, did not consider the potential ramifications that support for such articles in a law conveys, enhancing as it does the power of the state and limiting the freedom of vulnerable individuals. WHO Director-General Gro Harlem Brundtland has argued that the key values enshrined in human-rights legislation should inform health reform.[12] According to the 1984 Siracusa Principles, a set of principles under which departure from the 1966 International Covenant on Civil and Political Rights is recognised internationally, any restriction must be in accordance with the law, legitimate, and necessary, and the action must be the least restrictive alternative that is reasonably available, and its application must not be discriminatory. In essence, the Siracusa Principles expand upon and define more clearly Mill's "harm principle" that provides the ethical foundation for determining whether public-health programmes that involve coercive elements