The incidence of type 1 diabetes has been steadily increasing worldwide since the middle of the 20th century. The increase has so far been linear and predictable. However, in today’s Lancet Valma Harjutsalo and colleagues show that in Finland, which has the highest incidence of type 1 diabetes in the world, the incidence predicted for 2010 on the basis of data from the 1950s to the 1980s was exceeded by 2000.

A changing environment, infant and maternal diets in particular, would seem to be the most likely explanation for this alarming acceleration. Monitoring of the appearance of autoantibodies associated with underlying autoimmune destruction of islet β cells in genetically predisposed infants has identified several inconsistent dietary associations. Some studies have shown positive associations between diabetes and duration of breastfeeding and early introduction of cow’s milk or cereals; others have found no effect of cow’s milk or that cereals are protective. This inconsistency may reflect variations between populations or proxy effects, in which dietary habits reflect changes in other, as yet unidentified, factors.

An area so far neglected is the importance of intrauterine factors and their effect on early development. Placental transmission of viruses leading to type 1 diabetes (eg, rubella) is widely recognised, perhaps cereals, food toxins, and enteroviruses could similarly trigger islet autoimmunity through intrauterine exposure during pregnancy. A recent study of maternal diet during pregnancy found that of all the factors, only maternal consumption of root vegetables was associated with diabetes autoantibodies in offspring. Whereas consumption of root vegetables in infancy was associated with an increase in autoantibodies, their consumption during pregnancy was associated with delayed time to appearance of diabetes autoantibodies in offspring.

So what could the association with root vegetables mean? The answer may lie not in the food itself but with toxic contaminants. A class of Streptomyces toxins (plecomacrolics) can accelerate onset of autoimmune diabetes in the offspring of exposed NOD mice, which
shows that intrauterine environmental exposure can influence subsequent disease development. Species of Streptomyces infect potatoes and other root vegetables to cause common scab disease wherever root vegetables are grown. The toxins produced contaminate the vegetables and their products. Not only does the country have the highest incidence of type 1 diabetes, but in Finland 16% of infants also receive root vegetables exclusively as their first solid food, and a further 16% receive root vegetables with other food groups. Common potato scab is becoming more difficult to control because of increasing diversity of the causative organisms, thereby further increasing risk of population exposure to plecomacrolide toxins. The association of type 1 diabetes autoimmunity with root vegetables may reflect exposure of Finnish children to a higher burden of Streptomyces toxins, but this might not be restricted to Finland. These toxins could accumulate through the food chain and so contaminate milk, meat, and other products. Also, the industry practice of using vegetable peel and reject tubers to make vegetable by-products, such as starch and thickeners, increases the risk of exposure. Airborne microbial spores might also be a route of exposure. Causes of the increasing incidence other than Streptomyces are clearly possible.

The case for an environmental contribution to the cause of type 1 diabetes is compelling. However, identification of the probable agents is the ultimate challenge because they might be ubiquitous, and the hardest cause of a disease to identify is that which is universally present. There seems little doubt that we have underestimated the complexity of this form of diabetes—new ideas are clearly needed to stop the disconcerting acceleration of incidence.

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Lifestyle intervention, diabetes, and cardiovascular disease

Primary prevention of type 2 diabetes was first proposed in 1921 by Elliot Joslin who wrote: “There are entirely too many diabetic patients in the country...Therefore, it is proper at the present time to devote attention not alone to treatment, but still more, as in the campaign against the typhoid fever, to prevention. The results may not be quite so striking or as immediate, but they are sure to come and to be important.”

Almost 90 years later, we can still subscribe to Joslin’s view. Indeed, hard evidence from several trials unequivocally shows that lifestyle intervention can prevent or at least delay type 2 diabetes in individuals with impaired glucose tolerance. The effect is evident and of a similar magnitude in people from differing ethnic backgrounds and cultures.

In today’s *Lancet*, Guangwei Li and colleagues report 20-year follow-up results on incidence of type 2 diabetes and cardiovascular disease from the China Da Qing Diabetes Prevention Study. Originally, 33 clinics were randomly allocated to provide the participants (577 men and women with impaired glucose tolerance) with dietary, exercise, combined, or control interventions. After 6 years, diabetes incidence was 31%, 46%, or 42% lower than in the control group for dietary, exercise, and combined interventions, respectively. The researchers managed to follow-up...