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## Poliomyelitis: The Role of Diet in the Development of the Disease

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### Introduction

Poliomyelitis is an acute disease of the nervous system which can result in paralysis. The accepted dogma is that polio is a communicable disease, and that the infectious agent is the poliovirus, spreading mainly by excretion in the faeces and infection by mouth. Although much has been learned of the structure and replication of the poliovirus from *in vitro* studies, it has proved difficult to correlate any of these observations with *in vivo* processes; indeed, our understanding of the pathogenesis of polio has progressed surprisingly slowly over the past 30 years (1). Experts have explained this lack of progress by invoking the complexity of neurovirulence, a phenomenon thought to depend on the interactions of host and virus at many levels.

Recovery from polio apparently leaves a permanent immunity against further infection, and this has been exploited in vaccination programmes. The success of vaccination in the developed world has indeed seemed great; the number of cases in the US had fallen from 30,000 in 1955 to a total of 110 in the 5 years between 1969 and 1974. Despite this, polio is still an important disease, since, in the Third World, vaccination has not been accompanied by such a fall in the incidence of polio; only 15% of cases are detected and reported (2); the estimated number of paralytic cases in 1989 was 246,000 (3).

The importance of polio, and our failure to understand how the poliovirus causes it, should make us keep an open mind about its aetiology; but there are a number of anomalies in the accepted picture which should lead us to look for other influences which may be at work. Whilst I do not deny the existence of the virus, I believe that the evidence casts doubt on its accepted role as the only agent causing the disease. Indeed, I find it possible to take the heretical view that polio may not be an infectious disease at all, and that other elements of nutrition are the primary cause (4).

### *Anomalies in the history of polio*

*Anomalies in vaccination and contagion.* While it is true that epidemics have often stopped short after mass vaccinations, it is not possible to say what would have happened if these measures had not been taken; after all, every epidemic stops eventually. The results of studies of the effects of vaccination on immune status and polio incidence are confusing. Blind experiments are difficult to control, since, in the case of the Sabin vaccine, which has been the most commonly used since the 1960s, the vaccination state of one group of individuals affects those of others, by the transfer of attenuated live viruses. In addition, failure of immunisation, which has already been alluded

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to, has not yet been properly explained. In 1974 the WHO's only explanation for the poor response they saw to oral vaccines in Africa (5), was that there was some unidentified substance in the saliva or in the milk of the mothers of the subjects which was inhibiting viral multiplication.

Many times in the history of polio contagion was expected, but did not occur. Gatherings took place during epidemics, and no increase in disseminated cases was seen. In 1941, 3,600 boys from all over Switzerland gathered in Zurich for the National Youth Shooting Contest; at the time a severe epidemic was raging, but none of the boys nor their contacts fell ill. Similar circumstances, and the same 'gratifying but surprising outcome' prevailed during the Royal Visit to Western Australia in 1955 (6). Such gatherings certainly influenced the antibody titre in some of the participants, but did not seem to affect the number of cases. This is in marked contrast with our experience of other infectious diseases—what sensible person would have allowed a mass gathering during an epidemic of small-pox or typhus?

Other evidence of this 'reluctant virus' can be found in accounts of some of the major epidemics. During the New York epidemic of 1916 'no attendant, no physician, nurse or domestic assistant and no patient admitted to any of the hospitals for causes other than poliomyelitis during the epidemic, contracted polio' (7). Similar observations were made of the 1952 epidemic in Denmark (8).

The American clinician Wehrle (9) found no signs of polio infection among the staff of hospitals for communicable diseases, despite their being heavily exposed to virus from incontinent patients. Similarly, 73% of infants in Dehli were found to have no antibodies against polio (10), what is 'most astonishing—is that one can be healthy and happy without any antibody against polio—even in a country where polio is endemic'.

*A disease of the fit and well-fed.* The current explanation for the high incidence of polio after World War II is that the population was vulnerable because of lack of food. This seems unlikely, as there is a large body of evidence to show that polio preferentially strikes the strong and well-fed (11). This was noted as long ago as 1823 by John Shaw, a London surgeon, in his study of infantile paralysis (12), and Heine (13), wrote in 1840 of the good general health and strong 'blooming' constitutions of patients before the illness struck. An only child is more likely to be affected than one who has many siblings, and would therefore be expected to be less well nourished. In the Mau-

ritius epidemic of 1945, Chinese children were more severely affected than Creole and Indian children, despite having a more abundant diet (14).

*The shift from a sporadic to an epidemic disease.* Albert Sabin, writing in 1947, introduces another puzzle (15): 'No circumstance in the history of polio is so baffling as its change during the past fifty or sixty years from a sporadic to an epidemic disease. The greatest frequency and severity is in the very countries in which sanitation and hygiene have made their greatest advances.' The current opinion is that a high level of development, and the associated good sanitation, causes a delay in viral infection and induced immunity and therefore an increased incidence of polio epidemics. But this is not the case with other infectious diseases; might not some other factor be at work here?

#### *Sugar consumption as an explanation*

In my studies of polio epidemics and my search for a triggering factor, I came to the conclusion that, in each case, an epidemic could be illuminated in terms of sugar consumption. My attention was first turned towards sugar during my own polio attack, when I developed an aversion to it: this experience was shared by other sufferers, who were generally people with a sweet tooth. As I looked at examples of epidemics, the evidence for a link with certain sets of conditions of sugar manufacture and consumption became clear. In addition, if sugar were causally linked to polio, this would help to explain the anomalies outlined above: for instance, the well-nourished victims of polio are those whose diets are likely to be rich in sugar; and the change from a sporadic to an epidemic disease corresponds with the world-wide increase in the consumption of refined sugar. The accepted explanation, that the incidence of epidemics has to do with a high level of development, overlooks the fact that this very development goes together with a high intake of refined sugar.

*Sugar manufacture.* The successful establishment of a new cane—or beet-sugar industry is a tricky business. The basic problem is the need to plan ahead, while at the same time being faced with a number of imponderable factors, chiefly the weather. Farmers must be persuaded, for instance, to grow beet rather than more reliable crops, and if a regular supply of a certain amount of beets extending over a period of several years cannot be ensured, then disaster threatens, and

whole factories might have to be moved for lack of beets.

Sugar refining has long been considered an art, and its principles have changed little since it was first practised. The juice of cane or beet is clarified, filtered, and crystallised in evaporators. Centrifugation of the viscous mass separates the crystals from the adherent syrup, and produces the 'A' sugar. Subsequent crystallisations produce a less white sugar, then brown sugar, and finally molasses, the brown syrup which cannot be crystallised by ordinary means. In normal operation the primary product of the factory is 'A' sugar; the less valuable sugars are dissolved and reprocessed, lime and sulphuric acid first being added to bleach and then neutralise them. The market price of the 'A' sugar determines whether, and to what extent, this recycling is carried out. If the price is high, recycling is intensive, and molasses may also be desugared by precipitation with quicklime.

It often happens that the juice fails to clarify, and impurities remain in suspension; cane is more prone to such problems than beet. Typical causes are drought, disease, or attack by the cane borer, or under- or over-maturity. Beet give problems if they have been affected by the Yellow-Leaf Virus, or are decayed or frozen before harvesting. Clarification today is much less of a problem than it used to be, since the introduction of Bentonite, a clay alum colloid, which, though expensive, is a very good settling agent (16). With Bentonite (specifically labelled 'for frozen beet or deteriorated cane') refractory juices could be clarified without recycling or other treatments.

Other chemicals, such as anti-foaming agents, bleaches, and scale-preventors, are also necessary for the profitable operation of a sugar industry; fungicides and disinfectants, too, are used in large amounts to prevent fermentation. Under such harsh treatment, it would be surprising if some proportion of refined sugar were not changed into something other than pure sucrose. In fact, reducing sugars are converted into non-fermentable reducing substances (deoxysugars), during the last boilings and recyclings of cane juice in particular. Some organic acids, originating from the cane or the beet, precipitate during crystallisation, and form salts such as oxalates and aconitates.

Originally, the refined product was bagged at the factory, whereas now the sugar is carried by conveyor belts into vast silos. So, in former days, any batches which contained, for example, high concentrations of deoxysugars, would stay together, whereas now they are thoroughly mixed during bulk-handling, before being bagged for retail elsewhere.

#### *Sugar consumption and the link with polio*

*i) The developed world.* In a community with an average per capita consumption of sugar of 50kg per year, one age group in particular, that between 5 and 25 years old, has an annual consumption of over 100kg. Sugar consumption peaks during the summer months, and about 40% of the annual total is consumed between May and August. It is the 5–25 year-old age group which is also the most vulnerable to polio, and it is at the end of the summer that the disease is most prone to strike.

In times of shortage a nation's sugar stock may fall to an amount sufficient for one week or less, and refining is intensified as prices rise. This was particularly the case after World War II. During the First War, the production of beet-sugar alone was affected, whereas in the Second the cane-sugar industries of Indonesia and the Phillipines were hit as well. So, world production, which had been 10% short in 1918, was 25–30% below the pre-war level by the end of 1946. On top of this, demand was rising fast in countries such as Brazil and India. So these were years of production at any price, with the desugaring of the normal end-products of the refining process. And these, too were crucial years in the history of polio, with epidemics cropping up where they had never been before: in 1947 we see a severe epidemic in Great Britain, and in Austria the most serious outbreak in her history; the year 1952 was particularly notorious, with many victims in Germany, Denmark, Holland and the USA. After 1952 the incidence of polio tapered off; this occurred before any vaccination against the disease was practised, and coincided with a fall in the production pressure within the sugar industry. Two changes in the manufacture and handling of sugar could also have had an influence, the first of these was the introduction of bulk-handling, which has been discussed above, and the second was the use of activated carbon and Bentonite as clarifying agents.

If the sugar stocks of a country become very low, they are adjusted by an import, which I term 'complementary sugar'. In some part of the world there is always sugar ready for export, which can be delivered within a fortnight. This comparatively small quantity is recently refined, almost invariably bagged, and consumed immediately, for these reasons I believe it to be the most dangerous as a triggering factor in the development of polio. In the Northern Hemisphere, such shortages occur just before the sugar from the new harvest becomes available in September, which

is also the time when most epidemics peaked in this part of the world.

ii) *The developing world.* The developing world was traditionally considered the domain of the 'Stille Feiung', or silent infection; polio was endemic, with a few cases dispersed throughout the year, but no major epidemics. It was said that the people were infected at an early age due to the primitive sanitation, and that this infection occurred silently, without clinical evidence. Why these infections stayed so silent was never really explained.

After World War II, the picture changed radically. Epidemics occurred throughout the Third World, and the era of the 'Stille Feiung' was clearly over. At the same time a revolution was under way on the sugar front, as new industries were established everywhere. Governments of the newly independent states, though unable to fulfil all the needs of their people, could at least satisfy the demand for sugar, the first consumption commodity. For the villager, sugar was cheaply available, gave instant satisfaction, and could even keep the pangs of hunger at bay. In addition, a domestic sugar industry was a convenient object for taxation, and saved precious foreign currency. Thus the production pressure was within, rather than between countries.

As a consequence, sugar consumption shot up. In India it doubled, before the war India had been a sugar exporter, but by 1954, despite increased production, 800,000 tons had to be imported for home consumption. Between 1948 and 1958, world output rose by an average of 8% per annum.

There are a number of differences in sugar production between the developed nations and the Third World, which I consider important factors in the changing epidemiology of polio, and which, for an advocate of the poliovirus, may help to explain the comparative failure of vaccination seen by the WHO in its surveys of Africa. The most obvious difference is the sugar crop; in the West this is almost exclusively beet, while cane—the more difficult starting material—is the source in the poorer countries of the Southern Hemisphere. It is in cane in particular that the transformation of reducing sugars into the non-fermentable deoxy-sugars occurs (17), and this difference between cane and beet may explain the striking discrepancy between the incidence of polio in the West and the Third World, where the disease has persisted despite vaccination campaigns. Also manufacturers were much less likely to try to solve clarification problems with expensive agents such as Bentonite, preferring phosphoric acid, recycling and

desugarising. The last distinction is that the developing world failed to adopt bulk-handling methods; the equipment is expensive, so bulk-handling is economic only when large quantities of sugar are being handled where the saving of labour is important, so most developing nations will keep to the old, labour intensive method of bagging. Because of these factors, as the new factories sprung up, so new epidemics of polio arose. A huge number of cases were reported, but these figures represent less than half the real number; most parents could not afford to bring their children to hospital regularly, and only the clearly paralytic cases were notified.

#### *Further supporting evidence*

*Epidemics and intensive refining.* Before the Great War, Germany produced most of Europe's sugar, the great 'loess' sugar belt in Lower Saxony yielding 2.5 million tons a year. Of the other European nations, only France was independent, with beet fields in the North, and the Isle of Reunion for imports of raw sugar. To maintain the German industry, a vast and regular supply of beets was essential, and in 1911 a hot and dry summer cut this supply drastically. Output fell to 1.5 million tons; the Kaiser ordered that contracts be fulfilled, despite the poor harvest, so the refineries recycled again and again to squeeze the last crystals out of the molasses. But in the winter of the same year, epidemics arose over Europe. Even in the remote settlements of Norway children fell ill with seizures. In England, too, acute polio struck; in Stowmarket, with a population of only 275, 45 children were affected. The highest incidence in Germany herself was in the sugar-refining town of Braunschweig. In 1912, Germany's friends in South America began to suffer too. Only France, with her near normal output, seemed immune, and had fewer cases in 1911 than in the year before.

25 years later, under the policy of 'Guns before Butter', no imports of sugar were allowed into Germany, and so from 1933 the domestic industry pushed production to the limits. The beet harvest of 1936 was very poor, and despite the acquisition of 22 refineries in the newly annexed territories, sugar output was low. Intensive refining was stepped up in 1937/8, and in 1938 a major epidemic took place, with 5750 cases. A similar series of events occurred in Italy, who was following the same policies as her ally, and was followed by 6000 cases of polio in 1939.

After the war the recovery of the German industry was well under way, when in 1952 Yellow-Leaf Virus struck, cutting the yield by 30%, and also severely

affecting the Danish harvest. The virus reduces the amount of sugar in the beet and gives rise to by-products of abnormal metabolism. Infected beet decompose faster than healthy ones, and so processing and filtration are made difficult. After the austerity of the war, German demand was increasing. World sugar prices were high that year, so the refiners managed to produce 4000 tons of refined sugar by desugarising molasses. In the summer of 1952, a severe polio epidemic arose, with 9000 cases, spreading into Holland and Belgium. In Denmark, too, the Yellow-Leaf Virus was followed by polio, with an epidemic of 4000 cases in Copenhagen.

The New York epidemic of 1916 provides another good example: after failure of the Cuban harvest of 1915, and the rumours of American participation in the War, the price of raw sugar rose by 30%. The recycling and desugarising began, and, as the summer arrived, with it came a disease which paralysed the children. The doctors were helpless — this was the dreaded European disease they had heard of. Wealthy New Yorkers fled the city with their children, only to find that they fell ill at the summer resorts, or shortly after their return home — this was 'Reisepolio' where the stress of travelling seems to trigger the attack. In the summer there were 13,164 cases in New York State, with 3331 deaths; despite thorough inquiries, no clear source of infection could be found, and even infections from person to person could not be traced.

*Processing problems.* The difficulties of sugar production have already been described; it seems that technical problems in processing are often linked to polio epidemics. Between the Revolution of 1917 and 1926, most of the sugar factories of Russia had lain idle, so the factories were in disrepair, when, as part of the second 5-year plan, it was decided to revive production. For a Russian sugar campaign, the time limit for the harvest is the first severe frost of winter. The beets can stand frost as long as they remain in the soil, but in the Russian winter you might have to use a saw rather than a spade to uproot them for processing; this made it a nerve-racking business, especially with a boss like Stalin. Technical breakdowns beset the first campaign, and finally the factories had to process frozen beet. Despite the efforts of the refiners, output was extremely low. In 1927, polio epidemics cropped up in Russia, and wherever her sugar had been exported: the Eastern part of Germany, Romania, and even in Poland where, apart from a few cases in 1911, there had never been an epidemic before.

These are not the only instances where problems in processing may have triggered polio. Processing

difficulties in Jamaica in 1957 and 1960 were caused by strikes, and in both cases the problems were followed by epidemics. Similar events occurred in Eire between 1940 and 1949, where the chemicals needed for processing were scarce, and fertiliser (superphosphate) or bauxite were used as clarifying agents; in Trinidad in 1971 where stoppages and a prolonged harvesting period adversely affected the quality of the cane juice; and in Poland, where a particularly good harvest led to a long campaign, and processing of frozen beet.

*Complementary sugar.* Complementary sugar is necessary when domestic output has been low or exports have been too high, and is generally imported towards the end of the consumption year, just before new sugar from the home crop becomes available. In Europe, this happens between late August and mid October, when sugar is plentiful in the Southern Hemisphere, and relatively cheap, since it has not been stored for long. Such an import may have triggered the Italian epidemic of 1958, the only one of those years which struck in winter. There were 8377 cases and 1300 deaths, nearly three times the average annual number.

During the early part of World War II, a large sugar stock had built up in Mauritius, because of the lack of transport. In 1944, however, the position eased, and there were massive exports. A cyclone struck in mid-January, cutting the yield of sugar by half (18). Because of the excessive export in 1944, recently refined sugar had to be imported in the first months of 1945, and was consumed as soon as it arrived. An epidemic began in March, and by the end of May there had been over 1000 victims, most of whom were crippled. Excessive exports from Mauritius in 1951–2 again necessitated an import of fresh refined sugar from Durban, in South Africa; 324 cases of polio were reported that year.

The Danish epidemic of 1961 (with 365 cases) followed an import of 11,000 tons of refined Polish sugar to this traditionally self-supporting country. This outbreak was particularly unexpected, as 90% of the population was vaccinated at the time—the figure was 99% for children under 14 years old.

*Change of diet.* Populations which shift from eating brown to white sugar may be at particular risk of polio epidemics. After World War II there was a radical change in the diets of the poor of Jamaica, where brown sugar, or 'plantation white' formed up to a third of the diet of young children (19). Because of the world shortage, the entire stock was often exported,

including the consumption sugar for the local populace. Refined white sugar had to be imported, and, when this change in diet occurred, epidemics began to break out in Jamaica. A similar situation developed during 1934 in Cuba, described as a 'reservoir of virus', where polio was endemic. A glut on the home market brought about the change in diet here, which was followed by 434 polio cases around Havana. In 1958, in the Phillipines, sugar was scarce due to over-exporting, and prices high. Output in the villages was low because the refiners were buying up the cane, so the government forced the industry to provide refined sugar for domestic purposes; consumption of brown sugar fell by 90%, and that of refined rose by 25%. In the same year an epidemic developed, with 500 cases.

*Split populations.* Some populations are split into part which consumes refined, and part which consumes non-refined sugar. This was the case in South Africa: in both of the major epidemics here, in 1918, and 1944–45, the whites had an incidence of polio 10 times that of the blacks. Apartheid had not yet been introduced, and surely there was sufficient mixing of whites and blacks to allow viruses to cross-infect—black girls were even employed as wet nurses to white children. Indeed, any difference in incidence might be expected to be the other way round, since the blacks generally had poorer sanitation. However, the two population groups had different patterns of sugar consumption: the black majority consumed an average of 17kg/year of 2-Grade and mill-white sugar, while the whites ate an average of 50kg of refined sugar per year. The same circumstances pertained to Hawaii before Pearl Harbour—the white part of the population, with their high consumption of refined sugar, having twice the incidence of polio of the indigenous group, who consumed brown sugar.

*Elevation of blood sugar and polio.* A number of clinical, epidemiological, and laboratory observations suggest that elevated blood sugar levels are accompanied by an increased susceptibility to polio. In the 'forties an idea that flies were the vectors of polio was current, and programmes of fly eradication with DDT were begun; a first attempt was in an African village, but after the spraying an epidemic developed, where, up till then, polio had been sporadic disease. The same thing happened in the Rio Grande del Norte valley in Texas in 1948 (20), a severe epidemic developing in the nearby town of Matamoros. The 1954 Jamaican epidemic (21), with 759 victims, broke out after a DDT campaign aimed at eradicating mosquitos. The pharmacological action of DDT on man is to

increase the blood-sugar level; types of sugar which have been stored by the body, and which could give rise to polio, might be released by this elevation.

A woman is much more vulnerable to polio in the second half of pregnancy than at any other time. In addition, if she is a diabetic, her need for insulin increases after the 30th week, in some cases necessitating a doubling of the dose, apparently because of a sudden elevation of the blood sugar level. The coincidence with the increased susceptibility to polio is striking.

Mental and physical stress are also known to raise blood sugar, and to increase vulnerability to polio—'Reisepolio' has already been mentioned. The most famous case was that of President Roosevelt: on the day before his attack he attended a traumatic investigation of a senate commission, but it was the cold dip in the lake at his summer residence at Campobello which finally triggered the illness.

*The ginger paralysis epidemic of 1930–31.* During the Prohibition years an incident occurred in the USA, which shows that symptoms identical to those of polio can be produced by the action of a poison, without any involvement of a virus at all (22, 23, 24). At that time great ingenuity was exercised in the quest for alcohol. Alcoholic extracts of ginger were officially defined as non-potable; this view was not, however, universally shared, and large quantities of the fiery beverage were consumed. In early 1930 a great number of cases of paralysis were reported in Ohio and neighbouring Tennessee. It was found by interviewing the patients that almost all of them had drunk ginger about 10 days before the symptoms appeared. This led to a search for a contaminated batch of ginger, and by painstaking analysis it was shown that a phenol derivative was present in every sample which had given rise to the illness. The effects in laboratory animals of tri-orthocresyl phosphate ester (TOCP) were identical to those of the ginger extract; this compound, it was concluded, was the culprit.

The remarkable fact of this case is that the symptoms of the illness were in every respect identical to those of polio. Soreness of the leg muscles was followed by numbness in fingers or toes, and the next classical polio symptom to appear was bilateral foot drop; the subsequent 'disability in the hands and forearms was never so marked as in the feet and legs' (24). The milder cases could get about on crutches; the more severely affected were bedridden, and unable to feed themselves. In advanced cases paralysis extended to the thigh muscles. Superficial reflexes were normal, and the clinical picture was 'of a flaccid

paralysis for the most part of the distal muscles of the upper and lower extremities, clearly pointing to the involvement of the lower motor neuron remarkably localised to the lower lumbar and lower cervical regions in the cord' (24). Striking similarities to polio were also seen when calves were fed TOCP—their hooves dropped in a way identical to the classical foot drop of polio in man. Finally, post mortem examination showed degeneration of the anterior horns of the spinal cord: it was this which gave polio one of its early names—'anterior lameness'. However, because of the absence of sick children, and the clear involvement of a poison, no connection was made between the incident and polio.

The relevance of this episode to my story is simple. Here we have a compound which would not be expected to give rise to the complex and specific symptoms of polio; and yet that is what it does. The poison acts as an inhibitor of cholinesterases (25, 26), enzymes important in neurotransmission. The concentration of the TOCP in the paralytic ginger was very low: thus, it also has to be considered that this or similar compounds are now finding their way into the food chain, and so contributing to the toll of paralytic cases we describe as polio. This might either be by inadvertent production in the sugar refining process through the action of disinfectants and phosphoric acid, or because of their use as insecticides.

## Discussion

The circumstantial evidence presented above suggests that there may be a link between the consumption of refined sugar and polio.

I have suggested that some substance, produced during the refining process, may be responsible for polio; deoxysugars may be such substances. After ingestion of sucrose, insulin acts to remove sugar from the blood, promoting its storage as glycogen in the liver and muscles. Deoxysugars, however, taste like normal sugars, but are not recognised as such by the body. They fail to stimulate the secretion of insulin, and interfere with the normal storage and mobilisation of glucose. Administration of 60mg/kg body weight of 2-deoxyglucose can cause blood-sugar levels to rise to three times their normal value (27); this rise may be mediated by the increased levels of epinephrine which are also produced. Although I can supply no direct evidence that deoxysugars are implicated in polio, it has been observed that the lumbar fluid of polio victims is highly positive for these non-fermentable sugars (28).

Poliovirus may be present in a population, but it is my belief that it will not influence the occurrence

of any epidemic—the anomalies in contagion and the failures of vaccination speak against it. However, if the quantity of deoxysugars ingested were high (for example after intensive refining, after clarification difficulties, or when the sugar comes freshly refined and bagged), then the disease may well break out. Because deoxysugars are produced particularly in cane sugar refining, people of the developing world, who consume exclusively this kind of sugar, are more vulnerable to their effects. The body has no use for the deoxysugar, and may store it. If there were a build-up of such a store, as when DDT is stored in adipose tissue, or lead in the bone marrow, stress may act to release it. Or perhaps secretion of epinephrine induced by stress may increase the blood-level of these substances; the cases where blood-sugar is elevated and vulnerability to polio enhanced may be relevant here. These ideas are speculative, but they are also testable: different components of refined sugar could be separated and used in an animal model to measure their effect upon polio infection.

I hope that my suggestions might stimulate thought on this disease, which continues to claim more than 200,000 new victims each year. At the moment, production pressure is low in the West, not least because of the drive for healthier eating, and the introduction of artificial sweeteners. Thus there may be little risk of epidemics at present; however, because of this abundance, farmers are reducing their sowings, and it will only take one disastrous harvest for a glut to become a shortage, for production pressure to increase, and, possibly, for the risk to rise again. And I hope that, with further work on the ideas I have set forth here, the difference in polio incidence between the West and the Third World—a difference which has haunted epidemiologists from both sides for years—might finally be elucidated.

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