SIDS and the New Zealand Environment

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Abstract

Between 1981 and 1993, the Sudden Infant Death Syndrome (Cot Death) rate in New Zealand declined by 50 percent. Traditionally, New Zealand had experienced the highest death rates from this cause in the Developed World. The improving situation was widely attributed to the National Cot Death Prevention Programme which began in the early 1990s. This sought to persuade parents to modify their behaviour in four areas. Specifically, breast feeding was encouraged and smoking and infant bed sharing discouraged. Parents also were advised to ensure that the child slept on its back. However, despite improvements in these four risk-related behaviours, cot death mortality again appears to be rising in New Zealand. It is suggested here that this is because the root causes of cot death in New Zealand are widespread soil and associated dietary deficiencies in selenium and iodine. The impressive reduction in the Sudden Infant Death Syndrome rate in the early 1990s appears to have largely reflected greater consumption by the New Zealand population of selenium-enriched imported wheat and dairy products containing iodine from sanitizing solutions, rather than the benefits of deliberate risk-reduction behaviour.

Introduction

New Zealand has traditionally experienced one of the world's highest rates of Sudden Infant Death Syndrome (SIDS), or as it is often called in that country, cot death.[1,2] As a consequence, a National Cot Death Prevention Programme began in New Zealand population in the early 1990s. This programme placed its emphasis on changing parental behaviour, in an effort to reduce four previously identified risk factors for SIDS.[3] Specifically, attempts were made to encourage mothers to breast feed and not to smoke. Parents also were asked to ensure that infants slept on their backs and did not share a bed. This prevention programme appeared to be very effective and by December 1996, Mitchell and Tipene-Leach were able to claim that because of it, SIDS mortality in New Zealand had fallen from 4.2 per 1,000 live births in the period 1981 to 1983 4 to 2.1 by 1993, a 50 percent drop.

While not questioning that such a decline in SIDS occurred in New Zealand during the early 1990s, this author will now argue that this mortality drop had little to do with the National Cot Death Prevention Programme. Indeed, evidence will be presented to suggest that it was directly related to fortuitous, largely accidental dietary changes that took place, at this time, in New Zealand. More recently, some of these changes have been reversed and, as a consequence, SIDS rates may be beginning to rise again,[5] despite the ongoing prevention programme.

The Way Things Were

In 1988 Borman and coworkers[4] demonstrated that, during the period 1981 to 1983, the national New Zealand Sudden Infant Death Syndrome (cot death) rate was one of the highest in the Developed world, 4.2 per 1000 live births. Interestingly, such mortality had not been evenly distributed, but displayed a marked north-south gradient. As a consequence, the SIDS rate in the south of the South Island had been almost double that occurring in the north of the North Island. While research[6] established that SIDS typically is more common amongst the Maori (Paul, 1993), racial differences could not explain this national pattern. Indeed, since the majority of Maori lived in the North Island[7] their elevated SIDS rates could only have been helping to mask an even more pronounced national

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north-south SIDS gradient in infants of the non-Maori population. Further evidence to support an abnormally high SIDS rate on the South Island of New Zealand was provided in 1990 by Ford and colleagues[1] who described cot deaths in Canterbury, over the 20 year period 1969 to 1988. These researchers concluded that this city had experienced one of the highest rates ever recorded in the Developed World, 6.8 per 1000 live births from 1981 to 1985.

In the same year, Horvath[8] published his analysis of all the autopsies carried out on SIDS infants, in New Zealand, from1985 to 1987. His sample consisted of 629 out of the total infants 645 recorded as dying of this cause. Horvath's findings challenged the widely held view that asphyxia, particularly sleep apnea, was the primary cause of death in SIDS. Rather he showed that signs of heart failure had been noted in 457 (72%) of the cases studied. In contrast, cyanosis, the unequivocal hallmark of hypoxia had been recorded in only 31 (4.9%) of all autopsied infants. Horvath[8] concluded, therefore, that myocardial dysfunction, that is sudden infant heart failure, lay behind New Zealand's extremely high SIDS rate.

Three major conclusions can be drawn from the evidence just presented. Firstly, whatever variable or variables cause SIDS they must have been particularly common in the 1980s in New Zealand, occurring much more frequently than in other countries in the Developed World, where SIDS rates have traditionally been far lower. Secondly, since SIDS in New Zealand was experienced more often on the South Island than on the North Island these variables must also have displayed a similar north-south gradient. Thirdly, whatever caused SIDS in the 1980s must have been capable of triggering sudden heart failure in susceptible New Zealand infants.

SIDS: A Global Explanation

Interestingly, SIDS also displays distinct spatial distribution patterns in many other countries, including Australia where it again peaks in the south, that is in Tasmania.[2] In the United States, for example, it is more common in the north than the south and in the west than in the east. As a consequence, it occurs twice as often in the Pacific and Mountain Regions as it does in the Atlantic States.[2] In an attempt to explain this geographical pattern, this author[2,9,10] statistically compared US state SIDS mortalities for each of the years 1983 to 1987, and for the period as a whole, with 84 other disorders or diseases. By far the strongest Pearson's correlations discovered were between SIDS (for the period 1983 to 1987 as a whole) and the incidence of male military recruit goitre prior to the addition of iodine to US salt ($r= 0.75774$, $p<0.0001$). Beyond these SIDS-disease correlations, the author also compared such US cot death rates with the spatial distribution of 221 environmental variables. Interestingly, SIDS was found to be very common in regions of elevated soil selenium ($r= 0.54360$, $p<0.0001$). Stepwise multiple regression also was conducted for each year and for the entire five-year period using those medical and environmental data that had correlated with SIDS mortality, either negatively or positively, at the $p<0.001$ level. In all six regressions, male recruit goiter was the first independent variable to enter the initial equation; where it could explain between 57.4 percent and 42.2 percent of the SIDS variance.

On the basis of these analyses and a detailed review of the global literature, it was later suggested by Foster,[10] that the fundamental cause of SIDS is an infant iodine deficiency accompanied by either an excess or a deficiency of selenium. Interestingly, both high and low selenium intake have been shown to cause serious thyroid gland malfunctions in iodine deficient adults.[11,12] This appears to be because selenium is an essential component of deiodinase, the enzyme required to convert the thyroid hormone thyroxine (T4) to

triiodothyronine (T3). Both an excess or deficiency of selenium, combined with iodine inadequacy appear capable of disrupting this conversion. Interestingly, excess iodine can also cause thyroid disorders.

Iodine and Selenium in New Zealand

Iodine: Surveys conducted amongst New Zealand schoolchildren in the 1930's established that some 15 percent had goitre, a malfunction of the thyroid gland generally caused by iodine deficiency. The addition of iodized salt to diet reduced this figure to 0.1 percent by the 1950's.[13] Naturally, this programme did nothing for farm animals and goitre and high mortality amongst new-born livestock caused by iodine deficiency continued amongst unsupplemented New Zealand's livestock. Regions where soils are most iodine deficient are more common in the east than the west of New Zealand, and occur more frequently in the South Island than in the North. They are found in parts of Otago, Canterbury Plains, Westland and Marlborough, Hawke's Bay and an area between Wananui and Palmerston North14.

Beyond the use of iodized salt, after 1962, high levels of iodine also began to enter the New Zealand diet as the result of the addition of this mineral to animal feed and as a consequence of the use of iodophor sanitizing solutions by the dairy industry. Indeed, by 1991, Logan could describe New Zealand's food as being high in iodine, since surveys conducted in Wellington, Auckland and Dunedin showed intakes of iodine which were 2 to 3 times the 100 micrograms Recommended Daily Allowance.[15] It is clear that, in large part by accident, iodine levels in the New Zealand diet had changed from being far too low in the 1930's to perhaps being excessive by the late 1980's.

The situation, however, has since changed. Most dairy sanitizing solutions now no longer contain iodine and many people are avoiding the use of salt in an attempt to reduce or avoid hypertension. As a consequence, the dietary intake of iodine in New Zealand is declining. To illustrate, Thomson and co-workers[16-18] have been monitoring the iodine status of New Zealand residents as assessed by urinary iodide excretion and thyroid hormones levels. Their research has demonstrated that in Otago, Dunedin and Waikato the iodine status of New Zealand may no longer be considered sufficient "and may once again be approaching levels of intake associated with clinical I [iodine] deficiency." Thomson and colleagues predict that the situation is likely to worsen as iodine intake continues to fall and they anticipate the possible re-emergence of goitre as a medical problem in New Zealand.[17,18]

Selenium

There can be no doubt that the New Zealand diet has traditionally been very low in selenium. The only large population with a more depressed intake of this trace element occurs in the selenium-deficiency belt that crosses China from northeast to southwest.[19] The best record of the "original" selenium content of New Zealand soils are Maps 89 and 90 of the New Zealand Soil Bureau Atlas.[20] However, these illustrated total selenium, much of which was probably elemental or selenite and, as a consequence, was largely unavailable to plants and so did not enter the foodchain.[21] A better indicator of the natural selenium that was bioavailable to plants and animals, and therefore to humans, was the distribution of selenium-responsive disease that occurred prior to the widespread provision of this trace element to livestock. Fortunately, Andrews, Hartley and Grant[22] mapped the incidence of this disease in lambs, demonstrating that it was relatively rare in the North Island, but extremely common in the South Island, especially in the south. As a consequence, not only were livestock adversely affected but cereal grains grown in New Zealand generally

contained only about one-tenth of the selenium found in those imported from Canada and the United States. Grain from Australia was also usually more selenium-enriched than that from New Zealand.[23-24]

In summary, therefore, there was a natural, but marked, north-south selenium gradient in New Zealand that was paralleled not only by mortality from SIDS but also by death from two other diseases, (large bowel cancer and multiple sclerosis) that are thought to be associated with dietary deficiencies in this trace element.[15]

The situation is now more complicated because of the enthusiastic adoption of selenium fertilization by farmers. By 1998, some 1.2 million out of an estimated 4.5 million hectares of selenium-deficient New Zealand farmland were receiving fertilizers containing this element.[21] Beyond this, the deregulation of the wheat market has lead to a far greater consumption of flour made from grains imported from Australia and the United States. Flour was made from South Island wheat prior to 1988 and only contained about 15 micrograms per kilogram of selenium. By 1991, much of the flour was produced from imported wheat and so had a far higher selenium level of between 80 and 140 micrograms per kilogram. Winterbourn and colleagues[24] have shown that the deregulation of the wheat market and the use of imported grains has been parallelled by major changes in the plasma selenium status of New Zealanders. To illustrate, until 1987, Christchurch adults had mean plasma levels that ranged between 46 and 54 micrograms per litre. Mean plasma levels dramatically increased to between 66 and 70 micrograms per litre during the period 1988 to 1991. By 1992, the mean adult plasma selenium level in Christchurch had reached 80 microgram per litre, that is almost doubling in five years. It is clear, therefore, that selenium levels in the New Zealand diet are still greatly influenced by trade policies that control the levels of foreign wheat entering the country.

Impact of the National Cot Death Prevention Programme on Infant Iodine and Selenium

The author has argued elsewhere,[2,10] that SIDS risk factors such as the use of infant feeding formulae and cigarette smoking are not basic causes of cot death, but rather act as triggers which either cause greater infant demands for iodine and/or selenium, or reduce infant access to one or both of these essential trace elements. This will now be demonstrated for the four risk factors emphasized by the national Cot Death Prevention Programme in New Zealand.

1. Breast Feeding A study conducted by Dolamore and co-workers 25 in Christchurch, measured the levels of red cell and plasma selenium and of the selenoenzyme glutathione peroxidase, in 70 infants younger than one year old. It was established that there was a statistically significant difference between breast fed and formula fed infants, mean plasma selenium and glutathione peroxidase levels in formula fed infants being only about half of those occurring in infants who were breast fed. In addition, in formula fed children, red cell selenium was also significantly depressed. In summary, the "status of the infants reflected their diet, with the concentration of selenium in formulae being 3.9-5.2 micrograms per millilitre, compared with a mean of 13.4 micrograms per millilitre in breast milk." Clearly, infants who are breast fed are much less likely to become selenium deficient than those receiving formula. A second consideration is that many infant formulae contain soy, a goitrogen that reduces the availability of iodine to the infant. Soybean milk, for example, can produce hypothyroidism in susceptible infants.[26]

2. Cigarette Smoking: Similarly, one of the major reasons cigarette smoke may trigger SIDS is because it contains a goitrogen that interferes with iodine metabolism. The substance involved is thiocyanate, a metabolite commonly found in the blood of smokers. This chemical is formed by the

detoxification of the cyanide, absorbed from tobacco smoke. In pregnant women, thiocyanate enters the fetal blood stream resulting in the impaired production of iodinated thyroid hormones. The thyroid glands of fetuses being exposed to their mothers' cigarette smoke hyperfunction, causing an increased metabolic rate, resulting in lower thyrotrophin production and greater fetal caloric requirements.[27] A similar impact can be expected in breast fed infants of smoking mothers. This would explain the interesting observation made by Tonkin[28] that bed-sharing with a smoking mother in New Zealand increases the risk of SIDS. However, the SIDS rate is not affected by fathers smoking.

3. Sleeping on the Back: Interestingly, Mitchell[29] showed that in New Zealand the significance of a child sleeping on its stomach or back depended very much on the presence of any associated illness, latitude, season, thermal insulation and use of sheepskins. This suggests that rather than greater exposure to mattress gases, or those given off by urine, the face-down sleeping position influences the infants ability to regulate its own temperature. Thyroid hormones play a major role in human metabolism and temperature regulation.[30,31] It is likely, therefore, that sleeping on the stomach makes temperature regulation and breathing more difficult, increasing infant iodine and probably selenium requirements. This could explain the observations of Schluter and colleagues[32] that in Canterbury, after a prolonged period of cold minimum temperatures, infants were most at risk from SIDS on days on which a warmer minimum temperature was recorded. This is because fluctuations in external temperature increase the difficulty of infant thermoregulation and encourage the depletion of thyroid hormones.

4. Bed sharing with parents It seems likely that infant bed sharing with parents also triggers SIDS because it is responsible for causing marked temperature fluctuations that increase the need for more active thermoregulation by the infant. As previously discussed, fluctuating external temperatures stress the child, increasing the need for both iodine and selenium to maintain optimum body temperature and basic metabolism. Reid and Tervit[30] have suggested that brown adipose tissue synthesises fatty acids from glucose, playing a major role in heat production and, therefore, infant thermoregulation. Brown adipose tissue has the specific property of converting thyroxine to triiodothyronine, but to do so requires the selenoenzyme Type II deiodinase. There is a great increase in such conversion activity after exposure to sudden cold but this process is impaired by selenium deficiency. It seems probable that the use of selenium to produce heat in infants, that are deficient in this trace element, many render it unavailable for other metabolic processes. It is well established that selenium deficiency can trigger heart attacks in adults,[33] while supplementation reduces their frequency.[34] A deficiency, therefore, may be responsible for the very high rate of myocardial infarction seen by Horvath[8] in SIDS cases in New Zealand.

Clearly, there are links between the four modifiable risk factors, identified by the National Cot Death Prevention Programme and SIDS rates. Nevertheless, the geographical evidence strongly suggests that reducing these four factors could not have caused the majority of the decline in SIDS, experienced in New Zealand, in the early 1990s.[35] There is, for example, no suggestion that any of those behaviours were traditionally much more common amongst New Zealanders than other members of the Developed World. Indeed, research by Mitchell and colleagues,[36] comparing the prevalence of these four risk factors in New Zealand and the South West Thames region of the United Kingdom demonstrated that they could only explain 20 percent of New Zealand's excess SIDS risk. Beyond this, there appears to be no evidence of a north-

south gradient for any of these risky behaviours that could account for the traditional elevated SIDS mortality seen in the south of the South Island, nor is there much evidence that any of these four behaviours could trigger sudden infant myocardial infarction.

In contrast, a large percentage of the natural environment of New Zealand is both extremely iodine and selenium deficient. The author is unaware of any other country in the Developed World affected, on a similar scale, by such duel mineral deficiencies. Secondly, both of these mineral deficiencies display the same north-south gradient as SIDS, with the South Island being far more affected by them. Thirdly, in adults, selenium deficiency has been shown to be associated with heart attacks that can be prevented by supplementation with this trace element.[33,34,37]

Conclusion

It would appear that the decline in New Zealand SIDS deaths in the early 1990's was only partially due to the National Cot Death Prevention Programme.[38-39] Much of this mortality drop appears to have taken place because of an increase in dietary iodine, caused by iodophor sanitizing solution in the dairy industry, and the greater consumption of high selenium flour made from imported grain. The situation is altering and it seems likely that SIDS rates will begin to rise as iodine and selenium intake declines once more. Interestingly, New Zealand veterinarians and farmers have supplemented farm livestock for many years with selenium, especially on the South Island. If they do not, a deficiency of this trace element causes white muscle disease and sudden death in young animals.[40] Beyond this, iodine deficiency traditionally has been widespread in New Zealand, especially the South Island, causing goitre in farm animals and high-mortality in newborn livestock.[41] It seems strange that New Zealanders, armed with this agricultural knowledge, have been so slow to accept that these twin trace element deficiencies are also responsible for the country's high mortality from SIDS.

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