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GLOBAL IODINE DEFICIENCY DISORDERS IN THE LIGHT OF THE BIBLICAL FLOOD

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KEYWORDS

Aetiology, bio-geochemistry, disease, environmental iodine, Flood, goitrogens, iodine, iodine deficiency disorders, plate tectonics, sedimentary rocks, soil, thyrotoxicosis.

ABSTRACT

Iodine deficiency disorders are common throughout the world, caused biochemically by iodine deficiency, although that is sometimes exacerbated by the presence of antagonistic factors (goitrogens). Environmental iodine deficiency is assumed, but remains not-proven. Soil iodine levels, as well as water iodine, are not always directly related to the prevalence of the disorders, raising questions about the constancy and meaning of environmental deficiency. The role of the Flood in removing iodine from the environment has been crucial, leaving the world hovering on the edge of deficiency. It is unlikely that the uniformitarian paradigm can explain iodine deficiency as well as the Biblical Flood can. Despite the inferred enormous difference in pre-Flood and post-Flood iodine levels, the pre-Flood world probably did not suffer any iodine excess.

INTRODUCTION

The majority of countries in the world suffer from the iodine deficiency disorders; out of 183 countries listed in the latest WHO report [49] 123 (67%) suffer from the iodine deficiency disorders in some form or other (Table 1). The fact that many of the countries are listed as free of the disorders in the 1993 report can be attributed either to a deliberately raised intake of iodine or, sometimes, to other coincidental practices [35]. Many more places are thus susceptible to the disorders than at first meets the eye.

Goitre (%)	Africa	Americas	E Mediterranean	Europe	SE Asia	W Pacific	World
< 5.0*	6	18	8	13	1	14	63
5.0 - 19.9	23	9	5	16	7	3	63
20.0 - 29.9	9	3	3	11	2	3	31
≥ 30.0	7	2	4	2	1	2	18
No Data	0	3	2	6	0	0	11
<i>Total</i>	<i>45</i>	<i>35</i>	<i>22</i>	<i>48</i>	<i>11</i>	<i>22</i>	<i>183</i>

Table 1. Number of Countries with Goitre in Each WHO Region.

* < 5.0% prevalence of goitre is currently taken to mean that the iodine deficiency disorders are not a problem in these countries. Derived from [49].

It is generally accepted that the cause of the disorders is biochemical iodine deficiency [19], though environmental iodine deficiency is often presumed from this. The list of disorders the deficiency produces includes stillbirths, spontaneous abortions, congenital miscarriages, endemic cretinism (commonly characterised by mental deficiency, deaf-mutism, spastic diplegia, and lesser degrees of neurological defects), impaired mental function in children and adults, and goitre associated with decreased levels of circulating thyroid hormone [20]. Other more controversial effects of iodine deficiency are breast disorders such as fibrocystic disease [16]. Goitre itself is rarely a major health problem in a community, even with a prevalence greater than 70% in adult women [42]. It is the fetal wastage and the impaired mental functions that are so devastating to both the community and the individual. Goitre remains the visible marker of such disorders, but cretinism is the most devastating and important disorder.

There is no treatment for endemic cretinism, so prevention is important; but iodination of the diet does not necessarily eliminate the disorders. It seems that there is something lacking in our understanding of the aetiology of the iodine deficiency disorders. The question arises as to whether or not the role of environmental iodine is properly understood, or if there is another, missing, factor.

It is the aim of this paper first to explore the current understanding of the aetiology of the iodine deficiency disorders and then to discuss the role of the Biblical Flood in their origin. The Flood may offer a better understanding of the aetiology of the disorders than an old earth picture since it will be argued that environmental iodine levels were seriously and adversely affected by the Flood.

THE BIOCHEMISTRY OF IODINE

Iodine is absorbed by the gut and concentrated in the thyroid gland in the neck. In iodine deficient regions such is the affinity of the thyroid for iodine and the efficiency and specificity of its uptake that it can remove more than 90% of serum iodine on the first pass. In the thyroid, under pituitary-hypothalamic control, the iodine is incorporated into thyroxine (T4) by a series of steps. T4 is released into the bloodstream under a negative feed-back system whereby the level of T4 controls the level of thyroid stimulating hormone (TSH) released from the anterior pituitary gland in the brain. T4 is converted into the more active form, T3, by the removal of one iodine atom, usually in the target organ but also in the thyroid itself before release.

The thyroid hormones T3 and T4 control the rate of reactions in the cell nucleus in almost every cell in the body. Hence a deficiency of the hormones (hypothyroidism, myxoedema) slows the whole metabolism down while an excess (hyperthyroidism, thyrotoxicosis) causes hyperactivity. These effects may be a result of iodine deficiency, but may be caused by failures and abnormalities elsewhere in the metabolism and control of the thyroid.

After the hormones are de-iodinated most of the iodine is recycled, but a small amount is lost in the faeces. The major loss is via the urine, equalling the dietary intake, and is thus a simple but accurate way to measure dietary intake. The iodine deficiency disorders are found in regions where urinary iodine levels are consistently low. Less than 50 µg/day is the usually accepted figure to define iodine deficiency, although 100 µg/day is preferable. When the supply increases, the prevalence of the disorders drops, but there are discrepancies, which may be more important than realised. Nor is it always true that the same level of iodine deficiency produces the same rate of the disorders. The equation is not simply "iodine deficiency = iodine deficiency disorders".

The Iodine Deficiency Disorders

Goitre is a swelling of the thyroid resulting from any kind of malfunction of the gland; it is not specific to iodine deficiency. The term *endemic goitre* indicates the common swelling in iodine deficient regions. In such regions most people who suffer from an enlarged thyroid do so because of iodine deficiency. When there is little iodine available T4 levels are reduced. As a result there is an increased secretion of TSH from the pituitary gland in the brain, causing the thyroid to grow, and thus maximising the catch of iodine. Goitre is the most visible of the iodine deficiency disorders, and can be used to quantify the other disorders [9], the most devastating of which is endemic cretinism.

Endemic cretinism is a result of a neurological insult to the growing brain. It is not related to the thyroid size of the parents. A goitre probably takes a minimum of several months to develop, while the growing brain may be critically affected by an acute incident, so it is quite possible for an event to cause cretinism but not goitre. And it is possible for a woman in a goitrous region, whatever her thyroid status, to give birth to both cretins and non-cretins.

Thyroid hormones are associated with cognitive and motor measures related to the growing brain [33]. In early fetal life the maternal thyroid hormone levels appear to be important, since at that stage the child's thyroid is not sufficiently developed to manufacture its own hormones [34]. The child's gland becomes influential later as the growing thyroid becomes functional.

The clinical form of endemic cretinism depends on the timing of the insult to the growing brain, whether early in pregnancy, or towards the end of pregnancy and into early life. The severity of the condition is a

result of the depth of the insult. Cretinism is not a discrete disorder, and it may be that in severely affected regions, where the overt cretinism rate may reach 10-15% of the community, the brain development of almost everyone will be affected to some extent. This two-part insult can explain the wide variation in clinical picture, from children who can neither walk nor talk through to normality [43].

Prevention

Perhaps the worst aspect of endemic cretinism is that once a growing brain suffers an insult there is nothing that can be done to reverse the situation; prevention becomes all important. With endemic goitre the situation is not entirely similar as it can often be reversed by iodine.

The key to prevention is iodination of the diet. The most usual method is iodised salt, but iodination of other constituents of the diet such as bread or water has been successful. Where such means are difficult, for cultural or logistic reasons, iodinated oil by mouth or injection has a role, providing depots of iodine lasting 6 months or 2-4 years respectively, depending on dose.

Such iodination has accomplished much around the world. In particular, the western world has seen a significant reduction in endemic goitre and endemic cretinism this century, though the benefits of iodination by no means have been confined to the developed world. Many of the 60 countries without endemic goitre [49] are not so naturally, but as a result of increased dietary iodine supplies. It has been the success of such programmes, as much as anything, which has confirmed that the iodine deficiency disorders occur as a result of iodine deficiency. But two problems remain: (a) while iodine deficiency seems to be the biochemical cause of the disorders the success of the preventive programmes is not proof that the cause is environmental. (b) The very success of reducing goitre prevalence to 5% or less, and accepting this as an absence of the disorders, may blind us against asking why goitre still persists in such a situation.

THE GEOCHEMISTRY OF IODINE

The term *iodine deficiency disorder* was introduced in 1983 in a medical context, and referred to the underlying biochemical situation [19]. Unfortunately, it has been assumed that, since iodine is supplied naturally through food, environmental iodine deficiency must, therefore, occur in every region where the disorders are found [23, 49]. Such is the strength of this supposition that verification of the environmental deficiency is usually omitted in medical surveys. And so an important aspect of the disorders has been missed too often, namely that the prevalence of the iodine deficiency disorders and the level of environmental iodine do not correlate easily.

Soils and Plants

The literature on environmental iodine is quite sparse. In fact, a recent search for world-wide soil data revealed very little. The most recent review is by Fuge and Johnson [15]. Their figure for the average world-wide soil iodine level is 4-8 mg/kg, which is similar to Bowen's mean of 5 mg/kg [6]. Most of this data was analysed before modern techniques became available. Iodine is notoriously reactive and difficult to analyse; accurate modern techniques rely on the rate of the catalysis by iodine of the reaction between cerium (IV) and arsenious acid [45]. In the light of this, Fuge and Johnson [15] examined the available data for rock iodine and concluded that the earlier data was less accurate than the more recent (post-1950) data. Soil iodine is an order of magnitude greater than rock iodine, but it would be interesting to run a similar check on the soil data and be more certain of these means.

	Mass of depot (kg)	Concentration of iodine (mg/kg)	Global Amount of iodine (kg)
Igneous rocks	24×10^{21}	0.14	3.4×10^{15}
Sedimentary rocks	7.2×10^{21}	0.4	2.9×10^{15}
Hydrosphere*	1.42×10^{21}	0.05	7.0×10^{13}
Atmosphere <12km	5.3×10^{18}	0.000010	4.0×10^7
Biosphere	1.8×10^{16}	0.05	9.0×10^8

Table 2. Some Estimates Relating to Global Iodine Distribution.

* most of the iodine stored here is in the oceans. Adapted from [48].

Iodine is an unusual soil constituent in many respects, particularly in that the source of soil iodine is the atmosphere, not the bedrock [15]. Its retention and distribution in soil is governed by a number of factors: soil type [15, 50], the state of growth of the covering vegetation [1, 30], humus, animal biomass [5] and oxides of Al and Fe [47]. Far from being actively mobile in soils, as might be expected from its chemical nature, iodine can be difficult to displace. Whitehead [46] tried extracting iodine from soil by various methods, mobilising up to 34% by boiling water, thus demonstrating that there is a considerable residue of stable, bound iodine in soils. This raises the question of how soil iodine levels affect the genesis of the iodine deficiency disorders.

The eight-day half-life of ^{131}I is the longest half-life of the common isotopes of iodine. After nuclear bomb fallout, or the Chernobyl disaster, it has been found in humans. While much discussion has taken place after Chernobyl concerning the potential dangers, mainly thyroid cancer [18], it is not clear how much of the increased ^{131}I found in the body after such events was ingested through the diet, mainly milk, or how much was inhaled, since gaseous ^{131}I was two to four times commoner than the aerosol [32]. Radio-iodine activity in milk peaked on the fourth day after exposure [32], but there was an observed time lag of seven days in adults and nine days in children from the peak exposure to maximum biochemical activity. The figures differed from theoretical considerations [4]. If milk iodine is as important as it seems then this indicates how quickly iodine can move through the food chain.

Country	Water iodine $\mu\text{g/l}$	Goitre prevalence
Algeria	3.5	80
	4.5	70
Cameroon	2-4	58
	>4	non-endemic
Tunisia	3.9	36
	3.9	50
	6.9	1
Italy	0.35	11
	0.55	49
	0.82	48
	2.02	10
	2.26	49
	2.38	5-40
	9.09	38
500	7	
Michigan	0.0	65
	0.3	56
	7.3	33
	8.7	26

Table 3. Water Iodine Levels and Goitre Occasionally Correlate. Adapted from [41]

The source of biochemical iodine is the diet, either animal or plant, but since animal sources are originally plant in origin they can be considered together. Consequently, the soil-plant link would seem to be all important in the aetiology of the disorders. Surprisingly, little work done has been done on this link; that which exists suggests that the iodine level in the leaves, and possibly in the complete plant above ground-level, is not fully dependent upon soil iodine as taken up by the roots [17, 24]. It is possible that even some root iodine derives from that absorbed by leaves [51]. Whitehead and Truesdale [48] suggested that the atmosphere is a sufficient source of leaf iodine without any need to invoke soil influence. To further complicate the issue it is clear that plants methylate iodine and release methyl iodine into the atmosphere. Such volatilisation occurs easily from rice plants in a flooded paddy, but CH_3I also is volatilised from oats growing in dry soil. In both cases CH_3I is more easily volatilised from plants than from bare soil [29]. It appears that the role of soil iodine in the production of the iodine deficiency disorders may be little more than an assumption.

Rock and Water

At 0.08-0.50 mg/kg rock iodine levels are one-tenth of that in soil [7]. It is therefore unlikely that rocks are an important source of dietary iodine, or even of soil iodine. Iodine is found in greater concentration in sedimentary rocks (0.2-10.0 mg/kg), but while this means that on a global scale the sedimentary mass of iodine is almost equal to that of the igneous rocks [48], despite the much smaller mass of sedimentary rocks (Table 2), it is unlikely to be even of local importance since soil iodine derives from the atmosphere, not the parent rock.

The relevance of water iodine levels to the iodine deficiency disorders is not clear [43] (Table 3). However, water provides only 10-20% of the dietary intake of iodine; so clearly, like soil, water is not a major factor in the environmental background of the disorders. It is known, however, that the mean river iodine level is $5\mu\text{g/l}$ [15]. Values less than this may be low environmentally, but neither this mean nor the mean soil iodine quoted earlier gives us any basis on which to decide whether or not a region is deficient enough in iodine to produce disease. Indeed, it is true to say that, at the present state of knowledge, an environmental iodine level tells us very little about the likelihood of the occurrence of the iodine deficiency disorders.

Atmosphere and Sea

The atmosphere is the primary source of plant, and therefore dietary, iodine. Iodine is released into the atmosphere from the sea, the storehouse of iodine. Figure 1 gives a basic outline of the iodine cycle, while Table 2 summarises some concentrations. Iodine is concentrated in and released from the sea by macroalgae, particularly the brown seaweeds, in shallow water [15] and by microalgae in mid-ocean [44]. Once released into the atmosphere iodine is mixed in the troposphere and carried world-wide. The concentration decreases with increasing altitude [38], a factor worth further examination with regard to the mountainous distribution of the disorders.

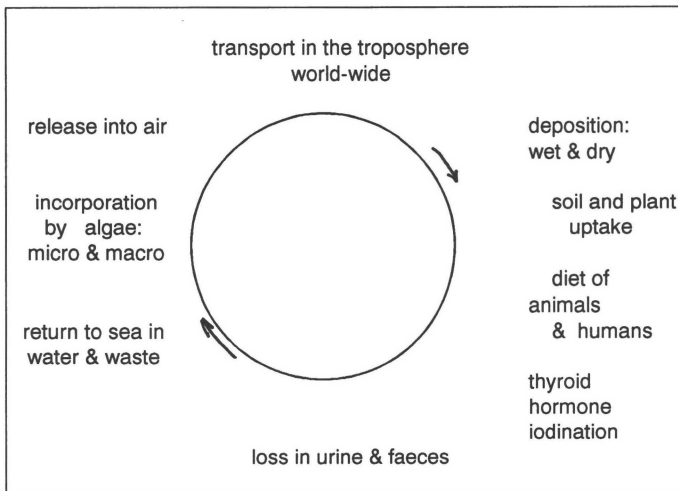


Figure 1. The Environmental Iodine Cycle

If environmental iodine levels are such poor indicators of the disorders it is reasonable to ask if environmental iodine is important in the genesis of the iodine deficiency disorders. The answer must be yes. Our diet is the source of our iodine supply, and as the supply increases (as measured by urinary iodine levels), either spontaneously across a geographical region or deliberately through time from a prophylactic programme, the prevalence of goitre and the other disorders decreases. The lack of correlation between environmental iodine and the prevalence of the disorders might mean we are measuring the wrong thing.

GEOGRAPHICAL SOLUTIONS

In an attempt to explain the global distribution of the iodine deficiency disorders several geographical hypotheses have been proposed.

Glaciation

The inadequacy of soil iodine to explain the prevalence of the disorders has been discussed already. However, there is a long-standing idea that some regions have become iodine deficient because glaciation stripped the soil away [40]. This hypothesis has been seriously questioned by earth scientists [12, 15]. The medical profession has yet to recognise this [49] and continues to rely on an early Swiss study [27, 28] which has deficiencies [43]. Perhaps the most damning aspect of the glaciation hypothesis is that the known extent of the iodine deficiency disorders far exceeds the known glaciated regions. It is time the hypothesis was abandoned.

Leaching

The leaching hypothesis has never been examined critically as a cause of the distribution of the iodine deficiency disorders, but has been assumed by both earth and medical scientists to be true. Although never formulated in the literature, the assumption is that a high input of water, usually in the form of heavy rain, will remove iodine already present in the soil. The chemistry of iodine suggests that it will be removed easily, and the finding of iodine in the leachate of soils has supported the hypothesis. However, it is likely that the through-put of iodine in soils is a reflection of a steady state, at least in some parts of the world. Figures quoted by Whitehead & Truesdale [48] indicate that in Berkshire (UK) and central Russia the annual losses through run-off were probably similar to the annual input from the atmosphere (20 and 45 g/ha respectively). Observations in Wales [31] and Scotland [10] further demonstrate this. Such a steady state is not dissimilar to the biochemical situation where dietary intake equals urinary loss. Leaching is unlikely to be an important factor in the aetiology of the disorders.

Regional Reasons

Alluvial plains, regions in the rain shadow of high mountains and mountain ranges are reputable suggestions for the environmental aetiology of the disorders [43]. However, the belief that flooding is a cause of the iodine deficiency disorders prevalent on alluvial plains assumes that leaching of iodine occurs. The rain shadow hypothesis remains to be expounded, while those living on mountain ranges are believed to suffer from the disorders because of either the previously unquestioned effects of heavy rainfall, or from the unexplained effect of "poor soil".

Distance from the sea is a well recognised factor, referred to in British school textbooks, but it is based only on small scale coastal studies [22, 46]. There has been no large scale continental investigation. The maritime influence on iodine probably decreases exponentially with distance [43].

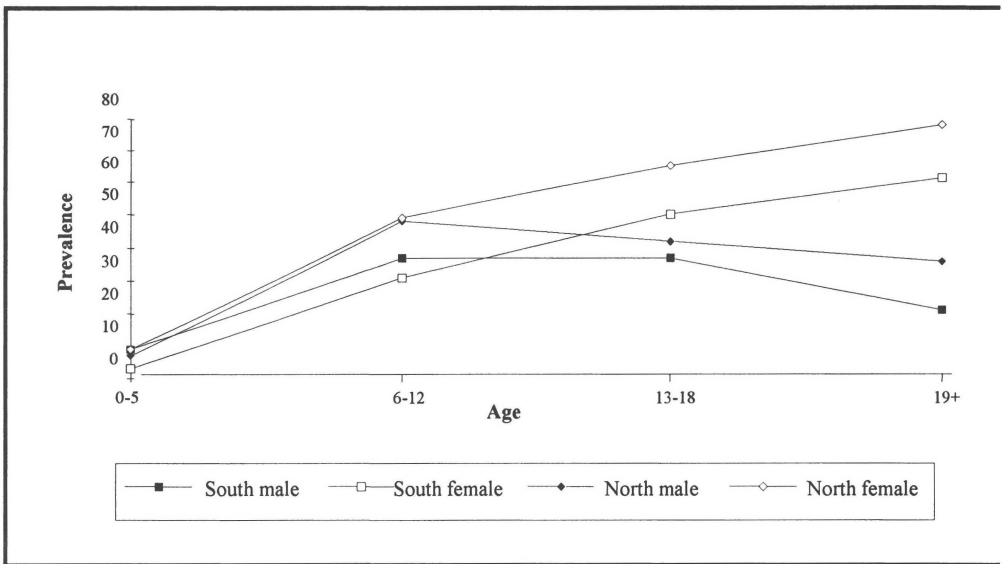


Figure 2. Goitre Prevalence in Northern Pakistan

Patients came from villages across district Baltistan, to the south and the north of the Indus-Tsangpo suture, which joins Asia to India geologically.

It is worth noting at this point that the iodine deficiency disorders are not present on all of the Pacific islands, thus further emphasising the inadequacy of both the distance from the sea and the leaching by heavy rainfall hypotheses. Hawaii has one of the heaviest annual rainfalls, yet its inhabitants have not suffered the disorders, whilst the inhabitants of Fiji, which has a much lower rainfall and similar proximity to the sea as Hawaii, have been known to be goitrous for many years [22].

Geology

It is possible that the inadequacy of our environmental understanding of the disorders is simply due to an as yet unidentified problem with environmental iodine. On the other hand, Fuge suggests that the solution may be geological, with iodine being so strongly bound in soils that it is neither available for volatilisation nor for atmospheric deposition on plants [14]. Equally, we must consider the presence of a goitrogen producing or enhancing biochemical iodine deficiency. Goitrogens are discussed in the literature [43], but have yet to find full acceptance in a unified theory of iodine deficiency.

Kelly and Snedden's monumental work [22] mentions geology several times as a possible factor in goitrogenesis. For example, they mention pre-Cambrian basement in Nigeria and limestone in Derbyshire as important regions of goitre occurrence. Derbyshire neck has been noted for generations, being recorded in 1769 [37]. However, the role of calcium is unclear, even disputed [25]; the link between limestone and goitre may be some other factor besides calcium. The early work by the Belgians in Zaire [11] has unanswered points with regard to differing prevalence rates of goitre overlying distinct geologies

43]. Recent work from northern Pakistan, straddling the suture between the Indian and Asian plates, has suggested that plate tectonics could provide a much needed unifying concept for the geological background to the disorders, through the concentration of a goitrogen by geological forces [42].

In Pakistan there was no difference in environmental iodine between the plates on either side of the suture, raising the possibility of a goitrogen [42]. On the subducting plate there was a high prevalence of goitre (61% in adult women, 21% in adult men) while on the non-subducting plate the prevalences were greater (78 and 36% respectively), a statistically significant difference ($p < 0.01$, 95% confidence interval or odds ratio 45-55%) (Fig. 2). A similar level of statistical significance was found when a parallel pair of studies [8, 39] to the west, performed four years apart but by the same team, were re-examined ($p < 0.01$, 95% confidence interval for odds ratio 33-55%) (Fig. 3).

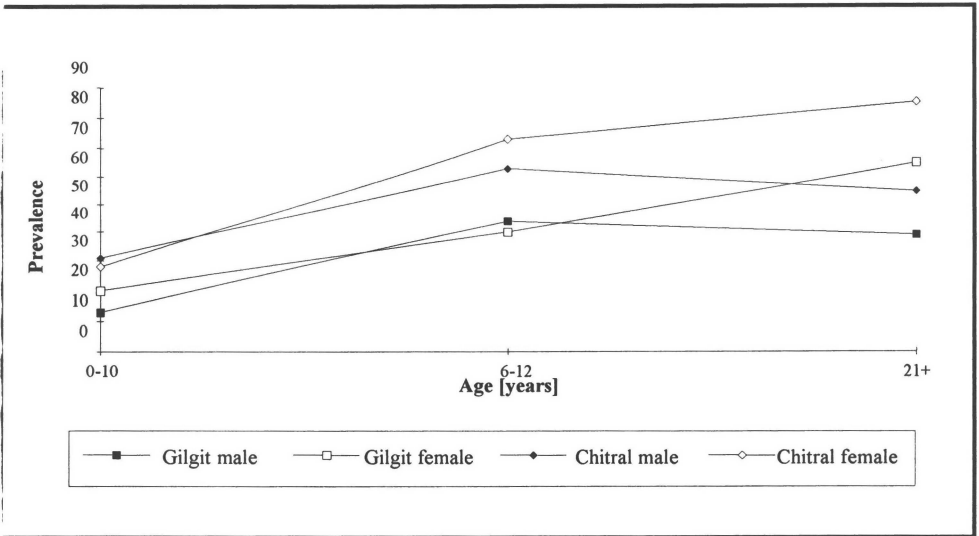


Figure 3. Similar Situation Further West in Pakistan

Gilgit is south of the suture, while Chitral is north of the suture.

Because of these different goitre prevalences it was proposed that the mineral assemblages characteristic of plate tectonics and concentrated in the non-subducting plate act as goitrogens [42]. This is not easy to confirm elsewhere, since comparable studies near suture zones on land are elusive. Nevertheless, the iodine deficiency disorders are found on all the non-subducting plates lying over currently active subduction zones. Obviously, most of these zones are off-shore, thus only the non-subducting plate is inhabited. But the lack of comparable populations does not weaken the hypothesis. Indeed, the hypothesis predicts that the subduction-related goitrogen will be found on some Pacific islands (e.g. Tonga) and not on others (e.g. Tahiti). It may be that some of the anomalies not explained by differences in environmental iodine levels will be explicable by plate tectonic-induced goitrogens, but much work remains to be done.

IODINE AND THE BIBLICAL FLOOD

Iodine Loss

We must turn our attention now to the effect of the Flood on the world iodine balance. By far the most devastating effect of the Flood on iodine status would appear to be the removal of iodine from the environmental cycle (Fig. 1).

It has been calculated that the sedimentary rocks world wide contain 2.9×10^{15} kg iodine, a mass similar to that spread throughout all the igneous rocks [48]. That is, approximately 3×10^7 times the iodine found in the whole of the current biosphere is locked away in the sedimentary layers. If, as appears likely, the sedimentary rocks are a result of the upheavals of the Flood, then it would seem sensible to assume that

any iodine found in them is also a result of the Flood cataclysm, the iodine being buried in and with the sediments which were produced by the scouring of the face of the earth by the Flood waters. Iodine despite its ubiquity, is an unusual element in that it is mainly associated with life, either plant or animal unlike, for example, the other halogens which occur in well recognised geochemical environments. Much of the iodine in the sedimentary rocks is associated with carbon, which suggests that such iodine, like the carbon, has come from the luxurious pre-Flood biosphere. With such an enormous loss of iodine from circulation, is it any wonder that the planet, along with its inhabitants, now teeters on the edge of iodine deficiency? Perhaps the surprise is that there is enough iodine left for normal life to flourish.

That the sedimentary rocks experienced a cataclysmic origin may be indicated by the strength of the iodine concentrations therein, since slow deposition of sediments at uniformitarian rates would almost certainly lead to the loss of iodine. Few modern sediments, besides deep marine sediments, have high iodine levels, a fact prompting Fuge to describe alluvial deposits as one cause of the disorders [13]. Volatilisation of iodine, though poorly understood, is thought to occur easily: Dutch soils developed on what used to be sea floor have been progressively losing iodine. Soils exposed since 1923, 1836, 1718 and 1300 contain 18.50, 12.00, 9.45 and 9.23 mg/kg respectively (quoted in [15]). In a uniformitarian environment over millions, even thousands, of years, such a half-life of 600 years would lead to a low level of iodine in any resulting rock formations if exposure occurred for any length of time, if the assumption of an exponential decrease is valid.

Replenishing Soil Iodine

Whitehead & Truesdale [48] have calculated that, with an annual rainfall of 80 cm (32 inches) containing 1.5 µg/l iodine, in 1000 years the soil iodine content to a depth of 15 cm would be increased by about 5.3 µg/kg soil if it were all retained. Dry deposition based on an atmospheric concentration of 15 ng/m³ and a deposition velocity of 0.2 cm/s would add 9.6 g/ha/year to the ground surface [48], or approximately 4.6 µg/kg soil to a depth of 15 cm in 1000 years if it were all retained (taking the average bulk density of soil to be about 1.4 g/cm³). This means that dry deposition is as important a source of iodine to soil (and plants) as wet. At these present rates of deposition it would therefore take some 5,000 years to reach the current average soil iodine content of about 5 mg/kg, ignoring the probably high initial deposition rate after the upheaval of the Flood. With a recent Flood about 3000 BC today's soil iodine levels are therefore quite achievable, even assuming the extreme position of total lack of iodine in the fresh post-Flood soils. Even if the iodine deposited in the top 15cm of a soil is not all retained it is still possible to attain today's soil levels if there was a high initial deposition rate in the early post-Flood years.

Iodine Toxicity and Thyroid Control

One question which may arise is: if the pre-Flood world contained so much excess iodine over the present situation, would there not be the possibility of toxicity? Could the early biosphere cope with all that iodine in it? Further investigation and calculation is needed to examine this point in detail, but bearing the following particulars in mind I believe that it is unlikely that a major problem will be identified.

It will make little difference if the association of iodine with carbon in the sedimentary rocks is related to animal or to plant life, since the iodine content of both is similar if we exclude animal thyroids (0.05-0.5 mg/kg). Much of the increased pre-Flood iodine was locked away in the biosphere, not being eaten by the early humans and animals. There is 7.0×10^{13} kg iodine currently in the hydrosphere. The shallower oceans pre-Flood would have contained less iodine than currently although the concentration may have been similar at 45-60 µg/l [7]. It is likely, though, that there was much more life and therefore much more iodine in the pre-Flood oceans because of higher temperatures. So, one way or another, on land and sea, the iodine would be found in the biosphere and not directly available in the diet. It does not follow that a vastly greater environmental supply of iodine would lead automatically to disease.

Indeed, the assumption that 'because the pre-Flood world had high iodine levels animal toxicity (thyrotoxicosis) is a likely result' is a misunderstanding based on the same false idea that low environmental iodine levels nowadays lead directly to the iodine deficiency disorders. While environmental iodine is crucial it should be clear that it is not always definitive in terms of health.

The important question is whether plant iodine was greater than current levels, or similar but spread across a greatly enlarged biosphere. If we assume that the daily dietary needs were similar to today in terms of calories, proteins, fats etc., then inhabitants of the pre-Flood world would not be eating

excessively more than what we now eat. As long as their food had similar levels of iodine compared with a good modern diet which does not induce the iodine deficiency disorders there would be no expectation of toxicity in the pre-Flood world, and we can safely assume that the excess iodine was to be found in an enlarged and enriched biosphere. It may even be possible to estimate from the sedimentary iodine levels how much greater that biosphere was.

A further point worth noting is that it may not be just the level of available iodine that is important in the production of disease, but the level experienced in childhood and any subsequent change into adulthood. There is evidence that the type of infant feeding (breast or bottle) affects the adult thyroid hormone levels [36]. This will determine how the individual responds to altered intakes of iodine, since the personal normal fluctuations of thyroid hormones are much less than the community normal ranges. In other words, the pituitary-hypothalamic-thyroid axis maintains a very tight control on the hormone levels, and is open to abuse if the iodine supply is continuously different from the calibration levels in infancy.

There are four topics of interest linked to this: (a) Thyrotoxicosis from excess iodine intake has been shown to be commoner in persons who have had a low iodine intake in childhood [2]. (b) It is possible that this could explain why currently some persons in goitrous regions suffer from goitre while others do not: their personal normal ranges initially were set higher due to an upswing in available iodine, with later downswings initiating a goitre. (c) It may be that this tight control of the thyroid hormones is (part of) the link between environmental and biochemical deficiency, for it seems that some link exists, insubstantial though it currently appears. (d) This programming and the disruption engendered by altered supplies of iodine argue for an inbuilt, pre-set level of minimal function. This is an integral aspect of a complex system; it argues for design and against Darwinian evolution [3].

It is possible that the first three topics are directly linked to the marginal iodine supplies caused by the upheavals of the Flood. Any movement of these supplies now has the potential to produce either endemic goitre or thyrotoxicosis, whereas originally the purpose would appear to be simply to respond to normal variations in iodine supply, not to respond to a supply which is often below the minimum necessary for healthy life.

Evolutionary Difficulties

Furthermore, if the present situations prevailed over a long time, then under uniformitarian conditions the existing low iodine circumstances would be usual, and the world would remain teetering on the edge of a long-term deficiency. The conditions for the production of the iodine deficiency disorders would then be the normal situation throughout history, raising questions concerning the effect of such continuously low conditions on evolution, and in particular the evolution of the thyroid.

In such a continuously low iodine environment the effect on the evolution of the thyroid mechanism would be similar to that affecting the calibration of the thyroid, where early iodine levels set the personal normal range for thyroid hormones for life. Any organism evolving in an iodine-poor environment would not experience the environment as deficient. In other words, the organism would accept the deficient environment as normal. As a result the thyroid would evolve able to utilise such a low supply. The iodine deficiency disorders would not develop, because they could not: there is no iodine deficiency in such a situation to act as a driving force.

It might be argued that fish can develop goitre in iodine-poor water, whether fresh [26] or salt [21], showing that they evolved in the iodine-rich sea. Mammals are therefore suffering from the move out of the sea into the iodine-poor environment of the land; the iodine deficiency disorders are simply a consequence of this move. Those suffering from the deficiency disorders are unfit and live in a poor environment, leading to their elimination. This is a rather simplistic view, both of evolution and of the disorders. All moving creatures need iodine, whether fish, reptile or mammal. Even birds and insects concentrate iodine, the former having similar thyroid function to mammals, although the purpose of iodine in insects is less clear.

Thyroid function is so central to the well-being of the animal that it is unlikely to have survived unchanged when so much else was evolving. Indeed, the pressure of the iodine-poor environment and the unfitness of the disordered should move evolution forward, towards organisms better adapted to deal with such pressures. There is no evidence that this has happened, despite many millions of years, but rather the

opposite. Iodine deficient animals abound, and the iodine deficiency disorders still produce many unfit creatures.

In fact, it is far more likely that iodine-poor environments would retard evolutionary change. The unfitness of those suffering from the disorders includes reduced reproduction. Miscarriage rates are higher, as is neonatal mortality, in regions suffering the disorders. The generalised reduction in IQ in affected communities is indicative of a negative affect, rather than something good. It would seem probable that evolution does not benefit from iodine deficiency.

CONCLUSIONS

- Iodine deficiency is primarily biochemical and not environmental.
- Environmental iodine supplies are marginal.
- The link between the biochemical deficiency and marginal environmental supplies of iodine could be due to programming of the thyroid in infancy.
- The marginal environmental supplies may be due to great losses of iodine in sediments deposited by the Biblical Flood.
- It is possible therefore that the Flood could be the primary cause of the iodine deficiency disorders.
- There may be a secondary mechanism inducing the iodine deficiency disorders through Flood-induced plate tectonics concentrating goitrogens, but this hypothesis needs further development and evaluation.

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