A PERSPECTIVE ON HIGH DOSE IODINE SUPPLEMENTATION – PART II – GUY ABRAHAM, MD AND HIS WORLD VIEW ON IODINE

INTRODUCTION

In part I of this series I departed significantly from the usual format of these newsletters by suggesting that issues of character and credibility need to be examined right along with the science when we are making decisions about whether we should let our nutritional recommendations veer from those that are generally accepted as true by the nutritional community. Specifically, as you may recall, I was highly critical of what appeared to be Dr. Guy Abraham’s personal vendetta against any researcher or clinician who disagreed with his stand on supplemental iodine. Furthermore, I felt strongly that the possibility exists that such obvious distain for those who disagree on the iodine issue could lead Dr. Abraham, who I would guess is as mortal and fallible as the rest of us, to view the body of research and clinical information on iodine with less than total objectivity.

Of course, with the above stated, an old saying needs to be kept in mind:

“Judge not lest ye be judged.”

In the last issue I also suggested that, in passing judgment on such a subjective issue, there exists the possibility that I may be overreacting. In turn, could my ability to accurately read and interpret Dr. Abraham’s various scientific discourses be impaired? So that you can best answer this question, in this issue I am going to do my best to set aside any thoughts on character and intent and focus purely on the scientific and research aspects of Dr. Abraham’s many articles on iodine that have been published within the last 4-5 years. While, as I mentioned in part I, many researchers have published, in my opinion, credible studies that refute Dr. Abraham’s position, I will reserve all but fleeting discussions of these studies for part III.

THE WORLD OF IODINE ACCORDING TO GUY ABRAHAM, MD

For ease of discussion I will be grouping Dr. Abraham’s various commentaries on iodine into seven major subsections. These subsections are:

- Is iodine deficiency a major health concern?
- Is iodine involved in major physiologic pathways outside of those related to thyroid function?
- Do dietary population studies exist that support the idea that iodine intake above RDA levels of 150 mcg per day is healthy?
- Does Dr. Abraham’s condemnation of those researchers who continue to support maximum daily iodine intake of 150 mcg per day have a scientific, biochemical basis?
- Do laboratory tests exist that can be used to clinically ascertain need for supplemental iodine?
- If it is true that daily intake of iodine should be higher than 150 mcg per day for most healthy and ailing populations, what exactly is the right amount and what is the best form?
- What clinical outcomes were observed by Abraham when using milligram dosing iodine?

While many of Dr. Abraham’s discussions focus on the basics of human iodine metabolism, I will not address them here. This is not only due to space considerations but due to the fact these discussions are relatively non-controversial and contain information that can be readily found in most basic nutritional biochemistry texts.

Is iodine deficiency a major health concern?

As you will see when I discuss papers written on the subject from other researchers, this question is actually quite non-controversial. For, many now feel that even after years of adding iodine to table salt, iodine deficiency is a significant issue not only in this country but worldwide. What specifically does Abraham have to say about prevalence of iodine deficiency? The following comes from the article “Optimum levels of iodine for greatest mental and physical health” (1):
“According to a recent editorial of the Journal of Clinical Endocrinology and Metabolism, one third of the world’s population lives in areas of I deficiency, which is the world’s leading cause of intellectual deficiency.”

The later claim is referenced by a major study on iodine nutriture in the US that was written by Hollowell et al and was published approximately ten years ago (2). As you will see in the abstract below, Abraham’s assertion that iodine deficiency is a concern in this country deserves our attention:

“Iodine deficiency in a population causes increased prevalence of goiter and, more importantly, may increase the risk for intellectual deficiency in that population. The National Health and Nutrition Examination Surveys [NHANES I (1971–1974) and (NHANES III (1988–1994)] measured urinary iodine (UI) concentrations. UI concentrations are an indicator of the adequacy of iodine intake for a population. The median UI concentrations in iodine-sufficient populations should be greater than 10 µg/dL, and no more than 20% of the population should have UI concentrations less than 5 µg/dL. Median UI concentrations from both NHANES I and NHANES III indicate adequate iodine intake for the overall U.S. population, but the median concentration decreased more than 50% between 1971–1974 (32.0 ± 0.6 µg/dL) and 1988–1994 (14.5 ± 0.3 µg/dL). Low UI concentrations (<5 µg/dL) were found in 11.7% of the 1988–1994 population, a 4.5-fold increase over the proportion in the 1971–1974 population. The percentage of people excreting low concentrations of iodine (UI, <5 µg/dL) increased in all age groups. In pregnant women, 6.7%, and in women of child-bearing age, 14.9% had UI concentrations below 5 µg/dL. The findings in 1988–1994, although not indicative of iodine deficiency in the overall U.S. population, define a trend that must be monitored.”

Thus, it appears that, even though we are not experiencing an outright iodine deficiency in this country, we are currently heading in the wrong direction in relation to iodine intake. In turn, how much iodine should the average citizen ingest? As you will see shortly, Abraham’s answer to this question appears to be the main reason why so much controversy about iodine nutriture exists right now.

Is iodine involved in major physiologic pathways outside of those related to thyroid function?

One of the main rationales used by Abraham to justify levels of daily iodine intake that are so much higher than RDA levels is his position that iodine utilization in the human body occurs in several different sites other than the thyroid. This suggestion by Abraham has, in turn, generated controversy primarily due to the fact that most conventional nutritional texts and instructors have long stated strongly that iodine’s only physiologic role in the human body is maintenance of thyroid function. Does Abraham present a strong case for the idea that we should start thinking of iodine as a “whole body” nutrient as opposed to a “thyroid” nutrient? While you certainly need to be your own judge on this issue, I do feel that Abraham supports this contention well.

In support of his hypothesis that iodide plays many extra thyroidal roles in human health, Abraham first presents some epidemiologic information (1):

“B.V Stadel, from the National Institute of Health, proposed in 1976 to test the hypothesis that the lower incidence and prevalence of breast dysfunctions and breast Ca; and the lower mortality rate from breast, endometrial and ovarian cancers observed in Japanese women living in Japan versus those women living in Hawaii and the continental US, was due to their I intake.”

In terms of more direct, intervention research, though, those studies that suggest an important relationship between iodine and breast health, particularly in relation to fibrocystic breast disease are, to me, most compelling. Abraham (1) states:

“Data are available, however, regarding the effects of I, ingested in daily amounts of several mg on subjective and objective improvements of fibrocystic disease of the breast (FDB). In 1966, two Russian scientists published their results regarding the effect of oral administration of potassium iodide in daily amounts of 5-10 mg in several mg on subjective and objective improvements of fibrocystic disease of the breast (FDB). In 1966, two Russian scientists published their results regarding the effect of oral administration of potassium iodide in daily amounts of 5-10 mg in 200 patients with ‘dyshormonal hyperplasia of mammary glands’. They postulated that this form of mastopathy was due to excess estrogens from ovarian follicular cysts which were caused by insufficient consumption of I. The duration of I supplementation of their patients varied from 6 months to 3 years. Within 3 months, there was significant reduction of swelling, pain, diffuse induration and nodularity of the breast. Out of 167 patients who completed the program, a positive effect was observed in 72% of them. In five patients with ovarian follicular cysts, there was a regression of the cystic ovaries following 5 months to one year of I supplementation. No side effects of I
supplementation was reported in those patients.”

Abraham (1) then goes on to discuss some compelling research on iodine and fibrocystic breast disease by Ghent et al (3):

“Ghent et al extended the Russian study further, using different amounts of different forms of I in women with FDB. Beginning in 1975, these Canadian investigators tested various amounts of various forms of I in three open trials. Lugol 5% solution was used in 233 patients for 2 years in daily amounts ranging from 31 to 62 mg I. They achieved clinical improvement in 70% of the patients. Thyroid function tests were affected in 4% of the patients and iodism was present in 3% of them. In 588 patients, using iodine caseinate at 10 mg/day for 5 years, only 40% success rate was achieved. In 1365 patients, using an aqueous saturated solution of iodine in daily amount based on body weight, estimated at 3-6 mg I/day, 74% of the patients had clinical improvements both subjectively from breast pain and objectively, from breast induration and nodularity. Iodism was present in only 0.1% in this last group. In a double blind study of 23 patients ingesting aqueous solution of iodine in amounts of 3 to 6 mg/day for a mean of 191 days, 65% showed objective and subjective improvement whereas in 33 patients on a placebo, 3% experienced worsening of objective signs and 35% experienced improved improvement in subjective breast pain….Although the percent of subjects reporting side effects in Ghent’s studies appear high ranging from 7% to 10.9%, the authors stated that the incidence of iodism was relatively low and most complaints were minor such as increased breast pain at the onset of I supplementation, and complaints about the unpleasant taste of iodine.”

Of course, after reading the preceding studies you may wonder whether the iodine is exerting more of a pharmaceutical effect or is functioning as an essential nutrient that has long been depleted in the breasts of the affected patients. In “The concept of orthioiodosupplementation and its clinical implications” (4), Abraham suggests that the answer is the latter:

“The mammary glands can effectively compete with the thyroid gland for peripheral iodine. Eskin, et al, measured the 24-hour radioiodide uptake in 57 clinically normal breasts, and in eight clinically abnormal breasts. The mean ± SD percentage uptake was 6.9±0.46% in the normal breasts and 12.5±1% in the abnormal breasts. These means were statistically significant at p<0.005. Considering that these measurements are representative of a single breast, and a woman has two breasts, the percentage uptake per patient is twice these amounts. This brings the 24-hour radioiodide uptake by the mammary glands of a woman in the same range as the 24-hour radioiodide uptake by the thyroid gland. The higher percentage uptake in the abnormal breasts suggests that the abnormal breasts were more deficient in elemental iodine than normal breasts.”

Do other parts of the body besides the thyroid and female breast metabolize iodine to a significant extent? According to Abraham (4) the following can be stated about the skin and iodine:

“In rats studied by Thrall and Bull, 20% of the iodide, but not iodine, administered orally was recovered in the skin. This suggests that the skin, like the thyroid gland, has a preference for iodide.”

Abraham (4) also presents suggestive in vitro and in vivo research that the following may also metabolize iodine:

- Human leukocytes
- Salivary glands
- Stomach cells
- Adrenal glands

Given that RDAs for iodine intake are strongly based on the assumption that the thyroid is the only part of the body that requires iodine to a significant extent, I feel that Abraham makes a strong argument that, because, in reality, other parts of the body appear to require iodine besides the thyroid, optimum daily intake of iodine should be higher than RDA levels. Of course, this brings us back to the central controversy. How much iodine is enough? I will comment more directly on this question shortly.

Do dietary population studies exist that support the idea that iodine intake above RDA levels of 150 mcg per day is healthy?

The primary epidemiologic basis for Abraham’s recommendations of milligram levels of supplemental iodine is the studies he references concerning the traditional iodine intake of Japanese populations. Abraham (1) states:

“Due to the large consumption of seaweeds in the Japanese diet, this population ingests several milligrams of I daily without ill effects and in fact with some very good results evidenced by the very low incidence of
fibrocystic disease of breast and of the low mortality rates for cancers of the female reproductive organs."

Furthermore, Abraham (5) suggests that because of this high iodine intake, the Japanese are very healthy overall:

‘Pittman, et al, were referring to mainland Japanese who consume a daily average of 13.8 mg (13,800 µg) of iodine from seaweed when they mentioned ‘groups ingesting diet unusually rich with iodine.’ Based on statistics generated some 20 years ago, mainland Japanese represent one of the healthiest nations on earth.”

How much seaweed and iodine does the average Japanese individual ingest? Abraham (1) continues:

“According to the Japanese Ministry of Health and Welfare, the average daily intake of seaweed is 4.6 gm. At an average of 0.3% I content (range = 0.08-0.45%), that is an estimated daily I intake of 13.8 mg. Japanese living in the coastal areas consume more than 13.8 mg.”

In discussing the rationale for the amount of supplemental iodine used for his various experiments, Abraham repeatedly uses levels of Japanese intake as justification. Of course, inherent in this justification is the assumption, as suggested by Abraham above, that the Japanese benefit from this level of iodine intake. As you will see in the next newsletter, not all Japanese researchers agree with Abraham’s hypothesis that everyone in Japan derives benefit from milligram levels of daily iodine intake. Why is this significant? Given that so much of Abraham’s justification comes from Japanese data, any evidence that weighs against a relationship between good health in Japanese populations and iodine intake represents a serious concern in terms of following Abraham’s recommendations concerning milligram dosing of supplemental iodine for the general population.

**Does Dr. Abraham’s condemnation of those researchers who continue to support maximum daily iodine intake of 150 mcg per day have a scientific basis?**

As I suggested in part I of this series, by far the most controversial aspect of Abraham’s writings on iodine is his condemnation of those clinicians and researchers who feel that iodine can only be used safely in microgram doses. Of course, as you know, I have made it clear that I feel Abraham’s manner of communication on this issue is clearly inappropriate. However, just because Abraham’s style of communication on this issue is a bit “unconventional” so to speak, I still feel that the scientific statements that led to Abraham’s passionate character attacks should be investigated as a separate issue. While many of Abraham’s papers contain criticisms of the iodine “establishment,” the paper that is clearly devoted to this issue is “The history of iodine in medicine part III: Thyroid fixation and medical iodophobia” (5). Therefore, I would like to begin my exploration of Abraham’s criticisms with this paper.

To begin this paper, Abraham discusses some of the earliest reports on the use of supplemental iodine, starting with the work by Marine:

“In the early 1920s, Marine reported a positive effect from iodide supplementation at 9 mg/day in the prevention of simple goiter among adolescent girls. That amount of iodine was based on research performed on farm and laboratory animals regarding the effect of iodine on thyroid function and also overall performance.”

Of course, during this time iodization of salt was instituted very successfully on a mass basis. Abraham (5) states:

“As a public measure to control goiter, iodization of table salt was implemented successfully in the US between 1971 and 1924. That is, iodization of table salt was successful in decreasing markedly the incidence of simple goiter in the supplemented population. Keep in mind that the amount of bioavailable iodine (0.05 mg/day) needed to prevent cretinism, endemic goiter, and hypothyroidism is 60 times less than the amount of iodide (9 mg/day) used by Marine in the original studies.”

Before the iodization of salt, how was the public obtaining iodine, other than from food? Abraham (5) elaborates:

“Prior to the iodization program, the public was relying on iodine preparations from apothecaries for their iodine needs. The recommended daily amount of iodine was 0.1-0.3 ml Lugol containing 12.5-37.5 mg elemental iodine.”

Interestingly, though, with the advent of salt iodization, use of the Lugol solution was largely abandoned, even though the levels of iodine obtained from salt was much less than that which was obtained from Lugol solution. Abraham (5) comments:
“Iodized salt was unfortunately used as a substitute for the previously recommended forms of iodine/iodide. The bioavailable iodide from iodized salt is only 10% of the estimated 0.75 mg iodide in iodized salt consumed per day. That amount, 0.075 mg of bioavailable iodide, represents less than 1% of the amount of iodide used in the Marine’s study (i.e., 9 mg) and also less than 1% of the recommended intake from Lugol solution.”

Then, in the 1930s, the idea of using supplemental iodine routinely took another major blow with the discovery of thyroid hormones and hormone replacement therapy. Abraham (5) states:

“With the availability of thyroid hormones in the 1930s, iodine was completely ignored by Thyroidologists in the treatment of iodine deficiency-induced goiter and hypothyroidism. A textbook, Diagnosis and Treatment of Diseases of the Thyroid, edited by Amy Rowland and published in 1932, contained chapters from 24 thyroidologists of that time. Although the most common cause of hypothyroidism and simple goiter worldwide is iodine deficiency, the recommended treatment of hypothyroidism was summarized in two sentences: ‘The treatment of hypothyroidism of any type consists merely in the substitution of thyroid extract for the deficient secretion. Any form of prepared gland or the active principle, thyroxin, may be used.’”

According to Abraham (5), another “…nail in the iodine coffin…” occurred when, in the late 1940s, two researchers named Wolff and Chaikoff performed animal research that suggested large doses of supplemental iodine would temporarily suppress thyroid function. This effect where large doses of supplemental iodine temporarily suppress thyroid function has become known as “the Wolff-Chaikoff effect.”

Eng et al (6) define the Wolff-Chaikoff effect more specifically in the quote below:

“Autoregulation in the thyroid refers to the regulation of iodine metabolism within the thyroid gland, independent of TSH. It was first reported by Morton et al in 1944, who observed that large amounts of iodide inhibited the formation of thyroid hormones by incubated sheep thyroid slices. Wolff and Chaikoff then reported that organic binding of iodide within the rat thyroid was blocked when the plasma iodide level achieved a critical threshold. This inhibition defines the Wolff-Chaikoff effect. They next demonstrated that this inhibitory effect of excess iodide was transient, lasting from 26-50 h, and that the thyroid escaped or adapted to prolonged iodide excess, resuming near-normal hormone synthesis.”

Concerning the research by Wolff and Chaikoff, Abraham (5) states the following:

“The first nail in the iodine coffin was the publication by Wolff and Chaikoff from UC Berkeley in 1948, describing their findings in rats administered iodide in increasing amounts by intraperitoneal injection. When serum inorganic iodide levels reach 0.2 mg/L, that is 10-6M, radiiodide uptake by the thyroid gland became undetectable. The correct interpretation would be: Iodide sufficiency of the thyroid gland was achieved when serum inorganic iodide levels reach 10-6M, as we previously discussed. But Wolff and Chaikoff concluded that serum inorganic iodide levels at a concentration of 10-6M blocks synthesis of thyroid hormones, resulting in hypothyroidism and goiter. These authors did not measure thyroid hormones in the rats studied. Hypothyroidism and goiter were not observed in those rats. This fictitious phenomenon became known as the Wolff-Chaikoff effect.”

According to Abraham (5), this research initially did little to stop physicians from using the large doses of iodine that had been discussed in the previous quotes. However, in 1969, Wolff published the paper “Iodide goiter and the pharmacologic effects of excess iodide” (7) This paper, in which Wolff discussed his research in terms of human application, had a major impact on the use of supplemental iodine by physicians. Abraham (5) states:

“The second and final nail in the iodine coffin was hammered in by Wolff in 1969. By 1969, Dr. Wolff had moved to the National Institute of Health. He arbitrarily defined four levels of ‘iodine excess.’ The first level of excess started with intake above 0.2 mg/day, and iodide intake of 2 mg or more was considered ‘excessive and potentially harmful.’ By the 1970s, physicians concluded that one must avoid inorganic, non-radioactive iodine like leprosy, unless it was incorporated into toxic, organic iodine-containing drugs. Then iodine could be tolerated because iodine could be blamed for the toxicity of these drugs.”

As I assume you can infer, Dr. Abraham does not agree with the way Drs Wolff and Chaikoff interpreted the results of their research and certainly does not agree with the clinical...
recommendations made by Wolff based on that research. Just how passionately does Abraham disagree with the findings of Wolff and Chaikoff and the recommendations by Wolff? Consider these statements from the paper written by Abraham entitled “The Wolff-Chaikoff Effect: Crying wolf?” (8):

“Shortly after the Axis powers capitulated and World War II came to an end, UC-Berkeley dropped a bombshell in 1948, which became known as the Wolff-Chaikoff (W-C) effect. Where the swords of many nations failed, the pens of two men succeeded. The W-C effect resulted in the removal of iodine from the food supply, and most likely caused a lot of misery and death in the US due to its negative impact on iodine consumption by the population and on the use of inorganic, non-radioactive iodine in medical practice.”

Abraham continues:

“To the disgrace and stupidity of the medical profession, US physicians swallowed the W-C forgery uncritically, which resulted in a moratorium on the clinical use of inorganic-non-radioactive iodine in effective amounts. However, this moratorium did not include toxic organic iodine-containing drugs and radioiodide. The iodophobic mentality prevented further research on the requirement for inorganic iodine by the whole human body, which turns out to be 100-400 times the very recently established RDA. Prior to World War II and the W-C publication, US physicians used Lugol solution safely, effectively and extensively in both hypo- and hyperthyroidism. Wolff and Chaikoff acknowledged the excellent and dramatic results achieved consistently with the use of Lugol solution in hyperthyroidism. But they postulated erroneously that these results were due to the fictitious W-C effect.”

As I hope you can see, of all the controversies concerning iodine supplementation discussed so far in relationship to Dr. Abraham, the Wolff-Chaikoff effect may be the most controversial and most important. For, if, as Abraham suggests, the effect of large doses of iodine on the thyroid is physiologic slowing due to iodine saturation, then it appears that we can, with a clear conscience, do as Abraham suggests by routinely supplementing iodine at levels that are several thousand percent higher than the RDA for iodine until saturation levels are reached. However, if, as Wolff-Chaikoff suggests, large doses of iodine are creating a transient disruption of thyroid function that can create or exacerbate hypo- or hyperthyroidism, then we need to continue to proceed very cautiously when contemplating milligram dosing of supplemental iodine.

While I must admit that I do find Abraham’s biochemical rationale against the Wolf-Chaikoff effect very compelling, I cannot yet accept it as fact. Why? As you will see in the next issue, clinical feedback from you plus a significant volume of research not reported by Abraham makes a very strong argument that Abraham’s biochemical rationale, even though it is very compelling, is, in fact, incorrect.

Do laboratory tests exist that can be used to clinically ascertain need for supplemental iodine?

**The iodine/iodide loading test** - To ascertain the need for milligram dosing of supplemental iodine, Abraham employs an iodine loading test. As noted in “The safe and effective implementation of orthiodosupplementation in medical practice” (9) a tableted form of iodine is used where each tablet contains 5 mg of iodine and 7.5 mg iodide as the potassium salt. In this paper Abraham briefly describes the loading test and how it is interpreted:
“We chose four tablets for the loading test. Sufficiency of the whole human body for iodine/iodide was arbitrarily defined as 90% or more of the ingested amount excreted in the 24-hour urine collection, using 50 mg of the iodine/iodide preparation (four tablets).”

In Abraham’s experience, what supplemental protocol was necessary to bring those patients who excreted less than 90% of the loading dose up to that level?

“Orthoiodosupplementation with one tablet a day required up to 18 months to achieve sufficiency in some patients, and in others, sufficiency was not achieved even after two years of supplementation with one tablet/day. With 3-4 tablets /day, however, sufficiency was achieved within three months in most cases. These data support keen observations of clinicians over the past century, regarding the amount of iodine/iodide needed for iodine/iodide supplementation, 12.5-37.5 mg elemental iodine from Lugol solution.”

It should also be noted that Abraham (5) recommends the following in order to maximize the accuracy of the loading test:

“The iodine/iodide loading test to assess whole body sufficiency for iodine becomes more accurate by implementing a complete nutritional program for one month prior to the loading test. In cases of iodine transport inefficiency, the high urinary excretion of iodide would give the false impression of iodine sufficiency. By correcting this inefficiency of the iodine transport system through nutritional intervention prior to performing the loading test, this test becomes more accurate.”

Finally, Abraham (5) mentions these caveats about the loading test:

“The loading test is not reliable in patients on antithyroid drugs which inhibit oxidation and organization of symported iodide in the target cells. This results in a high urinary excretion of iodide, and gives the false impression of whole body sufficiency.”

As with Abraham’s hypothesis concerning the Wolff-Chaikoff effect, I do find this loading test and its interpretation very compelling. However, the interpretation assumes that there is a direct correlation between the amount of iodine ingested and the amount excreted in the urine. Does this direct correlation, in fact, actually exist? As you will see in the next issue, a notable expert in the nutrition community has doubts about the validity of this correlation.

If it is true that daily intake of iodine should be higher than 150 mcg per day for most healthy and ailing populations, what exactly is the right amount and what is the best form?

As you probably know by now, the amounts of iodine that Abraham recommends and their impact on human health are the main issues that are stirring up so much controversy and strong emotions. However, because Abraham’s papers do not have specific sections that definitively state optimal amounts of iodine intake for specific populations, I find it difficult to answer this question succinctly. Fortunately, as had been discussed, Abraham is quite specific as to the optimal form of iodine supplementation. Therefore, in relation to optimal amounts, I would like to begin this discussion by reiterating that Abraham bases many of his attitudes concerning iodine nutriture on dietary habits of the Japanese. How does the US population compare with the Japanese in terms of iodine intake? Abraham (10) states:

“Currently, the average daily intake of I by the US population is 100 times less than the amount consumed by the mainland Japanese.”

With the assumption that the average daily intake of the US population is RDA amounts, it would follow that Abraham recommends 100 times the RDA to accomplish optimal iodine nutriture. As you will see in the following quote from Abraham (9), this is exactly what he recommends:

“Based on a review of the literature, we computed the daily amount of iodine/iodide needed for sufficiency of the thyroid gland and the whole human body. This amount, called orthoiodosupplementation, amounted to 100 times the RDA.”

Given that the current RDA is 150 mcg per day, the optimal amount of daily iodine intake recommended by Abraham is 15 mg per day. However, when he discusses amounts more specifically, he typically does not refer to this amount. Instead he speaks most often of 12.5 mg per day. Abraham (10) points out:

“Based on the information previously discussed, the optimal daily I intake for I sufficiency of the whole human body would be equivalent to 2 drops of Lugol solution.”

The author continues:

“Administration of I in liquid solution is not very accurate, may stain clothing, has an
unpleasant taste and causes gastric irritation. We decided to use a precisely quantified tablet form containing 5 mg iodine and 7.5 mg iodide as the potassium salt. To prevent gastric irritation, the iodine/iodide preparation was absorbed unto a colloidal silica excipient, and to eliminate the unpleasant taste of iodine, the tablets were coated with a thin film of pharmaceutical glaze."

Beyond the population studies on the Japanese, how did Abraham decide that 12.5 mg per day of iodine was optimal? The answer is he also made decisions on the iodine needs for organs in the body besides the thyroid. The author (10) notes:

“So far, the optimal daily requirement for I has been estimated at 6 mg of iodine for the thyroid gland and 5 mg of iodine for the mammary glands. The adrenal glands may also require adequate levels of I for normal function.”

Of course, it must be kept in mind that these recommendations are for the general population to maintain optimal, total body iodine levels. What about the patient who is already significantly iodine depleted? In these situations, Abraham recommends, as was suggested above, much larger doses that are administered only after the iodine loading test is performed. As an example of this approach to correction of iodine deficiency, please consider the following experiment performed by Abraham (4):

“Another group of six subjects, (three male and three female) were evaluated with 24-hour urinary iodide levels after ingesting one, two, and three tablets of the same preparation. The mean percentage excretions (± SD) were: 22±1.2% for one tablet, 23±2.8% for two tablets, and 25±12.3% for three tablets. In a third group of six subjects, urine iodide levels were evaluated following four tablets of the same preparation. The mean excretion rate was 39±17.2%. For the loading test, a single ingestion of four tablets was chosen because this dose resulted in the highest mean percent iodide excreted and in the widest interindividual variations.

Because of the improved overall well-being reported by the subjects who achieved 90% or more iodide excreted, sufficiency was arbitrarily defined as 90%. Implementation of orthoiodosupplementation, based on the loading test, revealed that sufficiency was not achieved in some subjects even after two years of iodine supplementation at 1-2 tablets/day. To achieve sufficiency within three months, most subjects required 3-4 tablets/day (37.5-50 mg).”

In the next installment in this series I will address in earnest the last and what might be the most controversial question of all:

**What clinical outcomes were observed by Abraham when using milligram dosing iodine?**

Did the patients in Abraham’s experiments experience any side effects relating to the Wolf-Chaikoff effect or any other factors? Did the patients experience positive outcomes? Is the protocol so safe that the public can self-administer milligram doses of iodine or does this approach to iodine nutrition always require supervision by a knowledgeable health care professional?

In addition, so that you can make your own decisions as to whether Abraham’s many claims of infinite intellectual, analytical, and clinical superiority over all others in the world of iodine biochemistry and supplementation truly have merit, I will review the writings of other researchers and clinicians. By examining these writings, I feel it should become readily apparent to all of us whether or not the positions on iodine taken by these authors are truly as worthless and, sometimes, malevolent as Abraham suggests.

**REFERENCES**