Use of major dietary iodine sources in married student households
by Elizabeth Kathleen Divers Jacobs

A thesis submitted to the Graduate Faculty in partial fulfillment of the requirements for the degree of
MASTER OF SCIENCE in Home Economics
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Abstract:
A survey was conducted to investigate the extent of iodized salt usage, reasons for the purchase of iodized or uniodized salt, from what sources information concerning the use of iodized salt is obtained, and the frequency with which fish, marine and shell, are served in households in the married student housing at Montana State University, Bozeman. Structured interviews were conducted with 66 household members responsible for choosing the food their families purchased.

Survey results showed that 72.7% of the households use iodized salt, and 83.3% have fish served at least twice a month. Uniodized salt was used and fish was served less than twice a month in 9.1% of the households . Over half of the respondents (54.5%) use iodized salt because it is better for their health, and 42.4% chose the type of salt they bought by picking up the first package they saw-. Mothers were the source of knowledge about iodized salt usage for the highest percentage of household members (43.6%), with schools a close second (40.0%).

It was concluded that a state-wide medical survey of goiter enlargement in Montana would be valuable to justify programs to increase iodized salt usage, such as education of mothers and school children.

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Signature Elizabeth Kathleen Davis Jacobs

Date August 14, 1970

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ELIZABETH KATHLEEN DIVERS JACOBS

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Approved:

Marjorie Kiser by Angelia Ohata
Head, Major Department

Andrea L. Pagenkopf
Chairman, Examining Committee

K. Goering
Graduate Dean

MONTANA STATE UNIVERSITY
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ABSTRACT

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Survey results showed that 72.7% of the households use iodized salt, and 83.3% have fish served at least twice a month. Uniodized salt was used and fish was served less than twice a month in 9.1% of the households.

Over half of the respondents (54.5%) use iodized salt because it is better for their health, and 42.4% chose the type of salt they bought by picking up the first package they saw. Mothers were the source of knowledge about iodized salt usage for the highest percentage of household members (43.6%), with schools a close second (40.0%).

It was concluded that a state-wide medical survey of goiter enlargement in Montana would be valuable to justify programs to increase iodized salt usage, such as education of mothers and school children.

CHAPTER I

INTRODUCTION AND PURPOSES

Introduction

Goiter is not a new disease: references to a swelling of the neck go back to the Chinese in the second millenium B.C. (1). There have therefore been reports of goiter for many centuries, but that does not mean the disease is no longer a problem: preliminary results of the 1968 National Nutrition Survey, conducted by the United States Public Health Service's Nutrition Program, indicate a 5% incidence of enlarged thyroids, or goiter (2).

Retardation, both physical and mental, occurs when generations are subjected to iodine deficiency (3). Bozeman, Montana, has extremely small amounts of iodine in its water supply (4), and a concomitant lack of iodine in the soil. There is little iodine to be found, therefore, in the food produced in the Bozeman area, so other sources of iodine must be used. Iodized salt and marine or shellfish are the two major nutritional sources of iodine (3).

Purposes

This study was undertaken to investigate the following trends among students living in married student housing at Montana State University: (1) the extent of iodized salt usage; (2) reasons for the purchase of iodized or uniodized salt; (3) from what sources information concerning the use of iodized salt is obtained; and (4) the frequency with which fish, marine and shell, are served. It was hoped that these trends would lead to conclusions regarding the following possibilities: (a) whether a state-wide goiter survey in Montana would be of value; and (b) if education on purposes and methods of goiter prophylaxis is necessary.

CHAPTER II

REVIEW OF LITERATURE

Before a discussion of the survey that was conducted, this chapter will present some background information about why there is a need for iodine by the human body and how much iodine is required. It is important to know how the body uses iodine, what may be the consequences of an iodine deficiency, and what methods are available for preventing these deficiency diseases, for they are rarely cured once they are well established in a human (1).

The Metabolic Need for Iodine by Humans

Use of Iodine by the Thyroid Gland

Iodine is essential in the manufacture of thyroid hormones, which affect a human's rate of growth and development. Thyroid hormones are produced in the thyroid gland.

The Thyroid Gland

In 1965 Wilson et al wrote: "All tissues and secretions of the body thus far analyzed have been found to contain iodine" (3: 167). Of the 20-50 milligrams of iodine found in the human body, however, 20-40% of it is located in the thyroid gland (5). The name "thyroid," which means "shield-shaped," was given to the gland in 1656 by Thomas Wharton (1,6). Figure 1 illustrates the placement of the thyroid

with respect to a human's trachea, or windpipe. Fibrous connective tissue supports and creates a framework for the gland. The active, functional tissue consists of a number of follicles which resemble the sacs of an orange section, except for being spherical and microscopic. Epithelial cells of the follicle wall manufacture thyroid hormones (Figure 2). A colloid fluid in the center of the cell stores the hormones (6).

Biosynthesis and metabolism of thyroid hormones

The biosynthesis and metabolism of thyroid hormones is a complex process that is not yet fully understood. Baumann, in 1895 (5) or 1896 (1), first demonstrated that iodine was a normal constituent of the thyroid gland. Iodine is consumed in organic or inorganic food forms, and readily absorbed into the blood stream from the small intestine (3). The blood ionizes iodine (I_2) to iodide (I^-). Iodide is the chemical form in which the mineral is transported from the blood into the thyroid gland (7,8,9,10). Iodide is then concentrated and stored in the colloidal matter of the thyroid cell (3,9,10). When the need for thyroid hormones arises, iodine is removed from storage and combined with thyroglobulin, a protein (3,8,9,10).

Thyroxine is the most abundant thyroid hormone. It is eventually formed by the addition of iodine to tyrosine, an amino acid, within the thyroglobulin molecule. Enzymes then release thyroxine from the

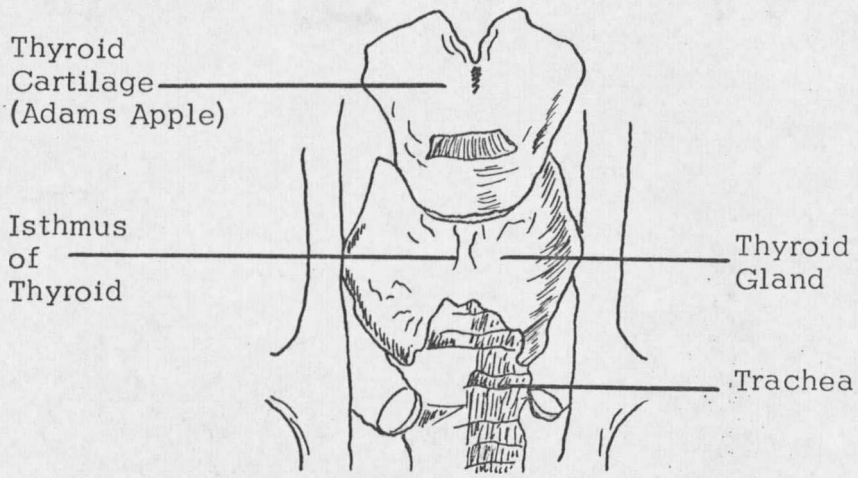

Figure 1--The Thyroid Body
(Sebrell, W. 1949. Iodine--a food essential. Pub. Health Rep. 64: 1077)

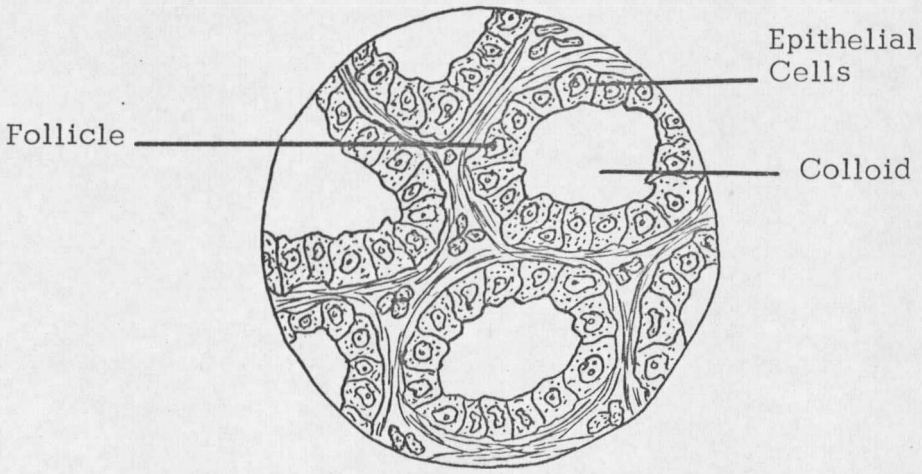

Figure 2--The Follicles of a Human Thyroid
(Sebrell, W. 1949. Iodine--a food essential. Pub. Health Rep. 64: 1077)

protein, and it is transported into the blood (8,9,10). There are three proteins that carry thyroxine in the blood to body tissues (11,12).

The catabolism of thyroxine exemplifies the body's ability to conserve its important constituents. While the breakdown of thyroxine usually begins in the liver, the kidney and muscle tissues may also initiate catabolism (12,13). The liver releases thyroxine from its carrier protein and secretes it into the intestines by way of the bile. Ninety-seven per cent of the thyroxine that reaches the small bowel is re-absorbed into the blood (12). This conservation of thyroxine means the thyroid gland only has to replace three per cent of the thyroxine it secretes.

Functions of thyroid hormones

"Investigating the mechanism of action of the thyroid hormones is somewhat like peeling the successive layers of an onion. Not only may it bring tears to the eyes, but after each successful step, one is left with the layer beneath" (14: 267). A discussion of how the thyroid hormones perform their functions is not, therefore, within the limitations of this study; what happens when thyroid hormones are or are not present will be discussed.

The major functions of thyroxine involve rates of energy metabolism and growth and development. Oxidation rates in the cells of a human body are affected by thyroxine levels (3,14). An increase in thyroxine secretion increases energy metabolism, and therefore basal

metabolic rate (3). Normal development at puberty is also dependent on proper thyroid function (15). A deficiency of thyroxine causes sterility in animals (3), and improper fetal growth in humans (16).

Consequences and Prevention of Iodine Deficiency

Simple and toxic goiter

The formation of a goiter is a mechanism by which the thyroid compensates for a lack of iodine (3). An iodine deficiency results in a lower level of thyroxine in the blood, sensitizing the pituitary gland, which activates the thyroid (17). A decrease in blood thyroxine, therefore, stimulates the thyroid. It enlarges to become more efficient, forming a goiter (3,17,18). Chatin, in the late nineteenth century, found a correlation between a lack of iodine intake and goiter (1). After Baumann's discovery of iodine in the thyroid, and a demonstration by Hopkins in 1912 that disease could be caused by a lack of trace elements, Chatin's theory was accepted.

Stages of goiter formation.--There are three stages of simple goiter, the last of which may eventually lead to toxic goiter. When there is a low iodine intake, the colloid center of thyroid follicles contains less iodine (6). The cells then increase in number and size to compensate. Parenchymatous goiter is the first stage (6). The thyroid's epithelial cells multiply abnormally (hyperplasia) when the iodine content of the thyroid goes below 0.1% (15). The result is a

hard, symmetrical goiter. Multiplication of the cells may continue until exhaustion, or it will stop and the follicles fill with colloidal matter. At this second stage the goiter is colloidal in nature, and very soft (6). The iodine content of the gland may be adequate for a thyroid of normal size, but there is not enough for the thyroid's increased size. After a first or second stage goiter, an adenomatous goiter may form (6). Adenomas are asymmetrical and uneven. They usually occur after thirty years of age in humans. After fifteen years of an adenomatous goiter, a toxic goiter may develop (6): the thyroid becomes hyperactive, so the thyroid hormones are present in the blood at toxic levels. Thyrotoxicosis can be fatal.

Etiology.--There are several etiological factors in simple or endemic goiter: dietary iodine deficiency, goiter-producing agents, or goitrogens in the diet, excessive intakes of calcium or fluorine, and heredity (19). A deficient intake of iodine may be absolute or relative (20,21). Absolute deficiency is the result of a very low dietary intake, whereas excess requirements for iodine by the body will cause a relative deficiency of iodine (21).

A low dietary intake of iodine may not be solely due to a bad choice of food sources. The physical environment of the area in question can be a large factor. Foods produced in certain areas of the world contain little or no iodine, as a result of low iodine levels in

the soil and water (20). After the last Ice Age, glaciers receded, sweeping away the iodine-rich soil. Then a process of replenishing the soil began, usually by air-borne iodine from the sea. It has been found that the shorter an area's post-glacial period, the greater the likelihood of endemic goiter (20). Areas with a great deal of rain and flooding lose soil iodine; cropping also depletes the soil's iodine content (20).

Soil and water content are not the only physical factors that limit dietary intake of iodine. A cold climate will indirectly influence the prevalence of endemic goiter as a result of an increased demand for thyroid hormones to regulate the body's metabolic rate (20). Iodine intake may not be sufficient to supply this increased requirement for thyroid hormones. Variations in sunlight and rainfall have been suggested as a factor that influences the goitrogenic properties of some foods (20). Proximity to the sea may also be important in the availability of sea foods, which have a high iodine content (20).

Inhabitants of some areas of goiter endemicity consume a very monotonous diet, which may be part of their cultural heritage. If the food is produced in an area with very little natural iodine, their diet will be deficient in this element (20).

Dietary intake of iodine may be adequate, and its absorption normal; yet thyroid enlargement will still occasionally occur. Extensive research has been done on goiter-producing agents in food (22).

Goiter has been produced in rabbits fed cabbage diets (3). Blum suggested that the goitrogen in cabbage may inhibit reactions in the liver that supply iodine to the thyroid gland (22). Clements and Wishart investigated possible reasons for the failure of a goiter prophylactic attempt with Belgian children (23). They found that dairy cows were consuming an uncooked forage, chou-moellier, that was known to contain a goitrogen. This forage was from the Cruciferae brassica family which also contains cabbage (23). The presence of a goitrogen in vegetable oils has been suggested: rats fed these oils had increased iodine requirements. Other foods that may contain goitrogens are: cauliflower, turnips, Brussel sprouts, kale (2), rutabaga, filbert nuts, walnuts, canned baked beans, string beans, and peas (25). Ground-nuts or peanuts (26), peaches, pears, strawberries, spinach, carrots, and perhaps soybeans are a few more of the many possible goitrogen-containing foods (20).

Under certain conditions, there is an increased need for iodine by the body (21,26). If iodine intake does not meet these excess requirements, a deficiency will result (27). During fetal life, puberty, pregnancy, lactation, and menopause, "Growth, differentiation, and energy transformations are the greatest" (27: 1463). These periods are when goiter most frequently develops. Other factors which may increase the body's need for iodine include infection, injury, and the presence of toxic agents in the body (27).

Excessive mineral intake and heredity are two etiological factors of goiter that have been investigated. There have been some reports of excessive calcium or fluorine intakes causing goiter in animals (19). However, Demarchi et al. found that areas in Iraq with hard water had no goiter endemicity (28). Extensive research has been performed on the factor of heredity in goiter etiology. Early conclusions indicate that heredity is a definite factor in susceptibility to thyroid enlargement (29). The inheritance of a susceptibility to goiter may differ in different families: either recessive or dominant genes, and sex-linked or non-sex-linked genes (29).

Incidence.--Incidence of goiter in certain areas of the United States is much greater than in other areas (Figure 3). In 1920 a goiter survey was made by the United States Draft Board (4). They considered goiters only so large that military collars could not be buttoned around the draftee's neck. Montana had the fourth largest ratio of goiters per 1,000 men drafted: 21.00 (4). In general, goiter is most prevalent in the Great Lakes region and the Pacific Northwest (4). Olesen compiled survey data on children in 43 states, and found a goiter incidence of ten to fifty times more than the Draft Board (4). This was mainly due to the smaller size of goiter reported by Olesen's data. A survey of 9,321 children in seven Montana counties revealed an incidence of thyroid enlargement as follows: 13.4% for boys, and 32.0% for girls (4,30,31). Goiter is usually considered to be more prevalent in