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Fluorine in Foods

Survey of Recent Data

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The analytical data relating to fluorine in foods have accumulated during the past decade to an extent that makes it desirable to assemble the data for purposes of comparison, as well as for an evaluation of the amount of fluorine ingested in the average human diet. With few exceptions, the more recent fluorine data are comparable as regards the analytical procedure since the Willard and Winter technique (*1*), or a slight modification (*2*, *3*, *4*, *5*), has become the most generally utilized fluorine method. While most analysts recognize the desirability of improvements in the fluorine determination (in view of an expected error of at least 10 percent, particularly in the analysis of organic materials), it has been some time since any radical changes have been made in the Willard and Winter procedure. Generally this fluorine method now calls for ashing the sample in the presence of an alkaline fixative at a low temperature, isolation of the fluorine by steam distillation using perchloric acid, and estimation of the fluorine in the distillate by microtitration with thorium nitrate or by comparative colorimetry.

Published results for fluorine in foods from various sources are compiled in table 1. In table 2, the data concerns the relation of the fluorine content of soil and water to the fluorine present in plant produce. Similarly, data concerning the effect of fluorine ingestion on fluorine in animal produce (meat, eggs, and milk) appear in table 1, sections a, d, e. In several of the publications cited, information is lacking as regards the "dry" or "fresh" condition of the material analyzed, and wherever it seemed desirable to supply such information the judgment of the author was based on the analytical figure. In general, results for meats, fish, eggs, milk, and wine are based on the materials as consumed. Other materials are reported on a fresh or a dry-weight basis, or both.

*From the National Institute of Dental Research, National Institutes of Health.

The results for animal tissues appear in table 1-a. The effect of sodium fluoride ingestion on fluorine in kidney tissue is notable. To produce this result, however, there was a pronounced induced fluorine toxicosis in the animal. The major portion of fluorine retained in the animal body is deposited in skeletal tissue. As shown in table 1-b, the fluorine in normal edible cuts of meat is of the order of 0.2–0.3 ppm. or less fluorine.

Table 1. *Fluorine in animal tissues, meats, fish, hen's egg, cow's milk, citrus fruits, noncitrus fruits, tea, cereals and cereal products, vegetables and tubers, miscellaneous substances and wine*

a. ANIMAL TISSUES

	Fluorine ppm		*Fluorine ppm*
Cow's liver, dry wt. (*6*):		Cow's kidney, dry wt. (*6*):	
No F added to ration	5.50, 5.80, 5.20	No F added to ration	6.9, 8.9, 10.1
F added to grain ration:		F added to grain ration:	
.022 percent	7.80, 8.50, 5.30	.022 percent	31.8, 25.7, 25.5
.044 percent	7.30, 8.30, 6.20	.044 percent	34.7, 33.5, 38.2
.088 percent	8.50, 7.70	.088 percent	43.0, 43.7
Chicken liver, fresh wt. (*7*):		Guinea pig kidney, fresh wt. (*12*)	0.06
NaF injected:		Cow's pancreas, dry wt. (*6*):	
63 mg	0.7015	No F added to ration	6.9, 10.3, 8.5
45 mg	1.209	F added to grain ration:	
90 mg	1.291	.022 percent	7.0, 8.2, 9.5
45 mg	trace	.044 percent	8.3, 9.0, 9.2
30 mg	1.010	.088 percent	9.1, 10.6
30 mg	1.131	Cow's heart muscle, dry wt. (*6*):	
Liver, fresh wt. (*8*)	1.43	No F added to ration	2.3, 2.7, 2.7
Do	1.59	NaF added to grain ration:	
Do	1.52	.022 percent	4.8, 3.7, 8.8
Guinea pig liver, fresh wt. (*12*)	0.40	.044 percent	7.5, 6.4, 9.7
Calf liver, fresh wt. (*12*)	0.19	.088 percent	9.4, 8.7
Beef liver (*10*)	0.99	Guinea pig heart, fresh wt. (*12*)	0.24

b. MEATS

	Fluorine ppm		*Fluorine ppm*
Chicken (*10*)	1.40	Pork chops (*10*)	0.98
Poultry, canned boned chicken (*11*)	0.63	Pork shoulder (*10*)	1.20
Beef (*26*)	2.00	Salt pork (*13*)	1.1
Round steak (*10*)	1.28	Salt pork (*10*)	3.33
Beef (*13*)	<0.20	Frankfurters (*10*)	1.67
Beef, fresh wt. (*11*)	0.29	Lamb (*10*)	1.20
Pork (*13*)	<0.20	Veal (*10*)	0.90
Pork, fresh wt. (*11*)	0.34	Mutton (*13*)	<0.20

c. FISH

	Fluorine ppm		*Fluorine ppm*
Fish, fillets (*11*)	1.49	Sardines—Continued	
Fish (*10*)	1.63	canned (*15*)	12.5
Mackerel:		in olive oil (*11*)	16.10
boned (*13*)	<0.2	Shrimp:	
with bones (*13*)	3.9	canned (*15*)	4.4
fresh (*14*)	26.89	edible portion (*11*)	0.93
dried (*14*)	84.47	Codfish:	
canned (*11*)	12.10	fresh (*15*)	7.0
Salmon:		salted (*15*)	5.0
canned (*13*)	4.5	Oysters:	
red, canned (*15*)	8.5	fresh (*15*)	0.65
pink, canned (*15*)	9.0	unspecified (*10*)	1.58
fresh (*14*)	5.77	unspecified (*15*)	1.5
dried (*14*)	19.34	Crab meat, canned (*15*)	2.00
canned (*11*)	4.16	Herring, smoked (*15*)	3.50
Sardines:		Tuna fish flakes, canned (*11*)	0.10
canned (*13*)	7.3		

NOTE.—Italic numbers in parentheses are references. See pages 1073–1074.

Table 1. *Fluorine in animal tissues, meats, fish, hen's egg, cow's milk, citrus fruits, noncitrus fruits, tea, cereals and cereal products, vegetables and tubers, miscellaneous substances and wine*—Continued

d. HEN'S EGG

(All analyses based on fresh weight)

	Fluorine ppm
Eggs (*10*):	
Whole	1.18
White	1.48
Yolk	0.59
Eggs (*7*):	
Hen No. 1 [1]	0.360, 0.368, 0.288, 0.165
Hen No. 2 [1]	0.294, 0.463, 0.441, 0.206
Egg (*8*):	
White	0.47, 0.32, 0.14, 0.00
Yolk	1.20, 0.42
Eggs, fresh (*12*)	0.15, 0.21, 0.22, 0.22, 0.42, 0.13, 0.20
Eggs, 1 dozen mixed (*11*)	0.12

	Fluorine ppm
No. F added to ration (*16*)):	
White	0.20, 0.30
Yolk	0.90, 1.20
F in ration (*16*):	
.035 percent:	
White	0.20
Yolk	1.80
.070 percent:	
White	0.20
Yolk	3.30
.105 percent:	
White	0.30
Yolk	3.00

[1] During 4- to 8-week period hens received intermittent intravenous injections of 30–90 mg. NaF.

e. COW'S MILK

No unusual fluoride in cow's ration or drinking water

	Fluorine ppm
Whole milk (*17*)	0.07, 0.09
Do	0.10, 0.15
Do	0.15, 0.17
Do	0.07, 0.22
Fresh milk (*10*)	0.38
Fresh milk, 1 qt. mixed (*11*)	0.09
Fresh milk (*12*)	0.55

	Fluorine pp
Commercial milk (*18*):	
Washington, D. C	0.22, 0.22, 0.26
Washington, D. C	0.14, 0.16, 0.13
Urbana, Ill	0.30, 0.13, 0.11, 0.10, 0.10, 0.10

Fluoride above normal in cow's ration or drinking water

	Fluorine ppm
F in drinking water:	
8 ppm (*18*)	0.26, 0.39, 0.19, 0.17, 0.23, 0.26, 0.18, 0.40, 0.49, 0.28, 0.29, 0.40
0.2 to 500 ppm (*19*)	0.20, 0.30, 0.50, 0.30
0.2 to 495 ppm (*19*)	0.00, 0.30, 0.40, 0.40, 0.40, 0.30, 0.40, 0.30
1.4 ppm (*12*)	0.97, 0.72, 0.91

	Fluorine ppm
F added to grain ration (*17*):	
.022 percent	0.15–0.20
.044 percent	0.11–0.15
.088 percent	0.14–0.26

f. TEA

	Fluorine ppm
Tea (*21*):	
Imported Indian	38.1
Imported Ceylon	8.7, 9.5
Indian Ceylon blends	28.5
Clipper	13.1
Anhwei	91.8, 54.3, 122.6
Amoy	52.7
Ting-ku	178.8
"Doubly scented"	91.7
1st grade Hunan	60.3
Yunnan	49.7
Jessamine	83.5
Hangchow, best grade	37.5
Hangchow, second grade	93.5
Cheap mixed	398.8
Tea (*15*):	
English breakfast	66.0
Gun powder	67.0
Oolong	41.0
Tea (*22*):	
White tea	6.80
Song, chian	9.89
Asiang-pain	43.20
Red tea	67.07
Lung-ching	70.70
Szechuan	85.63

	Fluorine ppm
Tea (*20*):	
Pu-er	91.25
Kocha	7.8
Makha	3.2
Hankow	4.1
Toko	9.2
Ajax	11.9
Maza wattee	29.4
Rajah	33.1
Lyons	18.4
Five roses	25.6
Gifto	18.4
Liptons	18.3
Indona	19.3
Fargo	18.4
Tea, average of ten samples (*11*)	97.0
Tea, infusion—15 gm. of tea were treated with 1,000 cc. boiling water, steeped 10 minutes and strained (*11*)	1.19
Liptons yellow label (*18*)	53.5
Orange Peko (*18*)	62.8
Tea, infusion—0.122 mg. of fluorine was extracted from one tea ball of 2.55 gm. tea (*18*)	62.8

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g. CITRUS FRUITS

	Fluorine ppm
Grapefruit:	0.36
edible portion (*10*)	0.36
fresh (*20*)	0.12
Lemon, fresh (*12*)	.028, .051, .174
Orange, edible portion (*10*)	0.34
Oranges (*20*):	
fruit, fresh	0.17–0.07
Brazil fruit, fresh	0.15
Brazil, peel, fresh	0.16
California, peel, fresh	0.18
Jamaica, peel, fresh	0.11–0.14
Oranges—Continued	
Palestine, fruit, fresh	0.04–0.15
Palestine, peel, fresh	0.11–0.15
Spanish, peel, fresh	0.07–0.13
Navel:	
Spain, fresh	0.15
S. Australia, fruit, fresh	0.06
S. Australia, peel, fresh	0.11
Pomelo (*20*):	
fruit, fresh	0.10–0.16
Florida, fresh	0.04–0.25
Florida, peel, fresh	0.13–0.25

h. NONCITRUS FRUITS

	Fluorine ppm	
	Fresh wt	*Dry wt*
Apples (*10*)	----------	0.42
Apples (*12*)	0.052	0.21
Apples (*20*)	0.22	-------
Apples (*13*)	0.035	0.13
Apples (*8*)	0.92, 1.10, 1.15	-------
Apples (*8*)	1.32, 1.30	-------
Apples, fresh only (*19*)	0.34, 0.77, 0.83, 0.87	-------
Apricots (*20*)	0.22	-------
Apricots (*12*)	0.02	0.08
Apricots (*12*)	0.06	0.24
Banana (*20*)	0.23	-------
Banana (*10*)	----------	0.65
Cherry (*20*)	0.25	-------
Cherries, black (*12*)	0.18	0.61
Currants (*20*)	0.12	0.69
Fig (*20*)	0.21	-------
Grapes (*20*)	0.16	-------
Grape juice (*12*)	0.093	-------
Grape seed (*12*)	0.105	-------
Gooseberries (*20*)	0.52	-------
Gooseberries (*12*)	0.11	0.72
Guava (*20*)	0.34	-------
Mango (*20*)	0.18	-------
Pawpaw (*20*)	0.15	-------
Pear (*20*)	0.19	-------
Prickly pear (*20*)	0.26	-------
Pears (*10*)	----------	0.70
Peach (*20*)	0.21	-------
Plum (*20*)	0.21	-------
Plum (*12*)	0.22	0.10
Pineapple (*20*)	0.14	-------
Pineapple tinned (*26*)	0.00	-------
Pomegranate (*20*)	0.20	-------
Quince (*12*)	0.06	0.37
Quince (*20*)	0.20	-------
Sweet melon (*20*)	0.20	-------
Strawberry (*20*)	0.18	-------
Watermelon (*20*)	0.11	-------

i. CEREALS AND CEREAL PRODUCTS

	Fluorine ppm	
	Fresh wt	*Dry wt*
Corn:		
unspecified (*22*)	0.62	0.70
unspecified (*23*)	---------	1.0–2.0
canned (*13*)	<.20	-----------
canned (*10*)	---------	.42
unspecified (*22*)	---------	5.09
yellow (*13*)	<.10	-----------
germ (*23*)	---------	8.0–11.0
germ (*13*)	---------	15.0
meal, as purchased (*11*)	.22	-----------
meal (*25*)	---------	2.00
flakes (*10*)	---------	1.33
Ralston (*10*)	---------	.58
Wheat:		
whole (*22*)	---------	.53
unspecified (*21*)	---------	1.70
unspecified (*13*)	<.10	-----------
unspecified (*27*)	.7	.8
bran (*27*)	.29	.33
bran (*13*)	<.2	-----------
germ A, commercial (*13*)	1.7	-----------
germ B, commercial (*13*)	4.0	-----------
germ, pure (*13*)	<.1	-----------
germ, pure (*27*)	.88	1.00
Cream of Wheat (*10*)	---------	.55
Flour:		
wheat, white (*22*)	.35	-----------
self-rising (*11*)	.45	-----------
whole wheat (*25*)	---------	1.32
white (*27*)	.27	.31
biscuit (*26*)	---------	.0
baking (*27*)	.31	.35
Bread:		
white (*11*)	---------	.54
white (*26*)	---------	1.00
white (*10*)	---------	.82
Biscuits (*26*)	---------	0.0–1.0
Do	---------	2.0–2.0
Ginger biscuits (*26*)	---------	2.0–2.0–2.0
Rice:		
unspecified (*27*)	.67	.76
whole (*13*)	<.10	-----------
unspecified (*21*)	---------	.70
middle (*22*)	.19	-----------
unspecified (*32*)	---------	.50
Soy beans (*21*)	---------	4.00
Soy beans (*11*)	1.33	-----------
Soy flour (*11*):		
low fat	---------	.52
with fat	---------	1.45
Buckwheat:		
unspecified (*34*)	---------	2.00
whole (*21*)	---------	1.70
bran (*21*)	---------	1.60
Millet (*21*)	---------	.20
Millet (*12*)	.60	.91
Oats:		
unspecified (*34*)	---------	3.0
crushed (*13*)	<.20	-----------
Mother's (*10*)	---------	.92
fresh (*27*)	.25	.29
Rye:		
from Norway (*18*)	---------	.34, .64, 1.30
unspecified (*12*)	.61	.69
Blackeyed peas (*11*)	.23	-----------
Chick peas (*11*)	.14	-----------
Cottonseed:		
meal (*13*)	12.0	-----------
meal (*23*)	20.0–31.0	-----------
hulls (*23*)	12.0–14.0	-----------
Spaghetti (*10*):		
canned	---------	1.15
dry	---------	.80
Macaroni, dry (*10*)	---------	.82

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j. VEGETABLES AND TUBERS

	Fluorine ppm *Fresh wt*	*Fluorine ppm* *Dry wt*
Aniseed (20)	0.4	--------
Amaranth (21):		
red	--------	8.5
green	--------	6.9
Asparagus, canned (10)	--------	0.48
Beans:		
string (10)	--------	0.64
string, canned (10)	--------	0.67
string (11)	0.13	--------
string, edible pods (28)	--------	3.2, 4.8
green (12)	0.15	1.01
light green (12)	0.11	0.73
lima, dry (10)	--------	4.51
lima, seeds (28)	--------	2.2
dry (22)	--------	1.04
dried (13)	--------	<0.20
navy, dry (10)	--------	1.70
Beets:		
unspecified (20)	0.2	--------
fresh (10)	--------	0.60
root (21)	--------	2.8
leaves, dry (21)	--------	3.80
tops (28)	--------	3.4
leaves (12)	0.38	3.45
roots (28)	--------	4.3
roots, sugar beet (28)	--------	3.3
string (12)	0.32	6.09
Cauliflower:		
fresh (10)	--------	0.45
flower (12)	0.12	0.86
leaves (12)	0.08	0.83
unspecified (20)	1.0	--------
Cabbage:		
large (22)	--------	9.34
foreign (22)	--------	15.38
fresh (10)	--------	0.70
unspecified (22)	0.13	--------
edible head (28)	--------	3.4
without leaves (12)	0.8	9.5
edible part (12)	0.15	1.5
unspecified (20)	0.3	--------
loose leaf (12)	0.38	1.31
loose leaf, stalk (12)	0.12	0.86
Carrots:		
unspecified (20)	0.4	--------
fresh (10)	--------	1.30
root (28)	--------	8.4
unspecified (13)	<0.20	--------
unspecified (21)	--------	6.92
Celery:		
unspecified (22)	--------	1.47
unspecified (11)	0.14	--------
powder (11)	0.10	--------
edible stalks (28)	--------	8.5
unspecified (12)	0.70	5.70
Cress (12)	0.24	4.38
Cucumber (20)	0.20	--------
Colza shoot, red (22)	--------	3.88
Colza (22)	--------	2.15
Eggplant (20)	0.4	--------
Endive (20)	0.2	--------
Garlic:		
green (22)	--------	17.72
unspecified (22)	--------	7.17
unspecified (20)	0.3	--------
Ginger plant (22)	--------	2.36
Kale (11)	0.16	--------
Kale (20)	3.0	--------
Leeks (22)	--------	3.90
Leeks (20)	0.1	--------
Lettuce:		
loose, head (28)	--------	11.3
cabbage (12)	0.30	4.45
prickly (12)	--------	5.18
fresh (10)	--------	0.42
Marjoram (12)	1.92	8.68
Mustard:		
greens (11)	0.15	--------
leaves, salted, dried (21)	--------	3.0–4.8
Onions:		
green (22)	--------	10.11
unspecified (20)	0.60	--------
unspecified (12)	0.24	2.40
Parsley:		
tops (28)	--------	11.3
unspecified (20)	0.8	--------
unspecified (12)	1.04	8.73
Parsnip, roots (28)	--------	5.5
Peas:		
unspecified (20)	0.6	--------
green (21)	--------	6.69
fresh (10)	--------	0.60
Potatoes:		
white (10)	--------	0.96
unspecified (13)	0.20	--------
whole (17)	6.4	22.0
peelings (12)	0.07	0.35
Irish, tuber (28)	--------	1.4
white, unpeeled (22)	0.16	--------
unspecified (21)	--------	0.73
unspecified (26)	--------	1.0
I, from Norway (18)	--------	0.9
II, from Norway (18)	--------	0.3
sweet, unpeeled (11)	0.13	--------
sweet (13)	<0.20	--------
sweet (10)	--------	1.08
Pumpkin (20)	0.10	--------
Radish (20)	0.8	--------
Rhubarb (20)	0.4	--------
Rutabaga:		
tops (28)	--------	7.0
roots (28)	--------	2.9
Spinach:		
fresh (10)	--------	1.11
unspecified (20)	1.8	--------
unspecified (22)	--------	7.97
unspecified (11)	0.21	--------
unspecified (11)	0.36	--------
unspecified (28)	--------	28.3
winter (12)	0.44	3.80
Squash, fresh (10)	--------	0.63
Shepherd's purse (4)	--------	2.26
Summer savory (12)	2.67	12.10
Tomatoes:		
unspecified (12)	0.24	2.40
fresh (10)	--------	0.53
fruit (28)	--------	0.0
Turnips:		
greens (22)	0.10	--------
tops (28)	--------	1.7
roots (28)	--------	2.6
unspecified (13)	<0.20	--------
unspecified (20)	0.30	--------
fresh (10)	--------	0.56
Watercress (20)	1.0	--------

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k. MISCELLANEOUS SUBSTANCES

	Fluorine ppm Fresh wt	Fluorine ppm Dry wt
Peanuts:		
unspecified (*10*)	--------	1.36
unspecified (*13*)	<0.20	--------
tops (*28*)	--------	1.7
kernel (*28*)	--------	1.5
Almonds (*12*)	0.90	0.90
Hazelnut (*12*)	0.30	0.30
Chestnut (*12*)	--------	1.45
shell (*12*)	--------	0.24
Coconut, fresh (*11*)	0.00	--------
Cocoa (*26*)	0.5, 0.50, 2.0	--------
as purchased (*11*)	0.92	--------
Plain chocolate (*26*)	0.50	--------
Milk chocolate (*26*)	0.5, 1.0, 2.0	--------
Molasses (*11*)	0.00	--------
Sugar (*10*)	0.32	--------
Honey (*26*)	1.00	--------
Gelatin (*26*)	0.00	--------
Glucose (*26*)	0.50	--------
Malt (*26*)	1.0, 1.5	--------
Powdered ginger (*26*)	1.00	--------
Baking powder (*13*):		
A	220.0	--------
B	19.0	--------
C	<0.1	--------
Coffee (*21*):		
Mocha, Arabian	1.6	--------
Del Monte, Brazilian	0.7	--------
E. B. C., Brazilian	0.2	--------
best raw Java	1.1	--------
Butter (*10*)	1.50	--------
Cheese (*10*)	1.62	--------
Pork and beans, canned (*10*)	1.40	--------

l. WINE

	Fluorine ppm
Chinese, Shao-shing (*21*):	
best grade	0.07
second grade	0.05
Chinese Lao, pai, chiu (*21*)	0.09
Port (*21*)	0.24
Beer (*11*)	0.20
Neuenberger (*12*)	0, 0, 0, 0, 0, 0.06, 0.08, 0.10, 0.10, 0.17, 0.18, 0.20, 0.24, 0.26, 0.34, 4.68, 6.34.
Walliser (*12*)	0, .03, .04, .07, 0.11, 0.12, 0.20, 0.21, 0.23, 0.23, 0.25, 0.25, 0.41, 0.47, 0.54, 0.54.
Franzosiseher (*12*):	
Weisswein, Cote d'or	0.22
Rotwein, Burgunder	0.31
Rotwein, Cote du Rhone	0.21
Do	0.07
Do	0.10
Italienischer, Rotwein (*12*)	0.21
Spanisher, Weisswein, Xers (*12*)	0.21
Algerischer, Weisswein (*12*)	0.26
Wine (*35*):	
variously dated	3.8, 3.3, 2.4
1938–1941	4.1, 2.1, 3.3, 2.7, (slight trace) 4.1, 3.3.
white Sta. Barbara, 1942	3.3
red, Sta. Barbara, 1942	5.0

Seafoods (table 1-c) are particularly interesting because they generally contain more fluorine than any other food, except tea, which obviously is not in a class with seafoods as an edible substance. Sea water may contain upwards of 1.2–1.4 ppm fluorine (*9*) and is the source of fluorine in seafood. The amount of bone remaining, particularly in canned fish, no doubt determines to a major extent the quantity of fluorine contained in the product.

The amount of fluorine in the hen's egg (table 1-d) is approximately 0.2–0.4 ppm. The fluorine results recorded in the table were obtained during an experiment (*16*) in which rock phosphate was a source of fluorine in the hen's ration for a period of 28 months. There was a definite increase of fluorine in the egg yolk, the fluorine being present almost exclusively in the acetone-insoluble portion of the fat-like substance of the egg yolk.

The fluorine content of cow's milk has never proved to be affected by fluoride in the cow's ration or drinking water (table 1-e). Normally cow's milk contains 0.10–0.20 ppm fluorine.

Fluorine in tea has been studied extensively (table 1-f). The data agree that tea is an unusual plant substance in its fluorine content. It has been reported that 75 percent or more of the fluorine in tea is extracted by boiling water (*18, 21*). It has been estimated also (*18*) that about 0.1 mg. of fluorine may be present in the hot water extract from one tea ball (2.5 gm. of tea contains 62.8 ppm fluorine in the dry tea).

The data for fluorine in citrus fruit (table 1-g), with three exceptions, were obtained by Hamersma (*20*). These results, based on the fresh or edible material, indicate the presence of about 0.10 ppm or less fluorine.

A number of noncitrus fruits (table 1-h) are also reported by Hamersma (*20*), the origin of most of his material being the Union of South Africa. In the United States, fluorine in apples has attracted much interest because of a presumed health hazard arising from a fluoride spray remaining on the apples (*24*).

The common cereals and cereal products have been analyzed extensively for fluorine (table 1-i) as would be expected for such important articles of the diet. The fluorine figures are quite variable, but for corn and wheat particularly and their edible produce, the values generally are extremely low, i. e., of the order of 0.10 or 0.20 ppm or less fluorine in the fresh material.

The seemingly high fluorine results for several vegetables and tubers (table 1-j) may be questioned in some instances, because of the possibility of soil contamination. The majority of results on the fresh weight basis, however, are in fairly good agreement, i. e., 0.10 to 0.30 ppm is about the average amount to expect.

A variety of results on a number of miscellaneous substances are shown in table 1-k. The majority of these materials, however, do not constitute a very important part of the average diet.

Fluorine in wine (table 1-l) has been studied recently by von Fellenberg (*12*), whose laboratory is in Switzerland, and by de Almedia (*35*). The results reported by de Almedia appear to be unusually high. Sodium fluoride formerly had some use in cleaning wine tanks and casks, but there is no hazard from this type of fluoride usage at this time. Although consumption of wine, particularly among Europeans and South Americans, may be unusually high, there is at present no knowledge that wine causes an unusual dietary fluorine intake.

Effect of Soil and Water Fluorine on Plant Fluoride

The data in table 2 answer the question of the relation of fluorine in plants to the fluorine in the water and soil in which the plants grow.

With few exceptions, and these seem to apply mostly to roots and tubers, fluorine in plant produce is not readily affected by fluorine in the soil and local water. According to Bartholomew (*33*), nutrient solutions containing fluorine up to 10 ppm may cause large increases of fluorine in cowpea roots, but the tops are increased in fluorine only when the quantity of fluorine in the roots is very large. Fluorine in

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Table 2. *Fluoride content of plants grown in soil or water containing added fluoride (all figures are ppm of fluorine)*

Reference 19 (dry weight):	*No fluoride added to soil*	*Calcium fluoride added to soil*
Corn	trace	trace
Hogari	0.00	2.1
Soybeans	.83	1.2
Wheat	.69	1.0
Wheat, stalks and leaves	3.70	7.2
Alfalfa:		
1st cutting	7.0	15.0
2d cutting	4.5	10.8
3d cutting	5.0	---------
4th cutting	6.0	11.3

Reference 19 (fresh weight):	*No fluoride added to soil*	*Sodium fluoride added to soil* 800 ppm	1600 ppm	3200 ppm
Beets	1.7	6.5	------	17.7
Carrots	1.0	3.0	0.4	1.3
String beans	.0	------	.0	------
Yams	.0	7.6	------	8.2
Tomatoes	.7	.5	2.3	1.2

Reference 29 (air dry basis):	*No fluoride added to soil*	*Phosphate fluoride fertilizer added to soil*		
Wheat grain	0.82	0.94	0.99	0.32
Do	.30	.48	.46	.26
Oat grain	1.25	1.75	.70	1.66
Alfalfa hay	No consistent or greatly increased fluorine content.			
Mixed hay				
Clover and timothy hay				
Oat straw				
Wheat straw				
Cowpea hay				

Reference 34:[1]	*Mineral fluorides added to soil*
Lawn grass	No appreciable effect on fluorine content.

Reference 21:[2]	*No fluoride added to soil* Fresh wt	Dry wt
Wheat	0.20	----------
Rice	0.90	----------
Barley	0.02	----------
Soybeans	0.67	----------
Cowpeas	0.43	----------
Green beans	0.17	----------
Green peppers	0.14	----------
Mustard leaves	----------	26.55
Turnip leaves, salted	----------	4.04
Tea, fresh[3]	1.75–7.8	----------
Tea, roasted	15.0–71.0	----------

[1] Fluorine a normal constituent of vegetation in study area (western Pennsylvania).
[2] Study area "near fluorine area" in China.
[3] Picked in later summer instead of usual springtime, suggesting increase of fluorine with age of leaves.

Table 2. ***Fluoride content of plants grown in soil or water containing added fluoride (all figures are ppm of fluorine)***

Reference 11 (dry weight):[4]	*No fluoride added to soil*
Wheat	0. 76–1. 15
Wheat flour	0. 50

Reference 20 (fresh weight):	*Fluorine in water for plants*[5]
Cabbage	6. 4
Salad	12. 1, 28. 3
Salad seed	21. 3
Mealies	<1. 0, 0. 7, 0. 1
Beetroot	6. 4, 3. 1
Beetroot leaves	9. 1, 0. 6
Carrots	9. 1, 5. 4
Carrot leaves	6. 9, 56. 6
Tomatoes	2. 8, 6. 4, 1. 8
Leeks	54. 7
Orange leaves	25. 7
Radish	12. 0
Radish leaves	132. 0
Guavas	2. 1
Beans	2. 8
Pumpkin	3. 4
Pumpkin peel	<0. 1

	Fluorine (ppm) in local water supply			
Reference 10:[6]	*0.0–0.8*	*1.0, 2.0, 3.0*	*3.1–6.0*	*6.1–18.0*
Squash, white	0. 42, . 36	0. 07	0. 11, 0. 26	0. 36, 0. 36
Squash, green	0. 32	0. 08	0. 13, 0. 24	--------
Tomatoes, green	--------	--------	--------	0. 41
Tomatoes, red	0. 62	--------	0.31,0.29, 0. 16	0. 47
Onions, green	--------	--------	0. 72	0. 25
Onions, white	--------	0. 52, 0. 51	--------	0. 12
Beets	--------	0. 65	0. 53	0. 32, 0. 73
Carrots	--------	0. 63, 0. 43	0. 72, 1. 34	0. 74
Beans, string	0. 16	0. 30	0. 43	--------
Lettuce	--------	0. 27	--------	--------
Potatoes	--------	--------	--------	0. 81
Cucumbers	--------	--------	0. 15	--------
Okra	0. 20	--------	0. 42	--------
Chard	--------	--------	0. 85	--------
Turnip tops	--------	--------	0. 69	--------

	Fluorine in nutrient solutions	*Fluorine ppm*	
Cowpeas (Reference 33):	*ppm*	*In roots*	*In tops*
NaF in nutrient solution	0. 25	13. 7, 58. 0	0. 0
	0. 50	8. 0, 13. 7	0. 0
	1. 00	39. 1, 60. 0	0. 0
	3. 00	237. 5	0. 0
	3. 00	550. 0	8. 0
	10. 00	826. 0	26. 2
	10. 00	1, 086. 0	40. 0
CaF_2 in nutrient solution	0. 25–0. 50	0. 0	0. 0
	7. 72	84. 3	0. 0
	7. 72	78. 3	11. 0
Na_2SiF_6 in nutrient solution	0. 25–0. 50	0. 0, 13. 7	0. 0
	1. 00	37. 4, 42. 7	0. 0
	10. 00	1, 970. 0	415. 0
	10. 00	1, 116. 0	475. 0

[4] Wheat produced in Deaf Smith County, Tex.
[5] Water for plants contained 4.0–12.7 ppm fluorine. Study area in South Africa.
[6] Results are presumably on fresh-weight basis.

wheat changed little when calcium fluoride was added to soil plots (*19*). In studies of control vs. fluoride plots (*19*), fluorine in beets and yams increased notably when an excess of sodium fluoride was applied to the soil. Fertilization of soil with phosphates and slags containing fluorine may increase fluorine in drainage waters (*29*), but plant fluoride was not increased (*29, 30*). As much as 2,300 ppm fluorine was added to the soil in one experiment (*30*), whereas an average figure for fluorine in surface soils is about 292 ppm (*28*). Results of analyses of grains and forage crops from fluoride areas frequently show unusually high fluorine concentrations, but this may be caused by contamination with soil dust. The evidence regarding soil fluoride and its effect on fluorine in plants shows generally a negative effect.

Although all evidence points to the contrary, the fluorine in local water supplies has been suggested frequently as influencing fluorine in plant life. Machle, Scott, and Treon (*10*) found no correlation between fluorine in certain food plants and fluorine in the local water supplies of Arizona. Wheat produced in Deaf Smith County, Tex., a fluoride water area, did not show an unusual fluorine content (*11*). Hamersma (*20*) has presented results regarding the effects of water containing 4.0–12.7 ppm fluorine on vegetables produced in small private gardens. A number of his results are unusually high for fresh materials and a residue of fluoride on the plant materials is suggested by his notation that the garden was watered by hose. As in the case of soil fluoride, there is no indication that fluorine in the local water supplies affects food fluoride. Sources of fluoride-bearing potable waters are, with few exceptions, deep wells, and these waters are not used for irrigation purposes. It is not to be expected, therefore, that normal use of water in fluoride water areas would add fluoride to the local plant produce.

Fluorine may be increased in foods cooked in fluoride waters, as the following results presented by Smith, Smith, and Vavich (*19*) indicate:

Food	*Cooked in Distilled water*	*Cooked in water containing*	
		5 ppm fluorine	*24 ppm fluorine*
	ppm	*ppm*	*ppm*
Pinto beans	2.0	--------	37.1
Beets	0	1.0	--------
Potatoes	.5	--------	9.7
Cabbage	0	3.6	--------
Carrots	2.3	3.2	--------
Cauliflower	0	4.2	--------
Oatmeal	.9	--------	22.8
Spinach	2.0	4.0	--------
Italian squash	.2	3.8	--------
Brussel sprouts	.2	2.9	--------

Assimilation of Natural Fluorine

The body's assimilation of "natural" fluorine in foods has been subjected to limited investigation. In addition to being largely extracted from a 2-percent infusion of tea leaves, the fluorine in tea, according to Reid (*21*), is capable of producing the characteristic striations in the incisor teeth of rats. Fluorine from various sources was administered at levels of 9–12 ppm to rats by Lawrenz and Mitchell (*31*) who found that green-tea fluoride was only about 5 percent less well assimilated than was sodium fluoride or calcium fluoride. An average of 31.7 percent of the fluorine in green tea was retained as compared with 33.7 percent retention from sodium fluoride.

Results on the rats' metabolism of the fluorine contained in canned fish are reported as follows by Lee and Nilson (*14*):

Source of fluorine in rat's diet	*F in diet*	*Total F ingestion*	*Percent stored*
	ppm	*mg.*	
Salmon (fresh)	5. 77	13. 87	19. 75
Salmon (dried)	19. 34	12. 10	20. 25
Mackerel (fresh)	26. 89	50. 75	21. 47
Mackerel (dried)	84. 47	49. 20	24. 24

This percentage of fish fluoride body storage by the rat is somewhat low when compared with the usual retention of inorganic fluorides by the growing rat (*31, 32*), but the quantities ingested in this study (*14*) are also relatively high.

In general it appears that natural fluorine in fish, tea, and other foods is largely available for assimilation. This conclusion is indicated also indirectly by urinary excretion data mentioned later on in this discussion.

Discussion

In a previous article (*36*) the total food-borne fluorine in the diets, exclusive of drinking water, of children 1–12 years old was estimated to be 0.25 mg.–0.55 mg. daily. Uncertainties surrounding food-fluorine analytical data at that time (1943) suggested that estimates of dietary intake should be based on 0.1, 0.2, 0.5, and 1.0 ppm fluorine in the dry weight of average foods. The analytical fluorine data accumulated since then do not substantially change these quantities—0.25–0.55 mg. fluorine daily in food—although the lower values, 0.25–0.30 mg. fluorine in the daily food, exclusive of drinking water, are probably more representative and in accord with the analytical data for fluorine in foods. Other fluorine analyses of entire diets exclusive of drinking water have indicated 0.25–0.32 mg. fluorine in the average daily food alone (*37*). Another study indicates 0.45 mg. fluorine in the average diet (*38*) where the drinking water is practically fluoride free.

It may be pointed out that 0.25–0.30 mg. of dietary fluorine may be applicable to average daily diets throughout the United States and perhaps other parts of the world. Thus Machle et al. (*10*) and McClure and Kinser (*39*), studying the urinary excretion of fluorine, found the urinary fluorine analysis to be a valuable criteria of the daily water-borne fluorine intake. In widely scattered areas in the United States, where the drinking water contained only traces of fluorine, the urinary fluorine may be attributed largely to food-ingested fluorine and is quite uniform, i. e., 0.2–0.3 ppm (*39*). This observation was regarded as indicative of a uniform content of fluorine in average daily diets, regardless of the locality. Similar urinary fluorine data were obtained recently with respect to Oslo, Norway, where the drinking water contains about 0.1 ppm fluorine (*18*).

Disregarding certain extreme industrial exposures, it is generally true that drinking waters containing upwards of 1.00 or more ppm fluorine are the source of the major quantities of dietary fluorine. As regards water containing 1.00 ppm fluorine, it is estimated that 1.0–1.5 mg. fluorine (based on an estimated 1,000–1,500 cc. water consumed daily) are ingested daily by an average adult via drinking water and water added to cooked foods (*36*). In the case of children 1–12 years old, drinking water containing 1.00 ppm fluorine will contribute an estimated 0.4–1.1 mg. fluorine daily above the fluorine in food (*36*). This added quantity of fluorine ingested during the crown calcification period of tooth life—through ages 8 to 10, or through ages 12 to 16 if the third molar teeth are to be considered—is the estimated amount of water-borne fluorine now associated with the partial alleviation of dental caries (*40*). The advantages to dental health surrounding the use of drinking water containing 1.00 ppm fluorine has justified investigation of the dental health value of a direct addition of sodium fluoride to community water supplies (*41*) and has suggested also the direct addition of a fluoride supplement to children's diets during formative tooth life (*36*).

The importance of fluorine in preventive dentistry has been widely discussed and thus far remains irrefutable (*40*). Many problems, however, have yet to be resolved regarding the most efficacious utilization of fluorides in dental caries prevention. Where fluorides are not present in drinking water and cannot be provided via a community water supply, serious consideration seemingly may be given to the advantages of a direct dietary fluoride supplement. For purposes of dental health it appears that during formative tooth life the average child's diet should contain an additional dental optimum supplement of a fluoride equal to about 1.00 mg. of fluorine daily. The accumulated data on the fluorine content of foods indicate that the average child's diet does not provide a dental optimum quantity of fluorine.

Summary

A survey of recent analytical data for fluorine in foods has been compiled. The majority of foods found in the average diet contain from 0.2–0.3 ppm or less fluorine in the food as consumed. Tea and seafoods are notable exceptions, the former containing upwards of 75 to 100 ppm fluorine in the dry tea, whereas seafoods may contain 5–15 ppm fluorine. Cow's milk contains about 0.1–0.2 ppm fluorine. Fluoride added to the cow's ration or drinking water has no influence on the milk-fluoride. Fluorine in soil and water has little or no influence on the fluorine content of edible plant produce. Although the data are limited, it appears that natural food-borne fluorine is largely available for body assimilation.

Exclusive of drinking water, the average diet appears to provide 0.2–0.3 mg. of fluorine daily. However, it has been observed that an additional intake of fluorine during formative tooth life, via drinking water containing 1.00 ppm or slightly more fluorine, is a distinct dental health advantage.

It is justifiable, therefore, to consider the possibility of a direct dietary fluoride supplement where the drinking water does not provide a dental optimum quantity of fluorine.

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Iodine—A Food Essential

By W. H. Sebrell, M. D.*

In a cooperative effort, the salt industry, medical profession, public health authorities and the Federal Government are seeking to protect and improve the health of American citizens by an educational campaign aimed at getting them to use a nutritionally improved salt.

The average United States citizen enjoys better health than the average citizen of most other countries. His food supply is also better than average, but many people in this country do not get the full recommended allowance of all the dietary essentials. It is well known that deficiency in any of these essentials results in ill health and disease.

We know that iodine in appropriate amounts, like iron, calcium, copper, and many other chemical elements, is essential throughout life. Also it is known that many people in all sections of this country do not get enough iodine from their food to meet their normal requirements. All doctors know that long-continued iodine deficiency may result in serious disease, or at least in a chronic state of border-line malnutrition. Therefore, in connection with the food-essential iodine it is necessary to discuss the deficiency disease, goiter, which results from lack of sufficient iodine.

It has been demonstrated many times in this country and abroad that iodine deficiency is easily corrected and better health achieved through the daily routine use of table salt to which tiny amounts of iodine have been added by the manufacturer. This salt is as pure as, and tastes no different from, ordinary salt. It can be obtained at no extra cost or inconvenience to the consumer, and, once acquainted with the facts, he can voluntarily insure his supply of this food essential.

Function and Importance of Iodine and Endocrine Glands

The newest studies of the human body emphasize the human being as a "whole man." For example, recent research proves that the pituitary gland at the base of the brain produces a secretion which stimulates the thyroid gland, and that the thyroid secretion can inhibit or stop this pituitary secretion. So important is this situation that Salter in 1940 called it the "pituitary thyroid axis."

The pituitary and thyroid glands often are called ductless glands or glands of internal secretion, because their secretions pour directly

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into the blood or the lymph, instead of running through a tube or duct.

The pituitary (or hypophysis) is a small gland which is fastened to the base of the brain. Its anterior lobe has been dubbed the "master gland" because of its importance to the body and its influence on other glands. Eleven different physiological effects or influences on body performance have been discovered in this anterior pituitary gland. At least six of these effects seem to be from distinct hormones. The action of these hormones can be classified into two groups. One group deals with effects on other endocrine glands, such as the thyroid-stimulating hormone, nicknamed TSH, mentioned already, and hormones which stimulate the adrenals, the parathyroids, the pancreas, and the sex glands. The other group of pituitary hormones acts on a variety of tissues.

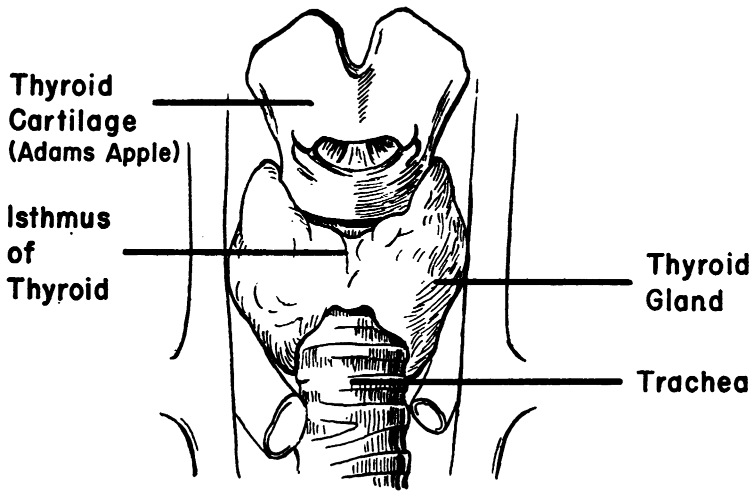
The thyroid gland is a large U-shaped gland in the neck. When it becomes enlarged, it is recognized as goiter. The word "thyroid" means "shield-shaped," and was given to the gland in 1656 by a London physician, Thomas Wharton. Dr. Wharton poetically told how the thyroid contributes to the beauty of the neck by filling up the vacant spaces around the larynx, particularly in women. More matter-of-factly, the thyroid gland has been likened to a pair of Brazil nuts on either side of the windpipe. A band of thyroid tissue, called the isthmus, connects the two lobes. Sometimes the thyroid is described as a thickened U in appearance. Sometimes an additional lobe, called the pyramid of Lalouette runs upward from the upper border of the isthmus. The weight of the thyroid gland in the male varies from 20 to 60 grams, or about 1 to 2 ounces. The normal thyroid in women usually is a little heavier.

Its rich supply of blood and lymph attests to the importance of this gland to the entire body.

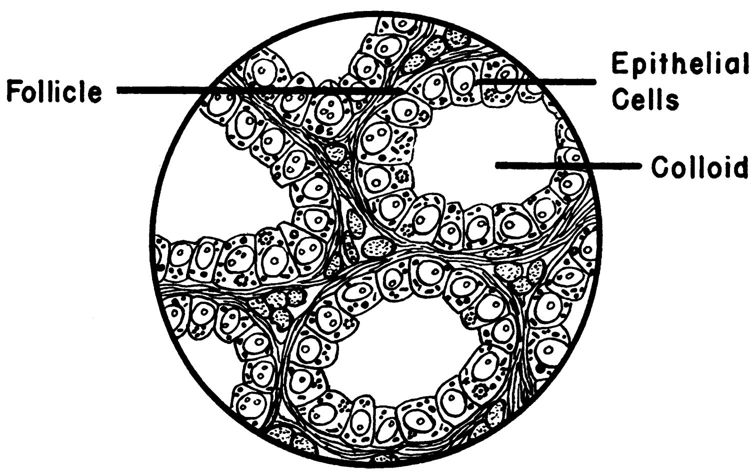
The microscopic appearance of the thyroid tissue shows why Dr. J. H. Means called the thyroid both a factory and a warehouse. Two kinds of tissue comprise the gland. The supporting tissue is a fibrous connective tissue, which makes the framework and the capsule of the gland.

The active, or functional, tissue consists of a number of follicles (also called vesicles, acini, or alveoli). These tiny follicles resemble sacs within an orange section, except, of course, that they tend to be spherical and are microscopic in size. It would take about 250 follicles to cross the diameter of a dime.

Each tiny follicle consists of a single layer of cells forming the outside. These epithelial cells are the actual factory which manufactures the thyroid hormone. In the center of each follicle is the colloid fluid, which is the storage or warehouse form of the thyroid hormone or secretion. In normal health, each sphere is plumply full of colloid.



THYROID BODY



FOLLICLES OF A HUMAN THYROID

When iodine is eaten in the form of iodized salt, or iodine-rich food, such as oysters, salmon, or tuna fish, it travels through the blood in the form of a salt, an iodide. The thyroid gland, of all the body tissues, has an affinity for iodine. When the thyroid gland absorbs the iodide, it is oxidized, that is oxygen is added, by an enzymatic process. This releases iodine in a form which readily combines with an amino acid, tyrosine, to form the long-named chemical diiodotyrosine. Two molecules of this latter compound combine to form thyroxine. Thyroxine is the thyroid hormone. After manufacture, this hormone, thyroxine, may be carried by the blood stream to produce striking effects in many parts of the body. Or it may be stored as a colloid, thyroglobulin, in the centers of the follicles to be called upon when needed.

Course of Food Iodine in the Body

Food iodine	**Iodized salt, seafood**
↓	
Alimentary canal	**iodine absorbed by body**
↓	
Blood stream	**iodine carried to the thyroid**
↓	
Thyroid gland	**iodide (iodine) + tyrosine = Thyroxine + protein = Thyroglobulin**
↓	
Thyroglobulin	**used as hormone or stored as colloid**

Just as the mineral calcium is essential for the formation of bones and the mineral iron is necessary to form hemoglobin for blood, so iodine is absolutely essential for the formation of thyroid hormone, or thyroxine—no iodine, no thyroxine. The thyroid normally contains from 15 to 20 milligrams of iodine. In normal adults, a supply of about 75 milligrams of iodine per year will maintain the thyroid in normal condition.

Some of the normal functions of the thyroid gland are quite popularly known. The thyroid hormone controls the rate of heat production in the body by controlling the rate of cell oxidation. Heat production is coupled with energy liberation from the cells. Basal metabolism tests measure thyroid activity by measuring the rate of heat production in a resting individual.

Other functions of the thyroid are to aid in the stimulation of normal growth of bones, hair, and skin; the normal development of the brain; the stimulation of sexual development at puberty; the maintenance of a normal pregnancy; and the production of an adequate milk supply during nursing.

Types of Goiter

The word "goiter" means an enlargement of the thyroid gland. As most diseases of the thyroid gland are accompanied by an increase in size of the gland, in popular language a goiter usually means a large and conspicuous thyroid gland. Shakespeare, in The Tempest, writes of men "whose throats had hanging at them wallets of flesh." But goiter was known in ancient times when the water from certain wells was said to produce goiter.

Simple Goiter

Simple goiter is by far the commonest form of goiter, and occurs in all parts of the world. But its distribution is not even. Goiter areas exist, often separated only from the next area by a change in the watershed, or the supply of salt.

Simple goiter is sometimes called benign goiter, because it often disappears when a particular strain on the body, such as puberty or pregnancy, is past. Simple goiter also has been called "physiologic goiter," because it so often is associated with a physiological period or situation, notably puberty, but also pregnancy, lactation, and infectious diseases with fever. But leading authorities have objected to the use of the term physiological, implying normal, because, if the individual going through the period of stress is given a really adequate amount of iodine, enlargement and goiter are prevented. Also, cases do occur when a simple goiter does not subside, but progresses to a more dangerous form.

The enlargement which occurs in simple goiter may be accompanied by a mild degree of hypothyroidism, or underactivity. The enlargement represents an effort to manufacture more adequate amounts of thyroxine.

Simple goiter begins with a lessening in the amount of iodine in the colloid and an increase in the size and number of the thyroid cells. The first stage is called parenchymatous goiter. "Parenchyma" means the essential or functional part of an organ. As seen in the thyroid structure, the factory or working part is composed of the thyroid epithelial cells. When hyperplasia or abnormal multiplication of thyroid cells spreads evenly through the whole gland, the resulting goiter is symmetrical, and firm. Parenchymatous goiter may go on to a complete exhaustion, or wearing out of the cells. Or the increasing process may stop, and the follicles become filled with colloid.

When the latter happens, a colloid goiter results. Colloid goiters are usually symmetrical and somewhat soft. They may be very large, because each of the many additional follicles becomes distended with colloid. The total quantity of iodine in the entire gland may be close to normal, but because of the enlarged size, the ratio of iodine per gram of gland tissue is low.

Another kind of goiter is called an adenomatous or nodular goiter. Either parenchymatous or colloid goiter may gradually change to adenomatous goiter, which is the commonest type of goiter after the age of 30. Asymetrical, or uneven, bulges or nodules form. They may increase in size from that of cherry stone to plum stone or larger.

Simple goiter is so called because it does not cause any toxic or poisoning symptoms. Its importance from a medical standpoint is due to the fact that nodular or adenomatous goiter may frequently have its origin in a preexisting colloid goiter, and when this occurs nodular goiter may subsequently become toxic or poisonous; also, symptoms due to pressure on neighboring structures such as the windpipe may occasionally be bothersome in patients with colloid goiter. From a health standpoint, colloid goiter is completely preventable by taking a small but adequate amount of iodine in the food regularly. Where iodized salt is used from infancy, simple goiter, with very few exceptions, is avoided.

Goiters Associated With Hormone Deficiency

Cretinism

A cretin is a child who is dwarfed by lack of sufficient thyroid secretion during fetal life. A similar but less severe condition, myxedema, may occur by the development of an insufficiency of thyroid secretion which occurs at any time after birth. Cretins, in the past, were found in districts where goiter was common. Among these areas are the Himalayas, the Pyrenees, the Alps, the Andes, South America, and the Great Lakes and St. Lawrence regions and northwestern States in North America. So many of these unfortunates were found in certain localities in the Alps that government aid was necessary to support them. However, cretins do appear occasionally in all these areas.

In cretins, the mental, physical and sexual development is greatly retarded. If these cretin dwarfs live to adulthood, they retain their childhood body build, and may not mature sexually. If untreated, their mentality may be arrested at a low level, making them unable to support themselves. The metabolism is very low, the skin has a typical dry, thick appearance, and deaf mutism is common. At autopsy, abnormal or very small thyroid glands are found.

If thyroid extract is given to cretins at an early age, marked improvements result but complete recovery does not always occur. Cretinism should be prevented by assuring an adequate supply of iodine for the mother before and during pregnancy.

Myxedema

When destruction or degeneration of the thyroid gland occurs at any time after birth the hypothyroid condition called myxedema results. Myxedema can be produced in animals by removal of the thyroid gland. Sir William Gull of England in 1874 described the defects in his patients which were associated with degeneration of the thyroid. Gull noticed several effects of hypothyroidism. A loss of mental and physical vigor; dry, brittle hair; an apathetic, lethargic reaction to mental stimuli; and a peculiar thickening of the skin are typical. Today we know by basal metabolism tests that a low metabolic rate exists. The thickening of the skin was thought to be due to the deposit of mucin, and the word myxedema means mucous swelling. Recent study indicates that the deposit is a semifluid albuminous substance, about like egg white.

The outlook for the adult with myxedema is hopeful today. Three months of administration of thyroid extract make a dramatic improvement. Thyroid extract will control the symptoms of myxedema entirely, and keep the patient in good health.

Hyperthyroidism

Increased function, called an overactive, or hyperactive state, may occur in the thyroid gland. Some unknown factor, perhaps a severe emotional shock, or an infection, may start a hitherto normal thyroid on a mad race to produce thyroid hormone. Perhaps an interruption in the amicable relations existing in the pituitary-thyroid axis is the direct cause. But if something in the pituitary starts the thyroid into trouble, that something has yet to be explained.

Hyperthyroidism, or toxic goiter, may begin in a previously healthy individual with enlargement of the gland and simultaneous poisoning symptoms of rapid pulse, palpitation, tremor, nervousness, restlessness and irritability. This type of toxic goiter is called primary or exophthalmic goiter, or Graves' disease.

A severe primary toxic goiter may soon be accompanied by a pop-eyed condition, medically called exophthalmos. The metabolic rate goes up, perhaps as high as 80 percent or more above normal, and with rapid burning of fuel goes a rapid loss in weight. The animation increases to an abnormal nervousness which becomes very trying to the patient and everyone about. There may be difficulty in breathing upon exertion and severe heart symptoms. Fortunately, various methods of cure for exophthalmic goiter are known today.

Another type of toxic goiter is called secondary, because it follows a simple goiter as a complication. Usually hyperthyroidism does not occur until after an average of 15 years of simple goiter. The pop-eyed, or exophthalmic, condition does not occur and the onset is more gradual than in exophthalmic goiter. The heart shows signs of poisoning by a rapid pulse and the metabolic rate soars. The simple adenomatous goiter becomes a toxic adenomatous goiter for unknown reasons. The tragedy of toxic adenomatous goiter lies in its preventability by known methods. If we avoided simple goiter, through adequate iodine intake, we would have no secondary toxic goiter.

Iodine, and Its Role in Normal and Abnormal Thyroid

In reviewing the thyroid gland and its function, recall how the thyroid cells took the iodine from the blood as an iodide and manufactured the thyroid hormone from it for body use or for storage. When the body is given more iodine than it needs from a diet exceptionally rich in seafood, the excess passes out in the normal person without any effect on the body.

But some entirely different situations can produce an iodine hunger in the body, and then unfortunate results follow. One situation is lack in the diet of the minimum iodine requirement.

Another situation is temporarily increased demand for iodine, which may make a previously sufficient iodine supply dangerously inadequate. The well-known situations of increased demand are puberty, pregnancy, and lactation. Since the thyroid hormone is closely associated with the gonads, or sex glands, it is natural that sexual development should make an increased call for thyroid hormone, which only can be met with iodine.

Since pregnancy is the growth period for the expected baby, a similar special demand for thyroid hormone is logical. Many obstetricians today are giving additional iodine to all their pregnant patients.

Because lactation, or the period of nursing a baby, is also a period of supplying growth for an individual, a logically extra demand for thyroid hormone occurs at this time. Proof of the need for extra iodine is seen in the enlargement of the thyroid which often exists in puberty, pregnancy, and lactation. Infections, poor diet, and poor sanitation also may make additional demands for iodine. Certain foods, notably members of the cabbage family, if taken in abnormal quantities, may do the same, and it is possible that they may cause goiter if the supply of iodine is small.

Drs. Curtis and Fertman have found that normal adults confined in bed on a monotonous diet required approximately 1 microgram of iodine per kilogram of body weight per day. An additional 2 micrograms daily are needed to take care of ordinary activities with some for reserve. Taking all needs into consideration the Food and Nutrition Board of the National Research Council gives the daily requirement of iodine for an adult as 0.15 to 0.30 milligrams and states that this need can be met by the regular use of iodized salt. They call attention to the special importance of its use in adolescence and pregnancy.

When the single layer of flat or cuboidal cells around the edge of the thyroid follicles (see figure) are offered enough iodine for their manufacturing needs, they sit quietly in their places like decent diners at a good home or restaurant. But when the amount of iodine is scanty, either from lack of iodine in the diet or because of special demands, a wild boarding-house grab ensues. Each epithelial cell seems to be stretching for all the possible iodine it can capture from the diminishing colloid supply. The cells change from their normal shape to an elongated, columnar shape. This hypertrophy, or overgrowth of individual cells, is soon followed by a hyperplasia or increase in the total number of cells. If a necessary food were suddenly rationed, a protective mother might call all her children and rush the whole family to the store to get as much as possible. These two changes in the thyroid, the stretching out of the individual cells, and the increase in the total number of cells, can be seen under the microscope.

When the rationing is over, either because the special demand ceases, or the diet is improved by additional iodine, the rush stops, but the thyroid gland never is quite the same again. For all practical purposes, the gland will function normally, and may return to its normal size. But the involution, or return to normal, is really a resting or colloid state. A woman who eats just enough food iodine for usual living may have a series of thyroid enlargements with each pregnancy.

The Subclinical Picture

It is easy for individuals to look the other way if they do not recognize that the iodine problem is their own problem. Many people who take vitamin tablets and are careful about getting an adequate supply of vitamins neglect the iodized salt which will insure a health minimum of iodine for the normal person. It has been seen how the thyroid secretion with its essential iodine affects growth, health, a normal skin, and an alert mentality. Doctors have found, in practicing obstetrics, that even where goiters were not involved, the giving of extra iodine decreased the number of miscarriages and increased the number of mothers who had an adequate milk supply. How much fatigue of the adolescent may be due to iodine-hunger is only a guess.

With today's emphasis on positive health, many medical authorities are actively endorsing the use of food iodine, as a simple, cheap, easy insurance against the possible handicaps of a subclinical iodine deficiency.

Why Iodine May Be Inadequate in Diets

From ancient times, goiter has been known to exist in certain regions. Endemic is a word used to describe a condition which exists all the time in a certain place. The draft boards of World War I found two endemic goiter areas in the United States, one centering around the Great Lakes Region, and the other in the Pacific Northwest. In areas where endemic goiter exists, the soil, water, and vegetation are poor in iodine. Early opinion pointed a finger of warning at certain supplies of drinking water. Analysis of drinking water in United States cities shows a wide variation in iodine content, from 0.01 micrograms per kilogram (γ/kg. or parts per billion) in Duluth, Minn., and Spokane, Wash., to 73.30 γ/kg. in San Dimas, Calif.

The iodine content of plants can be increased by adding iodine-containing salts to the soil, and the iodine content of milk can also be increased by feeding suitable rations to the cow. Such practices have been considered and have been declared to be utterly impractical in meeting the goiter problem. They are too inefficient, uneconomical, and difficult to put into operation on a national scale.

Seafood is a good source of iodine, although the iodine content varies with the variety and with the iodine content of the sea water from which it comes. Seafood-eating people, even in a goitrous area, are remarkably free from goiter.

Certain sea weeds such as kelp have a very high iodine content, and dried preparations in the form of tablets or powder have been widely promoted for their health value. These products have no important nutritive value other than that of the iodine they contain. While it may be possible to adjust the intake of these products so as to provide for a suitable uniform supply of iodine, the requirements for this element can be more easily and safely met by the use of iodized salt.

Some natural salt deposits contain iodine, but others do not. A dramatic demonstration of man's accidental conversion of a naturally healthy area into a goitrous area occurred in the Kanawha River Valley of West Virginia. Dr. O. P. Kimball reported the change which took place. Prior to 1900, goiter was exceedingly rare, according to local physicians. Prior to this date, the table salt came from local salt wells. It was a crude, coarse salt with brown particles. After 1900 a sparkling white salt, which the people preferred, was shipped in. This white salt contained no iodine, but it pushed the crude salt off the market. During the next quarter of a century, the goiter rate rose sharply. By 1922, a goiter survey showed that about 60 percent of adolescent girls in that Valley had goiter.

Repeatedly, civilized man has demonstrated that he is technologically ahead of his own welfare. But after harming himself, he often works out the cure. The classic story of the polishing of brown rice into white rice is a good example. The loss of thiamine (vitamin B_1) caused the development of the deficiency disease, beriberi, among those using white rice as a large item of diet. Prevention and cure were found in eating the rice polishings, or the unpolished brown rice.

Similarly, whole grain bread was robbed of iron and vitamins when white flour became popular. Now we are putting back some of the health-giving properties of the whole grain by "enriching" our white flour with several vitamins and iron.

A similar situation has happened with salt. When processing of salt became a national industry, the local salt deposits which contained iodine were no longer used. The majority of our salt today comes from iodineless sources. Even in processing sea salt, the iodine is removed. To some extent, modern progress has neutralized the lack of iodine in commercial salt by refrigerated shipping of seafood to the interior parts of our country. But everyone does not eat fish frequently. Shipping of canned and fresh frozen vegetables and fruits from regions where the soil is rich in iodine has helped to relieve the goiter problem. However, the shopper in the city grocery has no idea whether the vegetables and fruits she buys are rich in iodine or deficient in it.

Incidence of Goiter and Relation to Iodine

Because individuals cannot tell whether their natural supply of iodine is adequate, some plan of giving everyone the food equivalent of iodine has been tried in many parts of the globe for many years. The most popular and most practical way has been to add an infinitesimal (0.01 percent) amount of necessary iodine to table salt.

The name iodized salt came into use more than 25 years ago for table salt containing iodine equivalent in amount to that which would result from adding 0.02 percent of sodium or potassium iodide to ordinary table salt. About a decade ago it was demonstrated that the addition of small quantities of certain substances would greatly retard the loss of iodine that sometimes occurs in packaged iodized salt. Use of these so-called stabilizers has made it possible to reduce the quantity of iodide from 0.02 to 0.01 percent of the salt with assurance that the use of such salt will provide an adequate intake of iodine.

Since iodine occurs in nature in some sources of salt, iodized salt is properly regarded as a natural food. It is an improved salt.

Goiter occurs throughout the world, wherever the supply of iodine is inadequate. A goiter map of Europe was made in 1883, and McCarrison and Eggenberger have prepared maps showing world-wide goiter conditions.

To counteract this goiter prevalence from iodine deficiency, Boussingault in 1831 suggested that iodized salt be sold by the government of Colombia, South America, to prevent goiter. The use of iodine-containing sea salt or the addition of potassium iodide to rock salt was advocated in 1855 by Kostl.

Dr. O. P. Kimball of Cleveland, Ohio, has told of his personal experience with the effect of iodized salt on goiter incidence in Michigan. In 1924, the salt manufacturers agreed to manufacture an iodized salt, and the Wholesale Grocers' Association agreed to handle only iodized salt for table use in Michigan. Michigan lies in the Great Lakes goiter belt. At the time iodized salt was started, a survey placed the incidence of goiter in Michigan at 38.6 percent. A reexamination for goiter was made in 1928 after 4 years of using iodized salt. The incidence of goiter was found to have decreased to 9 percent.

Even more startling proof of the importance of food iodine in salt came from Calumet, Mich. During the depression, approximately two-thirds of the families in this copper-mining community were on relief. Relief officials endeavored to save money by buying bag salt (noniodized) for those on relief. The result was an upshoot in the goiter rate, occurring only in the families using the iodine-lacking salt. Dr. Kimball reported that of children not using iodized salt 60 percent had goiter, and in the same community of those who had remained on iodized salt only 3 percent had goiter.

The safety factor in administering food iodine has been the object of research. Health authorities never advocate self-diagnosis or self-administration of medicine. Treatment of such diseased conditions as toxic goiter is the duty of the physician. Because large amounts of iodine have conspicuous effects upon goiter, studies have been made to assure the toxic goiter patient and his physician that the tiny preventive amounts of food iodine in iodized salt will be safe for the sufferer from toxic goiter. Dr. Kimball personally investigated stories of persons who had complained of iodized salt having caused toxic goiter or other toxic manifestations. He was not able to find any untoward effects from the use of iodized salt.

In 1945 and 1946, the Public Health Service made limited surveys of goiter incidence. They report "Although the population groups that were studied in Florida and Georgia were not in the so-called 'goiter belt', the prevalence of enlarged thyroid was high in both white and negro family groups. In Mitchell County, Ga., 31.3 percent of white women and 8 percent of the males had an enlarged thyroid gland. In Alachua County, Fla., 25.6 percent of the Negro females between 13–20 years of age were affected."

Goiter is a problem in the United States, today. Simple goiter may be prevented by eating food iodine. The use of iodized salt is the most effective way of combating this important public health problem.

Authorities advocating table use of iodized salt are: Forty-fifth Annual Conference, State and Territorial Health Officers with Surgeon General, Public Health Service; Study Committee on Endemic Goiter of the American Public Health Association; Council on Foods and Nutrition of the American Medical Association; Surgeon General, Public Health Service; Food and Nutrition Board of the National Research Council; Medical Research Council, Great Britain, Special Goiter Subcommittee; International Goiter Conference, held in 1927 and 1928.

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INCIDENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED AUGUST 6, 1949

A total of 2,449 cases of poliomyelitis was reported (an increase of 25 percent), as compared with 1,961 last week, a 5-year (1944–48) median of 932, and 1,237 for the corresponding week last year (an increase of only 2 percent). Weekly increases continued in that year, with one exception, until the week ended September 18, when the peak of incidence was reported. The highest incidence was reported earlier, however, in 10 of the past 22 years. During the current week declines were recorded in the West North Central and South Central areas. Current totals (last week's figures in parentheses) for the 4 areas reporting 75 percent of the week's total are as follows: Middle Atlantic 500 (244), East North Central 645 (489), West North Central 427 (509), West South Central 264 (270). States reporting currently more than 14 cases each are as follows: *Increases*—Maine 21 (15), Massachusetts 82 (33), Connecticut 32 (14), New York 390 (200), New Jersey 81 (27), Pennsylvania 29 (17), Ohio 99 (65), Indiana 96 (83), Illinois 250 (145), Wisconsin 53 (47), Minnesota 94 (91), Iowa 84 (83), North Dakota 48 (18), Nebraska 31 (23), Virginia 27 (13), West Virginia 26 (24), Mississippi 22 (12), Texas 121 (95), Idaho 33 (30), Colorado 33 (21), Washington 24 (16), California 112 (87); *decreases*—Michigan 147 (149), Missouri 110 (194), Kansas 46 (68), Kentucky 35 (47), Tennessee 36 (48), Arkansas 64 (73), Oklahoma 75 (96). A total of 10,743 cases has been reported for the year to date, as compared with 7,030 for the same period last year and a 5-year median of 3,992.

Of 30 cases of Rocky Mountain spotted fever (last week 40, 5-year median 40), 26 occurred in the South Atlantic and South Central areas (11 in Virginia, 5 in North Carolina, 4 in Kentucky), 2 in Pennsylvania, and 1 each in New Jersey and Utah.

During the week, 2 cases of anthrax were reported, in New York.

Deaths recorded during the week in 93 large cities in the United States totaled 8,829, as compared with 8,913 last week, 8,261 and 8,937, respectively, for the corresponding weeks of 1948 and 1947, and a 3-year (1946–48) median of 8,242. The total to date is 289,680, as compared with 291,322 for the corresponding week last year. Infant deaths totaled 736, last week 688, 3-year median 675. The cumulative figure is 20,115, same period last year 20,858.

Telegraphic case reports from State health officers for week ended August 6, 1949

[Leaders indicate that no cases were reported]

Division and State	Diphtheria	Encephalitis, infectious	Influenza	Measles	Meningitis, meningococcal	Pneumonia	Poliomyelitis	Rocky Mt. spotted fever	Scarlet fever	Smallpox	Tularemia	Typhoid and paratyphoid fever [e]	Whooping cough	Rabies in animals
NEW ENGLAND														
Maine				15			21		3				15	
New Hampshire							9						3	
Vermont				11			7					1		
Massachusetts	8	1		40			82		12			1	112	
Rhode Island				5	1	4	7					2	1	
Connecticut				24	1	24	32		2				43	
MIDDLE ATLANTIC														
New York	3	2	(c)	186	3	116	390		d 40			4	235	13
New Jersey	1	2	1	116	2	42	81	1	7		1	2	102	
Pennsylvania	2	1	(c)	102	8		29	2	13			3	129	1
EAST NORTH CENTRAL														
Ohio	2		1	97	1	43	99		26		1	15	123	8
Indiana	5	1	34	7		5	96		3			4	5	10
Illinois		1	1	104	2	82	250		5			3	163	1
Michigan [a]	4	1	3	59		5	147		16			2	63	2
Wisconsin		2	12	161		1	53						85	
WEST NORTH CENTRAL														
Minnesota	2			11	1	19	94		1					1
Iowa		2		5			84						6	2
Missouri	2		1	1	4	4	110		1		2	6	8	
North Dakota		5		14	1		48		1					
South Dakota				2	4	1	14		1				1	
Nebraska			5	8		1	31		11				12	
Kansas	1		18	5		5	46		2			1	9	
SOUTH ATLANTIC														
Delaware				1	1		3						4	
Maryland [a]			2	10		28	14	3	2			1	29	
District of Columbia				10	1	10	4						3	
Virginia	1		79	66	1	31	27	11	3		1	5	42	2
West Virginia	1		18	5	1	3	26		3			4	26	
North Carolina	7			13			11	5	4			3	16	
South Carolina	3		38	10	1	18	1		2			3	5	2
Georgia	11	2	17	26	3	122	13		3			5	5	7
Florida	2			12		6	18		3			2		2

Telegraphic case reports from State health officers for week ended August 6, 1949—Continued

Division and State	Diphtheria	Encephalitis, infectious	Influenza	Measles	Meningitis, meningococcal	Pneumonia	Poliomyelitis	Rocky Mt. spotted fever	Scarlet fever	Smallpox	Tularemia	Typhoid and paratyphoid fever [e]	Whooping cough	Rabies in animals
EAST SOUTH CENTRAL														
Kentucky	5			3	3	4	35	4	8			13	26	8
Tennessee	2		3	3		34	36	1	12		1	7	26	
Alabama	3		2	42	2	10	6		3			11	7	6
Mississippi [a]	7		1	9		22	22		4		2	7	2	
WEST SOUTH CENTRAL														
Arkansas	1		29	9		9	64		3		14	6	24	
Louisiana	1		1			10	4					6		
Oklahoma	1		11	6		7	75	1				3	8	4
Texas	8	1	208	85	8	194	121	1	8		1	6	148	13
MOUNTAIN														
Montana				7			5		1					
Idaho			2	8		3	33		[d] 3			2	2	
Wyoming				1	1		9				3	1		
Colorado			7	26	1	10	33		3			2	6	
New Mexico				10		3	11		1					
Arizona	1		11	4		4	6		2				7	1
Utah [a]				32		2	8	1	2				56	
Nevada				1			2							
PACIFIC														
Washington				32	1		24		13				15	
Oregon				13		7	6				5		24	
California	2	1	3	109	5	10	112		[d] 21			5	100	1
Total	86	22	508	1,526	57	899	2,449	30	248		31	136	1,696	
Median, 1944–48	190	19	489	1,387	57		932	40	673	1	20	165	2,519	
Year to date 31 weeks	4,134	333	76,375	585,374	2,250	54,139	[1] 10,743	377	58,007	39	758	2,047	35,106	
Median, 1944–48	6,823	294	190,686	545,980	4,331		3,992	335	85,740	261	595	2,241	60,231	
Seasonal low week ends	(27th) 9 July		(30th) 31 July	(35th) 4 Sept	(37th) 18 Sept		(11th) 19 Mar		(32d) 14 Aug	(35th) 4 Sept		(11th) 19 Mar	(39th) 2 Oct	
Since seasonal low week	366		508	637,767	3,094		[1] 9,820		80,705	49		1,587	45,139	
Median, 1943–48 [b]	669		489	580,926	5,835		3,729		124,311	344		1,766	87,590	

[a] Period ended earlier than Saturday.
[b] The median of the 5 preceding corresponding periods; for diphtheria, influenza, poliomyelitis, and typhoid fever the corresponding periods are 1944–45 to 1948–49.
[c] New York City and Philadelphia only, respectively.
[d] Including cases reported as streptococcal infection and septic sore throat.
[e] Including paratyphoid fever; currently reported separately, as follows: Rhode Island 2, Ohio 6, Virginia 1, South Carolina 2, Georgia 2, Florida 1, Kentucky 2, Alabama 1, Louisiana 1, Colorado 2, California 4. Cases reported as Salmonella infection, not included in the table, were as follows: New York 2.
Leprosy: Week ended July 30, Texas 1. *Relapsing fever*: Week ended July 30, Texas 4. *Anthrax;* New York, 2.
[1] Correction: Florida—Week ended Jan: 22, 4 cases (instead of 5); week ended July 30, 7 cases (instead of 8).
Hawaii Territory: Measles 12, poliomyelitis 1. Alaska: Influenza 1, pneumonia 4.

PLAGUE INFECTION IN LOGAN COUNTY, KANS., AND SWEETWATER COUNTY, WYO.

Under dates of August 5 and 3, respectively, plague infection was reported proved in a pool of 99 fleas from 8 prairie dogs, *Cynomys ludovicianus*, shot July 22 on a farm 8 miles south and 5 miles west of Russell Springs, Logan County, Kans., and in a pool of 38 fleas from 32 white footed mice, *Peromyscus maniculatus*, trapped July 9 at a location 5 miles northeast of Rock Springs, Sweetwater County, Wyo., on U. S. Highway 30.

This is believed to be the first demonstration of plague infection in Sweetwater County, Wyo.

FOREIGN REPORTS

CANADA

Provinces—Notifiable diseases—Week ended July 16, 1949.—During the week ended July 16, 1949, cases of certain notifiable diseases were reported by the Dominion Bureau of Statistics as follows:

Disease	Prince Edward Island	Nova Scotia	New Brunswick	Quebec	Ontario	Manitoba	Saskatchewan	Alberta	British Columbia	Total
Chickenpox		47		56	109	23	85	41	44	405
Diphtheria				9	1					10
Dysentery, bacillary				4						4
Encephalitis, infectious							1			1
German measles		2		22	8	3	20	34	3	92
Influenza		32			5	1				38
Measles		12		235	126	62	256	80	180	951
Meningitis, meningococcal		1								1
Mumps		23		22	63	9	1	4	13	135
Poliomyelitis		1		12	23	1	1		10	48
Scarlet fever		2		12	8	3		10	4	39
Tuberculosis (all forms)		1	6	82		24	18		108	239
Typhoid and paratyphoid fever				11			2		9	22
Undulant fever				2						2
Venereal diseases:										
Gonorrhea		10	11	77	63	30	11	29	84	315
Syphilis		4	4	83	45	8	5	2	13	164
Other									1	1
Whooping cough		2		85		7	2		1	97

Newfoundland cases: Diphtheria, 1; gonorrhea, 4; syphilis, 5.

FINLAND

Notifiable diseases—May 1949.—During the month of May 1949, cases of certain notifiable diseases were reported in Finland as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	9	Poliomyelitis	8
Diphtheria	98	Scarlet fever	302
Dysentery	5	Syphilis	72
Gonorrhea	623	Typhoid fever	16
Paratyphoid fever	319		

WORLD DISTRIBUTION OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

From consular reports, international health organizations, medical officers of the Public Health Service, and other sources. The reports contained in the following tables must not be considered as complete or final as regards either the list of countries included or the figures for the particular countries for which reports are given.

CHOLERA

(Cases)

NOTE.—Since many of the figures in the following tables are from weekly reports, the accumulated totals are for approximate dates.

Place	January-May 1949	June 1949	July 1949—week ended—				
			2	9	16	23	30
ASIA							
Burma	190	38	5	6			
Bassein	128	36	5	6			
Moulmein	2						
Rangoon		2					
Ceylon:							
Trincomalee	1	1					
China:							
Amoy				[1] 1			
India	47,479	5,832	1,438	945	818		
Allahabad	5						
Bombay	[2] 1	[2] 2			[2] 1		
Calcutta	3,969	221	62	63	67	56	
Cawnpore	45	58	11	2	5	5	
Cocanada						1	
Cuddalore	2						
Lucknow	20	5	4	2			
Madras	57	19	19	18	16	34	
Masulipatam		1					
Nagpur					1		
Negapatam	26						
New Delhi		[3] 2	2		7	3	
Raj Samand	10	30					
Tuticorin	14						
India (French):							
Karikal	55						
Pondicherry	100						
Indochina (French):							
Annam	53						
Cambodia	34	8					
Cochinchina	4	6					
Pakistan	20,696	[4] 722		2	1		
Chittagong	63	8		2	1		
Dacca	90	2					
Lahore	11						
Siam	8	1					
Bangkok	8						

[1] Suspected. [2] Imported. [3] Includes imported cases. [4] Preliminary figures.

PLAGUE*

(Cases)

Place	January-May 1949	June 1949	July 2	July 9	July 16	July 23	July 30
AFRICA							
Basutoland	36						
Belgian Congo	6	1	2	1	1		
Costermansville Province		1	1				
Stanleyville Province	6		1	[1] 2	1		
British East Africa:							
Kenya	2	2	1				
Tanganyika	15						
Madagascar	64	2					
Tananarive	3						
Rhodesia, Northern	2						
Union of South Africa	[2] 33	3	1	[2] 9	1	[2,3] 6	
ASIA							
Burma	[4] 404	1	1				
Mandalay	1						
Moulmein	[5] 6						
Rangoon	[4] 5	1	1				

See footnotes at end of table.

PLAGUE—Continued

Place	January-May 1949	June 1949	July 1949—week ended—				
			2	9	16	23	30
ASIA—continued							
China:							
Chekiang Province	7						
Wenchow	7						
Fukien Province	20						
Kiangsi Province	9						
India	24,293	369	[6] 73	[6] 151	[6] 63	[7] 9	
Indochina (French)	84	3					
Annam	17	1					
Cambodia	20						
Cochinchina	29	2					
Laos	3						
Java	5	1					
Siam	151	1					
EUROPE							
Portugal: Azores	4						
SOUTH AMERICA							
Peru:							
Lambayeque Department	7						
Lima Department	3						
Piura Department	6						
Venezuela:							
Aragua State	1						
OCEANIA							
Hawaii Territory: Plague infected rats [8]							

[1] Pneumonic plague. [2] Includes suspected cases. [3] In Cape Province, distributed as follows: Smuts Farm, Kuruman District, 1 case (suspected); Boskop Farm, Gordonia District, 2 cases (1 fatal); Glen Aden and Hemelstraat 3 cases (1 fatal). [4] Includes imported cases. [5] Corrected figure. [6] Preliminary figures. [7] In Calcutta only. [8] Plague infection has been reported in Hawaii Territory as follows: On Mar. 12, 1949, in a mass inoculation of 2 pools of tissue from 10 rats (8 and 2), taken on Maui Island; on Mar. 16, 1949, in mass inoculation of 3 pools of 29 fleas (7, 12, and 10) from rats trapped on the Island of Hawaii.

*During the period July 23–Aug. 6, 1949, 2 cases of bubonic plague were reported in the State of New Mexico in the United States—1 case in Taos County and 1 case in Sandoval County.

SMALLPOX

(Cases)

(P=present)

AFRICA							
Algeria	112						
Angola	[1] 327						
Belgian Congo	941	48					
British East Africa:							
Kenya	22	2					
Nyasaland	772	90	8	10			
Tanganyika	271						
Uganda	32						
Cameroon (British)	10						
Cameroon (French)	52	9					
Dahomey	228	42			[2] 10		
Egypt	3						
Eritrea	1						
Ethiopia	4						
French Equatorial Africa	21	48					
French Guinea	1						
French West Africa: Haute Volta	72	36			[2] 10	[3] 1	
Gambia	51	3		[4] 4			
Gold Coast	[5] 17						
Ivory Coast	194	17			[2] 32	[3] 8	
Morocco (French)	8						
Mozambique	111	16					
Nigeria	6,087	594	[6] 3	[6] 15			
Niger Territory	405	[7] 6					
Portuguese Guinea	1						
Rhodesia:							
Northern	4	1					
Southern	223	53					
Senegal	16						
Sierra Leone	101	[7] 6					

See footnotes at end of table.

SMALLPOX—Continued

Place	January-May 1949	June 1949	July 1949—week ended—				
			2	9	16	23	30
AFRICA—continued							
Sudan(Anglo-Egyptian)	[8] 67	[1] 64	12	4	16		
Sudan (French)	151	[7] 1					
Togo (French)	64	40			[2] 26		
Union of South Africa	190	P	P	P	P		
ASIA							
Afghanistan	35						
Arabia	[8] 35	[9] 3					
Bahrein Islands	[8] 45	1			5		
Burma	1,365	[10] 15	[10] 6	[10] 6	[9] [10] 7	[9] [10] 7	
Ceylon		[9] 1					
China	855	[11] 14	[11] 5	[11] 2	[11] 1	[11] 1	
India	48,607	4,626	[7] 1,082	[7] 520	[7] 301		
India (French): **Yanaon**	1						
India (Portuguese)	191	14	3				
Indochina (French)	2,198	37	31	17	7	29	
Iran	197	[7] 24	[7] 3				
Iraq	[8] 310	50	10	4	25		9
Israel	[12] 3						
Japan	95	19	1	4	1		
Korea (Southern)	544						
Lebanon	[8] 134	[7] 5					
Malay States (Federated)	43						
Manchuria					[13] 9		
Netherlands Indies:							
Java	[8] 4,413	1,100	319	321	186	245	
Riouw Archipelago	2						
Sumatra	[8] 68	[8] 26	[9] 3	9	9	8	
Pakistan	2,909	[7] 141					
Philippine Islands:							
Mindoro Island	11						
Romblon Province	[9] 4						
Tablas Island	2						
Portuguese Timor	4						
Siam	37						
Straits Settlements: **Singapore**	[8] 2						
Syria	320	37	9	5	32		
Transjordan	165	21	3	1		2	
Turkey. (See Turkey in Europe)							
EUROPE							
Belgium		1					
Great Britain: England and Wales	[8] 20						
Italy	[14] 93	[15] 5					
Portugual: **Lisbon**	3						
Spain	2						
Canary Islands	6						
Turkey	88	[7] 1					3
NORTH AMERICA							
Cuba: **Habana**	[8] 5	[9] 1					
Guatemala	1						
Mexico	34	10				[16] 1	
SOUTH AMERICA							
Argentina	[1] 58			3			
Bolivia	[17] 35						
Brazil	[1] 67	[16] 4	[16] 5		[16] 1		
Chile	[9] 2						
Colombia	[1] 1,314	[1] 362					
Ecuador	[1] 450	51					
Paraguay	[9] 2						
Peru	1,151						
Venezuela	1,090						
OCEANIA							
Guam	[18] 2						

[1] Includes alastrim. [2] July 1–10, 1949. [3] July 11–20, 1949. [4] In Bathurst. [5] Jan. 1–15, 1949. [6] In Lagos. [7] Preliminary figures. [8] Includes imported cases. [9] Imported. [10] In ports only. [11] In Shanghai only. [12] Includes 1 case reported for week ended May 28, 1949, in Jerusalem. [13] At Port Arthur. [14] Includes 90 cases of varioloid reported in Rome Jan. 1–May 27, 1949. [15] Varioloid reported in Rome. [16] Alastrim. [17] Jan. 1–Feb. 15, 1949. [18] May 1–31, 1949.

TYPHUS FEVER*

(Cases)

(P=present)

Place	January-May 1949	June 1949	July 1949—week ended— 2	9	16	23	30
AFRICA							
Algeria	43						
Basutoland	7						
Belgian Congo	[1] 41						
British East Africa:							
Kenya	1						
Nyasaland		4					
Egypt	169	2	1				
Eritrea	40	10	4	1			
Ethiopia	336						
Gold Coast	1						
Libya	134	23			2		
Madagascar: **Tananarive**	[1] 6						
Morocco (French)	11	3					
Morocco (Spanish)	1	1					
Sierra Leone	[1] 1						
Tunisia	55	2					
Union of South Africa	[2] 53	P	P	P			
ASIA							
Afghanistan	1,395				([3])		
Arabia: **Aden**	[4] 2						
Burma: **Rangoon**	[5] 1						
Ceylon: **Colombo**	[1] 3	[1] 1					
China	25						
India	[4] 192	4					
India (Portuguese)	18	1					
Indochina (French)	6	4					
Iran	123	7					
Iraq	[1] 26	3		3	1	1	7
Japan	83	5	1		2		
Korea	142						
Lebanon	[1] 1						
Pakistan	562	27	1				
Palestine	[6] 100						
Philippine Islands: **Manila**	[1] 1						
Straits Settlements: **Singapore**	[7] 2						
Syria	15	5			1		
Transjordan	50	3	2		1		
Turkey. (See Turkey in Europe.)							
EUROPE							
Belgium	1	[8] 4					
Bulgaria	305	40	4	3	6		
Czechoslovakia	17	3					
France	2	2					
Great Britain: Island of Malta	[1] 4				[1] 1		
Greece	[2] 26	5		1			
Hungary	17	3					
Italy	27	[1] 2					
Sicily	13						
Poland	210	11					
Portugal: **Lisbon**	4						
Rumania	417						
Spain	2						
Turkey	109	13	4	3	3	3	
Yugoslavia	130	26					
NORTH AMERICA							
Bahama Islands: **Nassau**		[1] 1					
Costa Rica [1]	15	1					
Cuba [1]	3						
Guatemala	13	14					
Jamaica [1]	6	4	4				
Mexico [2]	78	11	4	6	6		
Panama Canal Zone [1]	5	1					
Puerto Rico [1]	10	10	1	3	2	1	
SOUTH AMERICA							
Argentina [1]	1						
Bolivia	53						
Brazil	2						
Chile [2]	108	16	2	2	6		
Colombia [2]	1,178	266					

See footnotes at end of table.

TYPHUS FEVER—Continued

Place	January-May 1949	June 1949	July 1949—week ended—				
			2	9	16	23	30
SOUTH AMERICA—continued							
Curacao [1]	4	1					
Ecuador [2]	122	48					
Peru	506						
Venezuela [1]	23	2					
OCEANIA							
Australia [1]	66	16					
Hawaii Territory [1]	3	1					1

*Reports from some areas are probably murine type, while others include both murine and louse-borne types.
[1] Murine type. [2] Includes murine type. [3] Epidemic of louse-borne typhus fever reported in Afghanistan July 22, 1949. [4] Includes imported cases. [5] Apr. 1-30, 1949. [6] Approximate number reported in outbreak in villages in Hebron and Bethlehem districts in February 1949. [7] One case type unspecified, 1 case murine type. [8] Imported.

YELLOW FEVER

(C=cases; D=deaths)

AFRICA							
Belgian Congo:							
Stanleyville Province ... D	5						
Gold Coast ... C	[1] 3	1		1	[1] 5	[2] 2	
Akwatia ... C					3	[2] 1	
Birim District ... C	[2] 2					[2] 1	
Komenda Village [3] ... D	1						
Nkwanta Dunkwa Area ... D				1			
Nyakrom ... D					[2] 2		
Oseikrome Village [3] ... D		1					
Nigeria:							
Lagos ... D	[4] 2						
NORTH AMERICA							
Panama:							
Pacora ... C	[5] 8						
SOUTH AMERICA							
Brazil:							
Amazonas State ... D	1						
Para State ... D	[6] 3						
Peru:							
Cuzco Department ... D	[7] 1						

[1] Includes suspected cases. [2] Suspected. [3] Near seaport of Sekondi. [4] Cases admitted to Lagos Hospital from ship that arrived from two other ports in Nigeria—Warri and Burutu. [5] Reported Jan. 15, 1949. Date of occurrence Nov. 11-Dec. 30, 1948. Five cases, all fatal, confirmed; 3 suspected cases. [6] Includes 1 case reported for Apr. 12, 1949, at Santarem. [7] In Quincemil, Jan. 1-31, 1949.

DEATHS DURING WEEK ENDED JULY 30, 1949

[From the Weekly Mortality Index, issued by the National Office of Vital Statistics]

	Week ended July 30, 1949	Corresponding week, 1948
Data for 94 large cities of the United States:		
Total deaths	8,945	8,338
Median for 3 prior years	8,338	
Total deaths, first 30 weeks of year	281,632	283,670
Deaths under 1 year of age	687	694
Median for 3 prior years	679	
Deaths under 1 year of age, first 30 weeks of year	19,429	20,212
Data from industrial insurance companies:		
Policies in force	70,309,590	70,988,876
Number of death claims	12,398	12,250
Death claims for 1,000 policies in force, annual rate	9.2	9.0
Death claims per 1,000 policies, first 30 weeks of year, annual rate	9.4	9.7

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