16th April-Multi-Project

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Project Overview

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Project Description

16th April-Multi-ProjectDesc

Rubric

Test_1 (0 to 4)
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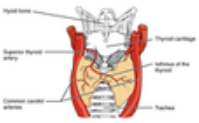
Kavita Hegade



Thyroid

Kavita Hegade

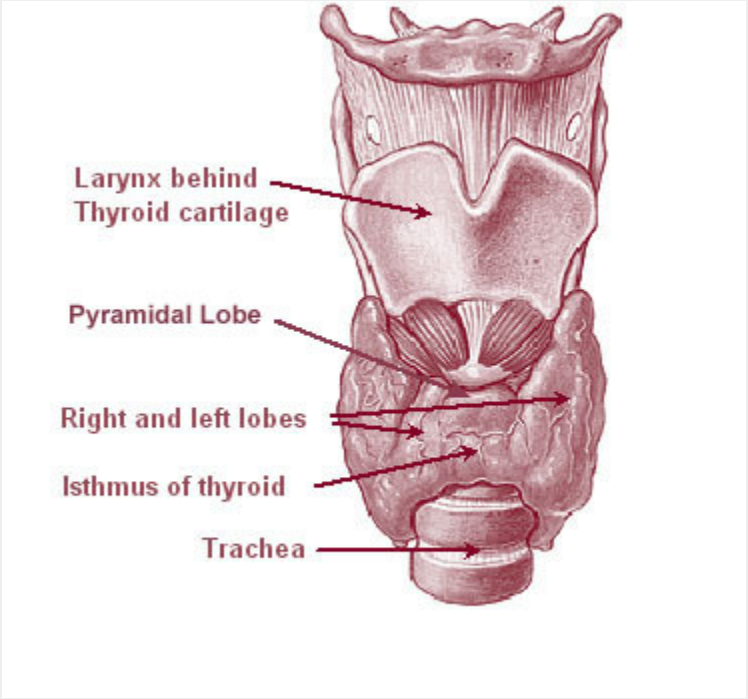
Apr 17, 2018 at 1:41 PM

Version 2

Thyroid

The thyroid gland, or simply the thyroid, is an endocrine gland in the neck, consisting of two lobes connected by an isthmus. It is found at the front of the neck, below the Adam's apple. The thyroid gland secretes thyroid hormones, which primarily influence the metabolic rate and protein synthesis. The hormones also have many other effects including those on development. The thyroid hormones triiodothyronine (T3) and thyroxine (T4) are created from iodine and tyrosine. The thyroid also produces the hormone calcitonin, which plays a role in calcium homeostasis.[1]

Hormonal output from the thyroid is regulated by thyroid-stimulating hormone (TSH) secreted from the anterior pituitary gland, which itself is regulated by thyrotropin-releasing hormone (TRH) produced by the hypothalamus.[2]



The thyroid may be affected by several diseases. Hyperthyroidism occurs when the gland produces excessive amounts of thyroid hormones, the most common cause being Graves' disease, an autoimmune disorder. In contrast, hypothyroidism is a state of insufficient thyroid hormone production. Worldwide, the most common cause is iodine deficiency. Thyroid hormones are important for development, and hypothyroidism secondary to iodine deficiency remains the leading cause of preventable intellectual disability.[3] In iodine-sufficient regions, the most common cause of hypothyroidism is Hashimoto's

thyroiditis, also an autoimmune disorder. In addition, the thyroid gland may also develop several types of nodules and cancer.

Development

In the development of the embryo, at 3–4 weeks gestational age, the thyroid gland appears as an epithelial proliferation in the floor of the pharynx at the base of the tongue between the tuberculum impar and the copula linguae. The copula soon becomes covered over by the hypopharyngeal eminence [17] at a point later indicated by the foramen cecum. The thyroid then descends in front of the pharyngeal gut as a bilobed diverticulum through the thyroglossal duct. Over the next few weeks, it migrates to the base of the neck, passing in front of the hyoid bone. During migration, the thyroid remains connected to the tongue by a narrow canal, the thyroglossal duct. At the end of the fifth week the thyroglossal duct degenerates and the detached thyroid continues on to its final position over the following two weeks.[17]

The fetal hypothalamus and pituitary start to secrete thyrotropin-releasing hormone (TRH) and thyroid-stimulating hormone (TSH). TSH is first measurable at 11 weeks.[18] By 18–20 weeks, the production of thyroxine (T4) reaches a clinically significant and self-sufficient level.[18][19] Fetal triiodothyronine (T3) remains low, less than 15 ng/dL until 30 weeks, and increases to 50 ng/dL at full-term.[19] The fetus needs to be self-sufficient in thyroid hormones in order to guard against neurodevelopmental disorders that would arise from maternal hypothyroidism.[20] The presence of sufficient iodine is essential for healthy neurodevelopment.[21]

Existing_20Project_20(13th_20April)_20(1).pdf
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The neuroendocrine parafollicular cells, also known as C cells, responsible for the production of calcitonin, are derived from neural crest cells, which migrate to the pharyngeal arches. This part of the thyroid then first forms as the ultimopharyngeal body, which begins in the ventral fourth pharyngeal pouch and joins the primordial thyroid gland during its descent to its final location.[22]

Aberrations in prenatal development can result in various forms of thyroid dysgenesis which can cause congenital hypothyroidism, and if untreated this can lead to cretinism.[18]

Gene and protein expression

Further information: Bioinformatics § Gene and protein expression

About 20,000 protein coding genes are expressed in human cells and 70% of these genes are expressed in the normal thyroid.[43][44] Some 250 of these genes are more specifically expressed in the thyroid with about 20 genes being highly thyroid specific. The corresponding specific proteins are mainly involved in thyroid hormone synthesis, such as thyroglobulin, TPO and IYD, and expressed in follicular cells. Other proteins elevated in the thyroid are calcitonin related proteins such as CALCA and CALCB, expressed in the parafollicular c-cells.

History

The presence and diseases of the thyroid have been noted and treated for thousands of years, although the gland itself has only been described and named since the renaissance.[87] The first recorded mention of the thyroid is in terms of goitre in Chinese texts circa 2700 BCE, of which there is general agreement.[87][88] In 1600 BCE burnt sponge and seaweed were used within China for the treatment of goitres, a practice which has developed in many parts of the world.[87][88] In Ayurvedic medicine, the book Sushruta Samhita written about 1400 BCE describes hyperthyroidism, hypothyroidism and goitre.[88] Aristotle and Xenophon in the fifth century BCE describe cases of Grave's disease, which receives its name over two millennia later owing to descriptions provided by Robert James Graves in 1834,[88] Hippocrates and Plato in the fourth century BCE provided some of the first descriptions of the gland itself, proposing its function as a

salivary gland.[88] Pliny the Elder in the first century BCE referred to epidemics of goitre in the Alps and proposed treatment with burnt seaweed,[87] a practice also referred to by Galen in the second century, referred to burnt sponge for the treatment of goitre.[87]

Embedded Media

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In 1500 polymath Leonardo da Vinci provides the first illustration of the thyroid.[87] In 1543 Anatomist Andreas Vesalius gave the first anatomic description and illustration of the gland.[87] In 1656 the thyroid received its name, by the anatomist Thomas Wharton.[87] The gland was named thyroid, meaning shield, as its shape resembled the shields commonly used in Ancient Greece.[87] The English name thyroid gland[89] is derived from the medical Latin used by Wharton – glandula thyreoidea.[90] Glandula means gland in Latin,[91] and thyreoidea can be traced back to the Ancient Greek word θυρεοειδής, meaning shield-like/shield-shaped.[92]

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Numerous authors described cretinism, myxoedema their relationship with the thyroid in the nineteenth century.[88] Charles Mayo coined the term hyperthyroidism in 1910,[87] Hakaru Hashimoto documented a case of Hashimoto's thyroiditis in 1912, and autoantibodies were demonstrated in 1956.[88] Knowledge of the thyroid and its conditions developed throughout the late nineteenth and twentieth centuries, with many modern treatments and investigative modalities evolving throughout the mid twentieth century, including the use of radioactive iodine, thiouracil and fine needle aspiration.[87]

Other animals

The thyroid gland is found in all vertebrates. In fish, it is usually located below the gills and is not always divided into distinct lobes. However, in some teleosts, patches of thyroid tissue are found elsewhere in the body, associated with the kidneys, spleen, heart, or eyes.[99]

In tetrapods, the thyroid is always found somewhere in the neck region. In most tetrapod species, there are two paired thyroid glands – that is, the right and left lobes are not joined together. However, there is only ever a single thyroid gland in most mammals, and the shape found in humans is common to many other species.[99]

In larval lampreys, the thyroid originates as an exocrine gland, secreting its hormones into the gut, and associated with the larva's filter-feeding apparatus. In the adult lamprey, the gland separates from the gut, and becomes endocrine, but this

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Thyroxine is critical to the regulation of metabolism and growth throughout the animal kingdom. For example, Iodine and T4 trigger the change from a plant-eating water-dwelling tadpole into a meat-eating land-dwelling frog, with better neurological, visuospatial, smell and cognitive abilities for hunting, as seen in other predatory animals. A similar phenomenon happens in the neotenic amphibian salamanders, which, without introducing iodine, don't transform into land-dwelling adults, and live and reproduce in the larval form of aquatic axolotl. Among amphibians, administering a thyroid-blocking agent such as propylthiouracil (PTU) can prevent tadpoles from metamorphosing into frogs; in contrast, administering thyroxine will trigger metamorphosis. In amphibian metamorphosis, thyroxine and iodine also exert a well-studied experimental model of apoptosis on the cells of gills, tail, and fins of tadpoles. Iodine, via iodolipids, has favored the evolution of terrestrial animal species and has likely played a crucial role in the evolution of the human brain.

References

1. Jump up ^ Guyton & Hall 2011, p. 907.
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4. ^ Jump up to: a b c d e f g h i j k l m n o p q r s t u v Gray's Anatomy 2008, pp. 462–4.
5. ^ Jump up to: a b c d e f g h i j k Elsevier's 2007, p. 342.
6. Jump up ^ Elsevier's 2007, pp. 342–3.
7. Jump up ^ Ellis, Harold; Susan Standring; Gray, Henry David (2005). Gray's anatomy: the anatomical basis of clinical practice. St. Louis, Mo: Elsevier Churchill Livingstone. pp. 538–539. ISBN 0-443-07168-3.
8. Jump up ^ Netter, Frank H. (2014). Atlas of Human Anatomy Including Student Consult Interactive Ancillaries and Guides (6th edition. ed.). Philadelphia, Penn.: W B Saunders Co. p. 27. ISBN 978-1-4557-0418-7.
9. ^ Jump up to: a b Page, C; Cuvelier, P; Biet, A; Boute, P; Laude, M; Strunski, V (July 2009). "Thyroid tubercle of Zuckerkandl: anatomical and surgical experience from 79 thyroidectomies". The Journal of laryngology and otology. 123 (7): 768–71. doi:10.1017/s0022215108004003. PMID 19000342.
10. ^ Jump up to: a b c Elsevier's 2007, p. 343.
11. ^ Jump up to: a b Cicekcibasi, AE; Salbacak, A; Seker, M; Ziylan, T; Tuncer, I; Buyukmumcu, M (April 2007). "Developmental variations and clinical importance of the fetal thyroid gland. A morphometric study". Saudi medical journal. 28 (4): 524–8. PMID 17457471.
12. Jump up ^ Kim DW; Jung SL; Baek JH; et al. (Jan 2013). "The prevalence and features of thyroid pyramidal lobe, accessory thyroid, and ectopic thyroid as assessed by computed tomography: a multicenter study". Thyroid. 23 (1): 84–91. doi:10.1089/thy.2012.0253.
13. Jump up ^ Dorland's (2012). Illustrated Medical Dictionary 32nd edition. Elsevier Saunders. pp. 999 redirect to 1562. ISBN 978-1-4160-6257-8.
14. Jump up ^ Fawcett, Don; Jensh, Ronald (2002). Bloom & Fawcett's Concise Histology. New York: Arnold Publishers. pp. 257–258. ISBN 0-340-80677-X.
15. ^ Jump up to: a b Young, Barbara; et al. (2006). Wheater's functional histology : a text and colour atlas (5th ed.). Oxford: Churchill Livingstone. pp. 333–335. ISBN 978-0-443-06850-8.
16. Jump up ^ Hazard, JB (July 1977). "The C cells (parafollicular cells) of the thyroid gland and medullary thyroid carcinoma. A review". The American Journal of Pathology. 88 (1): 213–50. PMC 2032150 [Freely accessible] . PMID 18012.
17. ^ Jump up to: a b Larsen, William J. (2001). Human embryology (3. ed.). Philadelphia, Pa.: Churchill Livingstone. pp. 372–374. ISBN 0-443-06583-7.
18. ^ Jump up to: a b c d Greenspan's 2011, p. 179.
19. ^ Jump up to: a b Eugster, Erica A.; Pescovitz, Ora Hirsch (2004). Pediatric endocrinology: mechanisms, manifestations and management. Hagerstwon, MD: Lippincott Williams & Wilkins. p. 493 (Table 33-3). ISBN 0-7817-4059-2.
20. Jump up ^ Zoeller RT (April 2003). "Transplacental thyroxine and fetal brain development". J. Clin. Invest. 111 (7): 954–7. doi:10.1172/JCI18236. PMC 152596 [Freely accessible] . PMID 12671044.
21. ^ Jump up to: a b c d "Iodine supplementation in pregnant and lactating women". World Health Organization.

Retrieved 2016-11-13.
22. ^ Jump up to: a b Sadler. Langman's Medical Embryology (13th ed.). pp. 285–6,293. ISBN 978-1-4511-9164-6.
23. ^ Jump up to: a b Davidson's 2010, p. 736.
24. Jump up ^ Guyton & Hall 2011, p. 932.
25. Jump up ^ Guyton & Hall 2011, p. 934.
26. ^ Jump up to: a b c d e Guyton & Hall 2011, p. 937.
27. ^ Jump up to: a b c d e Guyton & Hall 2011, p. 936.
28. Jump up ^ Guyton & Hall 2011, p. 935-6.
29. Jump up ^ Greenspan's 2011, p. 169.
30. ^ Jump up to: a b Bowen, R. (2000). "Thyroid Hormone Receptors". Colorado State University. Retrieved 22 February 2015.
31. Jump up ^ Greenspan's 2011, p. 178.
32. ^ Jump up to: a b Boron WF, Boulpaep E (2003). "Chapter 48: "synthesis of thyroid hormones"". Medical Physiology: A Cellular And Molecular Approaoch. Elsevier/Saunders. p. 1300. ISBN 1-4160-2328-3.
33. ^ Jump up to: a b Bianco AC, Salvatore D, Gereben B, Berry MJ, Larsen PR (2002). "Biochemistry, cellular and molecular biology, and physiological roles of the iodothyronine selenodeiodinases". Endocr Rev. 23 (1): 38–89. doi:10.1210/er.23.1.38. PMID 11844744.
34. Jump up ^ Melmed, S; Polonsky, KS; Larsen, PR; Kronenberg, HM (2011). Williams Textbook of Endocrinology (12th ed.). Saunders. p. 331. ISBN 978-1-4377-0324-5.
35. Jump up ^ How Your Thyroid Works: A Delicate Feedback Mechanism. Updated 2009-05-21.
36. Jump up ^ The thyroid gland in Endocrinology: An Integrated Approach by Stephen Nussey and Saffron Whitehead (2001) Published by BIOS Scientific Publishers Ltd. ISBN 1-85996-252-1
37. Jump up ^ Ganong's review of medical physiology Edition 25.
38. Jump up ^ Greenspan's 2011, p. 174.
39. Jump up ^ Greenspan's 2011, p. 177.
40. Jump up ^ Guyton & Hall 2011, p. 896.

Kavita Hegade

Feedback

Peer Reviewer: Kavita Hegade
Apr 17, 2018

Review

Overall Feedback Criterion: 2 of 4

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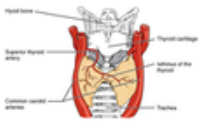

Thyroid

Kavita Hegade

Apr 17, 2018 at 12:53 PM

Version 1

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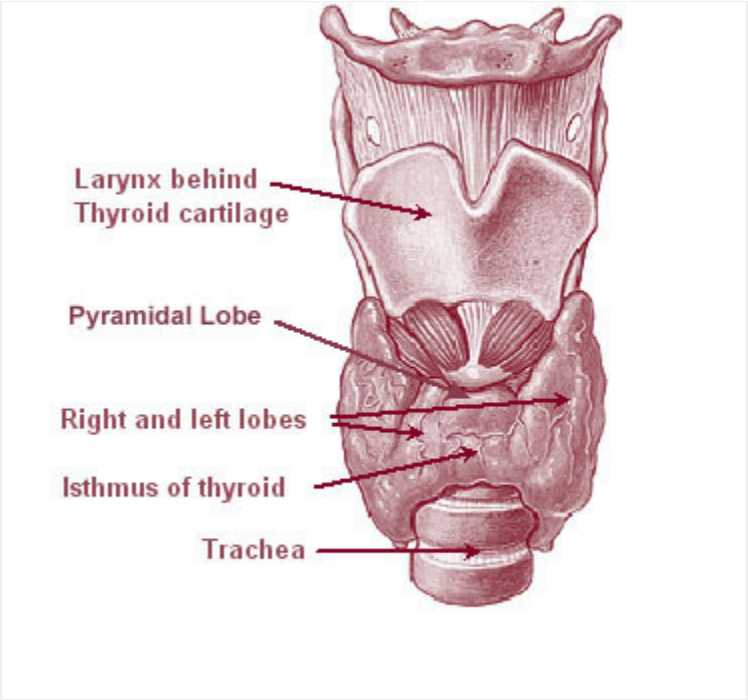

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12. Jump up ^ Kim DW; Jung SL; Baek JH; et al. (Jan 2013). "The prevalence and features of thyroid pyramidal lobe, accessory thyroid, and ectopic thyroid as assessed by computed tomography: a multicenter study". Thyroid. 23 (1): 84–91. doi:10.1089/thy.2012.0253.
13. Jump up ^ Dorland's (2012). Illustrated Medical Dictionary 32nd edition. Elsevier Saunders. pp. 999 redirect to 1562. ISBN 978-1-4160-6257-8.
14. Jump up ^ Fawcett, Don; Jensh, Ronald (2002). Bloom & Fawcett's Concise Histology. New York: Arnold Publishers. pp. 257–258. ISBN 0-340-80677-X.
15. ^ Jump up to: a b Young, Barbara; et al. (2006). Wheater's functional histology : a text and colour atlas (5th ed.). Oxford: Churchill Livingstone. pp. 333–335. ISBN 978-0-443-06850-8.
16. Jump up ^ Hazard, JB (July 1977). "The C cells (parafollicular cells) of the thyroid gland and medullary thyroid carcinoma. A review". The American Journal of Pathology. 88 (1): 213–50. PMC 2032150 [Freely accessible] . PMID 18012.
17. ^ Jump up to: a b Larsen, William J. (2001). Human embryology (3. ed.). Philadelphia, Pa.: Churchill Livingstone. pp. 372–374. ISBN 0-443-06583-7.
18. ^ Jump up to: a b c d Greenspan's 2011, p. 179.
19. ^ Jump up to: a b Eugster, Erica A.; Pescovitz, Ora Hirsch (2004). Pediatric endocrinology: mechanisms, manifestations and management. Hagerstwon, MD: Lippincott Williams & Wilkins. p. 493 (Table 33-3). ISBN 0-7817-4059-2.
20. Jump up ^ Zoeller RT (April 2003). "Transplacental thyroxine and fetal brain development". J. Clin. Invest. 111 (7): 954–7. doi:10.1172/JCI18236. PMC 152596 [Freely accessible] . PMID 12671044.
21. ^ Jump up to: a b c d "Iodine supplementation in pregnant and lactating women". World Health Organization. Retrieved 2016-11-13.
22. ^ Jump up to: a b Sadler. Langman's Medical Embryology (13th ed.). pp. 285–6,293. ISBN 978-1-4511-9164-6.
23. ^ Jump up to: a b Davidson's 2010, p. 736.
24. Jump up ^ Guyton & Hall 2011, p. 932.
25. Jump up ^ Guyton & Hall 2011, p. 934.

26. ^ Jump up to: a b c d e Guyton & Hall 2011, p. 937.
27. ^ Jump up to: a b c d e Guyton & Hall 2011, p. 936.
28. Jump up ^ Guyton & Hall 2011, p. 935-6.
29. Jump up ^ Greenspan's 2011, p. 169.
30. ^ Jump up to: a b Bowen, R. (2000). "Thyroid Hormone Receptors". Colorado State University. Retrieved 22 February 2015.
31. Jump up ^ Greenspan's 2011, p. 178.
32. ^ Jump up to: a b Boron WF, Boulpaep E (2003). "Chapter 48: "synthesis of thyroid hormones"". Medical Physiology: A Cellular And Molecular Approaoch. Elsevier/Saunders. p. 1300. ISBN 1-4160-2328-3.
33. ^ Jump up to: a b Bianco AC, Salvatore D, Gereben B, Berry MJ, Larsen PR (2002). "Biochemistry, cellular and molecular biology, and physiological roles of the iodothyronine selenodeiodinases". Endocr Rev. 23 (1): 38–89. doi:10.1210/er.23.1.38. PMID 11844744.
34. Jump up ^ Melmed, S; Polonsky, KS; Larsen, PR; Kronenberg, HM (2011). Williams Textbook of Endocrinology (12th ed.). Saunders. p. 331. ISBN 978-1-4377-0324-5.
35. Jump up ^ How Your Thyroid Works: A Delicate Feedback Mechanism. Updated 2009-05-21.
36. Jump up ^ The thyroid gland in Endocrinology: An Integrated Approach by Stephen Nussey and Saffron Whitehead (2001) Published by BIOS Scientific Publishers Ltd. ISBN 1-85996-252-1
37. Jump up ^ Ganong's review of medical physiology Edition 25.
38. Jump up ^ Greenspan's 2011, p. 174.
39. Jump up ^ Greenspan's 2011, p. 177.
40. Jump up ^ Guyton & Hall 2011, p. 896.

Kavita Hegade

Feedback

Peer Reviewer: Aakash Hs
Apr 17, 2018

Review

Test_1: 2 of 4

The thyroid may be affected by several diseases. Hyperthyroidism occurs when the gland produces excessive amounts of thyroid hormones, the most common cause being Graves' disease, an autoimmune disorder. In contrast, hypothyroidism is a state of insufficient thyroid hormone production. Worldwide, the most common cause is iodine deficiency.

Test_1: 3 of 4

The thyroid may be affected by several diseases. Hyperthyroidism occurs when the gland produces excessive amounts of thyroid hormones, the most common cause being Graves' disease, an autoimmune disorder. In contrast, hypothyroidism is a state of insufficient thyroid hormone production. Worldwide, the most common cause is iodine deficiency.

Test_1: 2 of 4

Hormonal output from the thyroid is regulated by thyroid-stimulating hormone (TSH) secreted from the anterior

pituitary gland, which itself is regulated by thyrotropin-releasing hormone (TRH) produced by the hypothalamus.[2]

Test_1: 3 of 4

The thyroid gland, or simply the thyroid, is an endocrine gland in the neck, consisting of two lobes connected by an isthmus. It is found at the front of the neck, below the Adam's apple. The thyroid gland secretes thyroid hormones, which primarily influence the metabolic rate and protein synthesis. The hormones also have many other effects including those on development. The thyroid hormones triiodothyronine (T3) and thyroxine (T4) are created from iodine and tyrosine. The thyroid also produces the hormone calcitonin, which plays a role in calcium homeostasis.[1]

Annotations

A1. Comment: *The thyroid may be affected by several diseases*

A2. Suggested Change - Check citation - suggestion?: *Thyroid hormones are important for development,*

A3. Suggested Change - Change word - suggestion?: *In addition, the thyroid gland may also develop several types of nodules and cancer.*

Feedback

Peer Reviewer: Mahalakshmi Maha1
Apr 17, 2018

Review

Test_1: 1 of 4

In contrast, hypothyroidism is a state of insufficient thyroid hormone production. Worldwide, the most common cause is iodine deficiency.

Test_1: 2 of 4

The thyroid may be affected by several diseases. Hyperthyroidism occurs when the gland produces excessive amounts of thyroid hormones, the most common cause being Graves' disease, an autoimmune disorder. In contrast, hypothyroidism is a state of insufficient thyroid hormone production. Worldwide, the most common cause is iodine deficiency.

Test_1: 2 of 4

Hormonal output from the thyroid is regulated by thyroid-stimulating hormone (TSH) secreted from the anterior pituitary gland, which itself is regulated by thyrotropin-releasing hormone (TRH) produced by the hypothalamus.[2]

Test_1: 4 of 4

The thyroid gland, or simply the thyroid, is an endocrine gland in the neck, consisting of two lobes connected by an isthmus. It is found at the front of the neck, below the Adam's apple. The thyroid gland secretes thyroid hormones, which primarily influence the metabolic rate and protein synthesis. The hormones also have many other effects including those on development. The thyroid hormones triiodothyronine (T3) and thyroxine (T4) are created from iodine and tyrosine. The thyroid also produces the hormone calcitonin, which plays a role in calcium homeostasis.[1]

Annotations

B1. Comment: *fgnfdsa*

B2. Comment: *The thyroid gland is found in all vertebrates.*

B3. Comment: *dfgbfdsa*

Feedback

Peer Reviewer: Kavita Hegade
Apr 17, 2018

Review

Overall Feedback Criterion: 2 of 4

asdffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffffff

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Iodine-131

Mahalakshmi Lakshmi

Apr 17, 2018 at 1:47 PM

Version 4

Iodine-131

Iodine-131 (131I) is an important radioisotope of iodine discovered by Glenn Seaborg and John Livingood in 1938 at the University of California, Berkeley.[1] It has a radioactive decay half-life of about eight days. It is associated with nuclear energy, medical diagnostic and treatment procedures, and natural gas production. It also plays a major role as a radioactive isotope present in nuclear fission products, and was a significant contributor to the health hazards from open-air atomic bomb testing in the 1950s, and from the Chernobyl disaster, as well as large fraction of the contamination hazard in the first weeks in the Fukushima nuclear crisis. This is because I-131 is a major fission product of uranium and plutonium, comprising nearly 3% of the total products of fission (by weight). See fission product yield for a comparison with other radioactive fission products. I-131 is also a major fission product of uranium-233, produced from thorium.

Due to its mode of beta decay, iodine-131 is notable for causing mutation and death in cells that it penetrates, and other cells up to several millimeters away. For this reason, high doses of the isotope are sometimes less dangerous than low doses, since they tend to kill thyroidtissues that would otherwise become cancerous as a result of the radiation. For example, children treated with moderate dose of I-131 for thyroid adenomas had a detectable increase in thyroid cancer, but children treated with a much higher dose did not.[citation needed] Likewise, most studies of very-high-dose I-131 for treatment of Graves disease have failed to find any increase in thyroid cancer, even though there is linear increase in thyroid cancer risk with I-131 absorption at moderate doses.[2] Thus, iodine-131 is increasingly less employed in small doses in medical use (especially in children), but increasingly is used only in large and maximal treatment doses, as a way of killing targeted tissues. This is known as "therapeutic use."

Iodine-131 can be "seen" by nuclear medicine imaging techniques (i.e., gamma cameras) whenever it is given for therapeutic use, since about 10% of its energy and radiation dose is via gamma radiation. However, since the other 90% of radiation (beta radiation) causes tissue damage without contributing to any ability to see or "image" the isotope, other less-damaging radioisotopes of iodine such as iodine-123 (see isotopes of iodine) are preferred in situations when only nuclear imaging is required. The isotope I-131 is still occasionally used for purely diagnostic (i.e., imaging) work, due to its low expense compared to other iodine radioisotopes. Very small medical imaging doses of I-131 have not shown any increase in thyroid cancer. The low-cost availability of I-131, in turn, is due to the relative ease of creating I-131 by neutron bombardment of natural tellurium in a nuclear reactor, then separating I-131 out by various simple methods (i.e., heating to drive off the volatile iodine). By contrast, other iodine radioisotopes are usually created by far more expensive techniques, starting with reactor radiation of expensive capsules of pressurized xenon gas.

Iodine-131 **Complete table of nuclides**	
General	
Name, symbol	**Radioiodine, 131I**
Neutrons	**83**
Protons	**53**

Iodine-131 Complete table of nuclides	
Nuclide data	
Half-life	**8.0197 days**
Isotope mass	**130.9061246(12) u**
Excess energy	**971 keV**

Iodine-131 is also one of the most commonly used gamma-emitting radioactive industrial tracer. Radioactive tracer isotopes are injected with hydraulic fracturing fluid to determine the injection profile and location of fractures created by hydraulic fracturing.[3]

Much smaller incidental doses of iodine-131 than those used in medical therapeutic procedures, are supposed by some studies to be the major cause of increased thyroid cancersafter accidental nuclear contamination. These studies suppose that cancers happen from residual tissue radiation damage caused by the I-131, and should appear mostly years after exposure, long after the I-131 has decayed.[4][5] Other studies can't find a correlation.[6][7]

Production

Most I-131 production is from nuclear reactor neutron-irradiation of a natural tellurium target. Irradiation of natural tellurium produces almost entirely I-131 as the only radionuclide with a half-life longer than hours, since most lighter isotopes of tellurium become heavier stable isotopes, or else stable iodine or xenon. However, the heaviest naturally occurring tellurium nuclide, Te-130 (34% of natural Te) absorbs a neutron to become tellurium-131, which beta-decays with a half-life of 25 minutes, to I-131.

A tellurium compound can be irradiated while bound as an oxide to an ion exchange column, and evolved I-131 then eluted into an alkaline solution.[8] More commonly, powdered elemental tellurium is irradiated and then I-131 separated from it by dry distillation of the iodine, which has a far higher vapor pressure. The element is then dissolved in a mildly alkaline solution in the standard manner, to produce I-131 as iodide and hypoiodate (which is soon reduced to iodide).[9]

131I is a fission product with a yield of 2.878% from uranium-235,[10] and can be released in nuclear weapons tests and nuclear accidents. However, the short half-life means it is not present in significant quantities in cooled spent nuclear fuel, unlike iodine-129 whose half-life is nearly a billion times that of I-131.

It is discharged to the atmosphere in small quantities by some nuclear power plants.[11]

Radioactive decay

I-131 decays with a half-life of 8.02 days with beta minus and gamma emissions. This nuclide of iodine has 78 neutrons in its nucleus, while the only stable nuclide, 127I, has 74. On decaying, 131I most often (89% of the time) expends its 971 keV of decay energy by transforming into the stable 131Xe (Xenon) in two steps, with gamma decay following rapidly after beta decay:

The primary emissions of 131I decay are thus electrons with a maximal energy of 606 keV (89% abundance, others 248–807 keV) and 364 keV gamma rays (81% abundance, others 723 keV).[12] Beta decay also produces an antineutrino, which carries off variable amounts of the beta decay energy. The electrons, due to their high mean energy [1][13]

Effects of exposure

Iodine in food is absorbed by the body and preferentially concentrated in the thyroid where it is needed for the functioning of that gland. When 131I is present in high levels in the environment from radioactive fallout, it can be absorbed through contaminated food, and will also accumulate in the thyroid. As it decays, it may cause damage to the thyroid. The primary risk from exposure to high levels of 131I is the chance occurrence of radiogenic thyroid cancer in later life. Other risks include the possibility of non-cancerous growths and thyroiditis.[2]

The risk of thyroid cancer in later life appears to diminish with increasing age at time of exposure. Most risk estimates are based on studies in which radiation exposures occurred in children or teenagers. When adults are exposed, it has been difficult for epidemiologists to detect a statistically significant difference in the rates of thyroid disease above that of a similar but otherwise-unexposed group.[2][15]

The risk can be mitigated by taking iodine supplements, raising the total amount of iodine in the body and, therefore, reducing uptake and retention in the face and chest and lowering the relative proportion of radioactive iodine. However, such supplements were not distributed to the population living nearest to the Chernobyl nuclear power plant after the disaster,[16] though they were widely distributed to children in Poland.

Within the USA, the highest 131I fallout doses occurred during the 1950s and early 1960s to children having consumed fresh sources of milk contaminated as the result of above-ground testing of nuclear weapons.[4] The National Cancer Institute provides additional information on the health effects from exposure to 131I in fallout,[17] as well as individualized estimates, for those born before 1971, for each of the 3070 counties in the USA. The calculations are taken from data collected regarding fallout from the nuclear weapons tests conducted at the Nevada Test Site.[18]

On 27 March 2011, the Massachusetts Department of Public Health reported that 131I was detected in very low concentrations in rainwater from samples collected in Massachusetts, USA, and that this likely originated from the Fukushima power plant.[19] Farmers near the plant dumped raw milk, while testing in the United States found 0.8 pico-curies per liter of iodine-131 in a milk sample, but the radiation levels were 5,000 times lower than the FDA's "defined intervention level." The levels were expected to drop relatively quickly[20]

Medical use

It is used in nuclear medicine therapeutically and can also be seen with diagnostic scanners if it has been used therapeutically. Use of the 131I as iodide salt exploits the mechanism of absorption of iodine by the normal cells of the thyroid gland. Examples of its use in radiation therapy are those where tissue destruction is desired after iodine uptake by the tissue.

Major uses of 131I include the treatment of thyrotoxicosis (hyperthyroidism) and some types of thyroid cancer that absorb iodine. The 131I is thus used as direct radioisotope therapy to treat hyperthyroidism due to Graves' disease, and sometimes hyperactive thyroid nodules (abnormally active thyroid tissue that is not malignant). The therapeutic use of radioiodine to treat hyperthyroidism from Graves' disease was first reported by Saul Hertz in 1941.

The 131I isotope is also used as a radioactive label for certain radiopharmaceuticals that can be used for therapy, e.g. 131I-metaiodobenzylguanidine(131I-MIBG) for imaging and treating pheochromocytoma and neuroblastoma. In all of these therapeutic uses, 131I destroys tissue by short-range beta radiation. About 90% of its radiation damage to tissue is via

beta radiation, and the rest occurs via its gamma radiation (at a longer distance from the radioisotope). It can be seen in diagnostic scans after its use as therapy, because 131I is also a gamma-emitter. (¼M=)

Because of the carcinogenicity of its beta radiation in the thyroid in small doses, I-131 is rarely used primarily or solely for diagnosis (although in the past this was more common due to this isotope's relative ease of production and low expense). Instead the more purely gamma-emitting radioiodine iodine-123 is used in diagnostic testing (nuclear medicine scan of the thyroid). The longer half-lived iodine-125 is also occasionally used when a longer half-life radioiodine is needed for diagnosis, and in brachytherapy treatment (isotope confined in small seed-like metal capsules), where the low-energy gamma radiation without a beta component makes iodine-125 useful. The other radioisotopes of iodine are never used in brachytherapy.

The use of 131I as a medical isotope has been blamed for a routine shipment of biosolids being rejected from crossing the Canada—U.S. border.[37]Such material can enter the sewers directly from the medical facilities, or by being excreted by patients after a treatment.

References

1. "UW-L Brachy Course". wikifoundry. April 2008. Retrieved 2014-04-11.
2. ^ Jump up to:a b c Rivkees, Scott A.; Sklar, Charles; Freemark, Michael (1998). "The Management of Graves' Disease in Children, with Special Emphasis on Radioiodine Treatment". Journal of Clinical Endocrinology & Metabolism. 83 (11): 3767–76. doi:10.1210/jc.83.11.3767. PMID 9814445.
3. Jump up^ Reis, John C. (1976). Environmental Control in Petroleum Engineering. Gulf Professional Publishers.
4. ^ Jump up to:a b Simon, Steven L.; Bouville, André; Land, Charles E. (January–February 2006). "Fallout from Nuclear Weapons Tests and Cancer Risks". American Scientist. 94: 48–57. doi:10.1511/2006.1.48. In 1997, NCI conducted a detailed evaluation of dose to the thyroid glands of U.S. residents from I-131 in fallout from tests in Nevada. (...) we evaluated the risks of thyroid cancer from that exposure and estimated that about 49,000 fallout-related cases might occur in the United States, almost all of them among persons who were under age 20 at some time during the period 1951–57, with 95-percent uncertainty limits of 11,300 and 212,000.
5. Jump up^ "National Cancer Institute calculator for thyroid cancer risk as a result of I-131 intake after nuclear testing before 1971 in Nevada". Ntsi131.nci.nih.gov. Retrieved 2012-06-17.
6. Jump up^ Guiraud-Vitaux, F.; Elbast, M.; Colas-Linhart, N.; Hindie, E. (February 2008). "Thyroid cancer after Chernobyl: is iodine 131 the only culprit ? Impact on clinical practice". Bulletin du cancer. 95 (2): 191–5. doi:10.1684/bdc.2008.0574. PMID 18304904.
7. Jump up^ Centre for Disease Control (2002). The Hanford Thyroid Disease Study (PDF). Retrieved 17 June 2012. no associations between Hanford's iodine-131 releases and thyroid disease were observed. [The findings] show that if there is an increased risk of thyroid disease from exposure to Hanford's iodine-131, it is probably too small to observe using the best epidemiologic methods available Executive summary
8. Jump up^ Chattopadhyay, Sankha; Saha Das, Sujata (2010). "Recovery of 131I from alkaline solution of n-irradiated tellurium target using a tiny Dowex-1 column". Applied Radiation and Isotopes. 68 (10): 1967–9. doi:10.1016/j.apradiso.2010.04.033. PMID 20471848.
9. Jump up^ "I-131 Fact Sheet" (PDF). Nordion. August 2011. Retrieved 2010-10-26.[permanent dead link]
10. Jump up^ "Nuclear Data for Safeguards, Table C-3, Cumulative Fission Yields". International Atomic Energy Agency. Retrieved 14 March 2011. (thermal neutron fission)
11. Jump up^ Effluent Releases from Nuclear Power Plants and Fuel-Cycle Facilities. National Academies Press (US). 2012-03-29.
12. Jump up^ "Nuclide Safety Data Sheet" (PDF). Retrieved 2010-10-26.
13. Jump up^ Skugor, Mario (2006). Thyroid Disorders. A Cleveland Clinic Guide. Cleveland Clinic Press. p. 82. ISBN 978-1-59624-021-6.
14. Jump up^ Council, National Research (11 February 2003). "Exposure of the American Population to Radioactive Fallout from Nuclear Weapons Tests: A Review of the CDC-NCI Draft Report on a Feasibility Study of the Health Consequences to the American Population from Nuclear Weapons Tests Conducted by the United States and Other Nations". nap.edu. doi:10.17226/10621. Retrieved 3 April 2018.
15. Jump up^ Robbins, Jacob; Schneider, Arthur B. (2000). "Thyroid cancer following exposure to radioactive iodine". Reviews in Endocrine and Metabolic Disorders. 1 (3): 197–203. doi:10.1023/A:1010031115233. ISSN 1389-9155. PMID 11705004.
16. Jump up^ Frot, Jacques. "THE CAUSES OF THE CHERNOBYL EVENT". Ecolo.org. Retrieved 2012-06-17.

17. Jump up^ "Radioactive I-131 from Fallout". National Cancer Institute. Retrieved 2007-11-14.
18. Jump up^ "Individual Dose and Risk Calculator for Nevada Test Site fallout". National Cancer Institute. 10/01/07. Archived from the original on 18 October 2007. Retrieved 2007-11-14.Check date values in: |date= (help)
19. Jump up^ "Low Concentrations Of Radiation Found In Mass. | WCVB Home - WCVB Home". Thebostonchannel.com. 27 March 2011. Archived from the original on 3 April 2012. Retrieved 17 June 2012.
20. Jump up^ "Traces of radioactive iodine found in Washington state milk" Los Angeles Times[dead link]
21. Jump up^ Sepe, S. M.; Clark, R. A. (March 1985). "Oxidant membrane injury by the neutrophil myeloperoxidase system. I. Characterization of a liposome model and injury by myeloperoxidase, hydrogen peroxide, and halides". Journal of Immunology. 134 (3): 1888–1895. ISSN 0022-1767. PMID 2981925.
22. Jump up^ Kowalsky RJ, Falen, SW. Radiopharmaceuticals in Nuclear Pharmacy and Nuclear Medicine. 2nd ed. Washington DC: American Pharmacists Association; 2004.
23. Jump up^ (PDF) https://www.eanm.org/scientific_info/guidelines/gl_paed_mibg.pdf?PHPSESSID%3D46d05b62d235c36a12166bf939b656c7. Retrieved 7 October 2011.Missing or empty |title= (help)[dead link]
24. Jump up^ Guidelines for Iodine Prophylaxis following Nuclear Accidents (PDF), Geneva: World Health Organization, 1999
25. Jump up^ "Information on Radiation, Health and the Thyroid, Including Iodine Testing, Potassium Iodide, and Thyrouūid Testing". Thyroid-info.com. Retrieved 2012-06-17.
26. Jump up^ "Potable Aqua Questions and Answers". www.pharmacalway.com. Retrieved 3 April2018.
27. Jump up^ LeMar, H. J.; et al. "Thyroid adaptation to chronic tetraglycine hydroperiodide water purification tablet use. Department of Medicine, Madigan Army Medical Center, Tacoma, Washington 98431". Journal of Clinical Endocrinology & Metabolism, Vol 80, 220-223, doi: 10.1210/jc.80.1.220 Copyright © 1995. Endocrine Society. Retrieved 20 Mar 2010.
28. ^ Jump up to:a b c d e f g Greer, Monte A.; Goodman, Gay; Pleus, Richard C.; Greer, Susan E. (2002). "Health Effects Assessment for Environmental Perchlorate Contamination: The Dose Response for Inhibition of Thyroidal Radioiodine Uptake in Humans". Environmental Health Perspectives. 110 (9): 927–37. doi:10.1289/ehp.02110927. PMC 1240994 [Freely accessible] . PMID 12204829.
29. ^ Jump up to:a b Wolff, J. (1998). "Perchlorate and the thyroid gland". Pharmacological Reviews. 50 (1): 89–105. PMID 9549759.
30. Jump up^ Barzilai, D.; Sheinfeld, M. (1966). "Fatal complications following use of potassium perchlorate in thyrotoxicosis. Report of two cases and a review of the literature". Israel journal of medical sciences. 2 (4): 453–6. PMID 4290684.

Mahalakshmi Lakshmi

Iodine-131

Mahalakshmi Lakshmi

Apr 17, 2018 at 1:16 PM

Version 3

Iodine-131

Iodine-131 (131I) is an important radioisotope of iodine discovered by Glenn Seaborg and John Livingood in 1938 at the University of California, Berkeley.[1] It has a radioactive decay half-life of about eight days. It is associated with nuclear energy, medical diagnostic and treatment procedures, and natural gas production. It also plays a major role as a radioactive isotope present in nuclear fission products, and was a significant contributor to the health hazards from open-air atomic bomb testing in the 1950s, and from the Chernobyl disaster, as well as being a large fraction of the contamination hazard in the first weeks in the Fukushima nuclear crisis. This is because I-131 is a major fission product of uranium and plutonium, comprising nearly 3% of the total products of fission (by weight). See fission product yield for a comparison with other radioactive fission products. I-131 is also a major fission product of uranium-233, produced from thorium.

Due to its mode of beta decay, iodine-131 is notable for causing mutation and death in cells that it penetrates, and other cells up to several millimeters away. For this reason, high doses of the isotope are sometimes less dangerous than low doses, since they tend to kill thyroidtissues that would otherwise become cancerous as a result of the radiation. For example, children treated with moderate dose of I-131 for thyroid adenomas had a detectable increase in thyroid cancer, but children treated with a much higher dose did not.[citation needed] Likewise, most studies of very-high-dose I-131 for treatment of Graves disease have failed to find any increase in thyroid cancer, even though there is linear increase in thyroid cancer risk with I-131 absorption at moderate doses.[2] Thus, iodine-131 is increasingly less employed in small doses in medical use (especially in children), but increasingly is used only in large and maximal treatment doses, as a way of killing targeted tissues. This is known as "therapeutic use."

Iodine-131 can be "seen" by nuclear medicine imaging techniques (i.e., gamma cameras) whenever it is given for therapeutic use, since about 10% of its energy and radiation dose is via gamma radiation. However, since the other 90% of radiation (beta radiation) causes tissue damage without contributing to any ability to see or "image" the isotope, other less-damaging radioisotopes of iodine such as iodine-123 (see isotopes of iodine) are preferred in situations when only nuclear imaging is required. The isotope I-131 is still occasionally used for purely diagnostic (i.e., imaging) work, due to its low expense compared to other iodine radioisotopes. Very small medical imaging doses of I-131 have not shown any increase in thyroid cancer. The low-cost availability of I-131, in turn, is due to the relative ease of creating I-131 by neutron bombardment of natural tellurium in a nuclear reactor, then separating I-131 out by various simple methods (i.e., heating to drive off the volatile iodine). By contrast, other iodine radioisotopes are usually created by far more expensive techniques, starting with reactor radiation of expensive capsules of pressurized xenon gas.

Iodine-131 **Complete table of nuclides**	
General	
Name, symbol	**Radioiodine, 131I**
Neutrons	**83**
Protons	**53**
Nuclide data	
Half-life	**8.0197 days**
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Excess energy	**971 keV**

Iodine-131 is also one of the most commonly used gamma-emitting radioactive industrial tracer. Radioactive tracer isotopes are injected with hydraulic fracturing fluid to determine the injection profile and location of fractures created by hydraulic fracturing.[3]

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Production

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The primary emissions of 131I decay are thus electrons with a maximal energy of 606 keV (89% abundance, others 248–807 keV) and 364 keV gamma rays (81% abundance, others 723 keV).[12] Beta decay also produces an antineutrino, which carries off variable amounts of the beta decay energy. The electrons, due to their high mean energy [2][13]

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Iodine in food is absorbed by the body and preferentially concentrated in the thyroid where it is needed for the functioning of that gland. When 131I is present in high levels in the environment from radioactive fallout, it can be absorbed through contaminated food, and will also accumulate in the thyroid. As it decays, it may cause damage to the thyroid. The primary risk from exposure to high levels of 131I is the chance occurrence of radiogenic thyroid cancer in later life. Other risks include the possibility of non-cancerous growths and thyroiditis.[2]

The risk of thyroid cancer in later life appears to diminish with increasing age at time of exposure. Most risk estimates are based on studies in which radiation exposures occurred in children or teenagers. When adults are exposed, it has been difficult for epidemiologists to detect a statistically significant difference in the rates of thyroid disease above that of a similar but otherwise-unexposed group.[2][15]

The risk can be mitigated by taking iodine supplements, raising the total amount of iodine in the body and, therefore, reducing uptake and retention in the face and chest and lowering the relative proportion of radioactive iodine. However, such supplements were not distributed to the population living nearest to the Chernobyl nuclear power plant after the disaster,[16] though they were widely distributed to children in Poland.

Within the USA, the highest 131I fallout doses occurred during the 1950s and early 1960s to children having consumed fresh sources of milk contaminated as the result of above-ground testing of nuclear weapons.[4] The National Cancer Institute provides additional information on the health effects from exposure to 131I in fallout,[17] as well as individualized estimates, for those born before 1971, for each of the 3070 counties in the USA. The calculations are taken from data collected regarding fallout from the nuclear weapons tests conducted at the Nevada Test Site.[18]

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Medical use

It is used in nuclear medicine therapeutically and can also be seen with diagnostic scanners if it has been used therapeutically. Use of the 131I as iodide salt exploits the mechanism of absorption of iodine by the normal cells of the thyroid gland. Examples of its use in radiation therapy are those where tissue destruction is desired after iodine uptake by the tissue.

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Because of the carcinogenicity of its beta radiation in the thyroid in small doses, I-131 is rarely used primarily or solely for diagnosis (although in the past this was more common due to this isotope's relative ease of production and low expense). Instead the more purely gamma-emitting radioiodine iodine-123 is used in diagnostic testing (nuclear medicine scan of the thyroid). The longer half-lived iodine-125 is also occasionally used when a longer half-life radioiodine is needed for diagnosis, and in brachytherapy treatment (isotope confined in small seed-like metal capsules), where the low-energy gamma radiation without a beta component makes iodine-125 useful. The other radioisotopes of iodine are never used in brachytherapy.

The use of 131I as a medical isotope has been blamed for a routine shipment of biosolids being rejected from crossing the Canada—U.S. border.[37]Such material can enter the sewers directly from the medical facilities, or by being excreted by patients after a treatment.

References

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3. Jump up^ Reis, John C. (1976). Environmental Control in Petroleum Engineering. Gulf Professional Publishers.
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6. Jump up^ Guiraud-Vitaux, F.; Elbast, M.; Colas-Linhart, N.; Hindie, E. (February 2008). "Thyroid cancer after Chernobyl: is iodine 131 the only culprit ? Impact on clinical practice". Bulletin du cancer. 95 (2): 191–5. doi:10.1684/bdc.2008.0574. PMID 18304904.
7. Jump up^ Centre for Disease Control (2002). The Hanford Thyroid Disease Study (PDF). Retrieved 17 June 2012. no associations between Hanford's iodine-131 releases and thyroid disease were observed. [The findings] show that if there is an increased risk of thyroid disease from exposure to Hanford's iodine-131, it is probably too small to observe using the best epidemiologic methods available Executive summary
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9. Jump up^ "I-131 Fact Sheet" (PDF). Nordion. August 2011. Retrieved 2010-10-26.[permanent dead link]
10. Jump up^ "Nuclear Data for Safeguards, Table C-3, Cumulative Fission Yields". International Atomic Energy Agency. Retrieved 14 March 2011. (thermal neutron fission)
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12. Jump up^ "Nuclide Safety Data Sheet" (PDF). Retrieved 2010-10-26.
13. Jump up^ Skugor, Mario (2006). Thyroid Disorders. A Cleveland Clinic Guide. Cleveland Clinic Press. p. 82. ISBN 978-1-59624-021-6.
14. Jump up^ Council, National Research (11 February 2003). "Exposure of the American Population to Radioactive Fallout from Nuclear Weapons Tests: A Review of the CDC-NCI Draft Report on a Feasibility Study of the Health Consequences to the American Population from Nuclear Weapons Tests Conducted by the United States and Other Nations". nap.edu. doi:10.17226/10621. Retrieved 3 April 2018.
15. Jump up^ Robbins, Jacob; Schneider, Arthur B. (2000). "Thyroid cancer following exposure to radioactive iodine". Reviews in Endocrine and Metabolic Disorders. 1 (3): 197–203. doi:10.1023/A:1010031115233. ISSN 1389-9155. PMID 11705004.
16. Jump up^ Frot, Jacques. "THE CAUSES OF THE CHERNOBYL EVENT". Ecolo.org. Retrieved 2012-06-17.
17. Jump up^ "Radioactive I-131 from Fallout". National Cancer Institute. Retrieved 2007-11-14.
18. Jump up^ "Individual Dose and Risk Calculator for Nevada Test Site fallout". National Cancer Institute. 10/01/07. Archived from the original on 18 October 2007. Retrieved 2007-11-14.Check date values in: |date= (help)
19. Jump up^ "Low Concentrations Of Radiation Found In Mass. | WCVB Home - WCVB Home". Thebostonchannel.com. 27 March 2011. Archived from the original on 3 April 2012. Retrieved 17 June 2012.
20. Jump up^ "Traces of radioactive iodine found in Washington state milk" Los Angeles Times[dead link]
21. Jump up^ Sepe, S. M.; Clark, R. A. (March 1985). "Oxidant membrane injury by the neutrophil myeloperoxidase system. I. Characterization of a liposome model and injury by myeloperoxidase, hydrogen peroxide, and halides". Journal of Immunology. 134 (3): 1888–1895. ISSN 0022-1767. PMID 2981925.
22. Jump up^ Kowalsky RJ, Falen, SW. Radiopharmaceuticals in Nuclear Pharmacy and Nuclear Medicine. 2nd ed. Washington DC: American Pharmacists Association; 2004.
23. Jump up^ (PDF) https://www.eanm.org/scientific_info/guidelines/gl_paed_mibg.pdf?PHPSESSID%3D46d05b62d235c36a12166bf939b656c7. Retrieved 7 October 2011.Missing or empty |title= (help)[dead link]

24. Jump up^ Guidelines for Iodine Prophylaxis following Nuclear Accidents (PDF), Geneva: World Health Organization, 1999
25. Jump up^ "Information on Radiation, Health and the Thyroid, Including Iodine Testing, Potassium Iodide, and Thyrouūid Testing". Thyroid-info.com. Retrieved 2012-06-17.
26. Jump up^ "Potable Aqua Questions and Answers". www.pharmacalway.com. Retrieved 3 April2018.
27. Jump up^ LeMar, H. J.; et al. "Thyroid adaptation to chronic tetraglycine hydroperiodide water purification tablet use. Department of Medicine, Madigan Army Medical Center, Tacoma, Washington 98431". Journal of Clinical Endocrinology & Metabolism, Vol 80, 220-223, doi: 10.1210/jc.80.1.220 Copyright © 1995. Endocrine Society. Retrieved 20 Mar 2010.
28. ^ Jump up to:a b c d e f g Greer, Monte A.; Goodman, Gay; Pleus, Richard C.; Greer, Susan E. (2002). "Health Effects Assessment for Environmental Perchlorate Contamination: The Dose Response for Inhibition of Thyroidal Radioiodine Uptake in Humans". Environmental Health Perspectives. 110 (9): 927–37. doi:10.1289/ehp.02110927. PMC 1240994 [Freely accessible] . PMID 12204829.
29. ^ Jump up to:a b Wolff, J. (1998). "Perchlorate and the thyroid gland". Pharmacological Reviews. 50 (1): 89–105. PMID 9549759.
30. Jump up^ Barzilai, D.; Sheinfeld, M. (1966). "Fatal complications following use of potassium perchlorate in thyrotoxicosis. Report of two cases and a review of the literature". Israel journal of medical sciences. 2 (4): 453–6. PMID 4290684.

Mahalakshmi Lakshmi

Feedback

Peer Reviewer: Mahalakshmi Lakshmi
Apr 17, 2018

Review

Overall Feedback Criterion: 2 of 4

Iodine-131 (131I) is an important radioisotope of iodine discovered by Glenn Seaborg and John Livingood in 1938 at the University of California, Berkeley.[1] It has a radioactive decay half-life of about eight days. It is associated with nuclear energy, medical diagnostic and treatment procedures, and natural gas production.

Iodine-131

Mahalakshmi Lakshmi

Apr 17, 2018 at 1:17 PM

Version 2

Iodine-131

Iodine-131 (131I) is an important radioisotope of iodine discovered by Glenn Seaborg and John Livingood in 1938 at the University of California, Berkeley.[1] It has a radioactive decay half-life of about eight days. It is associated with nuclear energy, medical diagnostic and treatment procedures, and natural gas production. It also plays a major role as a radioactive isotope present in nuclear fission products, and was a significant contributor to the health hazards from open-air atomic bomb testing in the 1950s, and from the Chernobyl disaster, as well as being a large fraction of the contamination hazard in the first weeks in the Fukushima nuclear crisis. This is because I-131 is a major fission product of uranium and plutonium, comprising nearly 3% of the total products of fission (by weight). See fission product yield for a comparison with other radioactive fission products. I-131 is also a major fission product of uranium-233, produced from thorium.

Due to its mode of beta decay, iodine-131 is notable for causing mutation and death in cells that it penetrates, and other cells up to several millimeters away. For this reason, high doses of the isotope are sometimes less dangerous than low doses, since they tend to kill thyroidtissues that would otherwise become cancerous as a result of the radiation. For example, children treated with moderate dose of I-131 for thyroid adenomas had a detectable increase in thyroid cancer, but children treated with a much higher dose did not.[citation needed] Likewise, most studies of very-high-dose I-131 for treatment of Graves disease have failed to find any increase in thyroid cancer, even though there is linear increase in thyroid cancer risk with I-131 absorption at moderate doses.[2] Thus, iodine-131 is increasingly less employed in small doses in medical use (especially in children), but increasingly is used only in large and maximal treatment doses, as a way of killing targeted tissues. This is known as "therapeutic use."

Iodine-131 can be "seen" by nuclear medicine imaging techniques (i.e., gamma cameras) whenever it is given for therapeutic use, since about 10% of its energy and radiation dose is via gamma radiation. However, since the other 90% of radiation (beta radiation) causes tissue damage without contributing to any ability to see or "image" the isotope, other less-damaging radioisotopes of iodine such as iodine-123 (see isotopes of iodine) are preferred in situations when only nuclear imaging is required. The isotope I-131 is still occasionally used for purely diagnostic (i.e., imaging) work, due to its low expense compared to other iodine radioisotopes. Very small medical imaging doses of I-131 have not shown any increase in thyroid cancer. The low-cost availability of I-131, in turn, is due to the relative ease of creating I-131 by neutron bombardment of natural tellurium in a nuclear reactor, then separating I-131 out by various simple methods (i.e., heating to drive off the volatile iodine). By contrast, other iodine radioisotopes are usually created by far more expensive techniques, starting with reactor radiation of expensive capsules of pressurized xenon gas.

Iodine-131 **Complete table of nuclides**	
General	
Name, symbol	**Radioiodine, 131I**
Neutrons	**83**
Protons	**53**
Nuclide data	
Half-life	**8.0197 days**
Isotope mass	**130.9061246(12) u**
Excess energy	**971 keV**

Iodine-131 is also one of the most commonly used gamma-emitting radioactive industrial tracer. Radioactive tracer isotopes are injected with hydraulic fracturing fluid to determine the injection profile and location of fractures created by hydraulic fracturing.[3]

Much smaller incidental doses of iodine-131 than those used in medical therapeutic procedures, are supposed by some studies to be the major cause of increased thyroid cancersafter accidental nuclear contamination. These studies suppose

that cancers happen from residual tissue radiation damage caused by the I-131, and should appear mostly years after exposure, long after the I-131 has decayed.[4][5] Other studies can't find a correlation.[6][7]

Production

Most I-131 production is from nuclear reactor neutron-irradiation of a natural tellurium target. Irradiation of natural tellurium produces almost entirely I-131 as the only radionuclide with a half-life longer than hours, since most lighter isotopes of tellurium become heavier stable isotopes, or else stable iodine or xenon. However, the heaviest naturally occurring tellurium nuclide, Te-130 (34% of natural Te) absorbs a neutron to become tellurium-131, which beta-decays with a half-life of 25 minutes, to I-131.

A tellurium compound can be irradiated while bound as an oxide to an ion exchange column, and evolved I-131 then eluted into an alkaline solution.[8] More commonly, powdered elemental tellurium is irradiated and then I-131 separated from it by dry distillation of the iodine, which has a far higher vapor pressure. The element is then dissolved in a mildly alkaline solution in the standard manner, to produce I-131 as iodide and hypoiodate (which is soon reduced to iodide).[9]

131I is a fission product with a yield of 2.878% from uranium-235,[10] and can be released in nuclear weapons tests and nuclear accidents. However, the short half-life means it is not present in significant quantities in cooled spent nuclear fuel, unlike iodine-129 whose half-life is nearly a billion times that of I-131.

It is discharged to the atmosphere in small quantities by some nuclear power plants.[11]

Radioactive decay

I-131 decays with a half-life of 8.02 days with beta minus and gamma emissions. This nuclide of iodine has 78 neutrons in its nucleus, while the only stable nuclide, 127I, has 74. On decaying, 131I most often (89% of the time) expends its 971 keV of decay energy by transforming into the stable 131Xe (Xenon) in two steps, with gamma decay following rapidly after beta decay:

The primary emissions of 131I decay are thus electrons with a maximal energy of 606 keV (89% abundance, others 248–807 keV) and 364 keV gamma rays (81% abundance, others 723 keV).[12] Beta decay also produces an antineutrino, which carries off variable amounts of the beta decay energy. The electrons, due to their high mean energy [3][13]

Effects of exposure

Iodine in food is absorbed by the body and preferentially concentrated in the thyroid where it is needed for the functioning of that gland. When 131I is present in high levels in the environment from radioactive fallout, it can be absorbed through contaminated food, and will also accumulate in the thyroid. As it decays, it may cause damage to the thyroid. The primary risk from exposure to high levels of 131I is the chance occurrence of radiogenic thyroid cancer in later life. Other risks include the possibility of non-cancerous growths and thyroiditis.[2]

The risk of thyroid cancer in later life appears to diminish with increasing age at time of exposure. Most risk estimates are based on studies in which radiation exposures occurred in children or teenagers. When adults are exposed, it has been difficult for epidemiologists to detect a statistically significant difference in the rates of thyroid disease above that of a similar but otherwise-unexposed group.[2][15]

The risk can be mitigated by taking iodine supplements, raising the total amount of iodine in the body and, therefore, reducing uptake and retention in the face and chest and lowering the relative proportion of radioactive iodine. However, such supplements were not distributed to the population living nearest to the Chernobyl nuclear power plant after the disaster,[16] though they were widely distributed to children in Poland.

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It is used in nuclear medicine therapeutically and can also be seen with diagnostic scanners if it has been used therapeutically. Use of the 131I as iodide salt exploits the mechanism of absorption of iodine by the normal cells of the thyroid gland. Examples of its use in radiation therapy are those where tissue destruction is desired after iodine uptake by the tissue.

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Mahalakshmi Lakshmi

Feedback

Peer Reviewer: Jenita Msu
Apr 17, 2018

Review

Test_1: 0 of 4

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Annotations

A1. Comment: *Iodine-131 is also one of the most commonly used gamma-emitting radioactive industrial tracer.*

Iodine-131

Mahalakshmi Lakshmi

Apr 17, 2018 at 12:03 PM

Version 1

Iodine-131

Iodine-131 (131I) is an important radioisotope of iodine discovered by Glenn Seaborg and John Livingood in 1938 at the University of California, Berkeley.[1] It has a radioactive decay half-life of about eight days. It is associated with nuclear energy, medical diagnostic and treatment procedures, and natural gas production. It also plays a major role as a radioactive isotope present in nuclear fission products, and was a significant contributor to the health hazards from open-air atomic bomb testing in the 1950s, and from the Chernobyl disaster, as well as being a large fraction of the contamination hazard in the first weeks in the Fukushima nuclear crisis. This is because I-131 is a major fission product of uranium and plutonium, comprising nearly 3% of the total products of fission (by weight). See fission product yield for a comparison with other radioactive fission products. I-131 is also a major fission product of uranium-233, produced from thorium.

Due to its mode of beta decay, iodine-131 is notable for causing mutation and death in cells that it penetrates, and other cells up to several millimeters away. For this reason, high doses of the isotope are sometimes less dangerous than low doses, since they tend to kill thyroidtissues that would otherwise become cancerous as a result of the radiation. For example, children treated with moderate dose of I-131 for thyroid adenomas had a detectable increase in thyroid cancer, but children treated with a much higher dose did not.[citation needed] Likewise, most studies of very-high-dose I-131 for treatment of Graves disease have failed to find any increase in thyroid cancer, even though there is linear increase in thyroid cancer risk with I-131 absorption at moderate doses.[2] Thus, iodine-131 is increasingly less employed in small doses in medical use (especially in children), but increasingly is used only in large and maximal treatment doses, as a way of killing targeted tissues. This is known as "therapeutic use."

Iodine-131 can be "seen" by nuclear medicine imaging techniques (i.e., gamma cameras) whenever it is given for therapeutic use, since about 10% of its energy and radiation dose is via gamma radiation. However, since the other 90% of radiation (beta radiation) causes tissue damage without contributing to any ability to see or "image" the isotope, other less-damaging radioisotopes of iodine such as iodine-123 (see isotopes of iodine) are preferred in situations when only nuclear imaging is required. The isotope I-131 is still occasionally used for purely diagnostic (i.e., imaging) work, due to its low expense compared to other iodine radioisotopes. Very small medical imaging doses of I-131 have not shown any increase in thyroid cancer. The low-cost availability of I-131, in turn, is due to the relative ease of creating I-131 by neutron bombardment of natural tellurium in a nuclear reactor, then separating I-131 out by various simple methods (i.e., heating to drive off the volatile iodine). By contrast, other iodine radioisotopes are usually created by far more expensive techniques, starting with reactor radiation of expensive capsules of pressurized xenon gas.

Iodine-131 **Complete table of nuclides**
General

Iodine-131 Complete table of nuclides	
Name, symbol	**Radioiodine, 131I**
Neutrons	**83**
Protons	**53**
Nuclide data	
Half-life	**8.0197 days**
Isotope mass	**130.9061246(12) u**
Excess energy	**971 keV**

Iodine-131 is also one of the most commonly used gamma-emitting radioactive industrial tracer. Radioactive tracer isotopes are injected with hydraulic fracturing fluid to determine the injection profile and location of fractures created by hydraulic fracturing.[3]

Much smaller incidental doses of iodine-131 than those used in medical therapeutic procedures, are supposed by some studies to be the major cause of increased thyroid cancersafter accidental nuclear contamination. These studies suppose that cancers happen from residual tissue radiation damage caused by the I-131, and should appear mostly years after exposure, long after the I-131 has decayed.[4][5] Other studies can't find a correlation.[6][7]

Production

Most I-131 production is from nuclear reactor neutron-irradiation of a natural tellurium target. Irradiation of natural tellurium produces almost entirely I-131 as the only radionuclide with a half-life longer than hours, since most lighter isotopes of tellurium become heavier stable isotopes, or else stable iodine or xenon. However, the heaviest naturally occurring tellurium nuclide, Te-130 (34% of natural Te) absorbs a neutron to become tellurium-131, which beta-decays with a half-life of 25 minutes, to I-131.

A tellurium compound can be irradiated while bound as an oxide to an ion exchange column, and evolved I-131 then eluted into an alkaline solution.[8] More commonly, powdered elemental tellurium is irradiated and then I-131 separated from it by dry distillation of the iodine, which has a far higher vapor pressure. The element is then dissolved in a mildly alkaline solution in the standard manner, to produce I-131 as iodide and hypoiodate (which is soon reduced to iodide).[9]

131I is a fission product with a yield of 2.878% from uranium-235,[10] and can be released in nuclear weapons tests and nuclear accidents. However, the short half-life means it is not present in significant quantities in cooled spent nuclear fuel, unlike iodine-129 whose half-life is nearly a billion times that of I-131.

It is discharged to the atmosphere in small quantities by some nuclear power plants.[11]

Radioactive decay

I-131 decays with a half-life of 8.02 days with beta minus and gamma emissions. This nuclide of iodine has 78 neutrons in its nucleus, while the only stable nuclide, 127I, has 74. On decaying, 131I most often (89% of the time) expends its 971 keV of decay energy by transforming into the stable 131Xe (Xenon) in two steps, with gamma decay following rapidly after beta decay:

The primary emissions of 131I decay are thus electrons with a maximal energy of 606 keV (89% abundance, others

248–807 keV) and 364 keV gamma rays (81% abundance, others 723 keV).[12] Beta decay also produces an antineutrino, which carries off variable amounts of the beta decay energy. The electrons, due to their high mean energy [4][13]

Effects of exposure

Iodine in food is absorbed by the body and preferentially concentrated in the thyroid where it is needed for the functioning of that gland. When 131I is present in high levels in the environment from radioactive fallout, it can be absorbed through contaminated food, and will also accumulate in the thyroid. As it decays, it may cause damage to the thyroid. The primary risk from exposure to high levels of 131I is the chance occurrence of radiogenic thyroid cancer in later life. Other risks include the possibility of non-cancerous growths and thyroiditis.[2]

The risk of thyroid cancer in later life appears to diminish with increasing age at time of exposure. Most risk estimates are based on studies in which radiation exposures occurred in children or teenagers. When adults are exposed, it has been difficult for epidemiologists to detect a statistically significant difference in the rates of thyroid disease above that of a similar but otherwise-unexposed group.[2][15]

The risk can be mitigated by taking iodine supplements, raising the total amount of iodine in the body and, therefore, reducing uptake and retention in the face and chest and lowering the relative proportion of radioactive iodine. However, such supplements were not distributed to the population living nearest to the Chernobyl nuclear power plant after the disaster,[16] though they were widely distributed to children in Poland.

Within the USA, the highest 131I fallout doses occurred during the 1950s and early 1960s to children having consumed fresh sources of milk contaminated as the result of above-ground testing of nuclear weapons.[4] The National Cancer Institute provides additional information on the health effects from exposure to 131I in fallout,[17] as well as individualized estimates, for those born before 1971, for each of the 3070 counties in the USA. The calculations are taken from data collected regarding fallout from the nuclear weapons tests conducted at the Nevada Test Site.[18]

On 27 March 2011, the Massachusetts Department of Public Health reported that 131I was detected in very low concentrations in rainwater from samples collected in Massachusetts, USA, and that this likely originated from the Fukushima power plant.[19] Farmers near the plant dumped raw milk, while testing in the United States found 0.8 pico-curies per liter of iodine-131 in a milk sample, but the radiation levels were 5,000 times lower than the FDA's "defined intervention level." The levels were expected to drop relatively quickly[20]

Medical use

It is used in nuclear medicine therapeutically and can also be seen with diagnostic scanners if it has been used therapeutically. Use of the 131I as iodide salt exploits the mechanism of absorption of iodine by the normal cells of the thyroid gland. Examples of its use in radiation therapy are those where tissue destruction is desired after iodine uptake by the tissue.

Major uses of 131I include the treatment of thyrotoxicosis (hyperthyroidism) and some types of thyroid cancer that absorb iodine. The 131I is thus used as direct radioisotope therapy to treat hyperthyroidism due to Graves' disease, and sometimes hyperactive thyroid nodules (abnormally active thyroid tissue that is not malignant). The therapeutic use of radioiodine to treat hyperthyroidism from Graves' disease was first reported by Saul Hertz in 1941.

The 131I isotope is also used as a radioactive label for certain radiopharmaceuticals that can be used for therapy, e.g. 131I-metaiodobenzylguanidine(131I-MIBG) for imaging and treating pheochromocytoma and neuroblastoma. In all of these therapeutic uses, 131I destroys tissue by short-range beta radiation. About 90% of its radiation damage to tissue is via beta radiation, and the rest occurs via its gamma radiation (at a longer distance from the radioisotope). It can be seen in diagnostic scans after its use as therapy, because 131I is also a gamma-emitter. (¼M=)

Because of the carcinogenicity of its beta radiation in the thyroid in small doses, I-131 is rarely used primarily or solely for diagnosis (although in the past this was more common due to this isotope's relative ease of production and low expense). Instead the more purely gamma-emitting radioiodine iodine-123 is used in diagnostic testing (nuclear medicine scan of the thyroid). The longer half-lived iodine-125 is also occasionally used when a longer half-life radioiodine is needed for diagnosis, and in brachytherapy treatment (isotope confined in small seed-like metal capsules), where the low-energy gamma radiation without a beta component makes iodine-125 useful. The other radioisotopes of iodine are never used in brachytherapy.

The use of 131I as a medical isotope has been blamed for a routine shipment of biosolids being rejected from crossing the Canada—U.S. border.[37]Such material can enter the sewers directly from the medical facilities, or by being excreted by patients after a treatment.

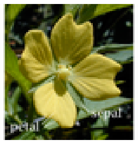
References

1. "UW-L Brachy Course". wikifoundry. April 2008. Retrieved 2014-04-11.
2. ^ Jump up to:a b c Rivkees, Scott A.; Sklar, Charles; Freemark, Michael (1998). "The Management of Graves' Disease in Children, with Special Emphasis on Radioiodine Treatment". Journal of Clinical Endocrinology & Metabolism. 83 (11): 3767–76. doi:10.1210/jc.83.11.3767. PMID 9814445.
3. Jump up^ Reis, John C. (1976). Environmental Control in Petroleum Engineering. Gulf Professional Publishers.
4. ^ Jump up to:a b Simon, Steven L.; Bouville, André; Land, Charles E. (January–February 2006). "Fallout from Nuclear Weapons Tests and Cancer Risks". American Scientist. 94: 48–57. doi:10.1511/2006.1.48. In 1997, NCI conducted a detailed evaluation of dose to the thyroid glands of U.S. residents from I-131 in fallout from tests in Nevada. (...) we evaluated the risks of thyroid cancer from that exposure and estimated that about 49,000 fallout-related cases might occur in the United States, almost all of them among persons who were under age 20 at some time during the period 1951–57, with 95-percent uncertainty limits of 11,300 and 212,000.
5. Jump up^ "National Cancer Institute calculator for thyroid cancer risk as a result of I-131 intake after nuclear testing before 1971 in Nevada". Ntsi131.nci.nih.gov. Retrieved 2012-06-17.
6. Jump up^ Guiraud-Vitaux, F.; Elbast, M.; Colas-Linhart, N.; Hindie, E. (February 2008). "Thyroid cancer after Chernobyl: is iodine 131 the only culprit ? Impact on clinical practice". Bulletin du cancer. 95 (2): 191–5. doi:10.1684/bdc.2008.0574. PMID 18304904.
7. Jump up^ Centre for Disease Control (2002). The Hanford Thyroid Disease Study (PDF). Retrieved 17 June 2012. no associations between Hanford's iodine-131 releases and thyroid disease were observed. [The findings] show that if there is an increased risk of thyroid disease from exposure to Hanford's iodine-131, it is probably too small to observe using the best epidemiologic methods available Executive summary
8. Jump up^ Chattopadhyay, Sankha; Saha Das, Sujata (2010). "Recovery of 131I from alkaline solution of n-irradiated tellurium target using a tiny Dowex-1 column". Applied Radiation and Isotopes. 68 (10): 1967–9. doi:10.1016/j.apradiso.2010.04.033. PMID 20471848.
9. Jump up^ "I-131 Fact Sheet" (PDF). Nordion. August 2011. Retrieved 2010-10-26.[permanent dead link]
10. Jump up^ "Nuclear Data for Safeguards, Table C-3, Cumulative Fission Yields". International Atomic Energy Agency. Retrieved 14 March 2011. (thermal neutron fission)
11. Jump up^ Effluent Releases from Nuclear Power Plants and Fuel-Cycle Facilities. National Academies Press (US). 2012-03-29.
12. Jump up^ "Nuclide Safety Data Sheet" (PDF). Retrieved 2010-10-26.
13. Jump up^ Skugor, Mario (2006). Thyroid Disorders. A Cleveland Clinic Guide. Cleveland Clinic Press. p. 82. ISBN 978-1-59624-021-6.
14. Jump up^ Council, National Research (11 February 2003). "Exposure of the American Population to Radioactive Fallout from Nuclear Weapons Tests: A Review of the CDC-NCI Draft Report on a Feasibility Study of the Health Consequences to the American Population from Nuclear Weapons Tests Conducted by the United States and Other Nations". nap.edu. doi:10.17226/10621. Retrieved 3 April 2018.
15. Jump up^ Robbins, Jacob; Schneider, Arthur B. (2000). "Thyroid cancer following exposure to radioactive iodine". Reviews in Endocrine and Metabolic Disorders. 1 (3): 197–203. doi:10.1023/A:1010031115233. ISSN 1389-9155.

PMID 11705004.

16. Jump up^ Frot, Jacques. "THE CAUSES OF THE CHERNOBYL EVENT". Ecolo.org. Retrieved 2012-06-17.
17. Jump up^ "Radioactive I-131 from Fallout". National Cancer Institute. Retrieved 2007-11-14.
18. Jump up^ "Individual Dose and Risk Calculator for Nevada Test Site fallout". National Cancer Institute. 10/01/07. Archived from the original on 18 October 2007. Retrieved 2007-11-14.Check date values in: |date= (help)
19. Jump up^ "Low Concentrations Of Radiation Found In Mass. | WCVB Home - WCVB Home". Thebostonchannel.com. 27 March 2011. Archived from the original on 3 April 2012. Retrieved 17 June 2012.
20. Jump up^ "Traces of radioactive iodine found in Washington state milk" Los Angeles Times[dead link]
21. Jump up^ Sepe, S. M.; Clark, R. A. (March 1985). "Oxidant membrane injury by the neutrophil myeloperoxidase system. I. Characterization of a liposome model and injury by myeloperoxidase, hydrogen peroxide, and halides". Journal of Immunology. 134 (3): 1888–1895. ISSN 0022-1767. PMID 2981925.
22. Jump up^ Kowalsky RJ, Falen, SW. Radiopharmaceuticals in Nuclear Pharmacy and Nuclear Medicine. 2nd ed. Washington DC: American Pharmacists Association; 2004.
23. Jump up^ (PDF) https://www.eanm.org/scientific_info/guidelines/gl_paed_mibg.pdf?PHPSESSID%3D46d05b62d235c36a12166bf939b656c7. Retrieved 7 October 2011.Missing or empty |title= (help)[dead link]
24. Jump up^ Guidelines for Iodine Prophylaxis following Nuclear Accidents (PDF), Geneva: World Health Organization, 1999
25. Jump up^ "Information on Radiation, Health and the Thyroid, Including Iodine Testing, Potassium Iodide, and Thyrouūid Testing". Thyroid-info.com. Retrieved 2012-06-17.
26. Jump up^ "Potable Aqua Questions and Answers". www.pharmacalway.com. Retrieved 3 April2018.
27. Jump up^ LeMar, H. J.; et al. "Thyroid adaptation to chronic tetraglycine hydroperiodide water purification tablet use. Department of Medicine, Madigan Army Medical Center, Tacoma, Washington 98431". Journal of Clinical Endocrinology & Metabolism, Vol 80, 220-223, doi: 10.1210/jc.80.1.220 Copyright © 1995. Endocrine Society. Retrieved 20 Mar 2010.
28. ^ Jump up to:a b c d e f g Greer, Monte A.; Goodman, Gay; Pleus, Richard C.; Greer, Susan E. (2002). "Health Effects Assessment for Environmental Perchlorate Contamination: The Dose Response for Inhibition of Thyroidal Radioiodine Uptake in Humans". Environmental Health Perspectives. 110 (9): 927–37. doi:10.1289/ehp.02110927. PMC 1240994 [Freely accessible] . PMID 12204829.
29. ^ Jump up to:a b Wolff, J. (1998). "Perchlorate and the thyroid gland". Pharmacological Reviews. 50 (1): 89–105. PMID 9549759.
30. Jump up^ Barzilai, D.; Sheinfeld, M. (1966). "Fatal complications following use of potassium perchlorate in thyrotoxicosis. Report of two cases and a review of the literature". Israel journal of medical sciences. 2 (4): 453–6. PMID 4290684.

Mahalakshmi Lakshmi



16TH-Test

Bindu Msu

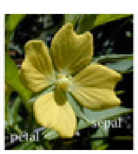
Apr 17, 2018 at 1:40 PM

Version 2

16TH-Test

The pulp papermaking process is said to have been developed in China during the early 2nd century CE, possibly as early as the year 105 CE,[1] by the Han court eunuch Cai Lun, although the earliest archaeological fragments of paper derive from the 2nd century BCE in China.[2] The modern pulp !#$%@®©¿ and paper industry is global, with China leading its production and the United States right behind it.The pulp papermaking process is said to have been developed in China during the early 2nd century CE, possibly as early as the year 105 CE,[1] by the Han court eunuch Cai Lun, although the earliest archaeological fragments of paper derive from the 2nd century BCE in China.[2] The modern pulp !#$%@®©¿ and paper industry is global, with China leading its production and the United States right behind it.The pulp papermaking process is said to have been developed in China during the early 2nd century CE, possibly as early as the year 105 CE,[1] by the Han court eunuch Cai Lun, although the earliest archaeological fragments of paper derive from the 2nd century BCE in China.[2] The modern pulp !#$%@®©¿ and paper industry is global, with China leading its production and the United States right behind it.The pulp papermaking process is said to have been developed in China during the early 2nd century CE, possibly as early as the year 105 CE,[1] by the Han court eunuch Cai Lun, although the earliest archaeological fragments of paper derive from the 2nd century BCE in China.[2] The modern pulp !#$%@®©¿ and paper industry is global, with China leading its production and the United States right behind it.The pulp papermaking process is said to have been developed in China during the early 2nd century CE, possibly as early as the year 105 CE,[1] by the Han court eunuch Cai Lun, although the earliest archaeological fragments of paper derive from the 2nd century BCE in China.[2] The modern pulp !#$%@®©¿ and paper industry is global, with China leading its production and the United States right behind it.

Bindu Msu



16TH-Test

Bindu Msu

Apr 17, 2018 at 1:35 PM

Version 1

16TH-Test

The pulp papermaking process is said to have been developed in China during the early 2nd century CE, possibly as early as the year 105 CE,[1] by the Han court eunuch Cai Lun, although the earliest archaeological fragments of paper derive

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Bindu Msu

Human digestive system

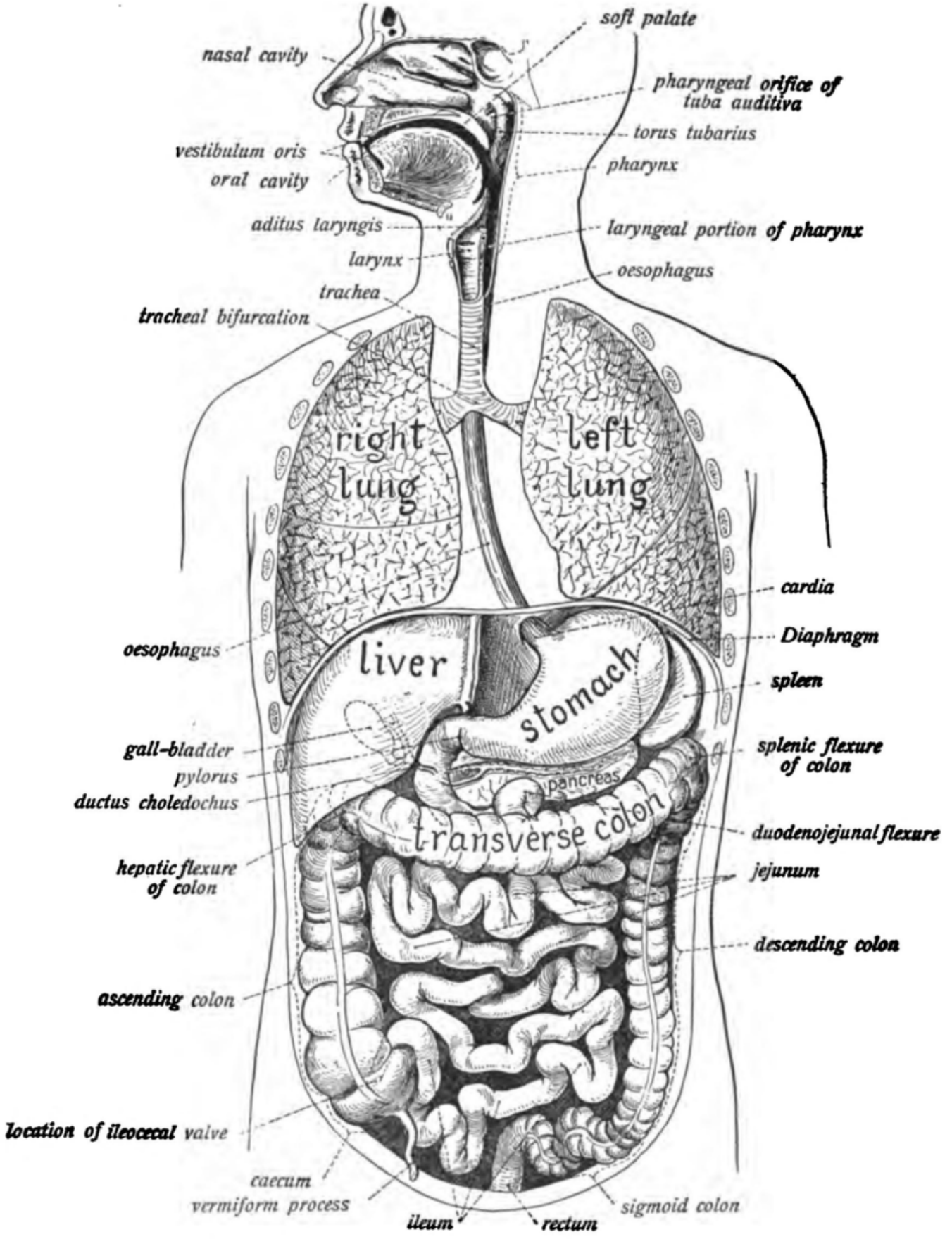
Shaikshavali Msu

Apr 17, 2018 at 1:41 PM

Version 2

Human digestive system

See also gastrointestinal tract.
"Digestive system" and "alimentary system" redirect here. For digestive systems of non-human animals, see Digestion.


soft palate
nasal cavity
pharyngeal orifice of tuba auditiva
torus tubarius
vestibulum oris
oral cavity
pharynx
aditus laryngis
laryngeal portion of pharynx
larynx
oesophagus
trachea
tracheal bifurcation
right lung
left lung
cardia
oesophagus
liver
Diaphragm
stomach
spleen
gall-bladder
splenic flexure of colon
pylorus
pancreas
ductus choledochus
transverse colon
duodenojejunal flexure
hepatic flexure of colon
jejunum
descending colon
ascending colon
location of ileocecal valve
caecum
vermiform process
sigmoid colon
ileum
rectum

The human digestive system consists of the gastrointestinal tract plus the accessory organs of digestion (the tongue, salivary glands, pancreas, liver, and gallbladder).[1] In this system, the process of digestion has many stages, the first of which starts in the mouth. Digestion involves the breakdown of food into smaller and smaller components, until they can be absorbed and assimilated into the body.

Chewing, in which food is mixed with saliva begins the process of digestion. This produces a bolus which can be swallowed down the esophagus and into the stomach. Here it is mixed with gastric juice until it passes into the duodenum where it is mixed with a number of enzymes produced by the pancreas. Saliva also contains a catalytic enzyme called amylase which starts to act on food in the mouth. Another digestive enzyme called lingual lipase is secreted by some of the lingual papillae on the tongue and also from serous glands in the main salivary glands. Digestion is helped by the mastication of food by the teeth and also by the muscular actions of peristalsis and segmentation contractions. Gastric juice in the stomach is essential for the continuation of digestion as is the production of mucus in the stomach.

Peristalsis is the rhythmic contraction of muscles that begins in the esophagus and continues along the wall of the stomach and the rest of the gastrointestinal tract. This initially results in the production of chyme which when fully broken down in the small intestine is absorbed as chyle into the lymphatic system. Most of the digestion of food takes place in the small intestine. Water and some minerals are reabsorbed back into the blood in the colon of the large intestine. The waste products of digestion (feces) are defecated from the anus via the rectum.

Components

There are several organs and other components involved in the digestion of food. The organs known as the accessory digestive glands are the liver, gall bladder and pancreas. Other components include the mouth, salivary glands, tongue, teeth and epiglottis.

The largest structure of the digestive system is the gastrointestinal tract (GI tract). This starts at the mouth and ends at the anus, covering a distance of about nine (9) metres.[2]

The largest part of the GI tract is the colon or large intestine. Water is absorbed here and the remaining waste matter is stored prior to defecation.[1]

Most of the digestion of food takes place in the small intestine.

A major digestive organ is the stomach. Within its mucosa are millions of embedded gastric glands. Their secretions are vital to the functioning of the organ.

There are many specialised cells of the GI tract. These include the various cells of the gastric glands, taste cells, pancreatic duct cells, enterocytes and microfold cells.

Some parts of the digestive system are also part of the excretory system, including the large intestine.[1]

Blood supply

The digestive system is supplied by the celiac artery. The celiac artery is the first major branch from the abdominal aorta, and is the only major artery that nourishes the digestive organs.

There are three main divisions – the left gastric artery, the common hepatic artery and the splenic artery.

The celiac artery supplies the liver, stomach, spleen and the upper 1/3 of the duodenum (to the sphincter of Oddi) and the pancreas with oxygenated blood. Most of the blood is returned to the liver via the portal venous system for further processing and detoxification before returning to the systemic circulation via the hepatic portal vein.

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The next branch from the abdominal aorta is the superior mesenteric artery, which supplies the regions of the digestive tract derived from the midgut, which includes the distal 2/3 of the duodenum, jejunum, ileum, cecum, appendix, ascending colon, and the proximal 2/3 of the transverse colon.

The final branch which is important for the digestive system is the inferior mesenteric artery, which supplies the regions of the digestive tract derived from the hindgut, which includes the distal 1/3 of the transverse colon, descending colon, sigmoid colon, rectum, and the anus above the pectinate line.

Nerve supply

The enteric nervous system consists of some one hundred million neurons[31] that are embedded in the peritoneum, the lining of the gastrointestinal tract extending from the esophagus to the anus.[32] These neurons are collected into two plexuses - the myenteric (or Auerbach's) plexus that lies between the longitudinal and the smooth muscle layers, and the submucosal (or Meissner's) plexusthat lies between the circular smooth muscle layer and the mucosa.[33][34][35]

Media embedded April 17, 2018
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Parasympathetic innervation to the ascending colon is supplied by the vagus nerve. Sympathetic innervation is supplied by the splanchnic nerves that join the celiac ganglia. Most of the digestive tract is innervated by the two large celiac ganglia, with the upper part of each ganglion joined by the greater splanchnic nerve and the lower parts joined by the lesser splanchnic nerve. It is from these ganglia that many of the gastric plexuses arise.

Development

Early in embryonic development, the embryo has three germ layers and abuts a yolk sac. During the second week of development, the embryo grows and begins to surround and envelop portions of this sac. The enveloped portions form the basis for the adult gastrointestinal tract. Sections of this foregut begin to differentiate into the organs of the gastrointestinal tract, such as the oesophagus, stomach, and intestines.[36]

During the fourth week of development, the stomach rotates. The stomach, originally lying in the midline of the embryo, rotates so that its body is on the left. This rotation also affects the part of the gastrointestinal tube immediately below the stomach, which will go on to become the duodenum. By the end of the fourth week, the developing duodenum begins to spout a small outpouching on its right side, the hepatic diverticulum, which will go on to become the biliary tree. Just below this is a second outpouching, known as the cystic diverticulum, that will eventually develop into the gallbladder.[36]

Clinical significance

Each part of the digestive system is subject to a wide range of disorders many of which can be congenital. Mouth diseases can also be caused by pathogenic bacteria, viruses, fungi and as a side effect of some medications. Mouth diseases include tongue diseases and salivary gland diseases. A common gum disease in the mouth is gingivitis which is caused by bacteria in plaque. The most common viral infection of the mouth is gingivostomatitis caused by herpes simplex. A common fungal infection is candidiasiscommonly known as thrush which affects the mucous membranes of the mouth.

There are a number of esophageal diseases such as the development of Schatzki rings that can restrict the passageway, causing difficulties in swallowing. They can also completely block the esophagus.[37]

Stomach diseases are often chronic conditions and include gastroparesis, gastritis, and peptic ulcers.

A number of problems including malnutrition and anemia can arise from malabsorption, the abnormal absorption of nutrients in the GI tract. Malabsorption can have many causes ranging from infection, to enzyme deficiencies such as exocrine pancreatic insufficiency. It can also arise as a result of other gastrointestinal diseases such as coeliac disease. Coeliac disease is an autoimmune disorder of the small intestine. This can cause vitamin deficiencies due to the improper absorption of nutrients in the small intestine. The small intestine can also be obstructed by a volvulus, a loop of intestine that becomes twisted enclosing its attached mesentery. This can cause mesenteric ischemia if severe enough.

A common disorder of the bowel is diverticulitis. Diverticula are small pouches that can form inside the bowel wall, which can become inflamed to give diverticulitis. This disease can have complications if an inflamed diverticulum bursts and infection sets in. Any infection can spread further to the lining of the abdomen (peritoneum) and cause potentially fatal peritonitis.[38]

Crohn's disease is a common chronic inflammatory bowel disease (IBD), which can affect any part of the GI tract,[39] but it mostly starts in the terminal ileum.

Ulcerative colitis an ulcerative form of colitis, is the other major inflammatory bowel disease which is restricted to the colon and rectum. Both of these IBDs can give an increased risk of the development of colorectal cancer. Ulcerative coliltis is the most common of the IBDs[40]

Irritable bowel syndrome (IBS) is the most common of the functional gastrointestinal disorders. These are idiopathic disorders that the Rome process has helped to define.[41]

Giardiasis is a disease of the small intestine caused by a protist parasite Giardia lamblia. This does not spread but remains confined to the lumen of the small intestine.[42] It can often be asymptomatic, but as often can be indicated by a variety of symptoms. Giardiasis is the most common pathogenic parasitic infection in humans.[43]

There are diagnostic tools mostly involving the ingestion of barium sulphate to investigate disorders of the GI tract.[44] These are known as upper gastrointestinal series that enable imaging of the pharynx, larynx, oesophagous, stomach and small intestine and lower gastrointestinal series for imaging of the colon.

See also

- Gastrointestinal physiology
- Gut–brain axis
- Neurogastroenterology

References

1. "Large intestine". Encyclopedia Britannica. 2016. Retrieved 1 October 2016.
2. Jump up^ Kong F, Singh RP (June 2008). "Disintegration of solid foods in human stomach". J. Food Sci. 73 (5): R67–80. doi:10.1111/j.1750-3841.2008.00766.x. PMID 18577009.
3. Jump up^ Maton, Anthea; Jean Hopkins; Charles William McLaughlin; Susan Johnson; Maryanna Quon Warner; David LaHart; Jill D. Wright (1993). Human Biology and Health. Englewood Cliffs, New Jersey, US: Prentice Hall. ISBN 0-13-981176-1.
4. Jump up^ Pocock, Gillian (2006). Human Physiology (Third ed.). Oxford University Press. p. 382. ISBN 978-0-19-856878-0.
5. ^ Jump up to:a b c Macpherson, G (1999). Black's Medical Dictionary. A & C. Black Ltd. ISBN 0713645660.
6. ^ Jump up to:a b c consultants Daniel Albert et al. (2012). Dorland's illustrated medical dictionary (32nd ed.). Philadelphia, PA: Saunders/Elsevier. ISBN 978-1-4160-6257-8.
7. Jump up^ Ten Cate's Oral Histology, Nanci, Elsevier, 2007, page 321
8. ^ Jump up to:a b Britannica Concise Encyclopedia. Encyclopedia Britannica, Inc. 2007. ISBN 1593392931.
9. ^ Jump up to:a b Ten Cate's Oral Histology, Nanci, Elsevier, 2013, page 275-276
10. Jump up^ Illustrated Anatomy of the Head and Neck, Fehrenbach and Herring, Elsevier, 2012, p. 157
11. Jump up^ Piludu, M; Lantini, MS; et al. (November 2006). "Salivary histatins in human deep posterior lingual glands

(of von Ebner)". Arch Biol. 51: 967–73. doi:10.1016/j.archoralbio.2006.05.011. PMID 16859632.
12. Jump up^ Maton, Anthea. Human Biology and Health. Prentice Hall 1993. ISBN 0-13-981176-1.
13. Jump up^ Edgar, WM (1992). "Saliva:its secretion, composition and functions". Br.Dent J. 172: 305–12. doi:10.1038/sj.bdj.4807861. PMID 1591115.
14. Jump up^ S Fagarasan; T Honjo (2003). "Intestinal IgA Synthesis: Regulation of Front-line Body Defenses". Nature Reviews Immunology. 3 (1): 63–72. doi:10.1038/nri982. PMID 12511876.
15. Jump up^ Pettit, John D.; Paul Moss (2006). Essential Haematology 5e (Essential). Blackwell Publishing Professional. p. 44. ISBN 1-4051-3649-9.
16. Jump up^ Bradbury, Jane (March 2004). "Taste Perception Cracking the code". PLOS Biology. 2 (3): E64. doi:10.1371/journal.pbio.0020064. PMC 368160 [Freely accessible] . PMID 15024416.
17. Jump up^ Jowett, Shrestha (1998). "Mucosa and taste buds of the human epiglottis". Journal of Anatomy. 193 (4): 617–618. doi:10.1046/j.1469-7580.1998.19340617.x. PMC 1467887 [Freely accessible] . PMID 10029195.
18. Jump up^ "pharyngeal plexus (anatomy)" [Paid subscription required] . GPnotebook.
19. Jump up^ Sherwood, Lauralee (1997). Human physiology: from cells to systems. Belmont, CA: Wadsworth Pub. Co. ISBN 0-314-09245-5. OCLC 35270048.
20. Jump up^ Dorland's Illustrated Medical Dictionary. Elsevier Saunders 2012. ISBN 978-1-4160-6257-8.
21. Jump up^ Guyton and Hall (2011). Textbook of Medical Physiology. U.S.: Saunders Elsevier. p. 784. ISBN 978-1-4160-4574-8.
22. Jump up^ Black's Medical Dictionary 39th Ed.1999
23. Jump up^ Drake, Richard L.; Vogl, Wayne; Tibbitts, Adam W.M. Mitchell; illustrations by Richard; Richardson, Paul (2005). Gray's anatomy for students. Philadelphia: Elsevier/Churchill Livingstone. p. 287. ISBN 978-0-8089-2306-0.
24. Jump up^ "Histology guide". Retrieved 22 May 2015.
25. Jump up^ Ahrens, Thomas; Prentice, Donna (1998). Critical care certification: preparation, review & practice exams. Norwalk, CT: Appleton & Lange. p. 265. ISBN 0-8385-1474-X.
26. Jump up^ Lower Gastrointestinal Tract at the US National Library of Medicine Medical Subject Headings (MeSH)
27. Jump up^ Cummings, JH; Macfarlane, GT (November 1997). "Role of intestinal bacteria in nutrient metabolism". JPEN. Journal of parenteral and enteral nutrition. 21 (6): 357–65. doi:10.1177/0148607197021006357. PMID 9406136.
28. Jump up^ O.D.E. 2nd. Edition 2005
29. Jump up^ Wood, Jackie D. (2009), "Gastrointestinal Physiology", in Rhoades, Rodney A.; Bell, David R., Medical Physiology: Principles for Clinical Medicine (3 ed.), Philadelphia, PA: Lippincott Williams & Wilkins, pp. 463–496
30. Jump up^ Essential Clinical Anatomy. K.L. Moore & A.M. Agur. Lippincott, 2 ed. 2002. Page 150

Shaikshavali Msu

Feedback

Peer Reviewer: Shaikshavali Msu
Apr 17, 2018

Review

Overall Feedback Criterion: 1 of 4

There are several organs and other components involved in the digestion of food. The organs known as the

accessory digestive glands are the liver, gall bladder and pancreas. Other components include the mouth, salivary glands, tongue, teeth and epiglottis.

Feedback

Peer Reviewer: Shaikshavali Msu
Apr 17, 2018

Review

Overall Feedback Criterion: 1 of 4

There are several organs and other components involved in the digestion of food. The organs known as the accessory digestive glands are the liver, gall bladder and pancreas. Other components include the mouth, salivary glands, tongue, teeth and epiglottis.

Human digestive system

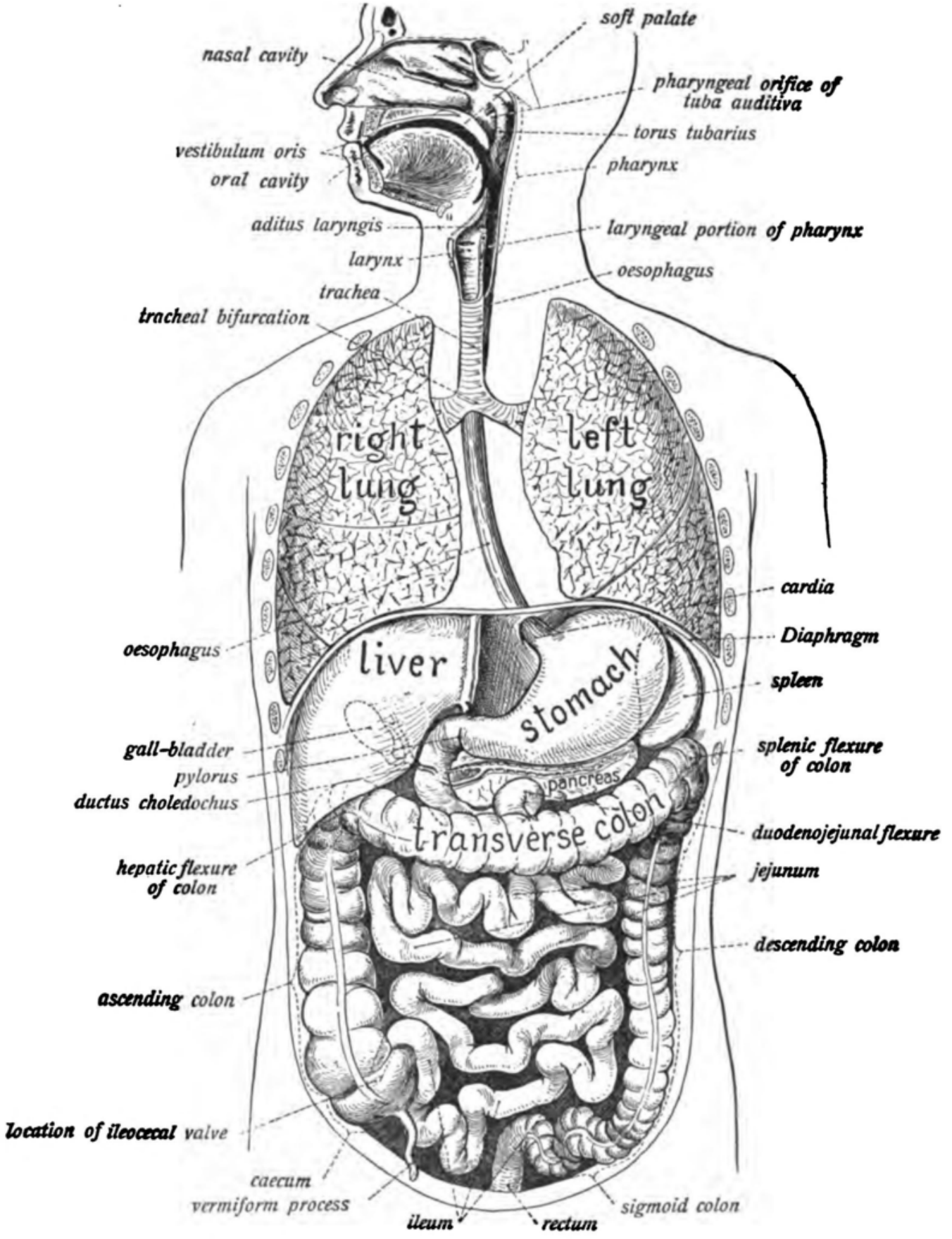
Version 1

Shaikshavali Msu

Apr 17, 2018 at 12:41 PM

Human digestive system

See also gastrointestinal tract.
"Digestive system" and "alimentary system" redirect here. For digestive systems of non-human animals, see Digestion.


soft palate
nasal cavity
pharyngeal orifice of tuba auditiva
torus tubarius
vestibulum oris
oral cavity
pharynx
aditus laryngis
laryngeal portion of pharynx
larynx
oesophagus
trachea
tracheal bifurcation
right lung
left lung
cardia
oesophagus
liver
Diaphragm
stomach
spleen
gall-bladder
splenic flexure of colon
pylorus
pancreas
ductus choledochus
transverse colon
duodenojejunal flexure
hepatic flexure of colon
jejunum
descending colon
ascending colon
location of ileocecal valve
caecum
vermiform process
sigmoid colon
ileum
rectum

The human digestive system consists of the gastrointestinal tract plus the accessory organs of digestion (the tongue, salivary glands, pancreas, liver, and gallbladder).[1] In this system, the process of digestion has many stages, the first of which starts in the mouth. Digestion involves the breakdown of food into smaller and smaller components, until they can be absorbed and assimilated into the body.

Chewing, in which food is mixed with saliva begins the process of digestion. This produces a bolus which can be swallowed down the esophagus and into the stomach. Here it is mixed with gastric juice until it passes into the duodenum where it is mixed with a number of enzymes produced by the pancreas. Saliva also contains a catalytic enzyme called amylase which starts to act on food in the mouth. Another digestive enzyme called lingual lipase is secreted by some of the lingual papillae on the tongue and also from serous glands in the main salivary glands. Digestion is helped by the mastication of food by the teeth and also by the muscular actions of peristalsis and segmentation contractions. Gastric juice in the stomach is essential for the continuation of digestion as is the production of mucus in the stomach.

Peristalsis is the rhythmic contraction of muscles that begins in the esophagus and continues along the wall of the stomach and the rest of the gastrointestinal tract. This initially results in the production of chyme which when fully broken down in the small intestine is absorbed as chyle into the lymphatic system. Most of the digestion of food takes place in the small intestine. Water and some minerals are reabsorbed back into the blood in the colon of the large intestine. The waste products of digestion (feces) are defecated from the anus via the rectum.

Components

There are several organs and other components involved in the digestion of food. The organs known as the accessory digestive glands are the liver, gall bladder and pancreas. Other components include the mouth, salivary glands, tongue, teeth and epiglottis.

The largest structure of the digestive system is the gastrointestinal tract (GI tract). This starts at the mouth and ends at the anus, covering a distance of about nine (9) metres.[2]

The largest part of the GI tract is the colon or large intestine. Water is absorbed here and the remaining waste matter is stored prior to defecation.[1]

Most of the digestion of food takes place in the small intestine.

A major digestive organ is the stomach. Within its mucosa are millions of embedded gastric glands. Their secretions are vital to the functioning of the organ.

There are many specialised cells of the GI tract. These include the various cells of the gastric glands, taste cells, pancreatic duct cells, enterocytes and microfold cells.

Some parts of the digestive system are also part of the excretory system, including the large intestine.[1]

Blood supply

The digestive system is supplied by the celiac artery. The celiac artery is the first major branch from the abdominal aorta, and is the only major artery that nourishes the digestive organs.

There are three main divisions – the left gastric artery, the common hepatic artery and the splenic artery.

The celiac artery supplies the liver, stomach, spleen and the upper 1/3 of the duodenum (to the sphincter of Oddi) and the pancreas with oxygenated blood. Most of the blood is returned to the liver via the portal venous system for further processing and detoxification before returning to the systemic circulation via the hepatic portal vein.

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[Permalink]

The next branch from the abdominal aorta is the superior mesenteric artery, which supplies the regions of the digestive tract derived from the midgut, which includes the distal 2/3 of the duodenum, jejunum, ileum, cecum, appendix, ascending colon, and the proximal 2/3 of the transverse colon.

The final branch which is important for the digestive system is the inferior mesenteric artery, which supplies the regions of the digestive tract derived from the hindgut, which includes the distal 1/3 of the transverse colon, descending colon, sigmoid colon, rectum, and the anus above the pectinate line.

Nerve supply

The enteric nervous system consists of some one hundred million neurons[31] that are embedded in the peritoneum, the lining of the gastrointestinal tract extending from the esophagus to the anus.[32] These neurons are collected into two plexuses - the myenteric (or Auerbach's) plexus that lies between the longitudinal and the smooth muscle layers, and the submucosal (or Meissner's) plexusthat lies between the circular smooth muscle layer and the mucosa.[33][34][35]

Media embedded April 17, 2018
[Permalink]

Parasympathetic innervation to the ascending colon is supplied by the vagus nerve. Sympathetic innervation is supplied by the splanchnic nerves that join the celiac ganglia. Most of the digestive tract is innervated by the two large celiac ganglia, with the upper part of each ganglion joined by the greater splanchnic nerve and the lower parts joined by the lesser splanchnic nerve. It is from these ganglia that many of the gastric plexuses arise.

Development

Early in embryonic development, the embryo has three germ layers and abuts a yolk sac. During the second week of development, the embryo grows and begins to surround and envelop portions of this sac. The enveloped portions form the basis for the adult gastrointestinal tract. Sections of this foregut begin to differentiate into the organs of the gastrointestinal tract, such as the oesophagus, stomach, and intestines.[36]

During the fourth week of development, the stomach rotates. The stomach, originally lying in the midline of the embryo, rotates so that its body is on the left. This rotation also affects the part of the gastrointestinal tube immediately below the stomach, which will go on to become the duodenum. By the end of the fourth week, the developing duodenum begins to spout a small outpouching on its right side, the hepatic diverticulum, which will go on to become the biliary tree. Just below this is a second outpouching, known as the cystic diverticulum, that will eventually develop into the gallbladder.[36]

Clinical significance

Each part of the digestive system is subject to a wide range of disorders many of which can be congenital. Mouth diseases can also be caused by pathogenic bacteria, viruses, fungi and as a side effect of some medications. Mouth diseases include tongue diseases and salivary gland diseases. A common gum disease in the mouth is gingivitis which is caused by bacteria in plaque. The most common viral infection of the mouth is gingivostomatitis caused by herpes simplex. A common fungal infection is candidiasiscommonly known as thrush which affects the mucous membranes of the mouth.

There are a number of esophageal diseases such as the development of Schatzki rings that can restrict the passageway, causing difficulties in swallowing. They can also completely block the esophagus.[37]

Stomach diseases are often chronic conditions and include gastroparesis, gastritis, and peptic ulcers.

A number of problems including malnutrition and anemia can arise from malabsorption, the abnormal absorption of nutrients in the GI tract. Malabsorption can have many causes ranging from infection, to enzyme deficiencies such as exocrine pancreatic insufficiency. It can also arise as a result of other gastrointestinal diseases such as coeliac disease. Coeliac disease is an autoimmune disorder of the small intestine. This can cause vitamin deficiencies due to the improper absorption of nutrients in the small intestine. The small intestine can also be obstructed by a volvulus, a loop of intestine that becomes twisted enclosing its attached mesentery. This can cause mesenteric ischemia if severe enough.

A common disorder of the bowel is diverticulitis. Diverticula are small pouches that can form inside the bowel wall, which can become inflamed to give diverticulitis. This disease can have complications if an inflamed diverticulum bursts and infection sets in. Any infection can spread further to the lining of the abdomen (peritoneum) and cause potentially fatal peritonitis.[38]

Crohn's disease is a common chronic inflammatory bowel disease (IBD), which can affect any part of the GI tract,[39] but it mostly starts in the terminal ileum.

Ulcerative colitis an ulcerative form of colitis, is the other major inflammatory bowel disease which is restricted to the colon and rectum. Both of these IBDs can give an increased risk of the development of colorectal cancer. Ulcerative coliltis is the most common of the IBDs[40]

Irritable bowel syndrome (IBS) is the most common of the functional gastrointestinal disorders. These are idiopathic disorders that the Rome process has helped to define.[41]

Giardiasis is a disease of the small intestine caused by a protist parasite Giardia lamblia. This does not spread but remains confined to the lumen of the small intestine.[42] It can often be asymptomatic, but as often can be indicated by a variety of symptoms. Giardiasis is the most common pathogenic parasitic infection in humans.[43]

There are diagnostic tools mostly involving the ingestion of barium sulphate to investigate disorders of the GI tract.[44] These are known as upper gastrointestinal series that enable imaging of the pharynx, larynx, oesophagous, stomach and small intestine and lower gastrointestinal series for imaging of the colon.

See also

- Gastrointestinal physiology
- Gut–brain axis
- Neurogastroenterology

References

1. "Large intestine". Encyclopedia Britannica. 2016. Retrieved 1 October 2016.
2. Jump up^ Kong F, Singh RP (June 2008). "Disintegration of solid foods in human stomach". J. Food Sci. 73 (5): R67–80. doi:10.1111/j.1750-3841.2008.00766.x. PMID 18577009.
3. Jump up^ Maton, Anthea; Jean Hopkins; Charles William McLaughlin; Susan Johnson; Maryanna Quon Warner; David LaHart; Jill D. Wright (1993). Human Biology and Health. Englewood Cliffs, New Jersey, US: Prentice Hall. ISBN 0-13-981176-1.
4. Jump up^ Pocock, Gillian (2006). Human Physiology (Third ed.). Oxford University Press. p. 382. ISBN 978-0-19-856878-0.
5. ^ Jump up to:a b c Macpherson, G (1999). Black's Medical Dictionary. A & C. Black Ltd. ISBN 0713645660.
6. ^ Jump up to:a b c consultants Daniel Albert et al. (2012). Dorland's illustrated medical dictionary (32nd ed.). Philadelphia, PA: Saunders/Elsevier. ISBN 978-1-4160-6257-8.
7. Jump up^ Ten Cate's Oral Histology, Nanci, Elsevier, 2007, page 321
8. ^ Jump up to:a b Britannica Concise Encyclopedia. Encyclopedia Britannica, Inc. 2007. ISBN 1593392931.
9. ^ Jump up to:a b Ten Cate's Oral Histology, Nanci, Elsevier, 2013, page 275-276
10. Jump up^ Illustrated Anatomy of the Head and Neck, Fehrenbach and Herring, Elsevier, 2012, p. 157
11. Jump up^ Piludu, M; Lantini, MS; et al. (November 2006). "Salivary histatins in human deep posterior lingual glands

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12. Jump up^ Maton, Anthea. Human Biology and Health. Prentice Hall 1993. ISBN 0-13-981176-1.
13. Jump up^ Edgar, WM (1992). "Saliva:its secretion, composition and functions". Br.Dent J. 172: 305–12. doi:10.1038/sj.bdj.4807861. PMID 1591115.
14. Jump up^ S Fagarasan; T Honjo (2003). "Intestinal IgA Synthesis: Regulation of Front-line Body Defenses". Nature Reviews Immunology. 3 (1): 63–72. doi:10.1038/nri982. PMID 12511876.
15. Jump up^ Pettit, John D.; Paul Moss (2006). Essential Haematology 5e (Essential). Blackwell Publishing Professional. p. 44. ISBN 1-4051-3649-9.
16. Jump up^ Bradbury, Jane (March 2004). "Taste Perception Cracking the code". PLOS Biology. 2 (3): E64. doi:10.1371/journal.pbio.0020064. PMC 368160 [Freely accessible] . PMID 15024416.
17. Jump up^ Jowett, Shrestha (1998). "Mucosa and taste buds of the human epiglottis". Journal of Anatomy. 193 (4): 617–618. doi:10.1046/j.1469-7580.1998.19340617.x. PMC 1467887 [Freely accessible] . PMID 10029195.
18. Jump up^ "pharyngeal plexus (anatomy)" [Paid subscription required] . GPnotebook.
19. Jump up^ Sherwood, Lauralee (1997). Human physiology: from cells to systems. Belmont, CA: Wadsworth Pub. Co. ISBN 0-314-09245-5. OCLC 35270048.
20. Jump up^ Dorland's Illustrated Medical Dictionary. Elsevier Saunders 2012. ISBN 978-1-4160-6257-8.
21. Jump up^ Guyton and Hall (2011). Textbook of Medical Physiology. U.S.: Saunders Elsevier. p. 784. ISBN 978-1-4160-4574-8.
22. Jump up^ Black's Medical Dictionary 39th Ed.1999
23. Jump up^ Drake, Richard L.; Vogl, Wayne; Tibbitts, Adam W.M. Mitchell; illustrations by Richard; Richardson, Paul (2005). Gray's anatomy for students. Philadelphia: Elsevier/Churchill Livingstone. p. 287. ISBN 978-0-8089-2306-0.
24. Jump up^ "Histology guide". Retrieved 22 May 2015.
25. Jump up^ Ahrens, Thomas; Prentice, Donna (1998). Critical care certification: preparation, review & practice exams. Norwalk, CT: Appleton & Lange. p. 265. ISBN 0-8385-1474-X.
26. Jump up^ Lower Gastrointestinal Tract at the US National Library of Medicine Medical Subject Headings (MeSH)
27. Jump up^ Cummings, JH; Macfarlane, GT (November 1997). "Role of intestinal bacteria in nutrient metabolism". JPEN. Journal of parenteral and enteral nutrition. 21 (6): 357–65. doi:10.1177/0148607197021006357. PMID 9406136.
28. Jump up^ O.D.E. 2nd. Edition 2005
29. Jump up^ Wood, Jackie D. (2009), "Gastrointestinal Physiology", in Rhoades, Rodney A.; Bell, David R., Medical Physiology: Principles for Clinical Medicine (3 ed.), Philadelphia, PA: Lippincott Williams & Wilkins, pp. 463–496
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Shaikshavali Msu

Feedback

Peer Reviewer: Swetha Msu
Apr 17, 2018

Review

Test_1: 1 of 4

Most of the digestion of food takes place in the small intestine.

Test_1: 1 of 4

The largest part of the GI tract is the colon or large intestine. Water is absorbed here and the remaining waste matter is stored prior to defecation.[1]

Test_1: 2 of 4

There are several organs and other components involved in the digestion of food. The organs known as the accessory digestive glands are the liver, gall bladder and pancreas. Other components include the mouth, salivary glands, tongue, teeth and epiglottis.

Test_1: 1 of 4

Peristalsis is the rhythmic contraction of muscles that begins in the esophagus and continues along the wall of the stomach and the rest of the gastrointestinal tract.

Annotations

A1. Suggested Change - Check punctuation - suggestion?: *Peristalsis*

A2. Comment: *There are several organs and other components involved in the digestion of food.*

Feedback

Peer Reviewer: Mahalakshmi Lakshmi
Apr 17, 2018

Review

Test_1: 1 of 4

Chewing, in which food is mixed with saliva begins the process of digestion. This produces a bolus which can be swallowed down the esophagus and into the stomach.

Test_1: 4 of 4

Chewing, in which food is mixed with saliva begins the process of digestion. This produces a bolus which can be swallowed down the esophagus and into the stomach. Here it is mixed with gastric juice until it passes into the duodenum where it is mixed with a number of enzymes produced by the pancreas. Saliva also contains a catalytic enzyme called amylase which starts to act on food in the mouth. Another digestive enzyme called lingual lipase is secreted by some of the lingual papillae on the tongue and also from serous glands in the main salivary glands. Digestion is helped by the mastication of food by the teeth and also by the muscular actions of peristalsis and segmentation contractions. Gastric juice in the stomach is essential for the continuation of digestion as is the production of mucus in the stomach.

Test_1: 3 of 4

Peristalsis is the rhythmic contraction of muscles that begins in the esophagus and continues along the wall of the stomach and the rest of the gastrointestinal tract. This initially results in the production of chyme which when fully broken down in the small intestine is absorbed as chyle into the lymphatic system. Most of the digestion of food takes place in the small intestine. Water and some minerals are reabsorbed back into the blood in the colon of the large intestine. The waste products of digestion (feces) are defecated from the anus via the rectum.

Test_1: 2 of 4

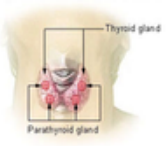
There are several organs and other components involved in the digestion of food. The organs known as the accessory digestive glands are the liver, gall bladder and pancreas. Other components include the mouth,

salivary glands, tongue, teeth and epiglottis.

Annotations

B1. Comment: *A major digestive organ is the stomach. Within its mucosa are millions of embedded gastric glands.*

Swetha Msu



Thyroiditis

Swetha Msu, Gokul pP

Apr 17, 2018 at 1:45 PM

Version 2

Thyroiditis

Thyroiditis is the inflammation of the thyroid gland. The thyroid gland is located on the front of the neck below the laryngeal prominence, and makes hormones that control metabolism.

Classification

Thyroiditis is a group of disorders that all cause thyroidal inflammation. Forms of the disease are Hashimoto's thyroiditis, the most common cause of hypothyroidism in the US, postpartum thyroiditis, subacute thyroiditis, silent thyroiditis, drug-induced thyroiditis, radiation-induced thyroiditis, acute thyroiditis, and Riedel's thyroiditis.[1]

Each different type of this disease has its own causes, clinical features, diagnoses, durations, resolutions, conditions and risks.

Causes

Thyroiditis is generally caused by an attack on the thyroid, resulting in inflammation and damage to the thyroid cells. This disease is often considered a malfunction of the immune system, and can be associated with IgG4-related systemic disease, in which symptoms of autoimmune pancreatitis, retroperitoneal fibrosis and noninfectious aortitis also occur. Such is also the case in Riedel thyroiditis, an inflammation in which the thyroid tissue is replaced by fibrous tissue which can extend to neighbouring structures. Antibodies that attack the thyroid are what causes most types of thyroiditis. It can also be caused by an infection, like a virus or bacteria, which works in the same way as antibodies to cause inflammation in the glands, such as in the case of subacute granulomatous thyroiditis (de Quervain).[4] Certain people make thyroid antibodies, and thyroiditis can be considered an autoimmune disease, because the body acts as if the thyroid gland is foreign tissue.[5] Some drugs, such as interferon, lithium and amiodarone, can also cause thyroiditis because they have a tendency to damage thyroid cells.

Diagnosis/investigation

The most common and helpful way to diagnose thyroiditis is first for a physician to palpate the thyroid gland during a physical examination. Laboratory tests allow doctors to evaluate the patient for elevated erythrocyte sedimentation rates, elevated thyroglobulin levels, and depressed radioactive iodine uptake (Mather, 2007). Blood tests also help to determine the kind of thyroiditis and to see how much thyroid stimulating hormone the pituitary gland is producing and what antibodies are present in the body. In some cases a biopsy may be needed to find out what is attacking the thyroid.

Treatment

Treatments for this disease depend on the type of thyroiditis that is diagnosed. For the most common type, which is known as Hashimoto's thyroiditis, the treatment is to immediately start hormone replacement. This prevents or corrects the hypothyroidism, and it also generally keeps the gland from getting bigger. However, Hashimoto's thyroiditis can initially present with excessive thyroid hormone being released from the thyroid gland (hyperthyroid). In this case the patient may

only need bed rest and non-steroidal anti-inflammatory medications; however, some need steroids to reduce inflammation and to control palpitations. Also, doctors may prescribe beta blockers to lower the heart rate and reduce tremors, until the initial hyperthyroid period has resolved.[6]

Epidemiology

Most types of thyroiditis are three to five times more likely to be found in women than in men. The average age of onset is between thirty and fifty years of age. This disease tends to be geographical and seasonal, and is most common in summer and fall.[3]

Hashimoto's thyroiditis

Hashimoto's thyroiditis was first described by Japanese physician Hashimoto Hakaru working in Germany in 1912. Hashimoto's thyroiditis is also known as chronic lymphocytic thyroiditis, and patients with this disease often complain about difficulty swallowing. This condition may be so mild at first that the disease goes unnoticed for years. The first symptom that shows signs of Hashimoto's thyroiditis is a goiter on the front of the neck. Depending on the severity of the disease and how much it has progressed, doctors then decide what steps are taken for treatment.

See also

- Hypothyroidism
- Hashitoxicosis
- Hyperthyroidism
- Hashimoto's disease
- Thyroid cancer

References

1. "Thyroiditis." www.thyroid.org. 2005. American Thyroid Association. 13 Mar. 2008. 8 Dec. 2015 <http://www.thyroid.org/wp-content/uploads/patients/brochures/Thyroiditis.pdf>.
2. "Thyroiditis." www.thyroid.org. 2005. American Thyroid Association. 8 Dec. 2015 <http://www.thyroid.org/wp-content/uploads/patients/brochures/Thyroiditis.pdf>.
3. Thyroiditis. FamilyDoctor.Org. 2011. American Academy of Family Physicians. <http://familydoctor.org/familydoctor/en/diseases-conditions/thyroiditis.html>.
4. De Groot, Leslie J., Nobuyuki Amino, and Akamizu Takashi. "Hashimoto's Thryoiditis." 30 Jan. 2007. Takashi Akamizu. 3 Mar. 2008 <http://www.thyroidmanager.org/chapter/hashimotos-thyroiditis/>.
5. Mather, M.d., Ruchi. "Hashimoto's Thryoiditis." Medicine.Net. 8 Sept. 2007. 9 Mar. 2008 <http://www.medicinenet.com/hashimotos_thyroiditis/article.htm>.
6. Ross, Douglas. "Beta blockers in the treatment of hyperthyroidism". UpToDate. Wolters Kluwer. Retrieved 27 July 2017.

Swetha Msu

Gokul pP

Feedback

Peer Reviewer: Swetha Msu
Apr 17, 2018

Review

Overall Feedback Criterion: 2 of 4

This disease is often considered a malfunction of the immune system, and can be associated with IgG4-related systemic disease, in which symptoms of autoimmune pancreatitis, retroperitoneal fibrosis and noninfectious aortitis also occur.

Feedback

Peer Reviewer: Swetha Msu
Apr 17, 2018

Review

Overall Feedback Criterion: 2 of 4

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Apr 17, 2018

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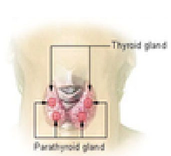

Thyroiditis

Version 1

Swetha Msu, Gokul pP

Apr 17, 2018 at 1:05 PM

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Classification

Thyroiditis is a group of disorders that all cause thyroidal inflammation. Forms of the disease are Hashimoto's thyroiditis, the most common cause of hypothyroidism in the US, postpartum thyroiditis, subacute thyroiditis, silent thyroiditis, drug-induced thyroiditis, radiation-induced thyroiditis, acute thyroiditis, and Riedel's thyroiditis.[1]

Each different type of this disease has its own causes, clinical features, diagnoses, durations, resolutions, conditions and risks.

Causes

Thyroiditis is generally caused by an attack on the thyroid, resulting in inflammation and damage to the thyroid cells. This disease is often considered a malfunction of the immune system, and can be associated with IgG4-related systemic disease, in which symptoms of autoimmune pancreatitis, retroperitoneal fibrosis and noninfectious aortitis also occur. Such is also the case in Riedel thyroiditis, an inflammation in which the thyroid tissue is replaced by fibrous tissue which can extend to neighbouring structures. Antibodies that attack the thyroid are what causes most types of thyroiditis. It can also be caused by an infection, like a virus or bacteria, which works in the same way as antibodies to cause inflammation in the glands, such as in the case of subacute granulomatous thyroiditis (de Quervain).[4] Certain people make thyroid antibodies, and thyroiditis can be considered an autoimmune disease, because the body acts as if the thyroid gland is foreign tissue.[5] Some drugs, such as interferon, lithium and amiodarone, can also cause thyroiditis because they have a tendency to damage thyroid cells.

Diagnosis/investigation

The most common and helpful way to diagnose thyroiditis is first for a physician to palpate the thyroid gland during a physical examination. Laboratory tests allow doctors to evaluate the patient for elevated erythrocyte sedimentation rates, elevated thyroglobulin levels, and depressed radioactive iodine uptake (Mather, 2007). Blood tests also help to determine the kind of thyroiditis and to see how much thyroid stimulating hormone the pituitary gland is producing and what antibodies are present in the body. In some cases a biopsy may be needed to find out what is attacking the thyroid.

Treatment

Treatments for this disease depend on the type of thyroiditis that is diagnosed. For the most common type, which is known as Hashimoto's thyroiditis, the treatment is to immediately start hormone replacement. This prevents or corrects the hypothyroidism, and it also generally keeps the gland from getting bigger. However, Hashimoto's thyroiditis can initially present with excessive thyroid hormone being released from the thyroid gland (hyperthyroid). In this case the patient may only need bed rest and non-steroidal anti-inflammatory medications; however, some need steroids to reduce inflammation and to control palpitations. Also, doctors may prescribe beta blockers to lower the heart rate and reduce tremors, until the initial hyperthyroid period has resolved.[6]

Epidemiology

Most types of thyroiditis are three to five times more likely to be found in women than in men. The average age of onset is between thirty and fifty years of age. This disease tends to be geographical and seasonal, and is most common in summer and fall.[3]

Hashimoto's thyroiditis

Hashimoto's thyroiditis was first described by Japanese physician Hashimoto Hakaru working in Germany in 1912.

Hashimoto's thyroiditis is also known as chronic lymphocytic thyroiditis, and patients with this disease often complain about difficulty swallowing. This condition may be so mild at first that the disease goes unnoticed for years. The first symptom that shows signs of Hashimoto's thyroiditis is a goiter on the front of the neck. Depending on the severity of the disease and how much it has progressed, doctors then decide what steps are taken for treatment.

See also

- Hypothyroidism
- Hashitoxicosis
- Hyperthyroidism
- Hashimoto's disease
- Thyroid cancer

Footnotes

1. ^ (190 keV, with typical beta-decay spectra present) have a tissue penetration of 0.6 to 2 mm.
2. ^ (190 keV, with typical beta-decay spectra present) have a tissue penetration of 0.6 to 2 mm.
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References

1. "Thyroiditis." www.thyroid.org. 2005. American Thyroid Association. 13 Mar. 2008. 8 Dec. 2015 <http://www.thyroid.org/wp-content/uploads/patients/brochures/Thyroiditis.pdf>.
2. "Thyroiditis." www.thyroid.org. 2005. American Thyroid Association. 8 Dec. 2015 <http://www.thyroid.org/wp-content/uploads/patients/brochures/Thyroiditis.pdf>.
3. Thyroiditis. FamilyDoctor.Org. 2011. American Academy of Family Physicians. <http://familydoctor.org/familydoctor/en/diseases-conditions/thyroiditis.html>.
4. De Groot, Leslie J., Nobuyuki Amino, and Akamizu Takashi. "Hashimoto's Thryoiditis." 30 Jan. 2007. Takashi Akamizu. 3 Mar. 2008 <http://www.thyroidmanager.org/chapter/hashimotos-thyroiditis/>.
5. Mather, M.d., Ruchi. "Hashimoto's Thryoiditis." Medicine.Net. 8 Sept. 2007. 9 Mar. 2008 <http://www.medicinenet.com/hashimotos_thyroiditis/article.htm>.
6. Ross, Douglas. "Beta blockers in the treatment of hyperthyroidism". UpToDate. Wolters Kluwer. Retrieved 27 July 2017.

Swetha Msu

Gokul pP

Feedback

Peer Reviewer: Kavita Hegade
Apr 17, 2018

Review

Test_1: 1 of 4

Thyroiditis is generally caused by an attack on the thyroid, resulting in inflammation and damage to the thyroid cells.

Test_1: 4 of 4

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Test_1: 2 of 4

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Test_1: 1 of 4

Thyroiditis is the inflammation of the thyroid gland. The thyroid gland is located on the front of the neck below the laryngeal prominence, and makes hormones that control metabolism.

Annotations

A1. Comment: *dfghhgfdsa*

A2. Comment: *FGN*

A3. Comment: *Treatments for this disease depend on the type of thyroiditis that is diagnosed.*

Feedback

Peer Reviewer: Shaikshavali Msu
Apr 17, 2018

Review

Test_1: 2 of 4

Thyroiditis is the inflammation of the thyroid gland. The thyroid gland is located on the front of the neck below the laryngeal prominence, and makes hormones that control metabolism.Thyroiditis is the inflammation of the thyroid gland. The thyroid gland is located on the front of the neck below the laryngeal prominence, and makes hormones that control metabolism.Thyroiditis is the inflammation of the thyroid gland. The thyroid gland is located on the front of the neck below the laryngeal prominence, and makes hormones that control metabolism.

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Annotations

B1. Comment: *Thyroiditis is a group of disorders that all cause thyroidal inflammation.*

Feedback

Peer Reviewer: Gowthami Palla
Apr 17, 2018

Review

Test_1: 3 of 4

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Annotations

C1. Comment: *sdffdsa*