

ESSENTIALS OF DENTAL CARIES, FOURTH EDITION

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ABSTRACT: *Essentials of dental caries*, fourth edition, provides a comprehensive coverage of the essentials of dental caries. Central to caries control management, the non-operative treatment of caries, for everyone with teeth, is daily plaque control by brushing, the use of fluorides, particularly with fluoride toothpaste, and a sensible, but not draconian, diet which minimizes the frequency and amount of sugar consumption. Some reservations can be felt about whether the authors's recommendations on the use of fluoride dental products are suitable for countries where exposure to high levels of fluoride is common, such as in India, and also the bland reassurance that careful study has never confirmed any of the claims that an increased ingestion of fluoride may result in a variety of diseases.

Keywords: Book review editorial; Dental caries; Fejerskov; Fluoride toothpaste; Kidd; Sugar in diet; Tooth brushing; Toxicity of fluoride.

The first and second editions of *Essentials of dental caries* were written by Edwina Kidd and Sally Joyston-Bechal in 1987 and 1997, respectively. Joyston-Bechal subsequently retired and Kidd, in 2005, wrote the 3rd edition on her own. For this 2016 4th edition,¹ she has combined with an old colleague and friend Ole Fejerskov with whom, along with Bente Nyvad, she co-edits the larger multi-author book *Dental caries: the disease and its clinical management*, the 480-page 3rd edition of which was published in 2015. This shorter 208-page text *Essentials of dental caries* was written with the hope that the dental student, and oral health care workers, would read it at the beginning of their studies and then progress to the larger book before qualification and at postgraduate level. The book is amply illustrated with over 100 colour photographs of teeth together with many further diagrams and various transmission and scanning electron microscope pictures of teeth and the dental biofilm.

The authors note the extreme importance of caries to dentistry with the consequences occupying most dentists and ancillary personnel for most of their time. Dental caries are the direct cause of 85% of the work of dentists in general dentistry with amalgam and composite fillings, sealants, cosmetic dentistry, pulp and root canal treatments, crowns and bridges, dentures, and dental implants. It

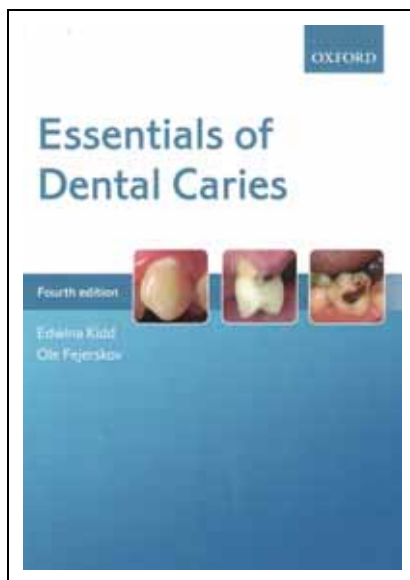


Figure 1. Cover of *Essentials of dental caries*, fourth edition, by Edwina Kidd and Ole Fejerskov.

^aKidd E, Fejerskov. *Essentials of dental caries*. 4th ed. New York, NY, USA: Oxford University Press; 2016. ISBN 978-0-19-873826-8. 208 pages. Available from: Oxford University Press £29.99.

noted that, if not dealt with, the caries lesion development and progression continues for the whole of life, and is the main reason for loss of teeth in populations with no or limited access to dental health care. In addition, the authors conclude that dental caries and restorative care are by far the predominant causes of tooth loss in high income countries. Dental caries are defined as a local chemical dissolution of a tooth surface brought about by metabolic activity in a microbial deposit, a dental biofilm, covering a tooth surface. The dental caries lesions result from a shift in metabolic activity accompanied by a gradual change in the ecology of the dental biofilm, where an imbalance in the equilibrium between the tooth mineral and the biofilm fluid develops. The lesions develop at relatively “protected sites” in the dentition where dental biofilms are allowed to accumulate and mature over time. These sites include pits, grooves, and fissures in occlusal surfaces, especially during eruption, approximal surfaces (the surfaces between adjacent teeth) cervical to the contact point/area, and along the gingival margin. The insertion of foreign bodies into the dentition may also result in protected areas. Some relative protection from caries occurs in the areas near the openings of the major salivary glands because of the buffering capacity and chemical composition of saliva.

The biofilm is characterized by continued microbial activity, with metabolism of salivary proteins and glycoproteins resulting in the very slow production of different types of acids producing continuous minute pH fluctuations. When dietary carbohydrates are added in excess, e.g., glucose, fructose, and sugar, distinct pH fluctuations occur. When the cumulative result of the numerous pH fluctuations over months or years is a net loss of calcium and phosphate, of an extent that makes the enamel sufficiently porous to be seen in the clinic, it may be diagnosed as “a white spot lesion.” At the apex of the lesion the mineral dissolution may reach the enamel-dentine border and continue into the dentine. When about 30–40% of the mineral in the enamel lesion is lost it becomes so porous and fragile that it may easily break apart and a cavity forms. Remineralization is fairly rare and it is only possible to reverse a lesion by starting the caries control measures while the lesion is in an active stage. Most often a white spot lesion remains as a scar in the tooth. No single type of microorganism can be claimed to be the primary cause of either root or enamel caries.

The contemporary soft diet and industrialized food, with the excessive spread of sugar and fat, are seen to be responsible for the dental misery of the last century. However, public health campaigns against the widespread use of sugars have not been very successful. Despite massive campaigns to reduce sugar use in Denmark, total consumption of sugar per capital remained high at approximately 50 kg/individual/yr. Nevertheless, Denmark experienced a more than 90% caries reduction, compared to the 1950s and 1960s because enormous resources were put into campaigns on oral hygiene and the use of fluoridated toothpastes.

Although the dentist may be paid for fillings, which are regarded as treatment, and minimally for preventive advice, the authors consider that caries control management is the non-operative treatment of caries, that it requires skill and time, and that it is worthy of payment. The approach involves daily caries control by

everyone with teeth by plaque control, the use of fluorides, and a sensible, but not draconian diet. Brushing habits should start as soon as the first deciduous teeth erupt with children being helped and supervised by an adult when brushing, even up to 12 yr of age. Teeth should be brushed twice daily, last thing at night and at one other time during the day. Children under 3 yr should use toothpaste containing no less than 1000 ppm of fluoride, the parent putting a small smear of paste onto a small brush. Children should not be allowed to eat or lick toothpaste from the tube. From about 3 yr onwards, the family fluoride toothpaste (1350–1500 ppm fluoride is indicated and now a pea-sized helping of paste is used. Children should always learn to spit out the toothpaste and not swallow it although very young children cannot spit effectively and will swallow most of what is on the brush. Rinsing with water after brushing should be discouraged in order to maximize the topical effect of the fluoride, “spit, don’t rinse.”

Until the 1980s, it was believed that fluoride had to be incorporated into the developing enamel to exert its maximum effect. We now know this is totally incorrect. Even with very low levels of fluoride intake, a certain level of dental fluorosis, an enamel hypomineralization, will be found with the dose-response relationship being clearly linear. The more fluoride ingested from birth, the more the dental fluorosis that may be expected on reaching adulthood and developing a permanent dentition. No association was found between the fluoride concentration in the enamel, as assessed by biopsies, and the caries experience. Painting sound enamel surfaces with 2% sodium fluoride solutions had a cariostatic effect without the fluoride being incorporated into the sound enamel apatite. Fluoride is considered to exert its clinical caries preventive effect by being present in the oral fluids during the dynamic inorganic chemical processes taking place during pH fluctuations at the interface between the biofilm and the tooth surface. The incorporation of fluoride into the enamel apatite was not the cause of the cariostatic effect but a result of numerous ongoing pH fluctuations, which gradually may result in mineral loss and lesion development. Fluoride was then seen to be able to be used with equal success in all age groups and applied in many different ways. Fluoride was not a magic solution that ‘prevented’ caries but an agent that could be used selectively in caries control because it was documented that if fluoride was available in slightly increased concentrations in the oral fluids, it will interfere with the chemical events at the surface of the tooth, so that the rate of caries lesion development is slowed down.

Although for many years a reduction of caries with water fluoridation in the USA of 50% was reported, since the late 1980s the difference between populations with or without water fluoridation has diminished to about 15%. In Europe, the best controlled study in the Netherlands, initiated in the middle of the twentieth century, was abandoned in the late 1970s because the difference in the caries rates between the fluoridated and nonfluoridated areas had gradually disappeared. A series of Cochrane reviews documented that the daily use of fluoride toothpaste was the most cost-effective method, although additional caries reductions could be obtained if other topically-applied fluoride treatments, e.g., varnishes, were combined with the fluoride toothpaste. However, the authors consider it is

important to stress that public water fluoridation is the most cost-effective way of obtaining a caries reduction to everyone in a society in a passive way, by which is meant that individual is not required to actively do anything to benefit.

The authors note that over the years, there have been many claims that increased ingestion of fluoride may result in variety of diseases, including increased hypothyroidism in the UK in populations exposed to increased fluoride levels in the water supply.² However, when such claims have been carefully studied, the results have never been confirmed. The authors comment with respect to the UK hypothyroidism study, that it has to be confirmed in other studies and populations if such a correlation truly exists and what the biological mechanisms might be.

The acute lethal dose of fluoride is given as approximately 15 mg F/ kg body weight (bw), although as little as 5 mg F/kg bw can kill some children. A dose of 5 mg F/kg bw should trigger immediate emergency treatment. Sublethal toxic effects can be produced by as little as 1 mg F/kg bw. The lethal dose of fluoride, at 5 mg F/kg bw, for a 5-yr-old weighing 20 kg would be 100 mg F (20 × 5) which would be contained in 100 mL of 1000 ppm fluoride toothpaste.

The authors comment that evidence that the frequency and amount of sugar consumption is linked to caries is irrefutable and that the dental profession should give dietary advice to all patients. The consensus recommendations are: (i) Drink plain water. Fruit juices, including those with no added sugar are cariogenic. Do not add sugar to drinks. (ii) Reduce the frequency and amount of sugars at all meals and between meals. (iii) Do not eat sweets. Restrict them to preferably 1 day a week. (iv) Avoid in-between meal snacks such as cakes, biscuits, and sweet soft drinks. Take an apple or carrot instead and drink plain water for thirst.

The authors note that the World Health Organization Nutrition Guidance Expert Advisory Group (WHO/FAO) has suggested that the daily consumption of free sugars should be limited to 5% of total calories. An earlier 2003 recommendation of a reduction to 10% of total calories was apparently successfully blocked from becoming WHO policy by the World Sugar Research Organization (WSRO), a trade organization representing more than 30 international members with economic interests in the cane and beet sugar industry, including the Sugar Association in the USA and Coca Cola. They note that dental caries are not a result of fluoride deficiency as fluorides can only modify the rate of mineral loss over time, while sugar consumption speeds up the metabolism in the dental biofilm. In summary, caries control depends on mechanically disturbing the formation and structure of the dental biofilm daily, using a fluoride containing toothpaste when brushing at least twice daily, and lowering the daily intake of fermentable carbohydrates.

The authors consider that increasing the number of dentists does not reduce oral diseases but results in more and more technically advanced restorative treatments to those who can afford them and leave the major parts of populations, often those having socio-economic challenges, without the help that can be provided. It should be possible to provide a much more cost-effective oral health system for the entire population, if current knowledge on the aetiology of dental caries and the

development of oral disease were to be applied in cost-effective disease control. The Danish Odder municipality dental health care programme and the Scottish Childsmile programme, neither of which involve water fluoridation, are given as successful examples, although the authors do not agree with the twice yearly application of fluoride varnish for all children aged 3 yr to young adults in the Scottish programme. The varnish is extremely costly and they reserve its use for those who need it.

The authors provide a comprehensive overview of dental caries. However, their comments on fluoride toxicity are not authoritative. Despite the authors' comment that other studies are required to determine what the biological mechanism of fluoride interfering with thyroid function, a considerable literature exists on the effects of fluoride in disturbing thyroid hormone metabolism and sonic hedgehog signalling.^{3,4}

Similarly, the authors make no reference to the current concerns about the developmental neurotoxicity of fluoride. Although they may accept the reassurance given in one recent report from New Zealand,⁵ that water fluoridation, at 0.7–1.0 mg F/L, poses no significant health risks, and deem the area as unworthy of comment, the view expressed in the corrected executive summary of the New Zealand report on the Choi et al. study⁶ that even a 6.75 IQ point difference, corresponding to 0.45 of a standard deviation, between children exposed to high and low levels of fluoride in drinking water is likely to be a measurement or statistical artefact of no functional significance, is not the consensus view of the majority of researchers on fluoride developmental neurotoxicity.

A study of 1.5–3.5-yr-old children in seven European countries found the amount of fluoride ingested from toothpaste ranged from 0.01 to 0.04 mg F/kg bw-day, corresponding for 18, 24, and 36 month-olds, weighing approximately 11, 12, and 14 kg, respectively, to daily fluoride intakes from toothpaste of approximately 0.11–0.44, 0.12–0.48, and 0.14–0.56 mg F/day, respectively.⁷ These values are approximately 2–12 times greater than the safe reference dose of 0.047 mg F/kg bw-day estimated with the Lowest Observed Adverse Effect Level/No Observed Adverse Effect Level (LOAEL/NOAEL) method.⁸ Similarly, the level of fluoride from water with a level of 0.7 mg F/L, at the 90th percentile of intake, for 8–13-yr-olds, of approximately 0.8 mg F/day is approximately 17 times greater than the safe reference dose of 0.047 mg F/day.⁸

Some resistance may be encountered in India to the recommendation to use fluoride toothpaste due to the difficulties there in finding safe water sources, with 1 mg F/L or less, with the rider in India that the “lesser the fluoride the better, as fluoride is injurious to health,”⁹ the presence of many fluoride containing foods in the diet,⁹ and the findings that fluoride ingestion may contribute to anaemia in pregnant women¹⁰ and in adolescent girls,¹¹ aged 10–17 yr. The view has been expressed that promoting fluoridation of dental products in India should be considered a crime.⁹ Similarly, the early diagnosis and complete recovery from fluorosis has been described by the practice of interventions including not using fluoridated toothpaste.¹²

The book provides a comprehensive coverage of the essentials of dental caries but some reservations can be felt regarding the bland reassurance that careful study has never confirmed any of the claims that an increased ingestion of fluoride may result in a variety of diseases.

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REFERENCES

- 1 Kidd E, Fejerskov. Essentials of dental caries. 4th ed. New York, NY, USA: Oxford University Press; 2016.
- 2 Peckham S, Lowery D, Spencer S. Are fluoride levels in water associated with hypothyroidism prevalence in England? A large observational study of GP practice data and fluoride levels in drinking water. *J Epidemiol Community Health*. 2015;69(7):619-24.
- 3 Shusheela AK, Bhatnagar M, Vig K, Mondal NK. Excess fluoride ingestion and thyroid hormone derangements in children living in Delhi, India. *Fluoride* 2005;38(2):98-108.
- 4 Spittle B. Short stature, bone deformities, cognitive impairment, delayed dental eruption, and dental fluorosis as examples of fluoride-induced developmental disorders involving disturbed thyroid hormone metabolism and sonic hedgehog signalling [editorial]. *Fluoride* 2016;49(2):95-101.
- 5 Eason C, Elwood JM, Seymour G, Thomson WM, Wilson N, expert panel appointed by the Royal Society of New Zealand; Bardsley A, report preparer; Prendergast KL, panel lay observer; Temple W, New Zealand reviewer; Coggan D, Ferguson MWJ, Reynolds E, international reviewers. Health effects of water fluoridation: a review of the scientific evidence. Auckland and Wellington; Office of the Prime Minister's Chief Science Advisor and Royal Society of New Zealand; 2014. Corrected and republished online on 2015 Jan 15. The published erratum was: Erratum: the previous version of the executive summary of this paper stated that the claimed shift of IQ from fluoride exposure was less than one IQ point; it should have stated less than one standard deviation. Updated 15 January 2015. Available from: <http://royalsociety.org.nz/expert-advice/papers/yr2014/health-effects-of-water-fluoridation/>
- 6 Choi AL, Sun GF, Zhang Y, Grandjean P. Developmental fluoride neurotoxicity: a systematic review and meta-analysis. *Environ Health Perspect* 2012 Oct;120(10):1362-8. [abstract in *Fluoride* 2012;45(3 Pt 2):311-2].
- 7 Cochran JA, Ketley CE, Duckworth RM, van Loveren C, Holbrook WP, Seppä L, Sanches L, Polychronopoulou A, O'Mullane DM. Development of a standardized method for comparing fluoride ingested from toothpaste by 1.5-3.5-year-old children in seven European countries. Part 2: Ingestion results. *Community Dent Oral Epidemiol* 2004; 32 (Suppl 1):47-53.
- 8 Hirzy JW, Connett P, Xiang QY, Spittle BJ, Kennedy DC. Developmental neurotoxicity of fluoride: a quantitative risk analysis towards establishing a safe daily dose of fluoride for children. *Fluoride* 2016;49(4 Pt1):379-400.
- 9 Susheela AK. A treatise on fluorosis. 3rd ed. Delhi: Fluorosis Research and Rural Development Foundation; 2007. pp. 16-8.
- 10 Susheela AK. Anaemia of pregnancy: an easily rectifiable problem [guest editorial]. *Fluoride* 2010;43(2):104-7.
- 11 Susheela AK, Gupta R, Mondal NK. Anaemia in adolescent girls: An intervention of diet editing and counselling. *Natl Med J India* 2016;29(4):200-4
- 12 Susheela AK, Mondal NK, Tripathi N, Gupta R. Early diagnosis and complete recovery from fluorosis through practice of interventions. *Journal of the Association of Physicians of India* 2014;62:564-71.