REVIEW

Effect of pregnancy on periodontal and dental health

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Clinical studies have shown that oral tissues can be affected by pregnancy. Pregnancy-related changes are most frequent and most marked in gingival tissue. Pregnancy does not cause gingivitis, but may aggravate pre-existing disease. The most marked changes are seen in gingival vasculature. Characteristic of pregnancy gingivitis is that the gingiva is dark red, swollen, smooth and bleeds easily. Women with pregnancy gingivitis may sometimes develop localized gingival enlargements. The gingival changes usually resolve within a few months of delivery if local irritants are eliminated. The inflammatory changes are usually restricted to the gingiva and probably do not cause permanent changes in periodontal tissues more often than those in the non-pregnant state. Although it is widely believed that pregnancy is harmful to the teeth, the effect of pregnancy on the initiation or progression of caries is not clear. Previous studies, however, indicate that the teeth do not soften, i.e. no significant withdrawal of calcium or other minerals occurs in the teeth. It is mainly the environment of the tooth that is affected. The number of certain salivary cariogenic microorganisms may increase in pregnancy, concurrently with a decrease in salivary pH and buffer effect. Changes in salivary composition in late pregnancy and during lactation may temporarily predispose to dental caries and erosion. Although their underlying mechanisms of action are not fully understood, pregnancy-related changes in the oral environment may have some untoward temporary or permanent effects on oral health. Most of these effects could be avoided by practising good oral hygiene. □ *Estrogens; gingiva; progesterone; saliva; tooth*

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In a woman's life, the major physiological and hormonal changes occur in pregnancy. All functions of the mother's body must adapt to the new condition. Although other hormonal changes also occur, the most significant hormonal change is the increased production of estrogens and progesterone. The production of these hormones gradually increases in pregnancy until the 8th month. During the last month of pregnancy, progesterone concentrations remain relatively constant, whereas estrogen levels continue to rise. The increased production of hormones during pregnancy is mainly due to the placenta, which takes over the production of progesterone and estrogens in early pregnancy, as the main source of these hormones from the 2nd trimester to term. Estrogen levels rise more than 100-fold from the beginning of pregnancy. Progesterone concentrations rise even more. The estrogen to progesterone ratio in the blood changes from 100:1 in early pregnancy to nearly 1:1 at term (1). During labor, when the placenta is withdrawn, a marked fall occurs in both progesterone and estrogens levels. Within 2–3 days of delivery, the hormone concentrations have reached their non-pregnant levels. Hormones classified as estrogens show differences in their biological activity, i.e. the main estrogen in pregnancy is estriol, which is a weak estrogen compared to estradiol-17β (estradiol), which is the main estrogen during the menstrual cycle. In addition to the type of the circulating hormones, the estrogens to progesterone ratio as well as the tissue concentration of the hormones are important for the tissue response. Progesterone and estrogens have several important functions in pregnancy. The main role of progesterone is the maintenance of pregnancy. Both estrogens and progesterone have effects on the vascular system, and they maintain the endometrium and prepare the mammary gland to secrete milk. In addition, they increase the basal metabolic rate, as more energy is needed for the growing fetus. Both progesterone and estrogens modulate the immune system during pregnancy. Some supposedly hormone-related changes have been reported to occur in the mouth during pregnancy. The subject's susceptibility to gingival problems increases, and it is widely believed that pregnancy damages the teeth.

Several studies have shown that pregnant women have more gingivitis than their non-pregnant peers (2, 3) with a prevalence ranging from 30% to 100% (3, 4). However, in a study of 530 pregnant women, Maier & Orban (5) found no significant difference in the prevalence of gingivitis between pregnant and non-pregnant women. The severity of gingival inflammatory changes has proved to be higher during pregnancy than after delivery (3, 5), although no significant changes occur in the amount of plaque (6, 7).

Clinical features of gingivitis in pregnancy

Gingivitis in pregnancy does not differ much histologically

from that in the non-pregnant state. Pregnancy gingivitis is a non-specific, vascularizing, and proliferative inflammation with large amounts of infiltrated inflammatory cells. Some clinical features of gingivitis may be more pronounced in pregnancy. Owing to the increased vascularity and extravasation of red blood cells, the gingiva is dark red and bleeds easily. Other clinical features are the edema-related smooth appearance of the gingiva, thickening of the gingival margin, and hyperplastic interdental papillae, which may result in the formation of pseudopockets.

The early works of Löe & Silness (3, 6, 8) showed that the first clinical signs of gingival inflammatory changes already appear in the 2nd month of pregnancy. The changes continue to occur until the 8th month of pregnancy, being worst at the 3rd and 8th months of pregnancy, after which some improvement occurs in the last month of pregnancy. After delivery, the gingival state was found to be similar to that of the 2nd month of pregnancy. The greatest relative increase in GI scores and mean probing pocket depths was found around the incisors. This is in line with the observations of Raber-Durlacher et al. (7), who found more gingval swelling and bleeding on probing in the anterior teeth region than in the molar and premolar regions, although the amount of plaque was higher in posterior teeth. The gingiva without pre-existing inflammation remains unaffected (8, 9), but the highly vascular gingiva may also bleed easily in women who have no significant signs of gingival inflammation (7).

In a cross-sectional study of 330 pregnant women, Ringsdorf et al. (10) found a positive correlation between tooth mobility during pregnancy and the severity of gingivitis. Rateitschak (11) showed that the upper incisors of periodontally healthy pregnant women are most mobile during the last month of pregnancy. The horizontal mobility of the teeth was thought to be a consequence of edema. Fluid retention may cause some extrusion of a tooth from its bony socket (11, 12). In addition, probing depth seemed to increase, which may also be due to edema in the gingiva (12, 13) and/or to hyperplastic gingival changes. In this case, deeper gingival pockets are not usually a sign of true periodontal destruction. True attachment loss probably requires a chronic inflammatory state of the gingiva lasting longer than those months of pregnancy when the gingival changes occur. Only a few longitudinal studies have been published on the effect of pregnancy at periodontal attachment levels. Tilakaratne et al. (14) and Cohen et al. (15) found no effect of pregnancy on periodontal attachment levels. Most studies have shown that the inflammatory changes referred to above are restricted to the gingiva (3, 14, 15).

The study designs have varied in these studies of gingival changes in pregnancy. The pregnant women participating in some studies have been clinically healthy (11), or all women in some studies have had some form of periodontal disease (3, 12). The results from the different studies are difficult to compare, since a healthy gingiva in pregnancy probably does not differ metabolically much from that of the non-pregnant state. On the other hand, the response of the gingiva to local irritants in pregnancy differs greatly from that in the non-pregnant state. Several studies have shown that pregnancy does not cause gingivitis. A plaque-free tooth surface with a healthy gingiva at the onset of the pregnancy shows no clinical signs of gingival inflammation in pregnancy if good oral hygiene is maintained. In addition, the inflammatory changes are usually restricted to the gingiva and probably do not cause permanent changes in periodontal tissues more often than those in the non-pregnant state. So far, no studies have compared periodontal health in women with children versus women with no children.

Etiology

Although the susceptibility of gingival tissues to inflammation in pregnancy seems to be connected to pregnancy-related hormonal changes, the exact mechanism by which these hormones increase gingival inflammation is not known. Gingival tissue is exposed to steroid hormones by the blood circulation and, to a lesser extent, by saliva. In addition, human gingival tissue contains steroid receptors (16–18), and steroid hormones are metabolized by gingival homogenates (19, 20). Steroid hormones may thus have a specific role in gingival physiology. Gingival vasculature and subgingival flora in particular seem to respond to increased hormonal levels. So far, most explanations for pregnancy-related gingival changes have suggested increased vascularity and vascular flow, directly or indirectly, as the main cause. Other proposed mechanisms include changes in the immune system or changes in connective tissue metabolism.

Gingival responses to microbial challenge during pregnancy

Periodontal diseases are characterized by inflammation, which leads to the destruction of tooth-supporting tissues if balance between the host and microbes cannot be maintained. Acute, non-specific inflammatory reactions and specific immune responses are involved in preventing the spread of inflammation deeper into tissues. Phagocytic cells, such as polymorphonuclear (PMN) leukocytes, macrophages, and lymphocytes, play a central role in host defense. Vascular changes are early events in host response to bacterial plaque. Blood vessels in periodontal tissues dilate and their permeability increases; PMN leukocytes migrate from the blood rapidly to the site of inflammation. The inflamed connective tissue is infiltrated by PMN cells and other migratory cells, such as macrophages and lymphocytes. The number of peripheral PMN cells increases during pregnancy (21), and the function of these cells, which represent non-specific immunity, is changed (21, 22). Although many immunological parameters are

depressed during pregnancy, in general pregnant women are not especially prone to infection. However, pregnant women are often infected by influenza and herpes viruses. T-lymphocytes appear to be more sensitive to hormonal changes than B-lymphocytes. The number of T-helper cells, a subset of T-lymphocytes, decreases slightly, and changes in their action have been shown during pregnancy (21). T-helper cells are important modulators of the immune response, since they are a major source of cytokines. They produce two functionally different types of cytokines. Th-1 type cytokines produce a proinflammatory response, whereas cytokines such as interleukin-6 (IL-6) produced by Th-2 cells have anti-inflammatory actions. Systemic immune responses during normal pregnancy are deviated toward secretion of Th-2 type cytokines (23). So far, the only study of immune responses in the gingiva of pregnant women has been that of Raber-Durlacher et al. (24), who showed increased numbers of T-cells and T-helper cells in gingival specimens from pregnant women during experimental gingivitis. The number of B-lymphocytes and macrophages was in turn decreased.

Connective tissue in pregnancy

Connective tissue seems to be a major target of hormones during pregnancy. The extracellular matrix, gingival vessels, migratory cells, and fibroblasts are all affected. Tissue damage causes the release of cytokines, which activate PMN cells, macrophages, fibroblasts, and epithelial cells to produce proteolytic enzymes, resulting in the breakdown of connective tissue extracellular components. IL-6 has an important role in inflammation. Its production has been shown to be modulated by steroid hormones. Progesterone (25) and testosterone (26, 27) decrease the IL-6 production of human gingival fibroblasts, which in turn may lower resistance to inflammatory challenges. Increased estrogen and progesterone concentrations have been shown to stimulate the production of prostaglandin E2 in the gingiva (28). The increased hormone levels during pregnancy may also exacerbate gingival inflammation (28).

Macrophages have multiple functions. In addition to their antimicrobial action, they play a significant part in tissue remodeling during inflammation. They are involved in the regulation of extracellular proteolysis. They produce plasminogen activator inhibitor type-2 (PAI-2), which is an important inhibitor of tissue proteolysis. Women with clinical symptoms of aggravated gingivitis have low concentrations of PAI-2 in their gingival crevicular fluid (GCF). Women whose response to plaque remains unchanged have high concentrations of PAI-2, which probably protects connective tissue from excessive breakdown (29, 30).

During pregnancy, the tissue concentrations and metabolism of steroid hormones change. Ojanotko-Harri et al. (31, 32) found that inflammation does not increase the metabolism of progesterone in pregnant women as it does in non-pregnant women, resulting in the accumulation of active hormone in gingival tissue. Increased levels of active progesterone in gingival tissue may be immunosuppressive and prevent the acute inflammatory reaction to plaque, allowing a chronic-type of inflammatory reaction and exaggerated appearance of inflammation in the gingiva (32). Dihydrotestosterone (DHT) has an anabolic effect on the gingiva. In health, gingival testosterone is converted by 5α-reductase to biologically more active DHT to a much lower extent in women than in men (33, 34). In an inflamed gingiva, this metabolism in men is similar to that in women (33). The anabolic effects of androgens may be involved in inflammatory repair of gingival tissue as well as in hyperplastic growth. Recently, Kasasa & Soory (35) found that the conversion of testosterone to DHT in periodontal ligament is much greater than in gingival tissue. Periodontal ligament has been suggested to have a role in the periodontal repair process (36). It is known that *Prevotella intermedia* is associated to a greater extent with gingival inflammation than periodontal breakdown (37). It converts testosterone to DHT faster than *Actinobacillus actinomycetemcomitans* or *Porphyromonas gingivalis*. Soory (37) suggested that less virulent bacteria may produce large amounts of DHT, which is involved in the repair of gingiva and alveolar bone (38). The conversion of estrone to estradiol is increased in chronically inflamed gingiva, resulting in the accumulation of the active hormone in the gingiva (39). Estradiol in turn is known to affect the ground substance homeostasis and collagen metabolism.

Cellular events in the gingiva during pregnancy are not fully understood. Balance between host response and bacteria seems to be largely maintained. Although there are some changes that can exacerbate gingival inflammation, there is no evidence so far that the progression of disease to supporting bone is increased during pregnancy. Most of the above studies have been made in vitro by testing cultured fibroblasts or peripheral blood monocytes. Different hormones have been tested separately for effects, although hormones do not act alone in vivo and the net effect may depend on the relative quantities of the different hormones. In addition to the altered tissue metabolism of steroid hormones, inflammatory cells and fibroblasts as well as their products seem have a central role in the modulation of gingivitis in pregnancy. However, several studies have shown that no pregnancy-related changes occur in the gingiva if it is clinically healthy (8, 9).

Microbiological changes in supra- and subgingival flora

Subgingival flora may be modified by pregnancy. Both longitudinal and cross-sectional studies of microbial changes have shown that the numbers of some microorganisms increase in pregnancy. The role of any specific bacteria in pregnancy gingivitis is difficult to determine. It

is known , however, that when the gingivitis is at its worst and the gingiva bleeds most easily, the number of Gram-negative rods is increased. Kornman & Loesche (40) found an increased ratio of subgingival anaerobic to aerobic bacteria. The proportion of *P. intermedia* has been found to increase during pregnancy, but the relation of *P. intermedia* to the clinical signs of gingival inflammation has been controversial (7, 40–43). *Prevotella* species can metabolize sex steroids and use them as growth factors (44). *P. intermedia* and *P. melaninogenica* can substitute estradiol and progesterone for menadione, which is an essential growth factor (44). Both decreased (45) and unchanged (46) responses of peripheral blood lymphocytes to *P. intermedia* have been shown in vitro. Pregnancy-related changes in PMN functions and other defense factors may help *P. intermedia* escape host defense mechanisms to some extent. It remains to be determined whether there are other species acting like *P. intermedia*, since relatively few periodontopathogens have been studied to date.

Altered immune responses, together with a better supply of nutrients in the deeper pockets, may provide a better milieu for some *Prevotella* species. In addition, blood from a bleeding gingiva may serve as an extra nutrient for microbes and gingival enlargement creates more anaerobic conditions in the gingival sulcus, favoring anaerobic flora anyway. These pregnancy-related changes of periodontopathogenic flora are probably temporary without any permanent change in subgingival flora.

Pyogenic granuloma in pregnancy

Pyogenic granuloma in pregnancy (formerly also known as *Epulis gravidarum*) is a particular form of an inflammatory gingival lesion in pregnancy, occurring in up to 5% of women during pregnancy (47). It is an edematous lesion having an interdental attachment and bleeding easily because of its excessive vascularity. In addition, the surface of the lesion may be ulcerated. Bone destruction around the teeth is rarely observed. The lesion usually occurs buccally on the upper anterior teeth. It can appear at any stage of pregnancy, but usually during the 1st or 2nd trimesters of the 1st pregnancy (47). It is usually painless, if not traumatized by the opposing teeth.

The etiology of pyogenic granuloma of pregnancy is largely unknown. In addition to plaque, other local or systemic factors, such as trauma or altered hormonal status, are probably involved in the initiation of this proliferative lesion. It is clinically and histologically identical to pyogenic granulomas in men and non-pregnant women. A mixture of both acute and chronic infiltrated inflammatory cells is seen in the highly vascular hyperplastic granulation tissue. Recently, Yuan et al. (48) showed that macrophages have a significant role in the inflammatory angiogenesis of pyogenic granulomas and possess estrogen receptors. The role of hormones in this connection remains to be resolved. The lesion can grow rapidly but is rarely larger than 2 cm in diameter; it usually disappears spontaneously after delivery in its mild form, if local irritants are eliminated. Surgical excision is usually done after delivery. In some cases, the lesion may cause functional problems or bleed profusely, even spontaneously, and is removed during pregnancy. If the granuloma is removed during pregnancy, it is readily recurrent during the same pregnancy. The lesion may cause permanent changes in the gingiva or additional local etiological factors can persist, since the lesion may occur in the same place in subsequent pregnancies (3, 47).

Effect of general health and smoking on the parodontium and oral mucosa during pregnancy

Some systemic diseases and disorders, as well as tobacco smoking, have been identified as risk factors for periodontal disease. Periodontal inflammation and destruction are increased in pregnant diabetic women compared to non-diabetic pregnant women (49), but the severity of diabetes does not seem to be related to the severity of gingivitis (50). During the second half of pregnancy, when iron requirements are high, iron deficiency may develop. In developed countries, expectant mothers are usually under strict medical control and severe anemia is rare. Oral signs of anemia include pale mucosa, atrophic oral epithelial cells, and smooth tongue due to the atrophy of fungiform and filiform papillae. Impaired wound healing and increased bleeding tendency may occur. During pregnancy, the levels of yeasts increase both in saliva and in the genital tract. Although vaginal candidosis is more common in pregnant women than in non-pregnant women, it is not clear so far whether the prevalence of oral candidosis is increased in pregnant, otherwise healthy women. However, the risk for oral yeast colonization in those infants whose mothers are heavily colonized is probably increased.

It is not clear how tobacco smoking predisposes to periodontal disease. It is likely that smokers have reduced capacity to maintain the inflammatory response to the microbial challenge. Pregnancy may further impair the inflammatory response in pregnant smokers. However, there are no studies on the relation between smoking during pregnancy and bacteria most commonly associated with periodontal disease. The association of smoking to the condition of alveolar bone has been studied mainly in postmenopausal women (51). However, Hildebolt et al. (52) studied 134 periodontally healthy postmenopausal women and found a direct association between alveolar bone loss and age, number of pregnancies, and tobacco smoking. It is not clear if these changes in alveolar bone are present before menopause or if parity predisposes to postmenopausal alveolar bone loss. Increased estrogen levels during pregnancy may protect alveolar bone from destruction.

Periodontitis and preterm birth

The altered metabolism of the whole body in pregnancy may reflect on the physiology of the gingiva and oral mucosa. On the other hand, periodontal infection can have effects on other parts of the body. An association between periodontal disease and preterm birth was suggested recently (53, 54). Pre-existing periodontal disease diagnosed in mid-pregnancy was found to increase the risk of preterm birth 4.5 to 7.1 times compared to the risk in periodontally healthy mothers (54). However, Mitchell-Lewis et al. (55) did not find any differences in clinical periodontal status between mothers with preterm births compared to mothers with full-time pregnancies. The mechanisms linking periodontal disease and preterm birth are not known. It is possible that locally produced inflammatory mediators such as prostaglandins and cytokines are carried to the uterus by the blood to cause uterine contractions.

Saliva and salivary microorganisms

Salivary glands, like other oral soft tissues, contain steroid receptors, which are typical of steroid responsive tissues (18, 56). Salivary glands are well vascularized, which means that their steroid supply is good. Salivary glands as well as other exocrine glands may be affected by pregnancy. The secretion of female sex steroid hormones in saliva is significantly increased during late pregnancy (57), reflecting the increase in serum hormone levels. Estrogens increase the proliferation and desquamation of epithelial cells, which may provide a better nutritional environment for bacteria in supragingival as well as subgingival sites (58, 59). The flow of GCF increases in gingival inflammation and provides access to serum-derived nutrients and hormones, especially for subgingival flora. Previous oral microbiological studies have mainly targeted on periodontopathogenic flora. Cariogenic flora has also been found to be modified during pregnancy. An increase in salivary *Streptococcus mutans*, yeast, and lactobacilli levels has been found in the 3rd trimester of pregnancy and during lactation (60). *S. mutans* has been found capable of metabolizing estradiol but much less so than *Streptococcus sanguis* (61).

Relatively few studies have been made of the flow rate of whole saliva in pregnant women. Longitudinal studies, however, have shown no significant changes in the flow rate of paraffin-stimulated whole saliva (63, 64, 77), while changes occur in salivary composition (Table 1). The pH and buffer effect (BE) values of paraffin-stimulated saliva have been found to decrease towards late pregnancy and promptly recover after delivery (63). Cross-sectional studies have shown that the pH of paraffin-stimulated and unstimulated saliva is lower in pregnant women than in non-pregnant women (65, 72). Lactation seems to have

Table 1. Studies of the effect of pregnancy on the flow rate and composition of saliva

Type of study	Source	Stimulation	No. of women		Change in salivary flow rate and composition	Author
			Pregnant	Non-pregnant		
c.s.	Whole	Paraffin	47	58	Flow rate ± 0	62
c.s.	Whole	Unstimulated	61	56	Flow rate n.d. pH↓	65
l.	Whole	Unstimulated	42		Flow rate n.d. Ca↓ P↓	66
c.s.	Whole	Unstimulated	45	45	Flow rate n.d. Nitrogen ↓	67
c.s.	Submand	Unstimulated	19	15	Flow rate↓	68
c.s.	Parotid	White-vinegar	40	20	Flow rate n.d	69
c.s./l.	Parotid	Lemon-candy	59/14		Flow rate ± 0 Na↓ K↑	70
c.s./l.	Submand	Lemon-candy	59/14		Flow rate ± 0 Na↓ K↑ Ca↓	
l.	Parotid	Unstimulated	26		Flow rate↓ K↑ Ca↑ Protein↑	71
l.	Parotid	Citric acid	26		Flow rate↓ Na↓ K↑ Ca↑ Protein ↑	
c.s.	Whole	Paraffin	50	50	Flow rate n.d. pH↓ Lactobacilli ↑	72
c.s./l	Whole	Paraffin	15/14	15/	Flow rate ± 0 IgA↑/Flow rate n.d. IgA↑	73
c.s./l	Parotid	Citric acid				
l.	Whole	Paraffin	16		Mutans streptococci ↑ Lactobacilli ↑ Yeasts ↑ IgA↑	60
					Flow rate ± 0 pH ↓ Buffer effect↓ Salivary peroxidase ↑	63
c.s.	Parotid	Citric acid	107	7	Flow rate ± 0 Protein ↓ Sialic acid ↓	74
c.s.	Whole	Unstimulated	40	9	Flow rate ± 0 Ca↑ Mg↑ P↑ Cl↑ during labor	75
c.s.	Whole	Unstimulated	45	15	Flow rate n.d. Protein↑ Amylase↑ in early pregnancy Sialic acid↑ Ca↓ P↓ in late pregnancy	76
l.	Whole	Paraffin	8		Flow rate ± 0 Buffer effect ↓	64
		Unstimulated			Flow rate ± 0	
c.s./l	Whole	Paraffin	9	9	Flow rate ± 0, Ca↓ , Na↓	77

l. = longitudinal.
c.s. = cross-sectional: Comparison was made between pregnant and non-pregnant women and/or between pregnant women in various phases of pregnancy.
n.d. = not determined.

no effect on BE, flow rate or pH values (63). Salivary calcium and phosphate contents are slightly lower in pregnant women than in non-pregnant women. The salivary levels of mutans streptococci and IgA concentrations (60, 73) have been found to increase in pregnancy, but these changes were not in line with hormonal changes, since the highest levels of mutans streptococci and IgA occurred during the 3rd trimester of pregnancy and during lactation. Any changes in dietary habits, i.e. smaller meals at more frequent intervals because of the increased energy demands of lactation, may favor the growth of acidogenic microbes also after delivery. The increased levels of mutans streptococci in the mouth can stimulate gut-associated lymphoid tissue, which in turn may lead to stimulation of the common mucosal immune system and further to increased production of IgA to saliva (78). Although in one study the levels of both mutans streptococci and total IgA increased significantly during late pregnancy and lactation, the specific salivary IgA antibodies against mutans streptococci did not change (79). Since most adults harbor mutans streptococci in their mouths, the increased antigen load does not change the levels of specific salivary IgA antibodies to species that are already part of indigenous flora (80). On the other hand, *S. mutans cells* may be transmitted to the child's mouth via the mother's saliva. This in turn may induce an IgG-type immune response in the child's serum. Natural immunization against *S. mutans* may thus occur before the primary teeth are colonized with mutans streptococci (81).

Caries

It is not definitely known whether dental caries incidence increases during pregnancy. Most studies of pregnancy and caries have been cross-sectional or short-term follow-up studies with conflicting results (82–84). As the development of caries usually takes several years, the possible pregnancy-related increase in caries incidence is difficult to estimate. However, Bánóczy et al. (85) found higher DMF indices in women with children than in women with no children. Earlier, it was assumed that calcium was withdrawn from the pulpal side of the teeth into the circulation in the same way as from the bones of a pregnant woman. Chemical analysis of human dentine of extracted teeth has shown that the total mineral content of the teeth seems to be the same in pregnant and non-pregnant women, i.e. the teeth do not soften (86). The calcium and phosphate concentrations in whole saliva seem to decrease during pregnancy (76, 77). Increased demineralization (lowered BE and pH) and decreased remineralization potential (lowered calcium and phosphate concentrations) together with increased salivary levels of mutans streptococci in late pregnancy and during lactation can increase the risk of caries. Morning sickness in turn may increase the risk of erosion.

Although human pulpal tissue was recently found to express estrogen and progesterone receptors (87, 88), no definite caries-promoting effects directly related to female sex steroids have been reported to date. Whether pregnancy affects pulpal responses to dental caries is not known, and there is no evidence available so far as to whether caries proceeds more rapidly in pregnancy than usual.

Owing to the multicausal etiology of caries, no single factor alone has any significant role in the development of caries. It seems that the effect of pregnancy is rather directed at the environmental factors of the teeth, such as changes in salivary gland function and salivary composition, than at the teeth themselves. Pregnancy-related changes in saliva may promote the development of caries in mothers with other risk factors.

Conclusions

Some pregnancy-related changes in gingival physiology and salivary composition may have adverse effects on oral health, not only on the gingiva but also on the teeth. The gingival changes are more readily recognizable, since the gingiva bleeds readily and the symptoms correlate relatively well with hormonal changes. The changes are usually restricted to the gingiva and usually subside some months after delivery if local irritants are eliminated. The prevalence of pregnancy gingivitis in industrialized countries is probably lower now than before thanks to better oral hygiene. The effect of pregnancy on dental health is more difficult to assess, since the initial caries lesion usually develops slowly to clinical caries. The most 'pregnancy-specific' change in saliva is the decrease in salivary BE and pH. These changes, together with increased levels of cariogenic bacteria, may have adverse effects on both the mother's and, later, on the child's dental health. Microbial tests on the saliva, together with salivary BE tests, may help in planning individual programs for the maintenance of good oral health during pregnancy and after delivery, as well as in the prevention of transmission of unfavorable microbial flora from mother to child (89). Fortunately, most pregnancy-related effects on oral health can be avoided by maintaining good oral hygiene.

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