Periodontal Health of Sudanese Women Taking Hormonal Contraceptives

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A Thesis Submitted for Fulfillment of MSC in Dental Public Health

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BDS, MSC, DDPH, RCS
Dedication

To my family especially my mother for her patience, love and blessings

To my husband, my daughters and my sons who had helped me in the time of need.
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Dedication

To my family especially my mother for her patience, love and blessings

To my husband, my daughters and my sons who had helped me in the time of need.

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Abstract

Hormonal contraceptives have become widely used as a form of birth control. Contraceptives induce a hormonal situation which simulate to some extent pregnancy. Women using hormonal contraceptives can be considered as a risk group for periodontal diseases, due to prolonged, sustained serum level of estrogen and progesterone. In previous studies the oral contraceptive users showed higher levels of gingival inflammation than non users. So the aim of this study is to investigate the periodontal health of Sudanese women taking hormonal contraceptives.

Methods:

144 women using hormonal contraceptives for more than 1 1/2 year, pills and injection and a matched control groups of 133 loop users (mechanical device without hormones) were randomly selected for the study. They were clinically examined for plaque levels (Plaque index: PLI), Gingival condition (Gingival: index GI), pocket depth and loss of periodontal attachment (LA).

Results:

Contraceptive users had a statistically significant higher level of plaque compared to the loop users (control) (P<0.00 25;) higher level of gingival inflammation (P = 0.0000;) statistically significant mean pocket depth (p=0.000, t test), and significantly higher LA (P = 0.000;). The conclusion is that follow long usage of hormonal contraceptive may be associated with increased prevalence of gingivitis and increased pocket depth and periodontal attachment loss. So it is important to advice contraceptive users on meticulous oral hygiene practices, together with regular dental check ups.
ملخص البحث

انتشار استعمال الهرمونات الأنثوية كموانع للحمل في شكل حبوب وم الحقنات ومن

الدراسات التي أجريت وجد أن من الأثار المترتبة من استعمال هذه الهرمونات هو

ملاحظة وجود ارتباط بين معدل انتشار أمراض ما حول الأسبوع أثناء فترة استعمالها

خصوصاً إذا استعملت لفترات طويلة. أجريت هذه الدراسة على السيدات المستعملات

لموانع الحمل بمركز تنظيم الأسرة الخرطوم (2) وتهدف هذه الدراسة لمعرفة ما إذا

كانت هناك علاقة بين استعمال هذه الموانع وأمراض ما حول السن (الثلثة).
لا تكون تطبيقية في 2 طريقة تشملNESS، والثبات، وتقتفي أهمية الدراسة. هذه الخاتمة في نصي ذلك لإزالة الصحة الطرقية الاستخدام على التدريب التثقيفية. هذا ويشمل الهرمونية الحمل موانع الاستعمال، صناعة الأسنان سطح على من المرى، وذلكل الأسنان للطب الدورية المرجعة مع ذلكل الأنسجة التهابية من يقلل.
1.1 Introduction:

Gingivitis and periodontitis are destructive inflammatory diseases of the periodontium. Epidemiological studies in many parts of the world have demonstrated a strong positive association between dental plaque and severity of periodontal diseases. However, various local factors which may favour plaque accumulation and systemic factors which may alter the host response to local irritants could influence the development and progression of the disease including calculus, malposed teeth, etc.

Several systemic factors have been associated with increased incidence and severity of periodontal diseases or modifying the course of that disease. Epidemiological studies have shown that over 90% of the variation observed in a population with respect to periodontal diseases can be accounted for by age and oral hygiene alone\(^1\). Other systemic factors can affect severity of involvement of the gingival and periodontal tissues like diabetes, malnutrition, blood diseases and drugs which may modify the course of the disease e.g. anti-epileptic, immunosuppressants, non-steroidal anti-inflammatory drugs and sex hormones (estrogen, progesterone) \(^2\).

There are several types of gingival diseases in which sex
hormones are considered to be either initiating or complicating factors. These types of gingival alterations are associated with physiological and hormonal changes and are characterized by non-specific inflammatory changes with predominant vascular component leading clinically to a marked haemorrhagic tendency\(^{(2)}\).

Experimental studies showed that progesterone administration to female dogs produces dilatation and increased permeability of gingival blood vessels which increases the susceptibility to injury and exudation but it does not affect the morphology of the gingival epithelium \(^{(3)}\).

Repeated injections of estrogen in female rats causes increased endosteal bone formation in the jaws and decreased polymerization of mucoply saccharide protein complexes in the bone ground substance \(^{(4)}\).

Elevated levels of estrogen and progesterone increase gingival exudation in female dogs with associated gingivitis most likely because of hormone induced increased permeability of gingival vessels \(^{(5)}\).

In human, puberty is frequently accompanied with an exaggerated response of the gingiva to local irritation. Gingival changes in pregnancy were also described earlier, even before any knowledge about hormonal changes in pregnancy was available \(^{(2)}\).
Pregnancy itself does not initiate gingivitis, which is caused by bacterial plaque (local irritant). Hormonal changes during pregnancy affects the gingival response to plaque and modifies the resultant clinical picture. The reported incidence of gingivitis in pregnancy in well-conducted studies varies from 50% to 100%. Pregnancy affects the severity of previously inflamed areas but it does not alter a healthy gingiva (2). The severity of gingivitis during pregnancy starts in the second and third trimesters when the levels of estrogen and progesterone are at their highest levels (6). Destruction of gingival mast cells by the increased sex hormones and the resultant release of histamine and the proteolytic enzymes may also contribute to the exaggerated gingival response to any local irritants (7).

Nowadays hormonal contraceptives have become a widely used form of birth control. Since all hormonal contraceptives act by artificially altering sex hormone levels, their influence on gingival inflammation has been studied. Hormonal contraceptives aggravate the gingival response to local irritants in a manner similar to that seen in pregnancy and when taken for a period of more than one and half years increase periodontal destruction (8).
1.2 Literature Review:

1.2.1. Epidemiology:

In a review of periodontal disease by an expert committee of WHO in 1961, periodontal disease was stated as one of the most widespread diseases affecting mankind. No nation, no area of the world is free from it, and in most, it has a higher prevalence affecting in some degree approximately half the child population and almost the entire adult population. Research and clinical evidence indicated that the damage caused to the supporting structures of teeth by periodontal disease in early adult life is irreversible, while in middle adult life it destroys a large part of the natural dentition and deprives many people of all their teeth long before old age. The total effect of periodontal disease on the general
health of the population is unassessable \(^{(9)}\).

1.2.1.1. Distribution of periodontal diseases:

WHO "Global Oral Data Bank" suggests that although gingivitis and calculus are more prevalent and severe in developing nations, there are fewer global differences in the prevalence and severity. Severe periodontitis, gingivitis and calculus are controlled by personal oral hygiene and professional dental care, so it is to be expected that they are less severe in economically developed nations as compared to developing and underdeveloped countries \(^{(10,11)}\).

1.2.1.2. Prevalence of periodontal diseases:

Data before 1980 is extremely difficult because the indices used to measure the condition are no longer considered valid.

Data from many parts of the world collected during the 1980s and 1990s showed that the prevalence of generalized severe periodontitis is in the range of 7% to 15% in almost all populations regardless of their state of economic development and oral hygiene or availability of dental care\(^{(12)}\).

1.2.1.3. Gender, race and periodontal diseases:

All surveys of periodontal health condition usually show that men have poorer periodontal health compared to women. It is well recognized that females may practice better oral health measures than males. The
common view may be that teeth are gender free, but how can this be when teeth exist in the body and that body differs if it is a male or a female. For many years, the primary acknowledged difference between males and females oral health was pregnancy gingivitis. But there are many areas where women's oral health may differ from that of men\(^{12}\).

WHO Global Oral Data Bank, which maintains data from many nations collected using the Community Periodontal Index Of Treatment Needs (CPITN) index, suggest remarkable uniformity of the condition around the world, overall, the evidence suggests that race and ethnicity in themselves can not be considered demographic risk factors of periodontitis, but the most determinant factors are oral hygiene practices and dental care\(^{10,11}\).

**1.2.1.4. Socioeconomic status and periodontal diseases:**

The levels of periodontal diseases, when recorded by Composite Indices have historically been related to socioeconomic status of populations (SES). Gingivitis and poor oral hygiene are clearly related to low SES but at less directly observed relationship between SES level and gingival health, but as a function of better oral hygiene among the more educated, and a greater frequency of dental visits among the more dentaly aware and more with dental insurance and income.

SES is complex and multifaceted variable that can include a variety
of cultural factors and it is virtually impossible to remove the effect of SES as a confounder in the race/ethnicity relationship (13,14).

1.2.2. Chronic inflammatory periodontal diseases

1.2.2.1. Etiology of Periodontal diseases:

Periodontal disease comprises a group of inflammatory conditions of the supporting tissues of the teeth that are caused by bacteria. In the 1900s it was believed that all bacterial species found in dental plaque were equally capable of causing disease and that periodontitis is a result of cumulative exposure to dental plaque. In 1960s following microscopic examination of plaque, indicated an association between specific bacterial species with specific types of periodontal diseases (15).

1.2.2.2. Dental Plaque:

Plaque can be defined as the soft deposits that form the biofilm adhering to the tooth surface or other hard surfaces of the oral cavity including removable and fixed restorations (16).

Plaque is classified as supra- gingival or subgingival based on its position on the tooth surface. Supra gingival plaque is found at or above the gingival margin. The subgingival plaque is found below the gingival margin between the tooth and the gingival sulcular tissue. The location of plaque is significant to
particular diseases of teeth and periodontium. For example, marginal plaque is of prime importance in the development of gingivitis, while supra-gingival plaque on teeth is associated and is critical in calculus formation and root caries, whereas tissue-associated subgingival plaque is important in soft tissue destruction a characteristic of different forms of periodontitis \(^{(17)}\).

The process of plaque formation can be divided into three phases, the formation of a pellicle coating on the tooth surface, initial colonization by bacteria, secondary colonization and plaque maturation. Initial bacteria colonizing the tooth surfaces are predominately gram positive facultative micro-organisms such as \textit{actinomyces viscosus} and \textit{streptococcus sanguis} \(^{(18)}\).

The plaque mass then maturates through the growth of attached bacterial species as well as colonization and growth of additional species. In this ecology succession of the biofilm, there is a transition for early aerobic environment characterized by gram positive facultative species to a highly oxygen-deprived environment in which gram-negative anaerobic micro-organisms predominate.

Secondary colonization by micro-organisms that do not initially colonize clean tooth surfaces occur, including \textit{prevotella intermedia},
prevotella loescheii, capnocytophaga species, Fuspobacterium nucleatum, and porphyromonas gingivalis.

These micro-organisms adhere to the cells of bacteria already present in the plaque mass(19).

1.2.2.3. Gingivitis:

The development of gingivitis has been extensively studied in a module system referred to as experimental gingivitis in man and initially described by Loe et al. (1965).(20) The initial microbiota of experimental gingivitis consists of gram-positive rods, gram positive cocci, and gram negative cocci. The transition to gingivitis is evident by inflammatory changes observed in the gingival tissue and is accompanied first by the appearance of gram negative rods and filaments, then by spirochetes and motile micro-organisms (21)

The periodontal microbiota is a very complex ecological system with many structural and physiologic interaction among resident bacteria and between bacteria and the host. It is clearly possible that levels of a particular species may be elevated as a result of environmental changes produced by the disease process and may not be a causative agent. For example, in studies on the development of gingivitis in humans at the time of puberty, it was found that only the proportion of capnocytophaga species increased prior to the development of gingivitis and bacteria
P. intermedia was recovered only after the onset of gingivitis. This suggests a causative role for capnocytophaga species and that environmental changes associated with disease can favor the emergence of species such as P. intermedia. Bacteria found in chronic gingivitis consists of a roughly equal proportions of gram positive (56%) and gram negative (44%) species as well as facultative (59%) and anaerobic (41%) micro-organisms. The gram-positive species are primarily S. Sanguis, S. mitis, A. viscosus, A. naeslundii and peptostreptococcus microorganisms. The gram negative micro-organisms are predominantly F. nucleatum, P. intermedia, V. Parvula, and Haemophilus and campylobacter spp. The term pregnancy gingivitis is used to denote acute inflammation of gingival tissues sometimes associated with pregnancy. This condition is accompanied by an increase in steroidal hormones in crevicular fluid and a dramatic increase in the level of P. intermedia which uses the hormones as a growth factor. Studies of gingivitis support the conclusion that disease development is associated with characteristic alterations in the microbial composition of dental plaque and are not simply due to an accumulation of plaque. Gingivitis is generally believed to precede the development of
chronic periodontitis; however, many individuals demonstrate long-standing gingivitis that never advances to destruction of the periodontium (25).

3.2. Effect of sex hormones on gingival tissue:

3.2.1. Experimental Studies:

In a series of studies on dogs, the effect of extraneous estrogen and progesterone was studied on clinically healthy gingival tissue injured by standardized surgical procedure. The regenerating gingiva was therefore selected as a test model. Lindhe and Branemark in (1967) found that application of intramuscular treatment with progesterone produces a change of blood flow of the plasma endothelial interface and of the cells of vein walls during regeneration of tissue. Thus distanced micro-circulation with micro-thrombi stasis and an increase of veinule diameter should be regarded as a consequence of an increase in the blood level of progesterone. This may in turn result in leakage from the vessels and edema of the perivascular tissue (7).

Lindhe, and Hugoson, (1969) studied the influence of estrogen and progesterone on gingival exudation of regenerating dento-gingival tissue. The experimental study was done on pseudo-pregnant female dogs. Before administration of extraneous estrogen and progesterone, clinically
healthy gingival tissue injured by standardized surgical procedure was prepared. The regenerating gingiva was therefore selected as a test model and the healing of the excised gingival area was followed by measuring the amount of gingival exudates (Brill 1959)\(^{(26)}\), and the depth of the crevice on days 4, 8, 15, 24, 29, 36 and 43 after surgery. After day 43 the gingivae were in the final phase of healing. The results indicated that during the first 4 days after surgery there was a marked increase in gingival exudation followed by a decrease throughout the following period up to day forty three. After that administration of female sex hormones was initiated. Each dog was given 1 mg estrogen intramuscularly, once every second day and 25 mg progesterone each day. The hormones were given during a 64 day period. Then regenerating gingivae were studied for further 20 days. The authors found that the gingival exudation increased and remained high as long as the hormones were given. When the hormone injections were stopped the gingival exudation decreased. The result was that the intramuscular injection of estrogen and progesterone increases the amount of exudates obtainable from chronically inflamed as well as from healing gingivae. This was the same as that noticed during pregnancy in human\(^{(5)}\).

Later Hugoson and Lindhe, (1971) studied the effect of extraneous
estrogen and progesterone on the regenerating gingivae using the same previous model. One week after initiation of the hormone injection and immediately before surgery the exudates for the estrogen progesterone period was twice as high as that for the control period. After day 15 there was a gradual decrease of the gingival exudation in the controls. The difference between the mean gingival exudates from the excised areas in the control period was significant\(^{(27)}\).

Again Hugoson and Lindhe, (1971) studied the influence of extraneous estrogen and progesterone on the histological and morphological appearances of healing gingivae of ten beagle dogs. They used a standardized surgical procedure. They found no effect on the morphology of the cervical epithelium or the oral epithelium. They also found that the administration of sex hormones did not seem to influence the number of fibroblasts and configuration of collagen fibers of the healing gingivae \(^{(28)}\).

Hugoson, et al (1971) in studying the revisualization of healing gingival tissues also found that administration of progesterone to female dogs produce dilatation and increased permeability of gingival blood vessels. This in turn increases the susceptibility to injury and exudation, but it does not affect the morphology of gingival or cervical epithelium
nor does it influence the number of fibroblasts or the configuration of collagen fibers of healing gingivae. The density of mononuclear inflammatory cellular infiltrate was found in the epithelium in both estrogen and progesterone treated dogs but in larger numbers in progesterone treated dogs. The exudate sample and score of PMN suggests an effect of progesterone primarily on structure and or function of the cervicular vessels. This finding was supplemented by the observation that the marginal vessels of progesterone treated dogs were wider and more tortuous than controls. However no prominent difference was found between the progesterone- treated and control animals in the number of vessels of regenerating cervicular plexus. The finding in studies on the dogs suggests that progesterone influence is limited only to the cervicular vessels (29).

3.2.2. Influence of hormonal variation on the periodontal health of women:
Hormones, exert significant influence on body physiology throughout life. Women, in particular, experience hormonal variation on both physiological and non physiological conditions, such as hormonal therapy or the use of hormonal contraceptives.

This variation affects women's oral health, as hormonal variation
affects the physiology of the host parasite interaction in the oral cavity.

Beckl in 1977, in a study of gynecological and obstetrics problems in connection with stomatology, found that hormonal changes during pregnancy produce changes in the whole organism. Every fifth to seventh pregnant woman showed hyperplastic gum changes with tendency to bleeding. Changes due to pregnancy in the oral cavity included lowered salivary flow rate, changes of PH towards acidity and an increase in estrogen contents of saliva, corresponding to the increase in plasma estrogen. Endocrinological changes during the menstrual cycle may give risk to recognizable cycle-dependant changes in the oral mucosa through changes in blood clotting mechanism may be minimal. The use of contraceptives (pills) lead to hypercoagulability of blood clotting system (30).

The effect of sex hormones on subgingival plaque was studied by Jensen, et al in 1981, One hundred and four women aged between 18-40 year were divided into three groups and the periodontal status of three groups; pregnant, taking oral contraceptives and non pregnant, were evaluated clinically and microbiologically for changes in their gingivae and any corresponding changes in subgingival microbial plaque, specially the percentages of fusobacterium species and Bacteroides species. They found statistically significant increased scores in the Gingival Index and
gingival crevicular fluid flow among the pregnant group compared to non pregnant women. The most dramatic microbial changes found were the increased proportion of Bacteroids species both in pregnant and oral contraceptive groups. Increased female sex hormones substituting for napthaquinone requirement for certain bacteroides were not likely responsible for this increase. No statistically significant difference was noted between the group taking contraceptives and non pregnant group. Although a 16 fold increase in Bactericides species was observed in the group taking oral contraceptives(31).

El Attar et al in 1982, studied the relationship between the concentration of female sex hormones and prostaglandin production by human gingivae in vitro, they found that estrodiol -17B and progesterone separately or in combination from C-arachidonic acid significantly affects the formation of prostaglandin in a fashion inversely related to it’s concentration. So the effects of both hormones could be stimulatory to prostaglandin synthesis in the gingivae of pregnant women. However prostaglandins are synthesized by activated microphage and to a lesser degree by polymorph nuclear neutrophils in response to inflammatory stimuli both of which seem to increase in number when the gingivae become inflamed (32).

Sooryamorthy and Grower (1989) studied the influence of sex
hormones on gingival tissue and its relationship to periodontal diseases. They found that the clinical changes seen in plaque induced gingivitis is accentuated by circulating levels of hormones via mechanisms such as partial immune suppression, increased fluid exudation, stimulation of bone resorption and stimulation of fibroblasts synthesis activity. High counts of *Bacteroides intermedius* have been observed in users of oral contraceptives and also during the second trimester of pregnancy, in the absence of overt gingival inflammation which is due to competition for binding between progesterone and napthaquinone, which have structural similarity and the latter is an essential nutrient for the microbe. They concluded that high counts of *Bacteroides intermedius* may be a more sensitive indicator of an altered systemic hormonal condition compared to the usual clinical parameters. The main hormonal effect accentuates false pocketing rather than initiating a change in attachment level, except in cases of progressive periodontal disease associated with plaque induced inflammation and bone loss (33).

Amar (1994) studied the influence of estrogen and progesterone on the periodontal environment of women. He found that microbial changes show increasing ratio of anaerobe to aerobic bacteria in estrogen and progesterone users and also there were increased numbers of *prevotella intermedia*. The vascular changes, include dilated gingival capillaries and
increase venule and capillary permeability on long acting progesterone. 

Progesterone stimulated endothelial cells and inhibits collagen production and progesterone decreases keratinization. Estrogen and progesterone increases epithelial glycogen and alter polymerization of ground substance and increase foliate metabolism. The immune changes include depressed neutrophil chemotaxis and phagocytosis, depressed antibody response and T cell response. Also estrogen and progesterone stimulates prostaglandin synthesis in microphages(34).

The influence of two different hormonal contraceptives on bacterial flora of gingival sulcus was studied by Klinger et al.(1998). In 14 women, an oral contraceptive containing 0.02mg ethinyI estradiol and 0.15mg desogestrel (preparation A) was used, and 15 women took a contraceptive containing 0.03mg ethinyl estradiol and 2.00 mg dienogest (preparation B) daily over a period of 21 days. There were no changes in clinical parameters of the teeth investigated during 3 weeks of the study. The periodontopathogenic bacteria Porphyromonas gingivalis and Actinobacillus actinomycetem comitans were never detected throughout the study. On the other hand, the periodontopathogenic species Prevotella intermediate was found in plaque samples of 22 women. The content of this microorganism showed only a little change between the pretreatment period and plaque sampling after 10 days of contraceptive treatment, but
striking increase occurred after 20 days of contraceptive treatment, especially in the preparation A group. In this respect, there was a significant difference between preparations A and B (35).

The modulation of androgen metabolism by estrogen and progesterone in human gingival fibroblast has been investigated by Tialkaratne and Soory in 1999. The results reinforced the potentially anabolic and catabolic roles of estradiol and progesterone, respectively.

This may partially explain the modulatory mechanisms involved in periodontal disease presentation during altered hormonal status and healing response in the inflamed periodontium (36).

On the other hand Morishuta et al (1999) studied the effect of hormones on the production of interleukin-1 from peripheral monocyte. IL-1 is a potent mediator of inflammation and is known to induce bone resorption. They also studied the effect of sex hormones on the function of monocyte, and demonstrated that prostaglandin E2 production was enhanced by progesterone and estradiol. As prostaglandin E2 has been shown to suppress the production of IL-1 by monocyte. It is possible that sex hormones also modify the production of IL-1 by regulating PGE2 production, furthermore the capacity of PGE2 to modulate IL-1 production
was examined by utilizing indomethacin which is an inhibitor of PGE2 synthesis. The results of the study showed that estradiol and progesterone suppress the production of IL-1 by human monocytes significantly, even in the presence of indomethacin. Thus it was concluded that the inhibition of IL-1 production was not the result of enhanced production of PGE2 induced by sex hormones.

Soory (2000) also discussed the effect of sex steroidal hormones glucorticoids and insulin deficiency on periodontal tissues and possible consequences on periodontal disease progression. The androgens, estrogen and progesterone have predominately anabolic functions in stimulating matrix synthesis, which is applicable to periodontal repair and medication-induced gingival overgrowth. The author concluded that estrogen and progesterone can contribute to pregnancy gingivitis, long term use of hormonal contraceptives can accentuate progression of periodontal disease.

3.2.3. Effects of puberty, pregnancy and hormonal contraceptives on the periodontium of women:
Hormones exert a significant influence on body physiology throughout life. Women in particular experience hormonal variation under both physiological and non physiological conditions such as hormonal therapy. The condition in which hormonal changes occur in women
3.2.3.1.Puberty:

At puberty, women change physically due to the production of the female sex hormones (estrogen and progesterone). This begins with the anterior pituitary secretions of gonadotropin hormones (follicle-stimulating hormone and lutenizing hormone) that cause ovaries to begin cyclical production and secretion of the hormones (estrogen and progesterone). Estrogen induces several of the developmental changes observed in women during puberty, and progesterone acts synergistically with estrogen to control menstrual cycle and inhibit follicitropin secretion by the anterior pituitary. In addition, progesterone affects both progestational changes in the endometrium and cyclical changes in the cervix of the uterus and vagina. These sex hormones also have other significant biological actions that can affect other organs and systems including the oral cavity.

In a longitudinal study of gingivitis and puberty, Stutcliffe, (1972) marked the initiation of changes from puberty and maturation into adulthood, which is associated with a major increase in the secretion of gonadotrophic hormones. In young women this results in a monthly cycle and menstruation. While plaque scores may remain relatively unchanged or low during puberty, the prevalence and severity of gingival inflammation
gradually increases with age resulting in what is known as pubertal gingivitis. (39)

Lawrence et al. in 1986, in a longitudinal study examined eighteen subjects processing normally through puberty (group 1) and a cross-sectional study of nine subjects with precocious puberty (group 2) to evaluate the effects of puberty on clinical and microbiologic parameters of periodontal health. The authors found no significant changes in the mean plaque index (PLI), gingival index (GI) or dark field microscopic counts of the associated microflora in group 1 from session to session. Individual subjects changing Black pigmented bacteroides (BPB) carrier status at a session also experienced change in GI when compared to the group as a whole. A positive correlation was observed between PLI and GI at all sessions in the study. No radiographic evidence of destructive periodontal disease was observed. A correlation was noted between the presence, but not the level of BPB and plasma estradiol concentration in group 2 subjects. Isolation of BPB, almost all P.intermedius, was not correlated with physical maturation in either group. During puberty, mild gingivitis may exist prior to detectable colonization by spirochetes or BPB. The authors concluded that hormones during puberty do not necessarily enhance the colonization of pathogens implicated in adult periodontitis (40).

3.2.3.2 Pregnancy:
During pregnancy, the levels of sex steroidal hormones are raised from luteal phase, until parturition. Pregnant women near or at term produce large quantities of estradiol, aestriol and progesterone 20, 80 and 300 mg/day, respectively (normal ranges are: 0.6, 3 and 19 mg/day for the three hormones, respectively). This prominent rise in hormonal levels during pregnancy appears to elevate the prevalence and severity of gingivitis.

Early works by Loe and Silless (1963) where they examined one hundred and twenty one pregnant women and sixty one post-partum women for occurrence and severity of periodontal diseases, are pioneering works. They found that 100% of the pregnant women showed signs of gingival inflammation. The prevalence and severity of gingival disease among pregnant women was significantly higher than in post-partum women. The increase was noticeable from the 2nd month of gestation and reached a maximum in the 8th month. During the last month of gestation a definite decrease occurred. After parturition the state of the gingivae became similar to that of the second month of pregnancy. The gingival pocket depth increased during pregnancy, then decreased after parturition indicating that the deepening was probably caused by enlargement of the gingivae rather than a true loss of attachment. The increase in occurrence and severity of gingival inflammation during pregnancy did not seem to
cause lasting injuries to the periodontium. The same investigators found that only one lady out of the one hundred and twenty one pregnant women presented with a gingival epulis (5).

Hugoson, in 1970 in a longitudinal study investigated the state of the gingivae and amount of bacterial plaque on the teeth in 26 women. The women were followed during pregnancy and after parturition. At the examination the amounts of bacterial plaque was graded according to PLI and measurement of the gingival exudate which were used to study gingivitis. The GI was also recorded and the depth of periodontal pockets was measured. The author found that all women had gingivitis and bacterial plaque on their teeth during pregnancy as well as after delivery. The severity of gingivitis gradually increased until parturition, after which it successively decreased. He also found that 20 weeks after parturition gingival inflammation was less severe than at the 12th week of pregnancy. Those women in whom the state of the gingivae was best at first examination and those whose gingivae was worst were studied separately. In both groups there was a roughly equal increase in gingivitis during pregnancy and a similar decrease after parturition. The relationship between gingivitis and bacterial plaque revealed no significant regression (41).

Hugoson, et al. in 1970, investigated sex hormones of pregnant
women by cytological analysis of vaginal smears in addition to the state of oral epithelium, from cytologic examination of oral smears during pregnancy and 8 weeks after delivery. The vaginal and oral smears were taken on the same occasion as the examination of gingivae and oral hygiene. At each examination differential counts were made of the cell population. No change was found in the oral epithelium during pregnancy similar to that found in the vagina. The severity of gingivitis in relation to the concentration of sex hormones in blood during normal pregnancy was investigated. A significant regression was found for both gingival exudation and gingival index with the varying amounts of sex hormones. The authors also found that gingivitis became more sever without any accompanying increase in the amount of bacterial plaque. The results of this study suggested that the physiological change in the concentration of sex hormones during normal pregnancy may influence gingival tissue anatomy and function.

Kornman and, Loeche (1980) showed that, during pregnancy, increased levels of progesterone and estrogen paralleled gingival conditions and proportions of _P.intermedia_. The shift of micro-organisms, represented by an increasing anaerobic- to aerobic ratio, is a result of change in the subgingival micro-environment caused by accumulation of active progesterone, whose metabolism is reduced during pregnancy and the
ability of *P. intermedia* to substitute an essential growth factor with progesterone and estrogen. Taken together, the effects of immunosuppression and influence of progesterone, increased the level of active progesterone in the gingiva and microbial shift towards increased proportion of *P. intermedia* which can exacerbate gingival response to microbial plaque in pregnant women. Although a significant proportion of pregnant women suffer from pregnancy gingivitis, this condition is self limiting and transient. Gingival tissues return to their original healthy state post-partum (42).

In an immunohistochemical study, Raber-dur- Laches et al (1993) suggested that increased CD4 positive cell found in oral and sulcular epithelium during experimental gingivitis in pregnant women may belong to the th-1 subset. This subset of CD4 cells are known to be cytotoxic to human leuckocyte antigen class II antigen- bearing cell (B cells and macrophage) which may result in diminished immunoresponsiveness during pregnancy gingivitis. Furthermore, the th-1 subset mediated delayed-type hypersensitivity and it's cytotoxicity to B-cell may reduce the production of antibodies against such bacteria as *P. intermedia*. This may in turn serve as another contributing factor that explain increased level of *P. intermedia* (43).
Tilakaratne and Soory (2000) in a study of a group of 47 pregnant women and 47 non-pregnant women as a matched control group found that despite of the similar scores of plaque in both pregnant and non-pregnant women, the GI of pregnant women was significantly increased, during the 1st and 2nd trimester compared to the control during the 3rd trimesters. GI was further increased but dropped at 3 month postpartum. The value of LA did not show significant differences from that of control, during any stage of pregnancy. The study showed that pregnancy had an effect only on the gingivae and not on the periodontal attachment level. The effect of estrogen and progesterone could give rise to a more florid response to the irritant effect of plaque, resulting in severe gingivitis (44).

Guillermo et al (1999) in a study of the influence of general health and socio-cultural variables on the periodontal condition of pregnant women found that the mean plaque index increased significantly when the professional care was low, education was low and previous maintenance was less frequent. The results illustrate the importance of establishing periodontal preventive measurements for pregnant women, even though their demographic and clinical characteristics do not differ from those of the general population (45).

3.2.3.3. Hormonal contraceptives:

Hormonal contraceptives are among the widely used
pharmaceuticals in the world today. Most oral contraceptives (birth control pills) are a combination of synthetic estrogens and progesterone. There are numerous currently various commercial preparations available. It is a relative conciliation, combination and sequences of estrogen and progesterone in these products, which distinguish the different type. Women who are taking these contraceptives are basically mimicking the hormonal condition of pregnancy in that they have increased levels of circulating estrogen and progesterone. The high plasma concentration of these hormones acts to suppress the secretion of the anterior pituitary hormone, follicle stimulating hormones (FSH) and lutenizing hormone (LH) by way of a negative feed back mechanism. This in turn prevents the development of Garfian follicle and subsequent ovulation; and thus contraception is achieved. In addition the high levels of estrogen and progesterone produce alteration in genital tract and endometrium which may contribute to the contraceptive effectiveness of these preparations.

The Oral contraceptives (OC) are of three main types:
(1) The combined oestrogen-progestogen preparations (with high, medium or low oestrogen contents).

(2) The combined sequential preparations with the doses of each steroid
varied throughout the cycle.

(3) The progestrogen-only preparations or mini-pills.

The oestrogens used are either ethnyloestradiolin doses of 20-50 mg, or mestranol in doses of 50-100 mg. The progestogens are either those derived from 19-norethisterone (eg norgestrel, norethisterone) or more rarely from 17-alpha-hydroxyprogesterone (eg megestrol) in doses ranging from 0.25 to 5.0 mg. The combined and sequential preparations are taken for 20-21 days followed by an interval of 7 days during which withdrawal bleeding occurs. Some of them includes six or seven tablets of lactose to be taken during this time so that the daily habit of taking a tablet is not broken.

The steroid hormone receptors in gingival tissue, were studied by Formicola et al (1970), and Lundgren (1973). They found that H-estradial or H-progesterone injected into ovariectomized guinea pigs, were localized in the gingivia to a greater extent than the uterus.

Southren, et al., (1978) provided evidence for specific cystosolic receptors for 5-DHT in human gingival tissue. There was, highly significant increase in the number of binding sites /mg protein in patients with gingival hyperplasia.

Wenk et al, in 1981 found evidence of specific receptors for
estradial in human and experimental animal's gingival tissue\(^{(51)}\).

Lindhe and Bjorn, (1967), showed that estrogen hormones commonly used as contraceptives in women, induce a hormonal situation which to some extent simulates pregnancy. The investigation was done on 115 women aged 18-35 years with excellent general health. The study of the gingival conditions of women before and during 12 months of regular use of contraceptive preparations was followed. Two types of contraceptives were used: Delpregnin and Gestadydral. The mesial aspects of the gingiva of 5 maxillary incisors and canines were examined before beginning the experiment and after 2, 6 and 12 months of hormone therapy. Intracrevicular sampling of the gingival exudate was performed according to Brill (1959). Both the Delpregnin-group and the Gestadydral-group showed an increase in the amount of exudate. There was a gradual increase in exudation during the first six months of hormone therapy. In the group using Gestadydral, no increase in the mount of exudate could be detected during the first six months, whereas a significant increase of the gingival exudate occurred between the last two examinations\(^{(52)}\).

Das, et al, (1971) studied the relationship of oral contraceptives and periodontal disease. They examined a 100 female patients taking oral contraceptives for PLI, GI and pocket depth around representative
teeth (incisors, premolars and molars). An equal number of patients not taking contraceptives was also examined as a control. The authors found that the GI and PLI were significantly higher among patients taking oral contraceptives. The incisors showed a higher GI than either premolars or molars. For pocket depths measurements variable results were obtained. Pocket depths in patients not taking oral contraceptives were in some cases higher than those taking oral contraceptives. They found, that scattered, mild gingivitis was common in patients not taking oral contraceptives, while localized or generalized gingivitis of either moderate or severe nature was common in patients taking oral contraceptives. There was no case of "pregnancy tumor" seen in any patient. Destructive periodontal disease was significantly higher in patients taking oral contraceptives.\(^{(53)}\)

Knight and Wade, (1974) concluded from their study of 89 women aged 17-23 years on oral contraceptives, that those women receiving hormones for more than 1\(\frac{1}{2}\) years exhibited greater periodontal destruction than those of comparable age in the control group. Hence the duration of hormonal therapy seems to affect periodontal attachment levels.

The authors postulated that this difference may be due to an
altered host resistance of the hormone treated group\textsuperscript{(8)}.

discovered that the addition of sex hormones to El Attar, (1976) gingival tissue caused a significant increase in gingival prostaglandin E2.

Since E-type prostaglandins are potent mediators of inflammation, explanation of the mechanism by which sex hormones increase inflammation may be possible\textsuperscript{(54)}.

Kenneth (1978), Investigated whether a particular brand of oral contraceptives and total accumulated exposure to oral contraceptives affects gingival inflammation in human. One hundred and sixty eighty female patients between the ages of 18 to 38 years were evaluated with an oral Debris Index and GI. The patients were divided into groups and subgroups according to their intake of oral contraceptives. They found that the group currently taking oral contraceptives had a higher mean gingival index than the group not taking oral contraceptives. The group taking oral contraceptives revealed a lower mean Oral Debris index than the control group. Further analysis revealed that while some brands of oral contraceptives produce more dramatic index change than others. No relationship appeared to exist due to the different progestrone or estrogen contents in the various brands. Increased accumulative exposure to oral contraceptive apparently had no effect on Oral Debris Index or gingival inflammatory indices \textsuperscript{(55)}.  

Jensen, et al, (1981) in a study aimed to investigate the effects of female sex hormones on subgingival plaque, examined the periodontal status of three groups of women, pregnant, taking oral contraceptives, and non-pregnant, clinically and microbiologically for changes of Fusobacterium species and Bacteroides species. The authors found that overall, the women in the three groups had relatively good gingival health. However, statistically significant increased scores were observed in the gingival index and the gingival crevicular fluid flow in the pregnant group compared to the non-pregnant group. The most dramatic microbial changes were the increased proportions of Bacteroides species both in the pregnant and the group taking oral contraceptives over the non-pregnant group. Increased female sex hormones substituting for the napthaquinone requirement of certain Bacteroides were most likely responsible for this increase. No statistically significant difference in the clinical parameters recorded was noted between the group taking oral contraceptives and the non-pregnant group, although a 16-fold increase in Bacteroides species was observed in the group taking oral contraceptives (31).

Zachariaser, (1993) found that the most common oral manifestation of elevated level of ovarian hormones, as seen in pregnancy and oral contraceptive users, is an increase in gingival inflammation with an
accompanying increase in gingival crevicular exudate. This gingivitis can be avoided or at least minimized by establishing low plaque levels at the beginning of pregnancy as well as the beginning of oral contraceptives therapy. It would appear that bacteria is not solely responsible for the gingivitis seen during these times, nor the ovarian hormone solely responsible for the condition. Data from numerous studies suggest that ovarian hormones alter the microenvironment of the oral bacteria so as to promote their growth leading to a shift in their population. So the most frequent oral manifestation of elevated levels of ovarian hormones is an increase in gingival inflammation with an accompanying increase in crevicular exudate.

Tialkaratne et al, (2000) conducted a study in hormonal contraceptive users as a "risk group" for periodontal disease, due to prolonged, sustained serum levels of oestrogens and progesterone. The study aimed to investigate the effect of hormonal contraceptives on periodontal tissues. The authors examined 32 women using hormonal contraceptives for less than 2 years, and a matched control group of 39 non-users were selected for the study. They were clinically examined for plaque levels and loss of periodontal attachment. The study found that contraceptives users for less than 2 years and 2-4 years duration and non-users had similar oral hygiene levels; yet the contraceptive users had
a significantly higher level of gingival inflammation, compared to the non-users. Usage of hormonal contraceptives for 2-4 years caused a significantly higher LA compared to that of controls. The study concluded that usage of contraceptive preparations containing oestrogen and progesterone resulted in hormonal changes similar to those seen in pregnancy, i.e. associated with increased prevalence of gingivitis. There were significantly higher LA with prolonged usage of hormonal contraceptives, compared with controls (57).

It is now well documented that pregnancy, puberty, menstrual cycle and oral contraceptive usage are associated with transitory, self limiting periods of gingival inflammation.

In Sudan only one study was carried out to investigate the issue of hormonal changes and periodontal diseases. Gismalla and Ghandour in 1998, studied the relationship between prevalence and severity of periodontal diseases among pregnant ladies at different stages of gestation and possible local etiological factors. The study group was 60 pregnant ladies at different stages of pregnancy, on the other hand 60 postpartum ladies were used as a control group. Both study and control groups were subdivided into two groups I and II. Subgroup II received oral hygiene instructions and 0.02% chlorhexidine
mouth wash once daily for eight weeks 20% of both study and control group had sex hormone (follicular stimulating hormone, estradiol) analysis using ELISA method. For all subjects the gingival index (GI), plaque index, probable pocket depth and attachment loss were recorded at days 0, 14, 28 and 56. The results of comparing plague index for study and control groups was statistically significant at day 0. Comparing the mean, plague index for the subgroup of the pregnant not treated and not pregnant not treated no statistically significant difference was found. On the other hand no statistically significant difference was found when the mean plaque index for pregnant treated and not pregnant treated were compared. Concerning the gingival index statistically significant difference was found between the study and control groups at all days. The mean gingival index for pregnant not treated was higher than that of not pregnant not treated subgroup at all days. On the other hand the mean gingival index for pregnant not treated was statistically significant when compared with not pregnant not treated at days 14 and 56. Comparing the mean attachment loss, of pregnant not treated and not pregnant not treated the difference was statistically significant. As for probable pocket depth of pregnant not treated and not pregnant not
treated subgroup indicated that the former showed a higher mean of probable pocket depth.

The results of comparison between mean gingival index and toothbrushing frequency for the pregnant treated subgroup showed no statistically significant difference even with increased toothbrushing frequency. The not pregnant treated subgroup showed reduction of gingival index with increased frequency of toothbrushing. The mean gingival index for the pregnant not treated showed higher reading when compared with that of pregnant treated subgroup.

Compared the mean plaque index and follicular stimulating hormone, estradiol levels for all subgroups statistically significant difference was found. The results of that study supports the earlier findings that mechanical plaque control is the best method of controlling plaque. Also it shows that pregnant ladies are having more gingival inflammation when compared with non-pregnant ladies even at comparable levels of oral hygiene standard. The investigators concluded that the matters needs further investigations.

So the present investigation was designed to study the periodontal health of Sudanese women taking hormonal contraceptives.
Material and Methods:

Following approval of the final protocol for the study, search started for the area where an acceptance sample fulfilling the criteria could be found.

Family planning clinics are found in different areas of Khartoum state, but they are in most cases part of centers for general medical care. Khartoum 2 centre is only for family planning services and it offers service to almost all women in Khartoum province.

The authorities in the center were contacted and a letter introducing the study was written. The management of the center welcomed the idea and staff cooperation was insured. The center was then visited, and the examination of the sample was planned.
Sample:

A group of contraceptive users combined pill (coc) and progesterone injectable every three months (Depoprovera) was randomly selected from women attending the family planning center in Khartoum 2 area during the period from May to August, 2002. A total of one hundred and forty four women aged 20-45 years using combined pills (Marvelon 28) each tablet contains 0.15mg deogestrol and 0.30mg ethyl estradiol (21 tablets and 7 placebo) and progestron only injectable (Depoprovera), containing hydroxy progestin acetate suspension, 150 mg/ml prepared for intramuscular injection deep into the gluteal muscle given once every three months.

The control group was one hundred and thirty three women from the same center aged 20-45 years using intrauterine contraceptive device copper T Model T cu 300 A (Mechanical device without hormones).

Both test and control groups were selected according to the following criteria:

1- Medically fit.

2- Have one child or more.

3- Usage of contraceptive for more than \(1\frac{1}{2}\) year.

In case of control or intrauterine contraceptive device the use is for the same period at least.

Clinical Examination:
For all test and control participants, the examination was done for the following teeth (6 teeth).

<table>
<thead>
<tr>
<th>Tooth Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper left first molar</td>
<td>26</td>
</tr>
<tr>
<td>Upper left central incisor</td>
<td>21</td>
</tr>
<tr>
<td>Upper left first premolar</td>
<td>24</td>
</tr>
<tr>
<td>Lower right first molar</td>
<td>46</td>
</tr>
<tr>
<td>Lower right incisor</td>
<td>41</td>
</tr>
<tr>
<td>Lower left first premolar</td>
<td>34</td>
</tr>
</tbody>
</table>

The examination was done by the same examiner and the observations were recorded in a special form with the help of a trained dental nurse (annex 1).

Both test and control groups were examined using the Plaque Index (PLI) according to Silness and Loe (59), Gingival Index according to Loe and Silness (6), probable pocket depth and attachment loss.

**Plaque Index (PLI) Silness & Loe (1964).**

A mouth mirror and a (University of Michigan-O probe) was used to assess the amount of plaque. The plaque scores were rated 0-3 according to the criteria of the Plaque Index, as follows:
<table>
<thead>
<tr>
<th>Score</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Absence of plaque deposits</td>
</tr>
<tr>
<td>1</td>
<td>Plaque seen after probing the gingival margin.</td>
</tr>
<tr>
<td>2</td>
<td>Visible plaque, seen by the naked eye</td>
</tr>
<tr>
<td>3</td>
<td>Abundance of plaque</td>
</tr>
</tbody>
</table>

The plaque score for the tooth was obtained by totaling the four plaque scores per tooth (on each surface) and the average is taken. The Plaque score per person is obtained by adding the plaque score for all teeth and dividing by the number of teeth examined.

**Gingival Index (GI) Loe & Silness** (6).

The University of Michigan (0) probe was used to assess the gingival index. Each gingival unit (buccal, lingual, mesial and distal of individual teeth is given score from 0-3 according to the criteria of the gingival index. The gingival score for each tooth and subject was then calculated and recorded as the gingival index.
### Score Criteria

<table>
<thead>
<tr>
<th>Score</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Normal gingiva</td>
</tr>
<tr>
<td>1</td>
<td>Mid inflammation mild change in color &amp; texture.</td>
</tr>
<tr>
<td>2</td>
<td>Moderate inflammation with redness, oedema and bleeding on probing</td>
</tr>
<tr>
<td>3</td>
<td>Severe inflammation with oedema, ulceration, and tendency to spontaneous bleeding.</td>
</tr>
</tbody>
</table>

Probable pocket depth and attachment loss:
Pocket depth around each of the six examined teeth was recorded using a graduated periodontal probe (University of Michigan (0) Probe). At the labial surface, the measurements were taken at the middle of the surface, while the mesial and distal pocket measurements were recorded buccally, labially to the contact points.

Efforts were made to direct the probe parallel to the long axis of the tooth. The measurement was done from the gingival margin to bottom of the pocket (Fig V).

Attachment loss for the same teeth was recorded by measuring the distance from the cemento-enamel junction up to the base of the pocket. Pocket depth and attachment loss measurements were recorded simultaneously.

All examinations were recorded with the patient sitting on a portable dental chair using artificial light.
Data was analyzed with the help of statistician the level of significance was predecided at the 5% level (P <0.05, and Confidence Interval at the 95% level).

Results

General Data:

One hundred and forty four contraceptive users (pills and injectable), aged 20-45 years were selected from women using hormonal contraceptives as birth control method at the Family Planning Center At Khartoum (2), as study group. The study group was divided into two sub
groups (OCP & Injectable). One hundred and thirty three loop users from the same age group were selected as a control group.

**Distribution of study and control groups according to age. (Table. 1).**

Table 1 is showing the distribution of the sample according to age the mean age and standard deviations of the study group was 31.4 ± 5.3. years.

The mean age and standard deviation of the control group was 31.9 ± 5.2. years The range of both study and control groups was 20 – 45 years.

Frequency: Distribution of plaque scores among O.C.P. and injection contraceptive users. (Tables. 2and3):

Table 2: Shows the frequency distribution of different plaque scores among oral and injectable contraceptive users. The plaque index for both study and control groups did not show any statistically significant difference as shown by the chi square test (P>. 0.7)- (Table.3).

**Frequency distribution of plaque scores among hormonal contraceptive users and the control groups (loop users). (Tables 4and 5).**
The frequency distribution of different plaque scores among OCP and injectable contraceptive users are shown in table (4). Table (5) shows comparisons of the plaque index which indicates statistically significant difference between the two groups as shown by the chi-square test \( P<0.0025 \).

**Frequency distribution of gingival inflammation among O.C.P and injectable contraceptive users. (Tables 6 and7).**

Table(6) indicates the frequency distribution of the different scores of the gingival index. The results of the gingival index showed that there is no statistically significant difference between O.C.P. and injectable contraceptive users \( P> 0.1 \).

Frequency distribution of gingival inflammation among hormonal contraceptive users and loop users (Tables 8 and 9)

The comparison of the gingival index among hormonal contraceptive users and loop users, indicated statistically highly significant difference between the two groups \( P = 0.00000 \) (table 9).

**Probable pocket depth of O.C.P and injectable contraceptive users (Table 10):**

Table 10 shows the mean and standard deviation of the probable pocket depth among O.C.P and injectable users. There was no
statistically significant difference between the means of the pocket depths of the two groups  (P=0.1).

Probable pocket depth among O.C.P and loop users (Table 11):
Comparing the probable pocket depth of O.C.P and loop uses (control) indicates statistically significant difference between the means of the two groups  P=0.000.

Attachment loss among O.C.P and injectable contraceptive (Table 12):
Table 12 is showing the mean attachment loss of the groups using the O.C.P and injectable contraceptive users. The difference between the means of the two groups was not statistically significant. P=0.607.

Attachment loss among hormonal contraceptive and loop users (Table 13).
The mean attachment loss among women using hormonal contraceptives was highly statistically significant compared to that of
the control group. (Loop users) P=0.000.

Table 1:

**Mean age and standard deviation of hormonal Contraceptive users and Control:**

<table>
<thead>
<tr>
<th>Group</th>
<th>No.</th>
<th>Age Years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study</td>
<td>144</td>
<td>31.4 ± 5.3</td>
</tr>
<tr>
<td>Control</td>
<td>133</td>
<td>31.9 ± 5.2</td>
</tr>
</tbody>
</table>

Table 2:

**Frequency distribution of Plaque scores among O.C.P. and Injectable Contraceptives users:**

<table>
<thead>
<tr>
<th>Criteria (score)</th>
<th>Contraceptive Used</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Oral</td>
</tr>
<tr>
<td></td>
<td>Injectable</td>
</tr>
<tr>
<td>No. (%)</td>
<td>No. (%)</td>
</tr>
</tbody>
</table>
Table. 3:
Comparison of plaque Deposits among O.C.P. and Injection Contraceptive Users:

<table>
<thead>
<tr>
<th>Plaque deposit</th>
<th>Oral Contraception</th>
<th>Injectable Contraception</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. (%)</td>
<td>No. (%)</td>
</tr>
<tr>
<td>Absent</td>
<td>(83)</td>
<td>(84)</td>
</tr>
<tr>
<td>Present</td>
<td>(17)</td>
<td>(16)</td>
</tr>
</tbody>
</table>

\[ \chi^2 = 0.1 \quad P > 0.7 \]

Table. 4:
Frequency distribution of plaque scores among hormonal contraceptive users and control (loop users):
<table>
<thead>
<tr>
<th>Plaque Index</th>
<th>Contraceptive Used</th>
</tr>
</thead>
<tbody>
<tr>
<td>Criteria (score)</td>
<td>Hormonal</td>
</tr>
<tr>
<td></td>
<td>No. (%)</td>
</tr>
<tr>
<td>Absence of deposit</td>
<td>(0)</td>
</tr>
<tr>
<td></td>
<td>(88.4)</td>
</tr>
<tr>
<td>Seen after Probing</td>
<td>(1)</td>
</tr>
<tr>
<td></td>
<td>(11.0)</td>
</tr>
<tr>
<td>Visible Plaque</td>
<td>(2)</td>
</tr>
<tr>
<td></td>
<td>(0.5)</td>
</tr>
<tr>
<td>Abundant Plaque</td>
<td>(3)</td>
</tr>
<tr>
<td></td>
<td>(0.1)</td>
</tr>
</tbody>
</table>

Table 5:
Comparison of plaque deposits among hormonal contraceptive Users and loop users:

<table>
<thead>
<tr>
<th>Plaque deposits</th>
<th>Hormonal users</th>
<th>Loop Users</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. (%)</td>
<td>No (%)</td>
</tr>
<tr>
<td>Absent</td>
<td>(83)</td>
<td>(88)</td>
</tr>
<tr>
<td>Present</td>
<td>(17)</td>
<td>(12)</td>
</tr>
</tbody>
</table>

\[ X^2 = 9.27 \quad P > 0.0025 \]
Table 6:
Frequency distribution of gingival inflammation among O.C.P and Injectable Contraceptives:

<table>
<thead>
<tr>
<th>Gingival Status Criteria (score)</th>
<th>Contraceptive Used</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Oral</td>
</tr>
<tr>
<td></td>
<td>No. (%)</td>
</tr>
<tr>
<td>Normal</td>
<td>(0)</td>
</tr>
<tr>
<td>Mild Inflammation</td>
<td>(1)</td>
</tr>
<tr>
<td>Moderate Inflammation</td>
<td>(2)</td>
</tr>
<tr>
<td>Severe Inflammation</td>
<td>(3)</td>
</tr>
</tbody>
</table>

Table 7:
Comparison of gingival Inflammation among O.C.P and Injectable Contraceptive Users:

<table>
<thead>
<tr>
<th>Gingival status</th>
<th>Oral Contraception</th>
<th>Injectable Contraception</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. (%)</td>
<td>No. (%)</td>
</tr>
</tbody>
</table>
Table 8:
Frequency distribution of gingival inflammation among hormonal contraceptive and loop users:

<table>
<thead>
<tr>
<th>Gingival Status Criteria (score)</th>
<th>Contraceptive Used</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hormonal</td>
</tr>
<tr>
<td></td>
<td>No. (%)</td>
</tr>
<tr>
<td>Normal</td>
<td>(0)</td>
</tr>
<tr>
<td>Mild Inflammation</td>
<td>(1)</td>
</tr>
<tr>
<td>Moderate Inflammation</td>
<td>(2)</td>
</tr>
<tr>
<td>Severe Inflammation</td>
<td>(3)</td>
</tr>
</tbody>
</table>

$X^2 = 2.23 \quad P > 0.1$

Table No. 9:
Comparison of gingival inflammation among hormonal contraceptive and loop users:

<table>
<thead>
<tr>
<th>Gingival Status</th>
<th>Hormonal Users No. (%)</th>
<th>Loop Users No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>(3.8)</td>
<td>(69.7)</td>
</tr>
<tr>
<td>Inflamed</td>
<td>(96.2)</td>
<td>(30.3)</td>
</tr>
</tbody>
</table>

\[X^2 = 786\quad P > 0.00000\]

Table. 10:
The mean and standard deviation of the probable pocket depths for oral and injectable contraceptive users:

<table>
<thead>
<tr>
<th>Contraceptive</th>
<th>No.</th>
<th>(X \pm SD\ MM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral</td>
<td>101</td>
<td>2.4 \pm 0.6</td>
</tr>
<tr>
<td>injectable</td>
<td>43</td>
<td>2.6 \pm 0.8</td>
</tr>
</tbody>
</table>

\[t = 1.635\quad P = 0.104\]
Table. 11:
The mean standard deviation of probable pocket depth among hormonal contraceptives and loop users:

<table>
<thead>
<tr>
<th>Contraceptive</th>
<th>No.</th>
<th>$X \pm SD \text{ mm}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hormonal users</td>
<td>144</td>
<td>2.5 ± 0.7</td>
</tr>
<tr>
<td>Loop users</td>
<td>133</td>
<td>1.9 ± 0.5</td>
</tr>
</tbody>
</table>

t-test = 8.399          p = 0.000

Table. 12:
The mean and standard deviation of attachment loss among OCP and Injectable contraceptive users:

<table>
<thead>
<tr>
<th>Contraceptive</th>
<th>No.</th>
<th>$X \pm SD \text{ mm}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral contraceptive (pills)</td>
<td>101</td>
<td>3.0 ± 1.0</td>
</tr>
<tr>
<td>Injectable contraceptives</td>
<td>43</td>
<td>3.0 ± 0.8</td>
</tr>
</tbody>
</table>

t-test = 0.515          p = 0.607
Table. 13:
The mean and standard deviation attachment loss among hormonal contraceptive and loop users:

<table>
<thead>
<tr>
<th>Contraceptive</th>
<th>No.</th>
<th>X ± SD mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hormonal</td>
<td>144</td>
<td>2.9 ± 1.0</td>
</tr>
<tr>
<td>Loop</td>
<td>133</td>
<td>2.0 ± 0.7</td>
</tr>
</tbody>
</table>

\[ t = 1.635 \quad p = 0.000 \]
Discussion
In this study a sample of 144 taking hormonal contraceptives both oral and injectable has been examined.

The women examined includes almost all those who fulfilled the criteria and arrived for care at the center during the period of study. On the other hand the control group was women of similar age and almost the same socioeconomic class, medically fit and using a mechanical contraceptive device. All confounding factors that may affect periodontal disease had been excluded including general health. The study and control group were almost matched. So any statistically significant differences should only be attributed to the use of hormones and the expected changes that may follow such medication.
1. Plaque index:

Comparing the plaque index among oral and injectable contraceptive users (both hormones) indicated statistically insignificant difference between the two subgroups ($X^2 = 0.1, \text{df}=1, P>0.7$). However, when the hormonal contraceptive users were compared for plaque accumulation with non hormonal control (loop users) (Table 4 & 5), the results indicated statistically highly significant differences between the two groups ($X^2 = 9.27, \text{df}=1, P>0.0025$). These results are consistent with many studies including Loe and Sillness (1963) and Das et al (1971). All those studies indicated increased plaque accumulation among women taking oral contraceptives (hormonal) compared to their matched control group who are not taking hormonal contraceptives. However, none of these studies examined the effects of injectable hormonal contraceptives on plaque accumulation. The results of this study indicated that both oral and injectable hormonal contraceptives have similar effects on plaque accumulation supporting the conclusions that it is the levels of circulating hormones that may have an effect on plaque bacteria this agrees with the conclusions of Jensen et al (1981).

2. Gingival index:
The gingival index of women in the subgroups using oral and injectable contraceptive are presented in (tables 6 and 7). The results indicated statically insignificant differences between the two subgroups. This result of no differences between the two subgroup may be due to the similarities in plaque levels and bacteria or may be a direct effect of the hormones on the inflammatory mediators and hence inflammation pathways as indicated by El-Attar (1976). However when hormonal contraceptives users were compared with non hormonal control group (Tables 8 and 9), there was statistically highly significant difference between the two groups ($X^2=786, df=1, P > 0.00000$).

This finding is in agreement with a number of studies including Lindhe and Anna Lisa Bjorn (1967), Kalkwarf (1978), and Soory et al (1989 and 2000).

All these studies concluded that oral hormonal contraceptives produce similar gingival changes as pregnancy and have been associated with increased prevalence of gingivitis and higher gingival cervicular flow rate. This study indicated that both oral and injectable hormonal contraceptive users have similar effects on gingival health.

However the effect of these hormones on gingival health could be due to an effect on inflammatory pathways as it has been mentioned or the influence of hormonal contraceptives on microbiological parameters in the
gingival sulcus. the contraceptives increase the percentage of anaerobic cultivated flora and higher sulcus – fluid flow, mean more nutrients for bacteria. On the other hand chemotaxis and phagocytosis were reduced by steroid hormones leading to a better growth of bacteria (Gisela et al.). Contraceptives could produce an exaggerated response due to increasing systemic progesterone. The important difference is that while hormone alteration is over in maximum of 9 month with pregnant women, those taking contraceptives their hormone levels were altered for much longer period producing may be more chronic gingival inflammation with greater possibility to proceed to chronic inflammatory periodontitis with pocket formation.

3. Probable pocket depth and attachment loss:

The means and standard deviation of the pocket depth of both oral and injectable hormonal contraceptive users is presented in (table 10). No statistically significant differences between the two subgroups was observed in this study using the t-test (p=0.104). this result is in conformaty with those of plaque and gingivitis. On the other hand there was statistically highly significant differences between the mean probable pocket depth of hormonal and loop contraceptive users (P=0.000). these results are consistent with those of Knight and Wade(1974), Tilakarant et
al (2000), who reported that women who use hormonal contraceptives for more than $1\frac{1}{2}$ years showed significantly higher pocket depth compared to their matched controls. However the attachment loss levels followed the same pattern as the probable pocket depth (Tables 10 and 11).

In general the results of this study clearly indicated that women who are medically fit and using hormonal contraceptives show more plaque accumulation, gingivitis, mean pocket depth and attachment loss than their controls who are using non-hormonal contraceptives mechanical device (loop).

However taking into consideration that bacterial plaque is the main cause of periodontal disease very low levels of plaque should be established and maintained during the period of hormonal contraceptive usage in order to reduce periodontal disease.
Conclusions

Women taking hormonal contraceptives (oral or injectable) show 1. comparable levels of plaque, gingivitis, and probable pocket depth and attachment loss.

Women who are using hormonal contraceptives show high 2. levels of plaque, gingivitis and periodontitis compared to the women who are using non-hormonal contraceptive methods (Loop), and the differences were statistically highly significant.
**Recommendations**

1. Effort should be made to advice women using hormonal contraceptives on meticulous oral hygiene measures in order to minimize periodontal diseases.

2. Oral hygiene services and professional dental cleaning should be part of any setup offering contraceptive services.

3. A detailed study should be carried on the changes in periodontal bacteria and the alterations in circulating hormones during hormonal contraceptives usage, among Sudanese women.
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Fig: (I) Gingival inflammation of upper and lower anterior teeth in C.O.C contraceptive user
Fig (II) Gingival inflammation of the upper anterior

Fig (III): Gingival inflammation the lower anterior
Fig (IV) Bleeding on probing
Fig.(V) : Measurement of pocket depth
Fig (VI) - Gingival inflammation of upper anterior teeth of injectable