

## **Periodontal Disease and its Association with a Greater Risk for Preterm Births and Low Birth Weight Infants in Spain**

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The possible link between periodontal disease and other systemic diseases, such as diabetes, cardiovascular disease or certain pregnancy and delivery complications, has sparked considerable interest in recent years.

Specifically, **preterm birth (PT)** and **low birth weight (LBW)** are two problems that are very important on a medical, social and economic level, and thus, their **possible association with periodontal disease** more than justifies research in this field.

The World Health Organization (WHO) defines **LBW** infants as **those who weigh under 2,500 g**, and **PT** as any delivery that takes place **before gestational week 37**. In developed countries, between 7 and 10 children out of every 100 are born prematurely. For example, in the USA in 2004, 12.5% of the 500,000 infants born that year were PT. This figure has increased by 16% since 1990 and by 30% since 1981, which can largely be attributed to the increase in multiple pregnancies and to an increase in the number of mothers who are 35 years or older (Martin et al, 2005). In Spain, 7-10% of all newborns are considered to be low birth weight infants (Pallás Alonso et al, 2003).

Approximately 75-80% of perinatal deaths occur in fetuses or infants born before week 37, and about 40% of these deaths occur in infants born prior to gestational week 32. Infant mortality and morbidity drop as gestational age increases. PT represents 78% of neonatal deaths in the USA, and is the main cause of perinatal death (death in the first 28 days of life) (Bibby and Stewart, 2004). PT infants, particularly those born very early, can suffer major consequences, both on the short and the long term. The most common diseases in PT children are bronchopulmonary

dysplasia, retinopathy and brain injuries. Moreover, PT children have a higher occurrence of developmental abnormalities than full term infants: motor, sensory, mental and growth-related conditions. (McCormick, 1985).

The aetiology of PT is clearly multifactorial. Risk factors are considered to be primary when they exist prior to pregnancy, or secondary when they develop during the pregnancy. Primary risk factors include: black mother, very young or very old mother, domestic violence, low socioeconomic status, stress or depression, smoking, cocaine or heroin intake, low body mass index, a history of PT, loss of the foetus in the second trimester, previous induced abortion, family history of inflammatory genetic polymorphism, chronic pulmonary disease, chronic hypertension, diabetes or kidney disease. Secondary risk factors include: lack of or inadequate prenatal care, *in vitro* fertilisation, low maternal weight gain in late pregnancy, iron deficiency anaemia; elevated levels of fetal fibronectin,  $\alpha$ -fetoprotein, alkaline phosphatase or stimulating colony factor granulocytes, early contractions, vaginal bleeding in the first or second trimester, bacterial vaginitis (particularly in the early stages of pregnancy), chorioamnionitis, placental abruption, *placenta previa*, hydramnios, preeclampsia and multiple foetuses (Muglia and Katz, 2010).

Adequate prenatal care should reduce the frequency of PT and/or LBW. However, in countries like Spain, where more than 95% of pregnant women receive optimum prenatal care, a decrease in PT has not been observed. Until now, there has been no reasonable explanation for why improved prenatal care has not had the expected impact in this situation, although it is most likely related to its multicausal nature. When some of the factors are controlled, others appear. Specifically, the **association between levels of gingival health and adverse pregnancy outcomes** has been studied in several different countries and populations. Most of these studies show that **inferior periodontal health in pregnant women**, mainly diagnosed with periodontitis, leads to a **higher risk for PT**. Quantification of this association varies

from study to study and from population to population, but could range **between 2.30 and 5.28 times more PT if the mother has periodontitis** (Radnai et al, 2006; Dasanayake et al, 2008). Studies are also being conducted to determine if treating gum infection during pregnancy might reduce the risk for prematurity. For now, this point has not been confirmed, although it has been established that treating these infections is safe for mother and child during the second trimester of pregnancy. This treatment also significantly improves gingival health (Michalowicz et al, 2006; Newnham et al, 2009).

In Spain, several research groups (Moreu et al, Marin et al, Agueda et al, Santacruz et al) have explored this topic, and have published their results in high impact international scientific journals.

In these studies, no **clear association** was observed between periodontal disease and adverse pregnancy outcomes, except for between **the prevalence of PT and maternal periodontitis (OR: 1.77)**, and **between LBW and some parameters of poor periodontal health** (pockets > 3mm, bleeding on probing), which in both cases was limited but statistically significant. Some of these specific risks increased as the pregnancy progressed. In addition, it was observed **that pregnant women may have a high percentage of periodontal disease, including both gingivitis and periodontitis (41.58%)**, associated with a high prevalence of periodontal pathogens.

Based on previous findings, **periodontal diagnosis in pregnant women may be quite relevant**, and protocols for **prevention and treatment of periodontal disease** should be incorporated into the healthcare programs offered to pregnant women throughout their pregnancies.

Although the treatment of periodontitis during pregnancy does not seem to reduce the incidence of PT or LBW, basic periodontal treatment during pregnancy seems safe both for mother and foetus alike.

Further study on the relationship between periodontal disease and PT and/or LBW should be conducted through new observational and interventional studies in our environment to determine whether this association is causal or incidental.

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