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A REVIEW

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Maternal nutrition and prevention of oral clefts

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Each year among 135 million new births in the world, about 3% are affected with major structural birth defects, called congenital abnormalities (CAs) (Andrew *et al.*, 2004). At present the total group of CAs is the major cause of infant mortality and disabilities among children in the industrialized countries. Therefore, the prevention of CAs is an extremely important public health issue. OFCs are among the most common birth defects with varying birth prevalence rates among populations, gender, and geographic region, occurring in 1-2/1000 live births (Vujkovic *et al.*, 2007). Its pathogenesis is multifactorial in that both genetic and lifestyle aspects such as nutrition are involved.

Inadequate maternal nutrition during pregnancy has been suspected as a cause of oral clefts in humans since atleast the early 1900s. In this respect, it is important to address that during pregnancy, specifically during the development of the lip and palate, the embryonic nutritional status is fully dependant on maternal food intake and metabolism. Due to increased needs, inadequate intake, decreased absorption, disturbances in embryonic transfer, or underlying genetic aberrations in the mother or embryo or both, maternal nutritional deficiencies during pregnancy may significantly affect the nutritional status of the embryo and gene expression and other developmental events in specific embryonic tissues (Krapels *et al.*, 2004).

Nutritional intake is also related to socio economic status and the increased frequency of oral facial clefts among the offspring of less educated women emphasizes the importance of maternal nutritional status on reproductive outcome. To date, research on the association between the maternal nutritional status and oral facial cleft has focused mainly on multivitamin and folic acid supplementation.

Neural tube defects (NTD) share similarities in the pathogenesis of oral facial clefts because both birth defects originate from disturbances in which neural crest cells are involved. Therefore, it is conceivable that nutritional factors implicated in NTD pathogenesis also apply to OFC.

At the time of conception and during the first trimester, the mother's nutritional status is important in the development of the lip and palate, as well as other craniofacial structures of the foetus. During this critical stage of development, several key nutrients have been implicated in the development of OFCs, some of which are folic acid, vitamin B-12, vitamin B-6, and zinc (WHO, 2000). Maternal ability to maintain adequate levels of vitamins B6 and B12 and fetal ability to utilize these nutrients are also seen as a factor in the development of oral clefts. When these nutrients are not metabolized properly, errors in DNA synthesis and transcription may occur leading to CAs.

Folic acid:

Folate, as a one-carbon donor, is involved in the biosynthesis of purines and pyrimidines and in homocysteine remethylation producing methyl groups for methylation of DNA, which is important for gene expression (Rooij *et al.*, 2004). The methylenetetrahydrofolate reductase (MTHFR) gene involved in the metabolism of folate is an example of OFC risk modification. The MTHFR enzyme catalyzes the conversion of 5, 10-methylenetetrahydrofolate to 5-methyltetrahydrofolate, an irreversible step, which is the predominant form of folate and the methyl donor for the

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