INTRODUCTION
Vitamin A deficiency (VAD) continues to curtail the lives and hopes of millions of people every year. This is happening more than 75 years after the development of our knowledge about the deficiency and about its treatment. In the words of Dr. Alfred Sommer (1994) of the John Hopkin's School of Hygiene and Public health: *Eduard DeMaeyer always complained that vitamin A deficiency was the "Cindrella" of diseases: nutritionists ignored it as a problem for blindness prevention, while those involved with blindness prevention ignored it as a problem of malnutrition. We now recognize it is appropriately central to all those working to prevent childhood deaths and disability.*

The origins of VAD in childhood are traced to poor vitamin A status of the mother during pregnancy and lactation. A logical approach to the prevention of VAD in the community must seek to address this basic cause. Pregnancy outcomes in women with low vitamin A status has been extensively studied although no link has been established between vitamin A deficient status and partial molar pregnancy (Berkowitz et al, 1995), premature rupture of membranes (Westney et al, 1994) or eclampsia (Ziari et al, 1996). Sharma et al (1996) reported lower vitamin A levels in placental abruption pregnancies than in normal pregnancies, but no cause-and-effect relation was established. Vitamin A and related compounds are transferred from mother to fetus via the placenta. Studies in animals reveal that this transfer is sensitive to maternal vitamin A intake, both chronic and acute (Rasmussen, 1997). It is postulated that growing fetus is preferentially supplied with available vitamin A in case of the maternal deficiency, as evident from the normal range of cord blood values (Venkatachalam et al, 1962). Further research is required in this subject of feto-placental transfer of the vitamin A. Circulating retinol values in mature newborns are always 50% lower than those in the mother, and fetal concentration of $\approx 1\mu$mol/l (286$\mu$g/l) are frequent and should not be considered indicative of a deficient status at this stage (Godel et al, 1996; Tamai et al, 1996). The situation is more critical for premature deliveries, because both serum and hepatic vitamin A concentration can be very low and may pose a direct threat to the child's health. Although vitamin A supplementation can be used, its ability to prevent and reduce lung injury such as bronchopulmonary dysplasia is still controversial (Shenai et al, 1987; ICGPD, 1996).

The fetus starts to accumulate vitamin A during the third trimester of pregnancy, and needs several months of sufficient intake after birth to build up an adequate hepatic store. In many countries, babies are breast-fed, in which case the vitamin A content of the breast
milk is influenced by the vitamin A status and serum concentrations of the mother during the last trimester of pregnancy (Ortega et al, 1997; Martinez et al, 1997). Blood vitamin A concentrations decline gradually in pregnancy due to inadequate dietary vitamin A intake especially in poor communities (Wallingford and Underwood, 1986; Panth et al, 1990). Colostrum and early milk are extremely rich in vitamin A, and even the milk of a mildly undernourished women may meet the physiological needs of the newborn during the first weeks (West et al, 1986; Humphrey et al, 1992; West et al, 1989; Fawazi et al, 1995). After this time, however, a rapidly growing infant may exhibit negative vitamin A balance, with severe consequences on health (Veronique Azais-Braesco and Gerard Pascal, 2000).

An adequate vitamin A status, one that is neither too low nor too high, is needed for harmonious fetal and child development (Veronique Azais-Braesco and Gerard Pascal, 2000). The WHO (1998) recommends that a daily vitamin A supplement taken during any part of the fertile period be limited to 10,000 IU (3000 RE). The Teratology Society of the US (1987) recommends that vitamin A supplements or total intake not exceed 8000 IU/day (2400 RE/d). In areas of endemic VAD, the benefit that pregnant women or their children may derive from vitamin A supplementation outweighs the potential risk of teratogenesis (Veronique Azais-Braesco and Gerard Pascal, 2000). Underwood (1994) has discussed maternal vitamin A supplementation at physiological doses as potential strategy to improve infant and child vitamin A status in developing countries. According to her, where health care infrastructures make contact with women during pregnancy and within the first month of postpartum, the potential exists for improving their status through diet and/or high dose supplementation. The preferred intervention is through a diet that provides a safe concentration of intake throughout pregnancy and lactation. Use of supplement during pregnancy presents a logistic problem in most developing countries because it is only safe to give a near physiological dose daily and only few health systems have such frequent contacts with mothers. Moreover, physiological doses do not require strict supervision like medical doses. Late in pregnancy, a vitamin A deficient mother may still obtain benefits from low-dose vitamin supplementation. Her status will improve and, in turn, influence to some extent her newborn's reserves at parturition (Underwood, 1994). Low doses (0.2-8.4 µmol, equivalent to about 200-8000 IU) of vitamin A given to
pregnant women and lactating women increases serum and breast milk retinol (Rasmussen, 1997).

Today, vitamin A supplementation is the most efficient way of correcting VAD. Dark green leafy vegetables (DGLV) and yellow-orange fruits (YOF) are the conventional inexpensive sources of betacarotene in Indian diets. They not only supply carotenoids, but also other nutrients, which can also contribute in some measure to better nutrition. Doubts have been raised about the effectiveness of carotene-containing foods in improving the vitamin A status of populations at risk. However, various workers in India and other countries (Nageswara and Narasing Rao, 1967; Lala and Reddy, 1976; Murthy et al, 1976; Devadas and Murthy, 1978; Devadas et al, 1978; Devadas et al, 1980; Hussain and El-Tohamy, 1988, Hussain and El-Tohamy, 1990; Annapurna et al, 1991; Bulux et al, 1994, Solomons, 1999; Ncube et al, 2001; Subbulakshmi, 2001) have demonstrated availability of plant carotenes to humans. More recently, stable-isotopically-labeled beta-carotene have been used to assess bioconversion in humans. The efficiency of the bioconversion of beta-carotene to vitamin A has been accepted to be six but this value may vary depending on vitamin A status and the amount of beta-carotene consumed (Nestel and Trumbo, 1999). However, in view of the shortfall in the DGLV and YOF production, there is an immediate need to simultaneously identify and exploit less conventional, but more potent food sources, of betacarotene. Two such additional food sources with great potential are the red palm oil and blue green algae, spirulina (Bamji and Rukmini, 1995).

The natives of Mexico, China, Japan, and Central Africa where spirulina grows naturally in lakes have used it as human foods for centuries. It has been approved as a food for human consumption by FAO and is cultivated in around 70 countries (Chamorro-Cevallos, 1980). It has a unique blend of nutrients that no single plant source can provide. It has a high amount of protein superior to plant proteins and comparable to milk proteins. It is an excellent source of many vitamins including β-carotene, riboflavin, thiamin, vitamin B₁₂, folic acid, vitamin E, and biotin. It is also a rich source of various minerals such as iron, calcium, phosphorus, magnesium etc. The natural β-carotene of spirulina is different from the synthetic β-carotene since it contains high percentage of 9-cis isomer compared to over 47% of all trans in synthetic form.
A thorough scan of the available literature reveals that extensive investigations have been carried out in the field of childhood VAD and supplementation with massive doses of synthetic vitamin A. Some data exist on prophylactic synthetic vitamin A supplementation for lactating mothers. Its major drawback in applying the same for pregnant women is the potential risk of teratogenesis. Having realized the poor vitamin A nutriture during pregnancy and lactation and its pernicious implications in childhood blindness with no scope to improve the status with synthetic vitamin A at pharmacological level, the only option is to consider using vitamin A at physiological levels. Interesting attempts have been made to replace vitamin A with provitamin β-carotene, which has never been associated with any teratogenic risk (Carlier et al, 1993). Data from India indicate that, unlike anemia, vitamin A deficiency in pregnancy is not associated with increased maternal morbidity and mortality. There is at this time, therefore, no program for universal supplementation to pregnant women (WHO, 1997a). Maternal daily supplementation at physiological doses has been speculated to have the similar effect as that of with single massive doses (Rasmussen, 1997; Underwood, 1994). However, there are insufficient studies to support this assumption. Moreover, the need for "multi-nutrient supplementation" is being increasingly felt for vulnerable populations such as that of pregnant and lactating women who are at risk of multiple-micronutrient deficiencies. In this regard, spirulina-a cyanobacterium holds a promise as supplementary food due to its exceptionally high betacarotene content and other major macro & micronutrients such as protein, iron, zinc etc. Although pregnant and lactating women in Central Africa and America have used spirulina for centuries as foodstuff, no scientific studies have been conducted to see the actual effect of its inclusion in diets. Kapoor and Mehta (1992) have studied beneficial effects of spirulina supplementation on milk volume and protein and fat contents of milk of lactating rats. However, effect on the vitamin A content has not been studied. Moreover, there are no studies conducted on spirulina supplementation and vitamin A status during human pregnancy and lactation. Preliminary research on effect of spirulina supplementation on serum retinol during pregnancy by the present author has given encouraging results.
The purpose of the present study, therefore, was to contribute to the field of vitamin A during pregnancy and lactation with the following specific objectives in mind:

1. To analyze spirulina for nutritional parameters

2. To estimate the dietary intake of pregnant and lactating women with specific reference to energy, proteins, vitamin A, iron and fat

3. To assess the serum total protein, hemoglobin, serum retinol and serum betacarotene of pregnant and lactating women

4. To conduct a well monitored spirulina supplementation study during pregnancy and lactation

5. To study the post-supplemental effect on vitamin A status during pregnancy and lactation by analyzing vitamin A content in maternal serum and breast milk

6. To investigate the effect of spirulina supplementation during pregnancy on fetal serum retinol and betacarotene levels and,

7. To evaluate the effect of spirulina supplementation on fetoplacental function (progesterone) during pregnancy