INTRODUCTION
Complement plays an integral role in host defense against infection. Evidence for this point of view comes from the observation that recurrent infection occurs in individuals born with deficiency of certain complement cascade and from careful analyses of complement function in vitro, showing that this system is required for many antimicrobial processes. The protective function of complement appears to be directed primarily against extracellular pathways namely the pyogenic and enteric bacteria, which are pathogenic because they resist phagocytosis. Antibody and complement through the process of opsonization overcome the resistance, with the possible exception of viruses (Mills and Cooper, 1978). Complement however does not play an important role in resistance of infection by intracellular parasites.

It is difficult to define complement system. Many denote complement as an auxiliary mechanism involved exclusively in activity of antibody or solely as an effector mechanism during inflammation.

Complement represents a biological system involved with the entire immune process and its major role may well be to modulate and regulate a large portion of that response.

The core of this system consists of at least 12 separate and distinct serum proteins. In addition,
there are a number of other serum proteins which are critical to the activation and modulation of these basic components. It was reported by Spitzer (1977) that all the proteins under consideration exist in plasma in an inactive or active state. When complement activity is initiated these proteins interact in a sequential orderly fashion.

The activity of one protein is dependent on its predecessor and often determines the fate of next component in sequence. The term cascade has frequently been applied to these interactions which seem to be a useful designation.

Complement is activated through two major pathways. The classical pathways is initiated by antigen antibody interaction or by a complex of C-polysaccharide and C-reactive protein . Components in the sequence are designated as C-142356789 and the alternate pathway in the order of activator (Antibody) - properdin system - C-356789.

The result of activation of either pathway is the fixation of C₃b and thereby opsonization of invading bacteria. Activation of either pathway also results in the release of C₃a and C₅a which serve as major chemotactic factor, and the fixation of late acting components to the organism which induce their lysis.

The concentration of C₃, C₄ and C₅ are lower in newborns than in adults and the adult levels are achieved at 3-6 months of age (Fireman et al, 1969). Similarly Stosselet et al (.1973), Feinstein and Kaplan (1975) and
Adamkin et al (1978) have reported defective opsonization and low level of $C_3$, $C_5$, properdin and component B in cord serum.

Ballow et al (1974) demonstrated deficiency of all nine complement components in cord sera relative to maternal serum. At 4th day of age, $C_{1q}$, $C_1$, $C_2$, $C_4$ and $C_7$ levels were increased markedly to maternal levels. $C_3$ was the only component which exhibited no significant change between birth and at 4th day of age. A marked deficiency of serum levels of $C_8$ and particularly of $C_9$ was evident at birth.

According to Johnston et al (1978) whole complement hemolytic activity also appears to be subnormal in approximately half of term infants with mean activity being about 70 to 90% of normal. Similarly they reported that the concentration of $C_{1q}$, $C_4$, $C_2$, $C_3$ and $C_7$ was 60% to 100% of adult concentration in term infants and somewhat less in preterm infants.

The fact that preterm and term infants are also deficient in $CH_{50}$ and complement component during the first month of life raises the possibility that the neonates might have impaired complement dependent biological functions, which accounts for the impaired resistance to infection especially in preterm babies.

It is in the light of these observations that the present venture is directed to study the complement
cascade in both mothers and their babies and to assess the concentration of these complement components in relation to birth weight and gestational age.