Prolactin is a polypeptide hormone of pituitary origin and induces pronounced physiological effects on growth, reproduction and osmoregulation in a number of species [1]. A variety of mammalian cells possess receptors for prolactin, including those of the mammary gland, liver, kidney, brain, prostate, testis and ovary [2]. Data is accumulating to suggest that prolactin may have a physiologic role in the regulation of humoral and cell-mediated immune responses. Specific prolactin receptors have been identified on human T and B lymphocytes [2-6]. However an exhaustive literature search revealed scant information [5] regarding the effects of prolactin on monocytes and neutrophils.

The present study was conceived to explore whether prolactin had any effect on the phagocytic leucocytes of peripheral blood, i.e. neutrophils and monocytes. This study was approved by the Institution’s Ethics Committee. Twenty milliliter blood was collected into heparinised tubes from the antecubital veins of 10 healthy, non-smoking, male volunteers after obtaining their written informed consent. The blood was layered on Ficoll-Hypaque (Sigma). Monocytes and neutrophils were isolated by the method of Boyum [7]. The monocyte count was adjusted to 6×10⁵ cells per aliquot. Each aliquot was incubated for 90 min with a suspension of Candida albicans (1 × 10⁶ organisms) and 0, 5, 10 or 20 µg of prolactin (Sigma). Care was taken to keep the total volume of suspension constant while incubating with different prolactin concentrations. Monocyte phagocytosis and fungicidal capacity were assessed using the modified method of Lehrer and Cline [8, 9]. The phagocytic capacity of neutrophils was studied using Staphylococcus aureus by the method of Wilkinson [10], where 1 × 10⁵ neutrophils were incubated with 1 × 10⁶ S. aureus and 0, 5, 10 or 20 µg of prolactin for 60 min. The results (expressed as mean ± SE) were analysed using Student’s t test.

The phagocytosis of neutrophils in the control aliquot was 31 ± 0.93%. When exposed to 5 µg of prolactin, it remained 32.57 ± 1.43%. A statistically significant increase in phagocytosis was obtained at prolactin concentrations of 10 µg (36.7 ± 0.93%, p < 0.001), 5, 10 or 20 µg of prolactin for 60 min. The results (expressed as mean ± SE) were analysed using Student’s t test.

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10 µg (p < 0.001) and 37.9 ± 1.18% at 20 µg of prolactin (p < 0.001). The fungicidal activity also increased from 47.21 ± 0.92% in the control cells to 50.22 ± 1.43% (NS) at 5 µg, 54.85 ± 1.15% (p < 0.001) at 10 µg and 58.37 ± 0.81% (p < 0.001) at 20 µg of prolactin.

This study shows that prolactin, a hormone only recently recognised to have immunostimulant properties [2-6] stimulates both neutrophils and monocytes in a dose-dependent fashion. Although prolactin receptors have been found on T and B lymphocytes [4-6] and monocytes [5], no studies have looked for the effects of prolactin on neutrophils. Our study suggests the possibility of existence of such receptors on neutrophils.

The phagocytic function of neutrophils and monocytes is expressed as the number of cells phagocytosing phils incubated with 10 µg prolactin, the number of phagocytosing cells per 100 cells counted increased from 31 ± 0.93 to 36.7 ± 0.93, which is an increase of 18.39%. Similarly for neutrophils incubated with 20 µg of prolactin, the increase was from 31 ± 0.93 to 38.85 ± 0.55 signifying an increase of 25.32%. In case of monocytes, phagocytosis increased by 15.2, 32.25, and 37.32% with 5, 10, and 20 µg of prolactin, respectively. Intracellular fungicidal capacity also increased by 16.18 and 23.64% with 10- and 20-µg concentrations of prolactin, respectively. Such a magnitude of increase in neutrophil and monocyte function is known to occur with bacterial infections [11], indicating biological significance. Hence, it is evident from our study that prolactin significantly affects the phagocytosing leucocytes in peripheral blood. This may have important implications in conditions where prolactin is naturally raised (pregnancy and lactation) or when its action is altered (patients receiving cyclosporin treatment).

References


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