INTRODUCTION AND AIM OF THE WORK

INTRODUCTION

Jaundice is the most frequent clinical problem in the neonatal period (**Koops and Battaglia, 1987**), being the visible manifestation of chemical hyperbilirubinemia (**Oski, 1991**).

The association beteveen breast milk feeds and neonatal hyperbilirubinemia was described in the mid sixty (**Gartner and Arias, 1966**).

Hyperbilirubinemia is observed more frequently in infants of diabetic mothers (IDMs) than those of non diabetic mothers. Although a number of hypotheses have been suggested, the pathogenesis remain uncertian (**Gowett and Schwartz, 1982**).

Neonatal intestinal beta-glucuronidase (βG) could cleave the ester linkage of bilirubin glucuronide, thus producing unconjugated bilirubin for intestinal absorption in the entrohepatic circulation causing increased serum indirect bilirubin (**Poland and Odell, 1971**).

In 1986 Gourley and Arend reported that the βG present in maternal milk is an important factor in breast milk jaundice. In aprevious report, higher values of serum βG are found in pregnant diabetic women in comparison with non diabetic women (**Ferra, et al., 1973**).

In the present study we have examined the hypothesis that the increased concentration of breast milk βG in diabetic mothers may be an additional cause of hyperbilirubinemia in their breast-fed infants.

AIM OF THE WORK

The aim of this work is to measure the level of β-glucuronidase concentration in the serum and milk of diadetic mothers and its relation to the degree of early neonatal indirect hyperbilirubinemia.