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شبكة المعلومات الجامعية
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شبكة المعلومات الجامعية التوثيق الالكتروني والميكروفيلم



شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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CANCEL

**MATERNAL PLASMA CORTICOTROPHIN-RELEASING
FACTOR (CRF) IN PRETERM LABOR**

Thesis

**Submitted for partial fulfillment of master degree in
Obstetrics and Gynecology.**

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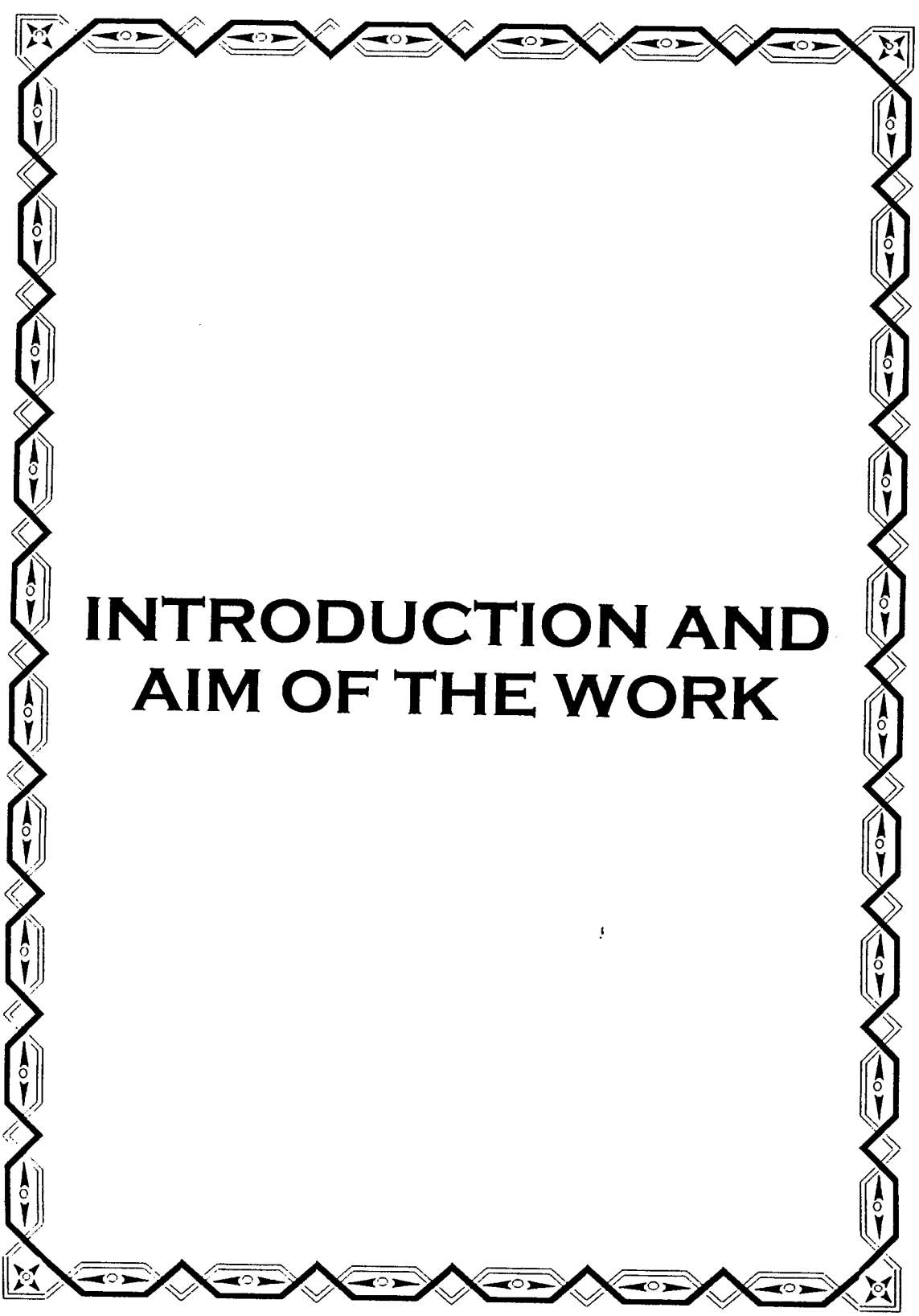
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INTRODUCTION AND AIM OF THE WORK



INTRODUCTION

An untimely birth is one of the major health hazards of human. Preterm birth is the greatest cause of neonatal morbidity and mortality; furthermore, the sequel of an untimely birth, for those infants who survive, often cause lifelong disabilities. A large number of permanently institutionalized persons are mentally or physically impaired because of an untimely birth.

Long mental and physical impairment is surely one of the greatest tragedies that can beset a person, his or her family, society, and even the economics of the world. An untimely birth may portend grave and horrendous impositions on the most innocent and vulnerable member to our society: the newborn. (*Cunningham, et al., 1993*).

Although preterm delivery is the leading cause of perinatal morbidity and mortality worldwide, the diagnosis and appropriate treatment of preterm labor remains an unresolved clinical problem. (*Roberts et al., 1990*). A growing body of evidence supports the view that preterm labor is a syndrome with multiple causes, (*Arias F, 1993*).

Corticotrophin releasing factor (CRF), a 41 amino acid hypothalamic neuropeptide, plays a major role in regulating

pituitary- adrenal function and the physiologic response to stress. (*Chrousos , 1992*).

During pregnancy, placenta, decidua and fetal membranes are additional sites of CRF synthesis from approximately 8 to 10 weeks' gestation onward. The expression of placental CRF rises exponentially during gestation, and it is released into maternal, fetal and amniotic compartments. (*Goland, et al1988*).

Placental CRF is identical to hypothalamic CRF in structure, immunoreactivity and bioactivity. However, there is one crucial difference in regulation between hypothalamic and placental CRF. In contrast to the negative control on hypothalamic CRF, glucocorticoids stimulates the expression of human CRF messenger ribonucleic acid in the placenta, establishing a positive feedback loop that results in elevated levels CRF, ACTH and cortisone during pregnancy. (*Wadhwa, et al., 1998*).

CRF may be implicated in the timing of delivery in at least two ways. First, Placental CRF may play a direct role in the physiologic characteristics of human parturition, Second, placental CRF may be a marker of antepartum risk conditions for preterm labour and an indirect predictor of