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# شبكة المعلومات الجامعية التوثيق الالكتروني والميكروفيلم



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# جامعة عين شمس

## التوثيق الإلكتروني والميكروفيلم

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# بعض الوثائق الأصلية تالفة



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بالرسالة صفحات

لم ترد بالأصل



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# *Plasma Fibronectin in Preeclampsia and Intrauterine Growth Retardation*

Thesis

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B 10444

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

اقْرَأْ بِاسْمِ رَبِّكَ الَّذِي خَلَقَ

خَلَقَ الْإِنْسَانَ مِنْ عَلَقٍ

اقْرَأْ وَرَبُّكَ الْأَكْبَرُ الَّذِي عَلَّمَ الْقُرْآنَ

عَلَّمَ الْإِنْسَانَ مَا لَمْ يَعْلَمْ

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# INTRODUCTION

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## INTRODUCTION

Fibronectins are large, usually dimeric glycoproteins (molecular weight approximately 450,000D), that are found both in a soluble form in plasma (plasma fibronectin) and in a larger non soluble form in the extracellular matrix (cellular fibronectin) (Moses et al., 1980, Rouslahti et al., 1988).

Plasma fibronectin is synthesized primarily by hepatocytes (Tamkun et al., 1983), and is involved in opsonic activities with the reticuloendothelial system and in clot stabilization (Schwarzbeur et al., 1984). Cellular fibronectin synthesized by fibroblast, endothelial cells, and astroglial cells among others, is involved in cell adhesion, migration, growth and differentiation (Rouslati et al., 1973). The various different fibronectins are derived from a single fibronectin gene and are composed of multiple domains, the sequence and the presence or absence of the various domains accounts for the different functional activities of different fibronectins (Moses et al., 1980).

There are at least 12 versions of fibronectin. although the fibronectin gene itself does not vary (Hynes et al., 1986). Different cells types (hepatic, fibroblast, and



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endothelial) may synthesize different fibronectin variants, one of these variants is a soluble plasma fibronectin containing an extra type III domain (ED I+), found predominantly in large vessel endothelial cells (Vartio et al., 1987), and in very small quantities in platelets (Paul et al., 1986). A growing body of evidence indicates that endothelial injury or activation is an important pathophysiologic feature of pre-eclampsia (Friedman et al., 1991). The Cellular iso form of fibronectin (cFN), is present in endothelial cells and endothelial matrix in vivo (Vartio et al., 1987), and is elevated in clinical situations characterized by substantial endothelial injury, such as vasculitis, sepsis and crush injury (Rouslahti et al., 1988). The pathogenesis of this endothelial injury is poorly understood, but appears to be related to antecedent trophoblastic hypoperfusion (Mosses et al., 1980). Impaired trophoblastic invasion of maternal spiral arteriols (which presumably results in reduced trophoblastic perfusion), has been observed in pregnancies complicated by both preeclampsia and intrauterine growth retardation (IUGR) (Rouslahti et al., 1973). Data demonstrating the presence or absence of endothelial injury in pregnancies with IUGR are limited.

# AIM OF THE WORK

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## AIM OF THE WORK

The aim of this study, is to determine the presence and the degree of endothelial injury, by measuring plasma concentrations of fibronectin, in pregnancies complicated by preclampsia and/or intrauterine growth retardation.



# REVIEW OF LITERATURE