

**Study of the Expression of Estrogen,
Androgen and Glucocorticoid Receptors
in Pregnant and Non Pregnant Females
with Recent Striae Distensae**

Thesis

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قالوا

سبحانك لا علم لنا
إلا ما علمتنا إنك أنت
العليم العظيم

صدق الله العظيم

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List of Abbreviations

Abbrev.

ACE	3-amino -9-ethyl carbozle
AF1	Activation function1
AF2	Activation function2
AP1	Activation protein 1
AR	Androgen receptor
BMI	Body mass index
DAB	Diaminobenzadine
DBD	DNA binding domain
DEJ	Dermo-epidermal junction
DHEA	Dehydroepiandrosterone
5α-DHT	5α-Dihydrotestosterone
ECM	Extracellular matrix
ER	Estrogen receptor
ERE	Estrogen response element
ERα	Estrogen receptor alpha
ERβ	Estrogen receptor beta
GA	Glycolic acid
GCS	Glucocorticoids
GR	Glucocorticoid receptor
GRE	Glucocorticoid response element
HBD	Hormone binding domain
H. & E.	Hematoxylin and Eosin

HsPs	Heat shock protein
K	Keratin
KDa	Kilo Dalton
LBD	Ligand binding domain
MAPK	Mitogen-activated protein kinase
MMPS	Matrix metalloproteinases
mRNA	Messenger ribonucleic acid
MG	multigravida
Nd:YAG	Neodymium Yttrium Aluminium Garnet laser
NS	Non- significant
NTD	N-terminal domain
NG	nulligravida
P	Phosphoralation
PBS	Phosphate puffered saline
PDL	Pulsed dye laser
SD	Striae distensae
T	Testosterone
TCA	Trichlor-acetic acid
TIMPS	Tissue inhibitors of metalloproteinases
UVA	Ultra violet A
UVB	Ultra violet B

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I- Introduction

Striae distensae are well defined, linear atrophic skin lesions secondary to connective tissue abnormalities (*Cambazard and Michel, 2006*).

The commonest sites are the outer aspect of the thighs and lumbosacral region in males, and thighs, upper arms, buttocks and breasts in females. In the early stages, striae may appear pink to red (striae rubra), which over time become atrophic and attain white color (striae alba) (*Burrow and Lovell, 2004*). They can also be distinguished into four distinct types; namely, striae alba, striae rubra, striae caeruleae, and striae nigra. Melanin pigmentary system may have a role in various colors of striae distensae (*Hermanns and Piérard, 2006*).

Multiple treatment modalities are available including tretinoin, glycolic acid, pulsed dye laser, CO2 laser, Intense pulsed laser, excimer laser, and others (*Elsaie et al., 2009*).

The pathogenesis is still unknown, but probably relates to changes in the structures that provide skin with its tensile strength and elasticity. Such structures include components of the extracellular matrix (ECM), like fibrillin, elastin, and collagen (*Watson et al., 1998; Thomas and Liston, 2004*).

They may be caused through loss of fibroblast synthesis capability and abnormalities to connective tissue, in addition to significantly decreased collagen, elastin, and fibrilin fibers. They may develop as an end result of various physiologic states, including pregnancy, adrenocortical excess and changes in body habitus, as seen in rapid weight change. A genetic predisposition is also presumed (*Viennet et al., 2005; cambazard and Michel, 2006*).

Striae are seen in 90% of pregnant women due to a combination of hormonal factors along with increased lateral stress on connective tissue (*Lawley and Yancey, 2003*). The action of estrogens in the skin is well-known to increase the thickness and elastic fibers in the papillary layer (*Punnonen et al., 2003*), increase of dermal collagen (*Sauerbronn et al., 2000*), interfere in the mechanism of wound repair and extracellular matrix reorganization (*Zecchin et al., 2005*), and participate with androgens in skin homeostasis (*Mills et al., 2005; Gilliver et al., 2003*).

Under the physiologic conditions, glucocorticoids may regulate the synthesis of glycosaminoglycans (*Smith, 1988*), while most relevant adverse effect of glucocorticoid therapy is skin atrophy through suppression of cutaneous cell proliferation and protein synthesis, which concerns keratinocytes as well as

dermal fibroblasts resulting in depressed collagen turn over. They may also intervene in regulation of proinflammatory cytokines, growth factors, matrix proteins, and matrix proteases which have impact on wound healing (*Schäcke et al., 2002*).

Some hormones, like estrogen, relaxin, and adrenocortical hormones are postulated to decrease the adhesiveness between collagen fibers and increase ground substance, which results in the formation of striae in areas of stretching (*Thomas and Liston, 2004*).

Aim of the work

The aim of this thesis is to study the expression of estrogen, androgen and glucocorticoid receptors in recent strain distensae in pregnant as well as non pregnant females to explore the proposed role of hormonal factor in the development of strain.