INTRODUCTION

Striae distensae (SD) or (Stretch Marks) are a common disfiguring skin disorder of significant cosmetic concern **(Mc Daniel, 2002).** Striae distensae are well defined, linear atrophic skin lesions and occure secondary to connective tissue abnormalities **(Cambazard and Michel,2006).**

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).**

Striae distensae are often found on the abdomen, thighs, buttocks, breasts, and sacro-lumbar region, and are often associated with the topical (**Kirtschig and Neve, 2006)** and systemic **(Cordeiro and Moraes, 2008)** use of corticosteroids, adolescence, pregnancy, infectious processes such as tuberculosis and typhoid fever, a rapid gain or loss of weight, Marfan and Cushing syndromes **,**tissue expansion **(Cho et al., 2006)** augmentation mammoplasty **(Mahhabir andPeterson 2001)** and tension-requiring skin sutures**.**Hence, their origin is multifactorial and, despite several studies, their physiopathogenic mechanisms are not entirely understood**(Raquel et al.,2010).**

Some authors believe that SD result from an initial inflammatory reaction that destroys collagen and elastic fibers, followed by the regeneration of collagen and elastic fibers in the direction imposed by mechanical forces.

Lesions appear as a result of the stretching and rupture of rigid cross-linked collagen and ‘‘elastic’’ unlinked collagen, thus permitting a limited degree of stretch and limited intradermal rupture **(Hibah et al., 2007).**

The inflammatory condition that appears seems to be an unresolved question; however the pathogenesis of SD 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), including fibrillin, elastin and collagen**(Raquel et al.,2010).**

Currently, the etiologic mechanisms involved in SD are usually classified under the headings of ‘‘Genetic Predisposition’’, ‘‘Biochemical or Hormonal Disorders’’ and ‘‘Mechanical Disorders’’.Studies have indicated the interference of estrogen in the mechanism of wound repair and extracellular matrix reorganization **(Zecchin et al., 2005)**and the participation of estrogens **(Mills et al., 2005)** and androgens **(Gilliver et al., 2006)**in skin homeostasis. It is also known that, under physiologic conditions, glucocorticoids regulate the synthesis of glycosaminoglycans in skin fibroblast culture**.** Moreover,topical corticosteroids reduce collagen synthesis and induce skin atrophy**(Raquel et al.,2010).**