INTRODUCTION

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Uterine leiomyoma is the most common benign smooth muscle tumor of the myometrium occurring as many as 30% of women over 35 years of age ***(Vollenhoven et al., 1990)***.

Up to 50% of women with leiomyomas complain of myoma-related symptoms, including pelvic discomfort, abnormal uterine bleeding, spontaneous abortion, and urinary frequency ***(Buttram et al., 1981)***.

Although the nature of the initial event is unknown, a role for ovarian steroid hormones in the growth of uterine leiomyomas is likely, because these tumors grow during the reproductive years,***(Marshall et al.,1997)*** increase in size during pregnancy and regress after menopause ***(Muram et al., 1980)***.

Furthermore, treatment with gonadotropin-releasing hormone (GnRH) agonists, which reduces ovarian hormone production, leads to a reduction in size of leiomyomas, but enlargement of leiomyomas occurs after therapy with GnRH agonists is discontinued ***(west et al., 1987)***. These findings suggest that leiomyoma growth is dependent on ovarian steroids.

The action of estrogen may be mediated in part by local growth factors, such as epidermal growth factor (EGF) and insulin-like growth factor 1(IGF-1), produced by the target cells ***(Nelson et al., 1992)***.

However, the mechanisms of action of ovarian steroids in the regulation of leiomyoma growth are not well defined yet ***(Andersen et al.,1995).***

It is possible that in tumors the death pathway may be suppressed extending the lives of the cells ***(Marx, 1993)***. Actually, apoptosis or programmed cell death is known to occur in tumors either spontaneously or in response to treatment.

Recent research efforts have focused on the function of protooncogene and tumor suppressor gene products in directing cell fate, interest has centered around the role of bcl-2 in controlling the survival and death of cells, data now support a role for bcl-2 protein as a cell death repressor ***(Yang et al., 1996).***

It is now evident that the bcl-2 protooncogene encodes a 26-KDa protein, localized to mitochondrial and perinuclear membranes, the product of the bcl-2 gene, when elevated in cells either in vivo or in vitro, prevents the normal course of apoptotic cell death in a variety of cells induced by tropic factor deprivation or other stimuli without altering proliferation ***(Reed, 1994).***

Reed, (1994) reported that in addition to extending the life span of certain cells, bcl-2 protein can promote cell replication by reducing the requirements for growth factors.