Introduction

Introduction

Cancer of uterine corpus is considered the commonest malignancy affecting female genital tract. Any approach to the control of endometrial cancers must include a consideration of the individuals at high risk. Endometrial hyperplasia has been found to be predispose to endometrial carcinoma ***(Disia and Creasman, 1993).*** Several investigators have provided well documented evidence that cystic, adenomatous and a typical hyperplasia if left untreated may ultimately progress to invasive cancer ***(Kistner, 1990)***.

Maintenance of homeostasis of tissue dynamics requires a balance between cell proliferation and apoptosis or programmed cell death. A variety of genes are considered to play an important role in caricinogenesis by blocking apoptosis ***(Nakamura et al., 1995).***

Because neoplastic growth occurs as a result of breakdown of the regulation of tissue dynamics an analysis of tumors from a

standpoint of cell proliferation and cell death may be important to evaluate the growth rate ***(Nakamura et al., 1997).***

The B-cell leukemia / Lymphoma –2 (bcl-2) gene, located at chromosome 18q21 was first identified at the breakdwon of the t (14;18) translocation found in the majority of follicular lymphomas and B-cell leukemias (Tsujimot et al., 1984- Bakhshi et at., 1985- Chenlevy et at., 1989). The bcl-2 gene has been shown to exten an inhibitory effect on apoptosis (programmed cell death) in many cell system ***(Bissonette et al., 1992- Krosmeyer, 1992)***

Although most initial studies of the bcl-2 oncogene were restricted to hemopoitic malignancies, the finding of bcl-2 expression in various epithelial tissues suggested a possible role in the biology of epithelial neoplasms (Lu et al., 1993).

Bcl-2 gene expression was identified in normal embryonic, fetal, and adult tissues, as well as in a variety of nonhematologic malignancies. Although the biologic mechanism of bcl-2 in these tissues is unknown, multiple studies have provided insight into a possible role of bcl-2 in normal morphogenesis, as regulator of

cellular events in normal tissue, and as a possible intial step toward malignancy in multistep carcinogenesis ***(Hockenberry et at., 1997 and Lu et at., 1993).***

The investigation of the role of bcl-2 in the pathogenesis of solid turmor is still in a comparatively early stage. Several recent studies have shown a strong correlation between bcl-2 and estrogen-receptor expression in both normal and neoplastic breast epithelium ***(Leek et al., 1994 and Bhargava et at., 1994).*** This further supports the hypothesis that bcl-2 expression is estrogen regulated and suggests that, in this instance, this protein might be involved in tumor pathogenesis. The possibility of estrogen regulation of bcl-2, along with the known association of hyperestrogenic states with the development of most endometrial carcinomas, has led us to investigate the role of bcl-2 in endometrial carcinogensis by quantifying and comparing bcl-2 expression by enzyme immunoassay (EIA) method in normal cycling endometrium, endometrial hyperplasias with and without atypia, and endometrial adenocarcinoma.