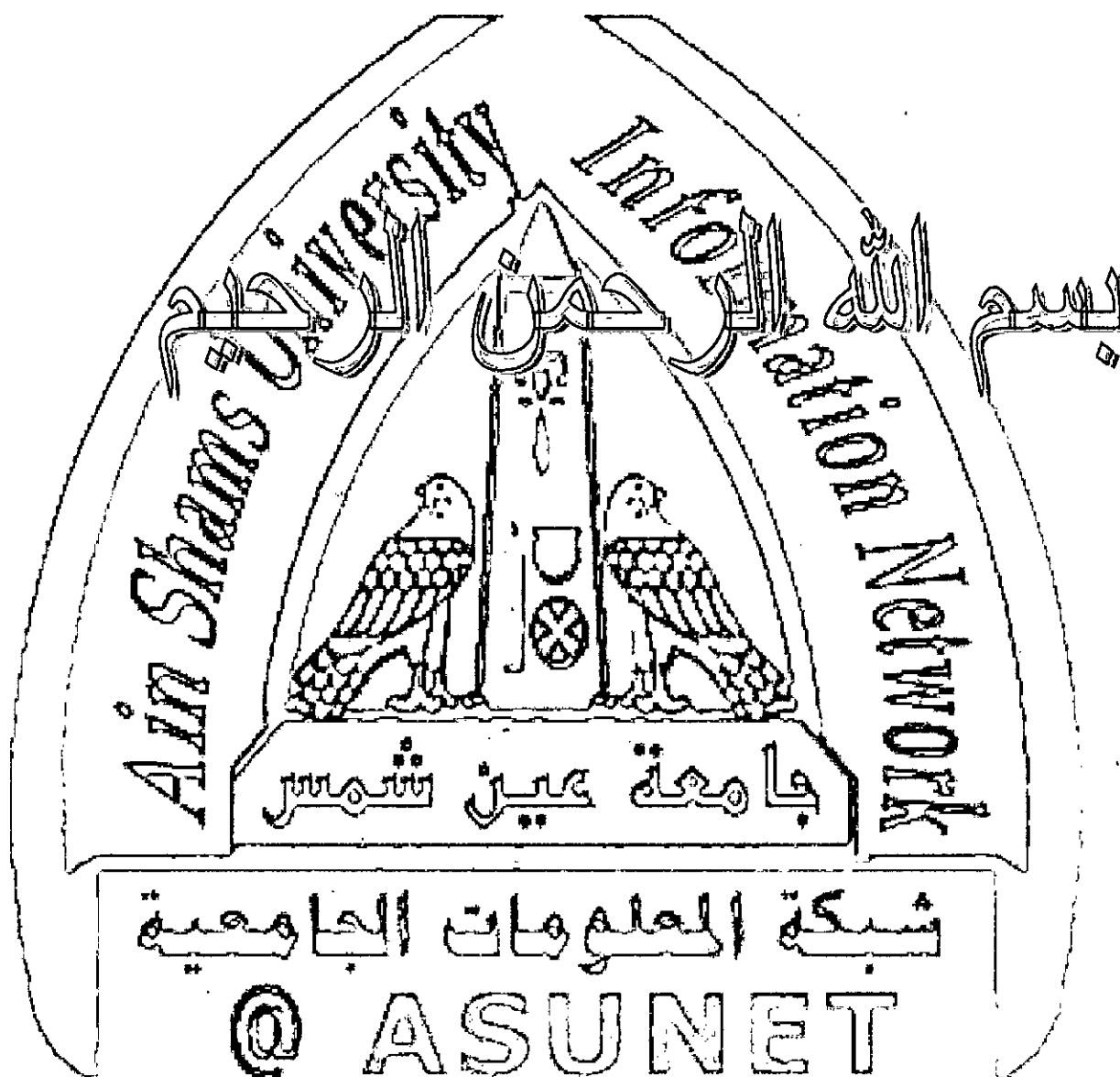




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**EXPRESSION OF Bcl-2 ONCOPROTEIN IN
ODONTOGENIC CYSTS
AN IMMUNOPATHOLOGICAL STUDY**

By

OMNEYA RAMADAN MAHMOUD, (B.D.S. 1995)

THESIS PROPOSAL

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***DEDICATED TO ALL MY
BELOVED FAMILY AND
ESPECIALLY TO MY
GUARDING ANGEL
YOUSSEF***

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INTRODUCTION

Cysts are the most common cause of chronic swellings of the jaws. Jaw cysts in which a carcinoma arises or an ameloblastoma develops as well as keratocysts and calcifying odontogenic cysts may be considered aggressive or potentially aggressive. Their aggressiveness is reflected by their destructive and infiltrative growth, their growth potential and their tendency to recur (Cawson and Odell, 1998).

There is a widespread interest in the comparatively rare squamous cell carcinoma arising in the odontogenic cyst due, to the belief of several investigators that it is one of the malignant neoplasms, which appear to arise from the specialized odontogenic epithelium lining cysts of the maxilla and mandible

Microscopic examination of several sections of cyst wall in instances of odontogenic cyst is urged as indispensable for detection of squamous cell carcinoma. The incidence of malignant changes in odontogenic cysts, including squamous cell and mucoepidermoid carcinomas has been estimated as varying from 1-2% (Stoelinga and Bronkhorst, 1988).

Apoptosis, or programmed cell death, plays a wide range of roles in embryogenesis and normal homeostasis as well as in oncogenesis and the pathology of tumours (Kumamoto, 1997).

Bcl-2 proto-oncogene is a member of a gene family that includes cell death suppressors and cell death promoters (Korsmeyer, 1999). Its gene product, bcl-2 protein, is a 26 kDa putative membrane-associated

protein located on the mitochondrial membranes, the endoplasmic reticulum, the nuclear membrane, and the mitotic nuclei in epithelial cell lines. Bcl-2 protein acts as a cell death suppressor that facilitates cell survival by regulating apoptosis. Over expression of bcl-2 protein occurs as a result of absence of regulation of its expression. The consequent hyperfunction causes cell immortalization and this has been reported in many human tumours (Lu et al., 1996).

Studies of bcl-2 emphasize the existence of multiple pathways in the generation of neoplasia (Korsmeyer, 1992).

Immunohistochemical studies have suggested that bcl-2 expression may be an early event in epithelial malignancies. Epithelial tumours often exhibit morphologically distinguishable stages from benign hyperplasia, dysplasia, carcinoma in situ to invasive carcinoma. It has therefore been postulated that bcl-2 expression prolongs the life span of cells, and increases their risk to acquire other changes, such as chromosomal abnormality and viral infection, resulting in malignant transformation or overt tumour progression (Lu et al., 1996).

Evolutionary new approaches in immunohistochemistry has allowed researchers to determine the normal developmental role of each of the bcl-2 gene family, and this promises to deliver insights into their oncogenic mechanism.