Nucleotide sequence of presenilin-1 cDNA fragment of Arabian camel, *Camelus dromedarius*

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ABSTRACT

The cDNA encoding camel presenilin-1 (PS1) fragment was cloned by reverse transcription-polymerase chain reaction (RT-PCR) using primers homologous to conserved sequences of PS1 of human, rat, lemur and mouse. The cDNA fragment, 402 bp in size was well conserved and found to be 79, 89, 91, 91 and 93% homologous to that of chicken, mouse, rat, lemur and human, respectively. The cDNA fragment encodes 130 amino acid protein fragment. The deduced amino acid sequence is also well conserved in various species, exhibiting 98% similarities with those of rat, lemur and human homologues. This cDNA fragment is quite significant as it is the most conserved portion of the PS1 in various animals and encodes four transmembrane regions (TM2, 3, 4, 5) of PS1. Moreover, more than 50% of the amino acid substitutions to which familiar Alzheimer's disease (FAD) have been linked are located in this region.

Key words: *Alzheimer's disease, camel, presenilin-1 cDNA*

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INTRODUCTION

Alzheimer's disease (AD) is the most common cause of dementia in elderly. The pathogenesis of AD is complex and the common clinical and neuropathological features can arise from several different genetic and non-genetic causes. Nevertheless, approximately 10% of all AD cases are estimated to be early-onset familial AD (FAD) and show autosomal dominant inheritance. Mutations in presenilin-1 (PS1) located on chromosome 14 have been linked with FAD (Schellenberg, 1995). More than 70 mutations have been reported in PS1 gene, their products cause dysfunction/death of vulnerable populations of nerve cells, resulting into clinical syndrome of progressive dementia (Price and Sisodia, 1998; Fraser *et al.*, 2000). In- spite of the significant number of recent studies focusing on AD and its causative factors, the underlying mechanism by which PS1 mutations lead to development of AD remains elusive (Fraser *et al.*, 2000; Saunders, 2001; Esler and Wolfe, 2001; Amtul *et al.*, 2002; Ponting *et al.*, 2002 and Zhou *et al.*, 2002). Although, various hypotheses have been put forward for the physiological function(s) of presenilin, yet it remains undefined and further studies are certainly