

# CURRENT RESEARCH AND DEVELOPMENT IN BIOTECHNOLOGY ENGINEERING AT IIUM

VOLUME II

Editors:

Ibrahim Ali Noorbatcha  
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Raha Ahmad Raus



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***(VOLUME II)***

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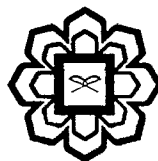
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**BIO-MOLECULAR COMPUTING IN DEGENERATIVE BRAIN RESEARCH**Ibrahim Ali Noorbacha<sup>1</sup> and Ahmad Faizul Shamsudin<sup>2</sup>

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**ABSTRACT**

Stress related genes are the ones that influence the production of GABA (Gamma Amino Butyric Acid) proteins which inhibit signal transmission between neurons in human brains. The bio-molecular mechanism of such gene-related causes is the state-of-the-art approach in brain research. This is to understand the puzzles of degenerative brain. In essence, it is part of a central dogma in the study of genes which relate genes to proteins and drugs for diseases. This chapter will illustrate the bio-molecular mechanisms of Degenerative brain (DBrain) which involve firing neurons and pseudo-genes that are both useful concepts in applying bio-molecular computing.

**Keywords:** GABA, Degenerative brain, pseudo-genes, bio-molecular computing

**INTRODUCTION**

The bio-molecular mechanism is one approach in understanding the underlying DBrain disease and related neurodegenerative disorders. One such mechanism involves the proteolysis of cell surface proteins and is referred to as ectodomain shedding. This is a basic cell biological process, which is not only essential in the pathogenesis of DBrain disease but also during embryonic development and in the immune system. The way how the ectodomain shedding process is regulated in the cell can be studied by using high-throughput functional genomics (expression cloning) to identify novel genes regulating ectodomain shedding. With a variety of molecular and cell biological methods we can analyze the molecular mechanism of their biological action of the novel genes and the potential role in DBrain disease.

Novel genes can be discovered from stress related DBrain disease. The stress related human GABA protein that belongs to a gene family is one of the major bio-molecular mechanisms for neural transmitter deficiency during neural degeneration (Hammond C., 2003). GABA inhibits the postsynaptic neuron from propagating nerve impulses from other neurons. GABA is derived by de-carboxylation of glutamate and appears to operate mainly in the brain. However, GABA is suspected of influencing a group of receptor genes in the degenerative brain. It is important to appreciate that it is the receptor that dictates the neurotransmitter's effect. GABA A receptors play a key role in controlling neuronal activity;