It is a first successful attempt to study all ADA genes together, association of Alzheimer disease with obesity and sex-biasness of the disease. With the objective to analyse broader view of the disease and associated biasness with women and obese people which is still not clear on molecular basis. This association was established through many clinical studies but underlying molecular mechanisms were yet to be explored. The most associated pathway with AD was found to be GnRH pathway which was not thought to be as significant as inflammation and presinilin pathways. It should be explored more to understand AD and subsequenty develop therapeutics for the disease. Gene's up- and down-regulated in the study were in co-relation with their function and role in AD pathogenesis hence pinpointing factors that may be useful for identification of new targets for improved AD therapeutics. Mitochondrially encoded *MT-TI* gene found to be up-regulated in all the AD samples, also associated with oxidative stress in the cell, is a proposed drug target for the disease.

The analysis has shown that 20% genes were common with both obesity and AD. The majority of those genes were found to be mainly implicated in signalling pathways. It highlights and enhances the importance of signalling in both the disease. Further expression study of obesity genes in AD samples reveal that total 153 genes were DGE. Above results justifies current study as majority of DGE obesity genes have direct or indirect role in AD.

Reduced expression of Y chromosome encoded genes in AD signifies the relevance of Y chromosome in AD. Interestingly none of the Y chromosome encoded genes were up-regulated in AD and its reduced transcriptional activity indicates the probable cause of women being more prone to AD. Genes found to be differentially expressed in AD samples were found to have significant role in AD etiology and suggests studies focusing on these molecules and genes i.e. *NLGN4Y* which plays significant role in cell adhesion. Loss of neurons and dysfunction of synapse is found to be responsible for AD etiology which implicates role of neural cell adhesion molecules in neurological disorders. It is possible that increased expression of Y chromosome encoded genes lead to promising therapeutic targets. Increased expression of mitochondrial genes in AD is in concurrence with earlier reported literature.

Transcription factors were identified to be differentially expressed out of which some regulates expression of genes which play significant role in AD hence it is suggested that all identified Tc factors should be explored in experimentation with respect to their association with AD.

According to gene expression analysis of all selected genes it was found that expression of majority of genes is reduced implying reduced transcriptional activity in the diseased brain which was in sync with the published literature. All analysis converges on one fact that AD is multifactorial disease because the associated genes were found to be of highly diverse nature according to their function, pathways associated and sequence similarity. Understanding how the DGE in the study are responsible for neuronal functioning and signaling would be highly intriguing and helpful in deciphering the AD mystery. These genes should be further explored for their detailed role in Alzheimer's in order to understand disease better and could be the promising therapeutic targets for the disease.