A.V.V.M. Sri Pushpam College (Autonomous)
Poondi– 613 503, Thanjavur-Dt, Tamilnadu
(Affiliated to Bharathidasan University, Tiruchirappalli – 620 024)

3.7.1 Number of Collaborative activities per year for research/ faculty exchange/ student exchange/ internship/ on –the-job training/ project work

Collaborating Agency:

Dr. V. BalachandarBharathiyar University, Coimbatore

Dr. S. GANESAN
Assistant Professor
PG & Research Department of Zoology
A.V.V.M Sri Pushpam College (Autonomous)
Poondi-613 503,
Thanjavur-Dt, Tamil Nadu, India.

Dr. V. BALACHANDAR
Assistant Professor
Department of Human Genetics and Molecular Biology, Bharathiar University
Coimbatore – 641 046
Tamil Nadu, India.

Date: 10.12.2015

LINKAGE

For the year 2015-2016

Between

1. Dr. S. Ganesan
   Assistant Professor
   PG & Research Department of Zoology
   A.V.V.M Sri Pushpam College
   (Autonomous), Poondi – 613 503.

&

2. Dr. V. Balachandar
   Assistant Professor
   Department of Human Genetics and
   Molecular Biology,
   Bharathiar University, Coimbatore – 641 046.

Considering the significance of the noble cause for the student community, we have come forward to collaborate with each other to exchange research knowledge, expertise, laboratory and library facilities to the process of scientific research and education in the field of medical science. The parties (mentioned above as 1. & 2.) have had preliminary discussion in this matter and have ascertained areas of broad consensus. The parties now therefore agreed to enter in writing these avenues of consensus, under a flexible linkage, and this project aims to fill the gap between knowledge demand and subject expertise related to the mentioned field.

Joint Responsibilities

- Sharing of laboratory facilities, library resources, database etc.,
- Joint Publication of research articles, books, magazines, bulletins etc.,
- Jointly organizing conferences, seminars, symposia and workshops.
- Submitting joint proposals for research funding from agencies like UGC, CSIR, DST and TNSCST.

10/12/2015

Dr. S. Ganesan

Dr. V. Balachandar

Dr. S.GANESAN M.Sc., M.Phil., Ph.D.,
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Screening of Genetic Mutations in Early Onset Parkinsonism Patients: A Family Based Study in Tamil Nadu Population

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KEYWORDS Chromosomal Abnormalities. Parkinson's Disease. Polymorphism. SNCA

ABSTRACT Parkinson's disease (PD) is a progressive motor system disorder which distress several parts of the brain, in particular substantia nigral area that controls balance and movement. The intend of the study was to identify the polymorphism in SNCA (α-Synuclein) and Parkin genes using PCR-RFLP and chromosomal analysis by GTG banding in 23 early onset PD patients who are below the age of 50. The results were analyzed and SNCA polymorphism was observed as missense mutation A53T with G–A transition whereas Parkin is also observed with G–A transition and change in amino-acid S167N. The chromosomal abnormality resulted with 22q11.2 deletion which is an increased risk factor in early-onset PD. Therefore, the resulted polymorphism is the foremost report in Tamil Nadu population and the researchers assure that this would be a unique study from the previous researches in India.

INTRODUCTION

Parkinson's disease (PD) is a type of movement disorder, which occurs due to the loss of the dopamine and it is the second most common neurodegenerative disorder following Alzheimer's disease. It affects substantia nigral area that controls balance and movement. The presence of intraneural inclusions, lewy bodies and loss of neurons in the substantia nigra are the distinguished pathological features (Fearnley et al. 1991). Usually, PD before the age of 50 is early-onset whereas after 50 named as late-onset disease. PD symptoms are subtle and occur gradually (Javier *et al.* 2015). The prevalence rate in North America and West European countries is about 150–200 per 100,000 (Zhang and Roman 1993) and lower in China, Japan and Africa (45–80 per 100,000). In India its occurrence is around 14–27 per 100,000 individuals (Singhal et al. 2003). It can be rarely inherited in families either as an autosomal dominant, autosomal recessive trait (Biswasa et al. 2006) or as sporadic cases. Hereditary and environmental factors increase the chance of PD and are likely to inflict a major socio-economic burden on aging populations.

Till now more than 16 PD related loci has been identified after the discovery of α-synuclein (SNCA) (Corti et al. 2011). The following five major genes have shown to cause PD: PARK2 (Parkin), PINK1, PARK7 seen in autosomal recessive PD whereas SNCA and LRRK2 genes seen in autosomal dominant form (Thomas and Beal 2007). PARKIN, DJ-1 and PINK 1 mutations have been detected in autosomal recessive juvenile Parkinsonism (Kitada et al. 1998; Funayama et al. 2002; Valente et al. 2004). Parkin and DJ-1 mutations are significant in causing familial PD (Healy et al. 2004; Hedrich et al. 2004).

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