**DOES ANXIETY CAUSE FREEZING OF GAIT IN PARKINSON’S DISEASE?**

There is strong evidence that anxiety is an important mechanism underlying freezing of gait (FOG) and supports the notion that the limbic system may have a profound contribution to freezing in PD, according to new research.

Anxiety has never been directly examined as a cause of FOG. This study used virtual reality to induce anxiety and evaluate whether it directly causes FOG. Fourteen patients with PD and freezing of gait (Freezers) and 17 PD without freezing of gait (Non-Freezers) were instructed to walk in two virtual environments: across a plank that was located on the ground (LOW) and across a plank above a deep pit (HIGH). Multiple synchronized motion capture cameras updated participants’ movement through the virtual environment in real-time, while their gait was recorded. Anxiety levels were evaluated after each trial using self-assessment manikins. Freezers performed the experiment on two separate occasions (in their ON and OFF state).

Freezers reported higher levels of anxiety compared to Non-Freezers (p<0.001) and all patients reported greater levels of anxiety when walking across the HIGH plank compared to the LOW (p<0.001). Freezers experienced significantly more freezing of gait episodes and spent a significantly greater percentage of each trial frozen when crossing the HIGH plank.

— *PloS One; 9*(9):e106561

**MORE DATA ON RARITY OF PARKIN (PARK 2) MUTATIONS IN EARLY-ONSET PD**

A new study “contributes to the growing body of evidence on the low frequency of the parkin mutations in the early-onset Parkinson’s disease suggesting the potential role of other genes in the pathogenesis of the disease.”

In it, parkin mutations were identified in five patients out of a total of 70 early-onset PD patients (age at onset ≤40 years); 75 controls were screened for the sequence variants and exon rearrangements in the parkin gene. The p.R334C point mutation was present in one patient, four patients had exon deletions. The detected mutations were observed in the heterozygous state except one homozygous deletion of the exon 4. No mutations were obtained in control subjects. A novel sequence variant p.V380I (c.1138G>A) was identified in one control. Non-pathogenic polymorphisms p.S167N and p.D394N were seen in similar percentage in patients and controls, polymorphism p.V380L was almost twice as frequent in controls as in patients.

— *PloS One; 9*(9):e107585.

**CANNABIDIOL MAY IMPROVE QOL IN PD**

New findings point to a possible effect of cannabidiol in improving quality of life measures in PD patients with no psychiatric comorbidities.

From a sample of 119 patients consecutively evaluated in a specialized movement disorders outpatient clinic, researchers selected 21 PD patients without dementia or comorbid psychiatric conditions. Participants were assigned to three groups of seven subjects each who were treated with placebo, cannabidiol (CBD) 75mg/day or CBD 300mg/day. One week before the trial and in the last week of treatment participants were assessed in respect to UPDRS; well-being and quality of life (PDQ-39); and possible neuroprotective effects. They found no statistically significant differences in UPDRS scores, plasma BDNF levels or H1-MRS measures. However, the groups treated with placebo and CBD 300 mg/day had significantly different mean total scores in the PDQ-39 (p = 0.05). The authors note studies with larger samples and specific objectives are required before definitive conclusions can be drawn.

— *J. Psychopharmacol. 2014 Sep 18*