**Dyskinesia Webinar Q&A**

**Young onset Parkinson’s (YOPD) and dyskinesia**

1. *Why do people with YOPD develop dyskinesia, even when they have a short disease duration?*
2. The exact reason is not entirely clear. One hypothesis is that the area that receives dopamine is more youthful and vigorous and remains relatively intact, so is more sensitive to the changes that lead to dyskinesia.

**Risk factors for developing dyskinesia**

1. *Why are some people more prone to dyskinesia than others?*
2. The answer seems mostly to be related to some combination of genetics, age, and treatment. The exact underpinnings of how these factors interact to cause dyskinesia is unknown.
3. *Do any of the following factors (age, genetics, body weight, use of PD drugs, gut malabsorption or depression) increase the likelihood of developing dyskinesia?*
	* Age – yes, younger onset of PD has higher risk of dyskinesia.
	* Genetics might be causative.
	* Body weight – those with lower body weight are more prone to dyskinesia, probably in part related to higher exposure to medication levels.
	* Use of PD drugs (e.g. entacapone) – dyskinesia is mostly related to levodopa exposure. Higher dose and longer duration of levodopa is associated with greater risk. Entacapone and similar medications boost levodopa and may increase dyskinesia risk in some individuals.
	* Gut malabsorption – no, this is not associated.
	* Depression – no, this is not associated.
4. *What is the highest/recommended safe L-Dopa dosage a patient can take daily?*
5. Every PwP is different. There is no single highest dose for a patient. The right dose is typically considered the dose that provides good control of symptoms for the individual without inducing side effects. In early PD, most patients are on around about ~300 mg of levodopa per day. Doses higher than 800-1000 mg per day are unusual but may be necessary in some patients. Levodopa doses in the Rytary formulation are different, and are typically 2-2.5 times higher.

**Non-motor symptoms (NMS)**

1. *How do we get more focus on treatment for NMS?*
2. We need more awareness and crucially patient voice telling government, charity and industry to focus their resource and money to management of NMS rather than yet more millions on genetic and motor symptoms research.
3. *Is DBS any good for dealing with non-motor symptoms?*
4. DBS is. Specifically helping sleep, pain and some aspects of urinary dysfunction. Panellist Professor Ray Chaudhuri has recommended some reading on this (Dafsari et al, 2018 and 2019), please see papers attached.

**Advanced Therapy**

1. *How effective are advanced therapies (eg. apomorphine) in reducing dyskinesia?*
2. Apomorphine can reduce dyskinesias. Panellist Professor Ray Chaudhuri has shared some reading on this (Euroinf 2 study, 2019), please see paper attached.

**Prevention:**

1. *My neurologist said if I stayed under 300mg per day it reduces risk of developing dyskinesia, is this true?*
2. Doses of levodopa above 400 mg/day are associated with an increased risk of dyskinesia. However, it is important to find balance. It may not make sense to stay on a low dose of levodopa if slowness and small movement becomes problematic.
3. *If it is a self-induced (to an extent) condition - then what do we do?*
4. It is due to both progression of the disease and its treatment. There is interest in whether long acting levodopa formulations can reduce the development and expression of dyskinesia. Amantadine is a moderately effective treatment for dyskinesia in those who can tolerate it. In addition, research continues to seek new treatments to reduce or prevent dyskinesia.