Creative Disability and the Shaking Palsy. Approaching a History of Parkinson's Disease

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Dorothy Porter

Parkinson's Disease gained notoriety in a Hollywood blockbuster film adaptation of Oliver Sacks's *Awakenings*, starring Robin Williams and Robert de Niro. Sack's 1973 book and the 1990 film, presented an account of survivors of the 1914-1924 encephalitis lethargica epidemic waking up from a catatonic state following large doses of L-Dopa.[1] In a particularly poignant moment in the movie, De Niro's character, Leonard Lowe, dances away his pathological muscle rigidity and spasms with his darling Paula. Sadly Leonard Lowe, like the non-fictional patients who experienced this moment in medical history, returns to his catatonic state but the idea of dance as a cure for symptoms of Parkinsonism has survived and is a subject important to the present article.[2]

In a book on Musicophilia in 2007, Oliver Sacks subsequently proposed that in fact musical rhythm may have a primal function in the brain which modern science is yet to fully explain.[3] Sacks illustrates his arguments with accounts of patients in 2007 with various types of frontal lobe lesions, such as Frontal Lobe Dementia, Alzheimer's and some Parkinson's Disease who developed new aptitudes for musical creativity after the onset of their disease. This led Sacks and others to speculate that creative inhibition was genetically coded into frontal lobe function and was disengaged through some pathological lesions which, so to speak, let the music out.[4]

In Sacks's captivating narratives neurological changes and creativity are tales of transformation brought about by illness. In *When Walls Become Doorways* (2006) Tobi Zausner recounted similar patients' narratives who experienced creative moments in their lives following illness such as the

[1] O. Sacks, *Awakenings* (Duckworth 1973).
[2] O. Sacks, *Musicophilia: Tales of Music and the Brain* (New York 2007) 270-283. See Oliver Sacks's discussion of dance therapy at Beth Abraham Hospital and exploration of the Tango, in particular, for Parkinson's patients.
[3] Ibidem.
[4] Ibidem, 162-170.

artist Henri Matisse.[5] Patient narratives do not have a prerogative, however, on defining and interpreting the relationship between illness and creativity. This is acutely the case for sufferers of neurological degeneration that brings about the assemblage of disorders that continue to be identified as Parkinson's Disease even though that terminology is itself challenged by the historical scientific and clinical tradition that created it.

This essay investigates how and why two cultural interpretations of the relationship between creativity and Parkinson's Disease have developed and changed over time. It interrogates the historical determinants of a neuro-psycho-biological discourse with the power to affect human consciousness and somatic experience through pharmacological manipulation according to its own edicts. The paper contrasts this discourse with Parkinson's Disease patients' narratives of the transformative affect of creativity allowing them to experience different ways of becoming human. The paper argues that the two counter cultures of creativity and Parkinson's Disease have been framed by a pivotal historical moment in which two critically significant changes occurred in the epistemological status and causal explanation of Parkinson's Disease. Firstly, when the ontology of Parkinson's Disease as a single entity became dispersed into a set of emergent interactions over a human life-course including risks of getting the disease and pre and post-diagnostic behavioral pathologies. Secondly, when neuroscientific investigation achieved this ontological dispersal through the explosion of the causal explanation of Parkinson's Disease as a motor disorder which was caused by the death of neurons in the brain that produce the neurotransmitter, dopamine. Finally the paper argues that contrasting representations of the relationship between being and becoming characterize the two historical cultural interpretations of the relationship between Parkinson's Disease and creativity. The paper demonstrates the historical rise of a clinical diagnosis of creativity in Parkinson's patients as a pathological symptom and the contrasting narrative of Parkinson's patients for whom creativity is an opportunity of experiencing a new way of what it means to be human. In the neuro-psycho-biological discourse the replacement of static ontology (being) with temporal emergence (becoming) has not eliminated reductionist reasoning about the relationship between creativity and disablement. The paper argues that a different epistemology of being and becoming is evident within patient narratives of the

[5] T. Zausner, *When Walls Become Doorways. Creativity and the Transforming Illness* (New York 2006) 21-25.

transformative affect of creativity represented in the understanding and the experience of human variety.

Neuroscience troubles ontology: exploding the Nigrastriatal Dopamine-Deficiency Paradigm of a Motor-Disorder.

Since 2008 The Jack Clark Professor in Parkinson's Disease Research at the University of Toronto (and Director of the Shulman Movement Disorder Clinic at Toronto Western Hospital), Anthony Lang, has been urging the clinical world to face up to the fact that there is no Parkinson's Disease but many manifestations of multiple aetiological syndromes with what historians might refer to as customs in common.[6] The discovery of multiple neuro-pathogenerative pathways resulting in motor and non-motor symptoms associated with Parkinson's has led Lang to identify the dawn of a new era in the clinical profile of what has until relatively recently been understood as a single nosology. What causes Lang to come to these conclusions is novel molecular understanding of neurodegeneration which has undermined the aetiological paradigm of nigrostriatal dopamine deficiency[7] and the decentralisation of motor disorder in the clinical profile of Parkinson's Disease (PD).[8]

A full historical account of the assembling of the nigrastriatal dopamine deficiency PD paradigm has been undertaken in another paper.[9] In this essay I am going focus on the historical process in which a clinical category of a Parkinson's patient personality was constructed and the pathologicalization of creativity within that paradigm.

[6] B. Patoine, 'Facing Up to the 'New Face' of Parkinson's Disease. An Interview with Anthony E. Lang', *The Dana Foundation* (2012) 1-3.

[7] See Fig 1: Dopamine Pathways in the Brain

[8] Milestones in Parkinson's disease—Clinical and pathologic features Glenda Halliday PhD, Andrew Lees MD, FRCP and Matthew Stern MD Article first published online: 27 May 2011 *Movement Disorders* 26 (May 2011) 1015-1021.

[9] D. Porter, 'A history of translational research and the discovery of the catecholamine function of dopamine', *Translational Medicine* (paper submitted).

Experimentation by the Professor of the Nobel Prize winner Arvid Carlsson at Lund University in the 1950s proved that the cause of Parkinson's Disease was the loss of dopamine in the brain. He demonstrated this causal relationship by using a neuroleptic drug, reserpine, to suppress dopamine in the brains of rabbits which made them catatonic. He revived the rabbits by injecting them with a large dose of the amino acid dopa proving that dopamine in the area of the brain known as the basal ganglia was responsible for determining physical motor control in the body.

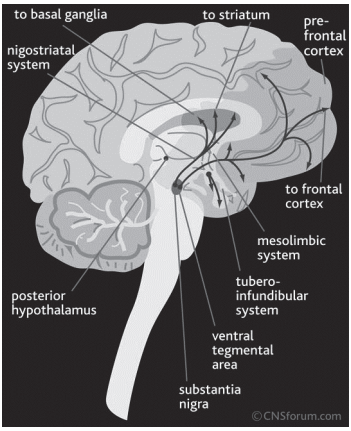

Fig. 1: 'The Dopamine Pathways in the Brain', *CNS Forum, Lundbeck Institute, Educational Resources* (2014) http://www.cnsforum.com/educationalresources/imagebank/normal_brain/neuro_path_da 21 augustus 2014.

The role of dopamine in behaviour, however, became an inevitable deduction once Carlsson had established the effect of behavioural drugs in suppressing dopamine release. Thus psychopathology must be the result of excessive dopaminergic activity.[10] The dopamine hypothesis of

[10] H.Y. Meltzer and S.M. Stahl, 'The Dopamine Hypothesis of Schizophrenia: A Review', *Schizophrenia Bulletin* 2.1 (1976) 19-76.

psychopathology became established and was contested throughout the latter half of the twentieth century but the dopamine system in the brain became definitively central to genomic research on cognition and emotional variation from the 1990s.[11] The role of the DA, that is dopamine molecules in the brain, system in cognitive and emotional variability, however, was given an entirely new profile with the development of Tridimensional Temperament System developed in the 1980s and 1990s by the Kantian transcendentalist philosopher-professor of psychiatry at Washington University, Claude Robert Cloninger.[12] In 1986 Cloninger argued in what he called a 'Unified Biosocial Theory of Personality' that heritable personality traits of novelty seeking, harm avoidance and reward dependence were correlated with dopaminergic activity in the basal ganglia, serotonergic activity and basal noradrenergic activity.[13]

The Tridimensional Temperament System provided a new methodological and epistemological framework for genomic correlation with personality and cognitive analysis.[14] Cloninger first developed his methodologies for developing predictive personality models when working in Sweden in the 1980s on alcohol dependence through 'separation studies', i.e. investigating cohorts of adopted children separated from their biological parents at birth.[15] By the end of the decade he codified his pyschobiological temperament model in a Tridimensional Personality Questionnaire (TPQ) and a Tridimensional Character Inventory (TCI).[16] These tools provided the

[11] D. Ball et al., 'Dopamine Markers and General Cognitive Ability', *Neuroreport* 9 (1998) 347-349; S. Nakajima e.a., 'The Potential Role of D3 Receptor Neurotransmission in Cognition', *European Neuropsychopharmacology* 23.8 (2013) 799-813.

[12] G. Hellinga e.a., 'Robert Cloninger' in: J. Aronson ed., *Personalities: Master Clinicians Confront the Treatment of Borderline Personality Disorder* (Northvale and London 2001) 99-120.

[13] C.R Clonginger, 'A unified biosocial theory of personality and its role in the development of anxiety states', *Psychiatry Developments* 1986 4.3 (1986) 167-226.

[14] Ibidem.

[15] E.J. Devor and C.R. Cloninger, 'Genetics of Alcholism', *Annual Review of Genetics* 23 (1989) 19-36.

[16] C.R. Cloninger e.a., 'The Tridimensional Personality Questionnaire: U.S. Normative Data', *Psychological Reports* 69 (1991) 1047-1057; idem, 'A Psychobiological Model of Temperament and Character', *Archives of General Psychiatry*' 50 (1993) 975-990; idem, 'Psychometric Properties of the Temperament

neurobiology of cognition and character with a new methodological framework of investigation.

Cloninger's dopaminergic hypothesis of character and temperament facilitated investigations into the genomic influence on dopamine dependent cognitive and emotional variation. From the earliest studies with twins a discourse on the inheritability of cognitive ability has been embedded in the psychobiology of intelligence.

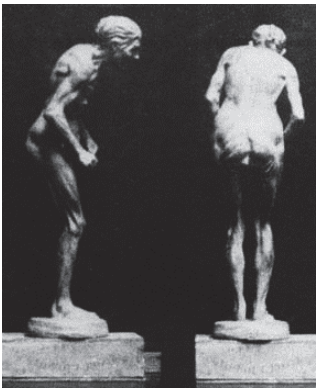

Fig. 2: Paul Richer: Figurine: 'Parkinson's Disease' (cc. 1882), *Viartis, History of Parkinson's Disease* (2014) http://viartis.net/parkinsons.disease/history.html (21/08/2014).

Since the completion of the human genome map the role of the dopaminergic system (DA) and its genetic variations have reframed this investigation. Candidate dopaminergic genes in cognitive variation that have been explored so far include catechol-*0*-methyltransfrerase (COMT)[17] and

and Character Inventory – Revised in a Belgian Sample', *Journal of Personality Assessment* 85 (2005) 1931-1946.

[17] A gene that codes for dopamine.

some of its nucleotide polymorphisms[18] related to prefrontal executive functioning in processes such as attention and working memory. COMT is an enzyme which plays a crucial role in the metabolism of the catecholamines by inactivating them in the synaptic cleft. Another candidate gene for intelligence that has been significantly investigated has been the D2 receptor gene (DRD2) and one of its polymorphic regions, DRD2 TAQ 1A.[19]

D2 receptor polymorphisms have also become central to genomic investigations of a subset of cognitive function, creativity, since the mid-2000s. Dopaminergic variation currently occupies the central focus of the neuromolecular gaze upon creativity defined and measured as levels of novelty seeking divergent thought using Cloninger's Tridimensional Character and Temperament System. Mesolimbic accounts of creativity have now gained sufficient confidence to begin claiming the capacity to be predictive. For example, Sapra, Beavin and Zak from Claremont Graduate University and Loma Linda Medical School in California recently claimed to have demonstrated what they believe is the gene-combo that produces successful careers amongst the top socio-economic 1% in their recent PLOS article on 'A Combination of Dopamine Genes Predicts Success by Professional Wall Street Traders'.[20]

Apart from facilitating the genomic investigation of heritable cognitive creativity variation Cloninger's Tridimensional Character and Temperament System has also been utilized in the genetic investigation into emotional variation.

The investigation into genomic variation in creativity centers upon the correlation of activity in the dopaminergic pathway in the brain that deals with reward learning, novelty seeking and pathological addiction with temperament and personality characteristics measured by the Tridimensional Temperament System.[21] This has had an emphatic influence

[18] The polymorphisms of the gene.

[19] M. Reuter e.a., 'The Influence of the Dopaminergic System on Cognitive Functioning: A Molecular Genetic Approach', *Behavioral Brain Research* 164 (2005) 93-99; Idem, 'Identification of first candidate genes for creativity: A pilot study', *Brain Research* 1069 (2006) 190-197.

[20] S. Sapra, 'A Combination of Dopamine Genes Predicts Success by Professional Wall Street Traders', *Plos One* 7.1 (2012) 1-7.

[21] T. Suhara, 'Dopamine D2 receptors in the insular cortex and the personality trait of novelty seeking', *Neuroimage* 13.5 (2001) 891-895; L.K. Teh e.a., 'Tridimensional

on the neuromolecular construction of a pre-morbid Parkinson's patient personality.

In 1913 Carl Dudley Camp[22], by then Clinical Professor in Nervous Diseases appointed at the University of Michigan posited that:

> It would seem that paralysis agitans affected mostly those persons whose lives had been devoted to hard work (…) The people who take their work to bed with them and who never come under the inhibiting influences of tobacco or alcohol are the kind that are most frequently affected. In this respect, the disease may be almost regarded as a badge of respectable endeavor.[23]

One hundred years of psychobiology have subsequently characterized Parkinson's patients as possessing distinctive personality traits of industriousness, seriousness and inflexibility. Cloninger's Tridimensional Character Framework has re-scripted this clinical/anecdotal epistemological system profoundly. The TPQ and TCI have become the major methodological tools for investigating a pre-morbid Parkinson's personality as a predictive device not only for determining predilection to the onset of motor-dysfunction but of progressive cognitive and behavioral transformation. [24]

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personalities and polymorphism of dopamine D2 receptor among heroin addicts', *Biological Research For Nursing* 14.2 (2012) 188-196; S.W. Savage e.a., 'Regulation of novelty seeking by midbrain dopamine D2/D3 signaling and ghrelin is altered in obesity', *Obesity (Silver Spring).* 11 (2014).

[22] R.N. Dejong, 'Carl Dudley Camp', *Journal of Nervous and Mental Disease* 122.1 (1955) 116.

[23] C.D. Camp, 'Paralysis Agitans, Multiple Sclerosis and their Treatment' in: W.A. White, S.E. Jelife and H. Kimpton ed., *Modem Treatment of Nervousand Mental Disease* 2 (Philadelphia 1913) 651-667; See also: C.T. Todes and A. J. Lees, 'The Pre-morbid Personality of Patients with Parkinson's Disease', *Journal of Neurology, Neurosurgery and Psychiatry* 48 (1985) 97-100; L. Ishihara, and C. Brayne, 'What is the evidence for a pre-morbid Parkinson's Personality: A Review', *Movement Disorders* 28.8 (2006) 1066-1072.

[24] See for example: R. Tomer and J. Aharon-Peretz, 'Novelty seeking and harm avoidance in Parkinson's disease: effects of asymmetric dopamine deficiency', *Journal of Neurology, Neurosurgery and Psychiatry* 75 (2004) 972-975; G. Pluck and R.G. Brown. 'Cognitive and Affective Correlates of Temperament in Parkinson's Disease', *Depression Research and Treatment* (2011). For critique of use of TPQ and TPI in these studies see: M. Poletti and U. Bonuccelli, 'Personality traits in patients

In 1993 Matthew Menza and colleagues from the Robert Woods Johnson Medical School at the University of Medicine and Dentistry New Jersey identified dopamine-related personality traits in Parkinson's disease using Cloninger's Tridimensional Personality Questionnaire. Menza e.a. claimed that low novelty seeking Parkinson's patients were reflective, stoic, slow-tempered, frugal, orderly and persistent. They were by contrast high scoring on Harm Avoidance. In 1995 the Menza team claimed to explain this correlation by confirming through PET scans of PD patients that low 18 F-Dopa (*i.e. Radioflurinated Dopamine)* striatal uptake correlated with low novelty scores.[25]

Subsequent studies such as those of Valterri Kaasinen e.a. from the University of Turku in Finland argued that Menza's results could simply be demonstrating the effects of long-term medication on advanced PD patients. But nevertheless, the neuromolecular pursuit of the predisposed PD low-creative, harm avoiding, fearful dullard has continued up to the present.[26] In 2011 Grahame Pluck and Richard Brown from Sheffield University and Kings College London attempted to extend the temperament model of predisposed PD personality through a study on cognitive attention capacity. Using the Cloninger epistemological and methodological framework Pluck and Brown claimed to have demonstrated characteristic apathy in PD patients that is unrelated to the affective disorders of depression and anxiety associated with the disease. They claimed instead that high apathy scores are correlated with characteristic cognitive function of the pre-morbid Parkinson personality. Pluck and Brown legitimize their claims by using historical studies confirming the existence of a pre-morbid Parkinson's personality stretching back to Charcot describing the low motivation in PD patients.[27]

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with Parkinson's disease: assessment and clinical implications', *Journal of Neurology* 259.6 (2012) 1029-1038.

[25] M.A. Menza e.a., 'Dopamine-related personality traits in Parkinson's disease', *Neurology* 43.3.1(1993) 505-508; idem, 'Personality correlates of [18F]dopa striatal uptake: results of positron-emission tomography in Parkinson's disease', *The Journal of Neuropsychiatry & Clinical Neuroscience* 7.2 (1995) 176-179.

[26] V. Kaasinen e.a., 'Personality traits and brain dopaminergic function in Parkinson's disease', *Proceedings of the National Academy of Science U.S.A.* 98.23 (2001) 13272-13277.

[27] G. Pluck and R.G. Brown, 'Cognitive and Affective Correlates of Temperament in Parkinson's Disease', *Depression Research and Treatment* (2001); G.C. Pluck and R.G.

Poor Parkinson's patients. Under the neuromolecular gaze PD patients are dogged by dopamine and never to be released. As Genevieve Aubert has pointed out the neurological patient has been studied and documented extensively through visual media.[28] The Parkinson's patient has been clinically visualized by Charcot's drawings of the propulsive and retropulsive gait which fascinated him and by Samuel Wilson's documentary films of patients at the Queens Square Neurological Institute in the 1920s and by contemporary MRI videos of dopaminergic activity in the brain.[29]

But perhaps the most emblematic representation of the Parkinson's patient is the frail, broken, pitiful figure depicted in a figurine by Paul Richer, Charcot's collaborator in the 1880s. Richer's image is an archetype of somatic degeneration vividly, sculptured into the imagination.[30] The stigmata of pitiful objectification of this pathetically broken creature is inescapably overwhelming.[31]

In this section I have tried to demonstrate how dopaminergic molecular-neuorogazing has prescribed inevitable destiny upon Richer's pitiful object. According to the dopaminergic neuromolecular gaze, Richer's figure was determined by dopaminergically coded inflexibility, stoicism, harm avoiding logic and dullard low creativity. According the genomic and psychobiological modeling of the Parkinson's personality, temperamental rigidity defines the Parkinson's patient long before he has any physical bracykinesia. And their predisposition to inhibited personality is inscribed as emphatically in their genetically determined lack of novel imagination as it becomes in their cogwheel motor movements and pill-rolling tremors.

In the next section of the paper I interrogate the history of how subsequent discovery of creativity in Parkinson's patients became

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Brown, 'Apathy in Parkinson's Disease', *Journal of Neurology, Neurosurgery and Psychiatry* 73 (2002) 636-642.

[28] G. Aubert, 'Neurological illustration: from photography to cinematography' in: S. Finger, F. Boller, K.L. Tyler ed., *Handbook of Clinical Neurology*, *History of Neurology* 95.3 (2010) 289-302.

[29] J. Volkmann, 'The Wilson Films – Parkinson's Disease', *Movement Disorders*, 26.14 (2011) 2475-2476.

[30] See Fig. 4: 'Parkinson's Disease Figurine', by Paul Richer (ca.1882)

[31] See: P. Richer, *Artistic Anatomy* (translated and edited by Robert Beverly Hale), (London 1986); P. Richer, Paul Marie Louis Pierre, *Attitudes et Mouvements* (Paris 1921).

pathologized according to the neuro-psycho-biological paradigm of a genetically predisposed Parkinson's personality.

Pathological Creativity

According to neuromolecular biopsychological theory, when genetically predisposed uncreative Parkinson's personalities demonstrate creativity it is a pathological symptom. A body of neurobiological and clinical observations articulated over the last decade has identified creativity in Parkinson's patients as another form of pathological disability.

In 2001 Anette Scharg, MD. PhD and Michael Trimble MD from University College London reported what they believed to be the first case of poetic talent being 'unmasked' by dopamine agonist treatment for Parkinson's.[32] Their patient was aged forty when he was first diagnosed with Parkinson's having a tremor in his left hand, dragging his left leg, and dystonia of the left foot. As symptoms increased the patient started treatment with the dopamine agonist lisuride and levadopa at age 44. His symptoms significantly improved and within the first month of treatment he began writing poetry for the first time in his life completing ten poems in the first year of treatment. His poems achieved significant publishing success and he won a prize in the annual contest of the International Association of Poets.[33]

The patient had always considered himself intellectually inferior to his siblings and family generally never having achieved an equivalent level of academic success. He did, however, have a maternal grandfather who was a published poet and he was related to a well-known Irish poet. No other changes occurred until after twelve years of treatment the patient began to show symptoms of depression, aggressive and volatile behavior along with grandiose ideas, paranoid delusions, extreme circumstantiality, overtalkativeness and pressured speech. After various therapeutic adjustments the patient returned to a stable emotional and cognitive state, with sustained control of motor symptoms. Throughout this period and to the end of his life the patient continued to write and publish his poetry.

[32] A. Shrag, 'Poetic Talent Unmasked by Treatment of Parkinson's Disease', *Movement Disorders* 16.6 (2001) 1175-1176.
[33] Ibidem.

Scharg and Trimble explored a wide range of possible explanations for their 'poet unmasked' including contradictory implications of the classic 'pre-morbid PD personality theory' but found none sufficiently satisfactory. They finally concluded that they could only speculate that the effect of dopaminergic and serotomergic drugs, either through cognitive enhancement, increased perception or a hypomanic syndrome in addition to selective frontocortical dysfunction, led to the release of previously inhibited creative power in this patient.[34]

If Scharg and Trimble were only tentatively speculating that perhaps their patient's art was a sort of hypomanic 'high' by the mid 2000s a more reductive neuropsychological explanation was being offered of PD visual art. In 2005, Ruth Walker and colleagues at the Veterans Affairs Medical Center New York and NYU School of Medicine described a patient who had possessed pre-morbid undeveloped 'artistic tendencies' for sketching that dramatically changed seven years after beginning dopamine agonist therapy.[35] At this point he started producing several pastel drawings a week – sometimes two a day. After moving to an assisted living facility he had even more time to devote to his art but at the same time began to show hypersexual disinhibition in what they describe as 'excessive flirting with women on the street and asking residents and staff of his assisted living facility to pose nude for his artwork'. [36] His disinhibition became controlled following what they describe as 'education' and his artwork began to be shown and sold in local galleries in the Bronx. In contrast to Schrag and Trimble, Walker e.a. describe their patient's artistic productivity as a pathological symptom of compulsive disorder equivalent to pathological gambling and hypersexuality associated with excessive dopamine stimulation. Thus, the Walker team identified their patient's creativity as the result of the overstimulation of the third dopaminergic pathway in the brain which is responsible for reward learning that can become pathologically transformed into addiction.[37]

In 2006 Anjan Chatterjee, Roy Hamilton and Prin X. Amorapanth from University of Pennsylvania were far more reluctant than the Walker team to 'reduce CSF's [their patient] art to the ravages of PD' even after

[34] Shrag, 'Poetic Talent Unmasked by Treatment of Parkinson's Disease', 1176.
[35] R. Walker e.a., 'Augmentation of Artistic Productivity in Parkinson's Disease', *Movement Disorders* 21:2 (2006) 285-286.
[36] Ibidem, 285.
[37] Ibidem, 286.

considering the pros and cons of defining it as an obsessive compulsive disorder.[38] CSD, the Chatterjee team's patient, was a 68 year old graphic designer whose motor symptoms had progressed over 15 years from 1992 while his agonist and Levadopa medications had been adjusted to compensate. A psychologist encouraged CSD to start painting as a therapeutic activity to relieve his depression in 2002. While initially producing work derivative of Van Gogh within a year he had begun producing uniquely original vibrant colored pencil abstract works using fine regular lines. He used his right hand in which his resting tremor was severe enough to significantly restrict his capacity to write. But his artistic work used large amplitude proximal movements [reaching rather than distal movements used in writing and grasping] that allowed him to feel 'a sense of bursting forth and tearing back walls'. CSD expressed an urgency to produce and utilized the sleep disruption produced by PD to awake early and get to work. His complete immersion into a visual image prevented him, he noted, however from working on more than one painting at a time. While Chatterjee e.a. note that CSD's preoccupation with his art could be interpreted as a compulsive obsessive disorder equivalent to addictive gambling they allow his own words to express its meaning for him:

> The train has left the station and I have just been served a delicious dinner in the café car. The train is picking up speed so I have to eat fast so I can finish my meal before we get to the last stop and I have to get off.[39]

The Chatterjee team, and Schrag and Timble were reticent to interpret their patients' artistic creativity as a pathological symptom. By the end of the decade, however, such clinical reticence had been overwhelmed by a neuro-psycho-molecular diagnosis of creativity in Parkinson's patients as a disabling pathology resulting from excessive dopamine levels in the VTA pathway producing addictive behavior. Jaime Kullisevsky, Javier Pagonabarraga and Merce Martinez-Corral from the University of Barcelona exemplify the diagnostic model of Parkinson's creativity as addiction.[40]

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[38] A. Chatterjee e.a., 'Art produced by a patient with Parkinson's disease', *Behavioral Neurology* 17 (2006) 105-108.

[39] Chatterjee e.a., 'Art produced by a patient with Parkinson's disease', 106.

[40] J. Kullisevsky e.a., 'Changes in aritistic style and behavior in Parkinson's disease: dopamine and creativity', *Journal of Neurology* 256 (2009) 816-820.

Kullisevsky began by noting the earlier reports by Walker, Scharg and Chatterjee. But in the case they reported their patient was a forty seven year old amateur painter before being diagnosed with PD whose interest in art diminished after he was first medicated and became depressed with mild apathy. He retired from his employment and nothing changed in his behavioral profile until two years later the dopamine agonist cabergoline was added to his levadopa treatment. His motor symptoms significantly improved and he began painting again but with a dramatically changed attitude toward his artistic endeavor. Prior to PD he took months to complete a painting and his work was detailed and figurative focused on accurately depicting reality. Now he started to produce one painting a week and his paintings grew more and more impressionistic emphasizing color and light rather than detail. The patient explained the change as a need to 'express his refreshed inner emotions'. Within nine months he painted everyday and started showing and selling his work in local galleries in Barcelona.[41]

Kulisevsky e.a. focused, however, on what they perceived as a degeneration into addiction as their patient began to continue painting through the night and his work pattern became disruptive to his family. They considered that his negative response to suggestions that he should stop painting was a symptom of his addiction to the activity. The patient regarded his art work as 'positive for him as he was able to move more easily and he felt emotionally relieved'[42] The Kulisevsky team added that no other behavioral changes occurred such as hypersexuality, domanine dysregulation syndrome or other impulsive behaviors.

Because they considered the patient's artistic focus socially disruptive they withdrew cabergoline over a period of six weeks during which the patient notably decreased his art work and became increasingly apathetic and depressed. They then increased levadopa but the patient did not resume painting. Then they decided to reinstitute cabergoline therapy at 4 mg per day and their patient immediately began painting again with the same intensity. They reduced cabergoline to 2 mg per day and he stopped painting at night. The clinical team believed they finally got the right therapy to manage what they interpreted as continuous dopaminergic stimulation

[41] Kullisevsky e.a., 'Changes in aritistic style and behavior in Parkinson's disease', 817.
[42] Ibidem.

through dopamine agonists producing 'a pathological usurpation of the neural mechanisms of creativity'.[43]

Using artistic creativity production as a measure of impulsive predisposition in Parkinson's patients has now become a standard methodology. Artistic work redefined as a Cloninger novelty seeking category has become firmly located within the dopaminergic pathway of addiction.

Fig. 3: G.M. Spurll, 'Zen and the art of painting', *Canadian Medical Association Journal*, 181.8 (2009) 175-179.

The research undertaken by an international team led by Atbin Djamshidian from Reta Lila Western Institute and University College London exemplifies this.[44] The Djamshidian team reported on their investigations the measurable 'proneness' of one group of PD patients over another to compulsive novelty seeking compared to healthy controls.[45]

[43] Ibidem, 819.
[44] A. Djamshidian e.a., 'Novelty seeking behaviour in Parkinson's disease', *Neuropsychologia* 49 (2011) 2483-2488.
[45] Ibidem.

One component of addictive proneness has been identified by Petra Schwingenschuh and her team at University College London and the Queen Square Institute as being a professional artist prior to the diagnosis of PD.[46] When pre-morbid PD patients defy the Cloninger/Menza stereotypical low creative, harm avoiding, unimaginative profile then they are likely to acquire 'Dopamine Dysregulation Syndrome' during therapy, i.e. self-overdosing and strategic deception to obtain more dopamine than they needed according to the Schwingenschuh team. The Schwingenschuh team report on four patients who had all been successful visual artists before diagnosis who developed dopamine addiction while being treated. They explain:

> Artists require a certain stimulus and drive to produce new and creative work. In the cases described here, we suggest that the compulsive seeking of, and overdosing with, dopaminergic drugs may arise from the patients' need to fuel this drive and enhance their creativity.[47]

Creative Disability

Creativity has become disabling in the neuro-psycho-biology of novelty seeking as hectic pathological compulsion and addiction. The neuro-pscho-bio narrative on Parkinson's and creativity dramatically contrasts, however, with a much longer established published discourse by patients about their transformative experiences of illness leading to creative ways in which to experience what it means to be human. Parkinson's patients' discourses describe the impact of illness upon their creativity in terms of humanistic meaning rather than biologically coded spirituality.

Johanne Vermette exhibited her paintings in an exhibition of Parkinson's patients art and writing mounted at the McGill Centre for Studies in Montreal in Aging in 2001.[48] Vermette was diagnosed with PD in

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[46] P. Schwingenschuh e.a., 'Artistic Profession: A Potential Risk Factor for Dopamine Dysregulation Syndrome in Parkinson's Disease?', *Movement Disorders* 25.4 (2010) 493-496.

[47] Ibidem, 493

[48] Johanne Vermette: MD, Parkinson's Patient, Artist, Susan Pinker, 'Art Movements', *Canadian Medical Association Journal* 166.2 (2002) 224. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC99284/ geraadpleegd, 22 januari 2002.

her late thirties. She is a professional artist, a mother and a physician. She believed that her painting style became enhanced since her diagnosis:

> The new style is less precise but more vibrant (…) I have a need to express myself more. I let myself go, sometimes painting with enraged fingers.[49]

Vermette is familiar with the theoretical view about the influence of her medication on her imagination but she clearly doesn't care. She perceives her practice of painting through the night as a way to manage the chronic insomnia that accompanies Parkinson's and allows her undisturbed time while her children sleep. Vermette sums up the experience of enhanced creativity as 'When I paint I'm in the best place, because I am only doing that. There's no planning, no hierarchy of actions, but just the urgency of living'. Urgency to paint for Vermette is not characterized as compulsive addiction but rather as calming release.

Similarly, in contrast to creativity defined as pathologically hectic compulsive addiction in the neuro-psycho-molecular paradigm, Parkinson's patient Gwendoline Spurll who is a hematologist at McGill University argues that artistic expression has given her a new Zen like quality in her life generally including her clinical practice often commented upon by her patients.[50] Spurll began painting in 2003 prior to her PD diagnosis when she focused on expressing the desolation she had felt during a period of her life twenty years earlier while struggling with infertility. Following her diagnosis of PD in 2005 she started to focus on the dialectics of joy and dismay.[51]

Dismay that her Parkinson's will one day force her to end her clinical career and probably leave her home and joy that she will at least be able to continue to paint probably throughout the duration of her degenerative condition:

> Does painting affect my practice as a physician', Spurll asks. 'I think so. My patients often describe my bedside manner as 'calm, even Zen.[52]

[49] S. Pinker, 'Art Movements', *Canadian Medical Association Journal* 166.2 (2002) 224.
[50] G.M. Spurll, 'Zen and the art of painting', *Canadian Medical Association Journal* 181.8 (2009) 175-179.
[51] Ibidem.
[52] Spurll, 'Zen and the art of painting', 178.

Spurll accounts for this as a sort of transference of her own experience in sitting down with a blank canvass and starting to paint. For her it induces, 'a calm state in my mind that removes me from the imperfections of my life – and it convinces me that patients can likewise accept whatever confronts them. Apparently this conviction is communicated to my patients through my calmness'.[53]

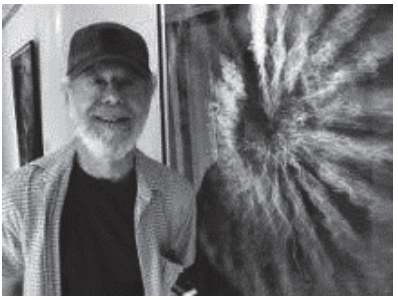

Fig. 4: Allan Babbit, 'Movement Disorder. Un-still Photography', http://www.abproductions.com/movement_disorder/movement_disorder_ss.html (21 augustus 2014).

Spurll reflects on the relationship of the onset of Parkinson's and the emergence of her creative interest in painting. Citing Sacks discussion of the disruption of self-expression inhibition in neurological patients with Frontal Lobe lesions such as Frontal Lobe Dementia she ponders whether this effect noted in Parkinson's patients may also be happening to herself.
'What does this mean?' Spurll asks, 'I don't know'. What she does know is that painting 'induces a flow state, and removes from my consciousness the constant knowledge of my physical limitations. It is like a meditation. It is my Zen'.[54]

A comparable patient experience was reported by Georg Ebersback, at the Beelitz-Heilstatten Parkinson's Institute in Germany, of a 61 year old woman who created a series of abstracted colored photographs

[53] Spurll, 'Zen and the art of painting', 178.
[54] Ibidem.

to express the meaning and help her manage hallucinations produced by her Parkinson's drug therapy.[55]

For Alan Babbitt, however, the relationship between his Parkinson disease and his creativity has nothing to do with therapeutics.[56] For Babbitt Parkinson's became an opportunity in much the same way as Henry Matisse described the impact of illness upon his artistic life-world over a century ago. For Babbitt PD stimulated the creation of an entirely new artistic genre which he calls Tremor Enhanced Photography.[57]

Babbitt has been a locally renowned San Francisco Bay Area Artist since the 1970s. And his initial interest in photography stemmed from the transformative visual experience it gave him having impaired vision from albinism, nystagmus, lazy-eye and profound myopia. When his mother and father gave him a Brownie Hawkeye camera at the age of seven for the first time he could see details close-up. Babbitt describes something far more profound, however, than just being liberated from his myopia:

> But then I discovered I could use it [photography] to see things people with good vision couldn't. Now that was satisfying! Turns out the truth is, when it comes to photography how well you see has only a little to do with how well you 'see'. [58]

Babbitt makes fun of his 'addiction' to photography admitting to becoming shamelessly 'high' on it, breaking trespass laws and getting a paper round in order to feed his habit. When the movement disorders of Parkinson's began to appear in 2003 for the first time 'darkness filled my viewfinder'. He lost interest in photography, rarely venturing out to shoot and bored with the results. One night on the Las Vegas Strip, 'of all places' he says in disbelief, 'I began to see the light (…) I began to let the tremor have its way as I clicked the shutter.'

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[55] G. Ebersbach, 'An Artist's View of Drug-Induced Hallucinosis', *Movement Disorders* 18.7 (2003) 833-834.

[56] See Fig. 7: Alan Babbitt with 'Ferris Wheel'.

[57] A. Babbit, 'Movement Disorder. Un-still Photography', http://www.abproductions.com/movement_disorder/movement_disorder_ss.html , geraadpleegd juni 2010.

[58] A. Babbit, 'The Photographer with Lousy Vision. Confessions of a Photo Addict', http://www.abproductions.com/about/about.html, consulted june 2010.

The experience was, 'Whoa!!!' 'The visual gumbo of flashing lights, muli-tinted neon and watery reflections became wonderful smears, blurs and streaks of color. Now THIS was fun!'[59]

Beyond the sheer exuberance of the work Babbitt describes an epistemological shift of understanding photography and PD.
'Liberated from the photo dogma of 'still'ness and sharpness, I've experienced a rush of creative freedom like never before. Rules? Who needs rules?' Babbitt puts it this way: 'The damn disease has given me a terrific gift!'[60]

Conclusion

I have argued that a behavioral paradigm of a genetically determined Parkinson's personality developed from the end of the twentieth century which identified the existence of pre-morbid patients with symptomatic low creativity and high harm avoidance using Cloninger [and Cloninger's subsequently derived] psychobiological dopaminergic measurement. As a result, when the genetically predisposed low creativity paradigm of the Parkinson's personality is disrupted by artistic expression in patients, creativity is redefined neuromolecularly as a pathological symptom of excessive dopamine stimulation and addiction.

Finally I have argued that the neuromolecular psychobiological characterization of the relationship between creativity and Parkinson's patients has a counter culture represented in the discourses articulated by patients of their experiences. In patient narratives, replacing ontological stasis with emergence changes the relationship between embodiment and becoming. As a result sufferers have changed the material experience of disability by creating the meaning of shifting embodiments for themselves and others. For example, by engaging, expressing and disseminating the dialectics of dread and subliminal exquisiteness through artistic work, unfolding the consequences of the mis-folding proteins that are causing their degenerative physical change.

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[59] Allan Babbit, 'Movement Disorder. Un-still Photography', http://www.abproductions.com/movement_disorder/movement_disorder_ss.html ,consulted 21 augustus 2014.

[60] A. Babbit, 'The Photographer with Lousy Vision. Confessions of a Photo Addict', http://www.abproductions.com/about/about.html, consulted june 2010.

If the meaning of disablement is disembodied then the essentialism of stigma – including the stigma of objectified pity might also be undermined. In Parkinson's patients' accounts of their experience of creativity, the doyen of disability theory, Paul Longmore's, vision of a new epistemology of ability becomes a material possibility.[61] For Parkinson's patients engaged in creative artistic work the essentialist concept of disability disappears into limitless opportunities for creating meaning not out of being but out of incessantly becoming human. And here, perhaps, the contemporary focus of post-humanist theory on emergent ontology may be of some interpretive value.[62] If post humanist theory has any value for interpreting the history of Parkinson's disease it is that it interrogates the stifling suffocating stability of the conceptualization of humanness in humanism.[63] If troubling ontology with emergent becoming can disrupt cultural essentialisms regarding Parkinson's disease then perhaps such ideational shifts can impact the clinical encounter in which it is defined and managed. Perhaps undermining cultural essentialisms and scientific reductionism might lend legitimacy to a broader expansive therapeutic creativity. In such a self-reflective clinical imaginary, counter intuitive therapeutics may garner less ridicule and gain more traction such as encouraging people with motor disorders to dance as prefigured by the Leonard Lowe character in *Awakenings*. Only this time the therapeutic treatment which is currently being reported in the clinical literature, is specifically the Tango cure for retropulsive gaits.[64]

[61] P.K. Longmore, *Why I Burned my Book and Other Essays on Disability* (Philadelphia 2003).

[62] C. Wolfe, *What is Post-humanism* (Minneapolis 2010); N. Badmington ed., *Post-Humanism* (Palgrave 2000).

[63] C. Wolfe, 'Introduction', *What is Post-humanism* (Minneapolis 2010) xi-xxiv.

[64] M.E. Hackney e.a., 'Effects of Dance on Movement Control in Parkinson's Disease: A Comparison of Argentine Tango and American Ballroom', *Journal Rehabilitation Medicine* 41.6 (2009) 475-481.