

# The Relationships between Gait Impairments and Activity Limitations in People with Depressive and Related Disorders Include: Depressive Pseudodementia, Hypochondriasis, Factitious Disorder, Cognitive Dysfunction and Normal Pressure Hydrocephalus

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## Abstract

According to Katzenschlager and Pirker walking speed is a sensitive indicator of general health status and it associated with life expectancy in older adults [1]. Accurate pathology diagnosis is the most important among patients exhibiting neurological disorders of gait. Several studies have investigated the relationships between gait dysfunction and participation restrictions in people with various neurological disorders. Studies show that depression has been associated with increased risk of gait impairments. This study reviewed existing evidence on gait dysfunction in neurological disease, including Depressive, Depressive Pseudodementia, Hypochondriasis, Factitious disorder, Cognitive dysfunction and Normal Pressure Hydrocephalus. The aim of this research study is to review the various neurological factors particularly relevant to depression diseases, affecting gait impairments.

**Keywords:** Gait Impairments; Depressive Pseudodementia; Hypochondriasis; Factitious Disorder; Cognitive Dysfunction and Normal Pressure Hydrocephalus.

## Introduction

According to Pirker and Katzenschlager 'the causes of gait disorders include neurological conditions (e.g. sensory or motor impairments), orthopedic problems (e.g. osteoarthritis and skeletal deformities) and medical conditions (e.g. heart failure, respiratory insufficiency, peripheral arterial occlusive disease and obesity)'(Pirker, et al.) [1]. Meanwhile, 'neurological gait disorders are a common cause of falls and mortality, particularly amongst the elderly' (Kaski and Adolfo, et al.) [2]. This study reviews the neurological aspects of gait impairments (Tan, et al. & Moon, et al.), with emphasis on Depressive (Brandler, et al. & Laboni and Flint, et al.), Depressive Pseudodementia (Brenner, et al.), Hypochondriasis, (Billier, et al.) Factitious disorder (Billier, et

al.), Cognitive dysfunction and Normal Pressure Hydrocephalus [3,4,5,6,7,8].

## Depressive

In 2013 Brandler and et al 'conducted a crosssectional study of the relationship between depressive symptoms and gait function, in an ambulatory community residing sample of 610 older adults (age 70 and older) who were free of dementia and MDD'(Major depressive disorder) (Brandler, et al.) [5]. They made conclusion that 'increasing depressive symptoms in community residing older adults are associated with quantitative gait dysfunction even in the absence of major depression or dementia' (Brandler, et al.) [5]. Simultaneously, Lord and et al have found that 'very mild depressive symptoms are associated with gait disturbance in early Parkinson's disease' (Lord, et al.) [9]. In other study (Hausdorff, et al.) have made conclusion that 'patients with MDD and patients with bipolar disorder display gait unsteadiness' (Hausdorff, et al.) [10].

## Genetic factors

'Recent studies have demonstrated impaired balance performance in patients with major depressive disorder (MDD)'(Deschamp, et al.) [11]. Multiple 'genetic factors play important roles in the development of MDD' (Major depressive disorder) according to Lohoff (Lohoff, et al.). 5HTT, 5HTTLPR (Brown & Harris, et al.) and SLC6A4 genes associate with MDD across numerous studies (e.g. Brown, et al., Luddington, et al., Kuzelova, et al.) [12,13,14].

## Depressive Pseudodementia

According to Kennedy: Depressive Pseudodementia is a term commonly used to describe a condition whereby a

patient experiences a cognitive deficit secondary to a primary mood disorder. (Kennedy, et al.) [15]. 'Cognitive deficits in Pseudodementia are characterized by poor effort and difficulties with attention and working memory' (Flemming and Jr, et al.) [16]. In other word 'Pseudodementia is a situation where a person who has depression also has cognitive impairment that looks like dementia' ((Steckl C, et al), Reversible Cognitive Disorder Pseudodementia) [17]. However, there are many studies showing that gait impairments coexist with depressive pseudodementia.

### Genetic Factors

'Long repeat sequences in the C9ORF72 gene have cropped up in cases of multiple system atrophy (MSA) and depressive pseudodementia' (ALZFORUM, et al.), C9ORF72 Repeats Expand into New Disorders Cause, or Coincidence?) according to Bieniek, et al. [18].

### Hypochondriasis

According to Wilhelmsen Hypochondriasis (Illness anxiety disorder and Somatic symptom disorder) 'describes a persistent preoccupation with the possibility of having one or more serious and progressive physical disorders' (Wilhelmsen, et al.), and 'defined as a chronic condition distinct from anxiety and depressive disorders' (Simon, et al) [19,20]. Hypochondriasis is distinguishable clinical condition (Hiller, et al.) however, according to (Weck, et al.) [21,22]. 'previous experiences with illness and traumatic childhood experiences did not prove to be specific risk factors for the development of hypochondriasis' Hypochondriasis can be accompanied by Major Depressive Disorder (MDD) (Kapfhammer, et al.). According to Diagnostic and Statistical Manual of Mental Disorders (DSM5) 'hypochondriasis and several related conditions have been replaced by two new, empirically derived concepts: somatic symptom disorder and illness anxiety disorder' (Mayo Clinic.) [23]. There is a strong correlation between the number of somatic symptoms and the likelihood of a depression or anxiety diagnosis (Croicu, et al.).

### Genetic factors

In 2010 Holliday and et al found that: HTR2A, SERPINA6 and TPH2 are associated with somatic symptoms score [24].

### Factitious disorder

According to (Uzuner, et al.) 'factitious disorder is characterized by deliberate production or imitation of physical or psychological symptoms in order to adopt the sick role' [25]. 'The etiology of factitious disorder is unclear' (Osterman, et al.), however, 'in the sense that the physical symptoms are prominently contributed by psychological factors, all somatoform disorders may be considered to be a subset of psychological factors affecting a physical condition' (Leight, et al.) [26,27]. According to Guzman and Correll 'patients with factitious disorder and comorbid depression have shown improvement with antidepressant therapy in addition to psychotherapy' [28].

### Genetic factors

Some studies report genetic estimates in factitious disorder:

According to Jaghab and et al (2006) 'magnetic resonance imaging (MRI) has detected abnormalities in the brain structure of some patients with chronic FD, suggesting that there may be biological or genetic factors in the disorder' [29]. In recent study Kreisl, et al. 'describe the novel constellation of a factitious disorder presenting as a supposedly genetically confirmed hereditary disease manifesting with abnormal movements' [30].

### Cognitive Dysfunction

Cognitive dysfunction refer to deficits in attention and motor perception, learning disability, shortterm and working memory and processing speed, problem solving functions, visual or auditory processing deficits (Lam, et al.) [31]. According to Stout and Paulsen 'changes to the motor system resulting from central nervous system damage or disease are virtually always accompanied by cognitive dysfunction' [32]. There are a variety of different types of cognitive dysfunction e.g. ALPS or Adultonset leukoencephalopathy with axonal spheroids and pigmented glia, cognitive decline, mild cognitive impairment (Bahureksa, et al.), and Alzheimer's disease dementia (Dorfman, et al.) [33]. Cognitive dysfunction is a common feature of Adultonset leukoencephalopathy with axonal spheroids and pigmented glia (ALSP) (Adams, et al.) [34]. This disease is recognized as both a cognitive and a movement disorder (Ikeuchi and et al 2017). Other feature of cognitive dysfunction is Mild Cognitive Impairment (MCI). In 2015 Callisaya and et al conducted a study to determine the relationships between cognitive decline and gait slowing. They made conclusion that 'decline in nonamnestic function (specifically executive function) was associated with decline in gait speed irrespective of the presence of baseline cognitive impairment' (Callisaya and et al 2015) [35]. In other study in 2017 Bahureksa and et al made a conclusion that existing studies 'provide evidence that Mild Cognitive Impairment (MCI) affects specific gait parameters' (Bahureksa, et al.) [36]. There are evidences of a direct cause relationship between the mild cognitive impairment and the development of Alzheimer's disease (Roed, et al.).

### Genetic factors

According to (Foulds, et al.) 'AdultOnset Leukoencephalopathy with Axonal Spheroids and Pigmented Glia (is) caused by a novel R782G mutation in CSF1R' [37].

### Normal pressure hydrocephalus (NPH)

According to Shprecher and et al (2008) 'normal pressure hydrocephalus (NPH) is a syndrome of gait dysfunction and enlarged cerebral ventricles in the absence of another cause' [38]. The characteristic psychiatric symptoms of normal pressure hydrocephalus are dementia and depression (Chopra, et al.) [39]. In 2016 (Israelsson, et al.) claimed that 'in many dementias, depression is overrepresented, but the prevalence of depression in shunted patients with idiopathic normal pressure hydrocephalus is unknown' [40].

### Genetic factors

There are increasing evidence that iNPH may have a genetic

component (Korhonen, et al.). In 2016 (Sato, et al.) demonstrated that a 'copy number loss in intron 2 of the SFMBT1 gene may be a genetic risk for shuntresponsive definite iNPH' [41-45].

## Methods and Materials

The narrative reviews of the literatures were conducted with combination of relevant keywords. We searched the IEEE, ScienceDirect, Springer, OMICS, PubMed, PubMed Health and FINNA databases.

## Results and Conclusion

There are convincing evidences about the relationship between gait impairments and Depressive, Depressive Pseudodementia, Hypochondriasis, Factitious disorder, Cognitive dysfunction and Normal Pressure Hydrocephalus are improving.

## Discussion

This study was reviewed literatures including biological and psychological aspect. However, Kinesigraphy is important functional gait imaging modalities for study the limb mobility and neural activities. Kinesigraphy is the interpretation of limb moment into mathematical form. Combining Kinesigraphical information with biological and psychological aspect of patient holds promise to produce and improve clinical facility. In this manner we can apply the image matching methods for the evaluation of gait impairments in patients with Depressive and related disorders.

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1. Pirker W, Katzenschlager R. Gait disorders in adults and the elderly A clinical guide, Wien Klin Wochenschr, 2017;129(3):81-95. doi: 10.1007/s0050801610964.
2. Kaski D and Bronstein AM. Treatments for Neurological Gait and Balance Disturbance: The Use of Noninvasive Electrical Brain Stimulation. Advances in Neuroscience. 2014;1-13. Doi: org/10.1155/2014/573862
3. Moon Y, Sung J, An R, Hernandez ME, Sosnoff JJ. Gait variability in people with neurological disorders: A systematic review and meta analysis, Hum Mov Sci. 2016;47:197-208. doi: 10.1016/j.humov.2016.03.010.
4. Tan D, Danoudis M, McGinley J, Morris ME. Relationships between motor aspects of gait impairments and activity limitations in people with Parkinson's disease: a systematic review, Parkinsonism Relat Disord, 2012;18(2):117-124. doi: 10.1016/j.parkreldis.2011.07.014.
5. Brandler TC, Wang C, OhPark M, Holtzer R, Verghese J. Depressive Symptoms and Gait Dysfunction in the Elderly. Am J Geriatr Psychiatry. 2012;20(5):425-432. Doi: 10.1097/JGP.0b013e31821181c6.
6. Laboni A, Flint AJ. The Complex Interplay of Depression and Falls in Older Adults: A Clinical Review, Am J Geriatr Psychiatry. 2013; 21(5):484-492. doi: 10.1016/j.jagp.2013.01.008
7. Brenner RP, Reynolds CF, Ulrich R. EEG findings in depressive pseudodementia and dementia with secondary depression. Electroencephalography and Clinical Neurophysiology. 1989;72(4):298-304.
8. Biller J and Espay AJ. Practical Neurology Visual Review. Lippincott Williams & Wilkins, a Wolters Kluwer business. 2013;
9. Lord S, Galna B, Coleman S, Burn D, Rochester L. Mild depressive symptoms are associated with gait impairment in early Parkinson's disease, 2013;28(5):634-639.
10. Hausdorff JM, Peng CK, Goldberger AL, Stoll AL. Gait unsteadiness and fall risk in two affective disorders: a preliminary study. BMC Psychiatry. 2004;4:39. Doi: 10.1186/1471244X439
11. Deschamps T, Ollivier VT, Sauvaget A, Bulteau S, Bourbousson MF, Vachon H. Balance characteristics in patients with major depression after a twomonth walking exercise program: A pilot study. 2015;42(4):590-593. Doi: org/10.1016/j.gaitpost.2015.07.057.
12. Lohoff FW. Overview of the Genetics of Major Depressive Disorder, Curr Psychiatry Rep. 2010;12(6):539-546. doi: 10.1007/s1192001001506
13. Brown GW, Harris TO. Depression and the serotonin transporter 5HTTLPR polymorphism: a review and a hypothesis concerning geneenvironment interaction. J Affect Disord. 2008;111(1):1-12. Doi: 10.1016/j.jad.2008.04.009.
14. Luddington NS, Mandadapu A, Husk M, Mallakh R. Clinical Implications of Genetic Variation in the Serotonin Transporter Promoter Region: A Review, Prim Care Companion J Clin Psychiatry. 2009;11(3):93-102.
15. Kennedy James 2015, Depressive pseudodementia - how 'pseudo' is it really? Old Age Psychiatrist 62.
16. Flemming KD and Jones Jr LK. Mayo Clinic Neurology Board Review: Clinical Neurology for Initial Certification and Moc. OXFORD UNIVERSITY PRESS. 2015;
17. Steck C. Reversible Cognitive Disorder - Pseudodementia. 2008;
18. ALZFORUM. C9ORF72 Repeats Expand into New Disorders-Cause, or Coincidence? 2014;
19. Wilhelmson . Hypochondriasis or Health Anxiety Encyclopedia of Human Behavior (Second Edition), 2012;385-391.
20. Simon, Gureje, Fullerton. Course of hypochondriasis in an international primary care study, General Hospital Psychiatry, 2001;23(2):51-55.
21. Hiller W, Leibbrand R, Rief W, Fichter MM. Differentiating hypochondriasis from panic disorder. J Anxiety Disorder. 2005;19(1):29-49. doi:10.1016/j.janxdis.2003.10.006
22. Weck F, Neng J, Göller K, Marbach M. Previous Experiences With Illness and Traumatic Experiences: A Specific Risk Factor For Hypochondriasis? Psychosomatics, 2014;55(4):362-371.
23. MayoClinic. Clinical updates. 2018;
24. Holliday KL, Macfarlane GJ, Nicholl BI, Creed F, Thomson W, McBeth J. Genetic variation in neuroendocrine genes associates with somatic

- symptoms in the general population: Results from the EPIFUND study. *J Psychosom Res.* 2010;68(5):469-474. doi:10.1016/j.jpsychores.2010.01.024
25. Uzuner S, Bahali K, Kurban S, Erenberk U, Cakir E. A pediatric case of factitious disorder with unexplained bleeding symptoms, *General Hospital Psychiatry*, 2013;35(6):679.
26. Osterman M, Koman LA, Osterman AL. Factitious Disorders, Conversion Reaction, and Malingering in Children, *The Pediatric Upper Extremity*. 2014;1602-1617.
27. Leigh H. Psychological Factors Affecting Physical Conditions, Somatoform Disorders, Conversion, Dissociation, and Factitious Syndromes, *Handbook of Consultation Liaison Psychiatry*. 2017;141151.
28. Guzman J and Correll T. Factitious Disorder. *Psychiatry*. 2008;11.
29. Jaghab K, Skodnek KB, Padder TA. Munchausen's Syndrome and Other Factitious Disorders in Children Case Series and Literature Review. *Psychiatry (Edgmont)*. 2006;3(3):46-55.
30. Kreisl WC, Lawrence R, Page E, Turner RS. 11 CPBR28 PET detects translocator protein in a patient with astrocytoma and Alzheimer disease. 2017; doi: 10.1212/WNL.0000000000003693.
31. Lam RW, Kennedy SH, McIntyre RS, Khullar A. Cognitive Dysfunction in Major Depressive Disorder: Effects on Psychosocial Functioning and Implications for Treatment, *Can J Psychiatry*. 2014;59(12):649-654. doi: 10.1177/070674371405901206
32. Stout JC, Paulsen JS. Assessing Cognition in Movement Disorders, *Mental and Behavioral Dysfunction in Movement Disorders*. 8599.
33. Dorfman M, Mirelman A, Hausdorff JM, Giladi N. Gait Disorders in Patients with Cognitive Impairment or Dementia. *Movement Disorders in Dementias*. 2014; 17-44. Doi: org/10.1007/978-1-4471-6365-72
34. Adams SJ, Kirk A, Auer RN. Adultonset leukoencephalopathy with axonal spheroids and pigmented glia (ALSP): Integrating the literature on hereditary diffuse leukoencephalopathy with spheroids (HDLS) and pigmentary orthochromatic leukodystrophy (POLD). *J Clin Neurosci*. 2018;48:42-49. Doi: 10.1016/j.jocn.2017.10.060.
35. Michele L, Christopher LC, Amanda GB, Amanda GW, Wardill TT, Srikanth VK. Longitudinal Relationships Between Cognitive Decline and Gait Slowing: The Tasmanian Study of Cognition and Gait. *The Journals of Gerontology*. 2015;70(10):1226-1232. doi:https://doi.org/10.1093/gerona/glv066.
36. Bahureksa L, Najafi B, Saleh A, Sabbagh M, Coon D, Mohler MJ. The Impact of Mild Cognitive Impairment on Gait and Balance: A Systematic Review and MetaAnalysis of Studies Using Instrumented Assessment. *Gerontology*. 2017;63(1):67-83 Doi.org/10.1159/000445831.
37. Foulds N, Pengelly RJ, Hammans SR, Nicoll JA, Ellison DW, Ditchfield A, et al. AdultOnset Leukoencephalopathy with Axonal Spheroids and Pigmented Glia Caused by a Novel R782G Mutation in CSF1R. *Sci Rep*. 2015;5:10042. Doi: 10.1038/srep10042
38. Shprecher D, DO, Schwalb J, Kurlan R. Normal Pressure Hydrocephalus: Diagnosis and Treatment, *Curr Neurol Neurosci Rep*. 2008; 8(5):371-376.
39. Chopra VK, Sinha VK, Das S. Normal pressure hydrocephalus presenting as psychotic depression : moderately successful treatment with a course of ect & pharmacotherapy : a case report. *Indian J Psychiatry*. 2002;44(1):71-75.
40. Israelsson H, Allard P, Eklund A, Malm J. Symptoms of Depression are Common in Patients With Idiopathic Normal Pressure Hydrocephalus: The INPHCRasH Study. *Neurosurgery*. 2016;78(2):161-168. Doi: 10.1227/NEU.0000000000001093
41. Sato H, Takahashi Y, Kimihira L, Iseki C, Kato H, Suzuki Y, et al. A Segmental Copy Number. 2016;
42. Hamilton M. Frequency of symptoms in melancholia (depressive illness). *Br J Psychiatry*. 1989;154:201-206.
43. Katon W, Lin EH, Koneke K. The association of depression and anxiety with medical symptom burden in patients with chronic medical illness. *Gen Hosp Psychiatry*. 2007;29(2):147-155.
44. Loss of the SFMBT1 Gene Is a Genetic Risk for Shunt Responsive, Idiopathic Normal Pressure Hydrocephalus (iNPH): A CaseControl Study, *PLOS ONE*. doi:10.1371/journal.pone.0166615.
45. Studenski S, Perera S, Patel K, Rosano C, Faulkner K, Inzitari M, et al. 'Gait Speed and Survival in Older Adults', *JAMA*, 2011;305(1): 50-58. doi: 10.1001/jama.2010.1923.

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