

Letter to the Editor

Relationship between Parkinson Disease and Nocardial Infections

Masood Ghorbani³, Mehdi Marashi², Mehdi Fatahi Bafghi^{1*}

¹Applied Biotechnology Researches center, Pajooheshgah, Baqiatallah University of Medical Sciences, Tehran, Iran

²Fachhochschule Frankfurt am Main, University of Applied Sciences, FB 4: Soziale Arbeit & Gesundheit, Pflege-Advanced Practice Nursing Nibelungenplatz 1, D-60318 Frankfurt am Main, Germany

³Department of Pathobiology, School of Public Health, Tehran University of Medical Sciences, Tehran, Iran

*Corresponding author: Mehdi Fatahi Bafghi, Department of Microbiology, School of Public Health, Tehran University of Medical Sciences, Tehran, Iran, Email: Mehdifatahi371@gmail.com

Received: March 09, 2015; Accepted: May 01, 2015;

Published: May 16, 2015

Dear Editor,

The genus *Nocardia* is Gram-positive, non-motile and partially acid-fast bacteria. These bacteria cause infection in both immunocompromised and immunocompetent individuals [1-3]. Hoped that his monograph entitled *An Essay on the Shaking Palsy*, in which he detailed six patients with “involuntary tremulous motion with lessened muscular power, in parts not in action even when supported, with a propensity to bend the trunk forward and to pass from a walking to a running pace”, would persuade neurologists that he had described an unrecognized disorder [4,5]. Jean Martin Charcot proposed that the syndrome should be called Parkinson’s disease (PD). PD is regarded as a sporadic disorder but few environmental causes or triggers have so far been recognized [6-8]. In PD, ageing is the major risk factor, although 10% of people with the disease are younger than 45 years of age. The incidence of PD seems to decrease in the ninth decade of life [7] which could be actual or related to under diagnoses of elderly people of that age, or could be a real decline. Risk of PD is more than other people [9,10] and this risk in men and postmenopausal women who are not taking hormone replacement increased [11,12]. The cause of this difference be related to dopamine’s role in reward pathways and to low premorbid novelty seeking personality traits [13] rather than to any protective effect of tobacco smoke, nicotine or caffeine [13,14]. Although some studies have shown the inverse association between risk to develop the disease and smoking [15]. In the brains of smokers oxidative stress can increase because of both nicotine and caffeine increase striatal dopamine release and the enzyme monoamine oxidase [16]. In literature were reported that infectious agents have a role as a risk factor for Parkinson’s disease for example *Helicobacter Pylori* and *Nocardia* spp. [17]. *Nocardia asteroides* reported by Eppinger of brain abscess in 1891 [18]. In a study was reported that *Nocardia asteroides* is cause syndrome similar to PD in mice. The number of literature, *Nocardia asteroides* is one of the risk factors in PD [19]. Tam *et al.* reported that *Nocardia asteroides* GUH-2 destroys substantia nigra

neurons [17]. Yassin and colleagues was first described (type VI drug pattern) in 2001 and found in the soil. *Nocardia cyriacigeorgica* may inhibit the Ubiquitin/proteasome system with produces a pigment [20,21]. Some organisms, such as *Streptomyces*, especially *Streptomyces Venezuela*, district protein degradation with produce material inhibitors [22]. In PD region-specific selective degenerate dopaminergic, neuromelanin-containing neurons from the pars compact of the substantia nigra. Cell loss in the locus coeruleus, dorsal nuclei of the vagus, raphe nuclei, nucleus basalis of Meynert and some other catecholaminergic brain stem structures including the ventro tegmental area also exists is happened [23]. In the Lewy body, the pale body and the Lewy neurite decrease nerve cells. Lewy bodies are subdivided into classical (brain stem) and cortical types on the basis of their morphology. The anatomy of brain-stem shape is a spherical structure with a hyaline core surrounded by a peripheral pale-staining halo and is composed histologically of 7–20-nm wide filaments with dense granular material and vesicular structures. Pale bodies are displacing neuromelanin and are the predecessor of the Lewy body [24]. Lewy bodies and irrespective of disease duration are part of constant proportion of nigral neurons. Lewy bodies also are continuously forming and disappearing in the diseased substantia nigra [25]. An abnormal, post-translationally modified and aggregated form of the pre-synaptic protein α -synuclein is the main component of Lewy bodies. The standard and most sensitive immuno-histochemical method for routine diagnostic purposes is α -synuclein antibodies stain Lewy bodies and Lewy neuritis. Reported that, the incidence of the disease rises steeply with age, from 17.4 in 100 000 person years between 50 and 59 years of age to 93.1 in 100 000 person years’ between 70 and 79 years with a lifetime risk of developing the disease of 1.5% [26,27]. In future, more studies are very necessary about confirm role of nocardiosis in Parkinson disease because it’s a question for researcher.

References

1. Bafghi MF, Eshraghi SS, Heidarieh P, Habibnia S, Nasab MR. Nocardiosis in immune disorder disease. *Malays J Med Sci.* 2014; 21: 75-76.
2. Bafghi MF, Eshraghi SS, Heidarieh P, Habibnia S, Nasab MR. DNA extraction from nocardia species for special genes analysis using PCR. *N Am J Med Sci.* 2014; 6: 231-233.
3. Bafghi MF, Heidarieh P, Soori T, Saber S, Meysamie A, Gheitoli K, et al. *Nocardia* isolation from clinical samples with the paraffin baiting technique. *Germes.* 2015; 5: 12-16.
4. Parkinson J. An essay on the shaking palsy. *Neurological Classics.* 1997: 88.
5. Kempster PA, Hurwitz B, Lees AJ. A new look at James Parkinson’s Essay on the Shaking Palsy. *Neurology.* 2007; 69: 482-485.
6. Tanner CM. Is the cause of Parkinson’s disease environmental or hereditary? evidence from twin studies. *Adv neurol.* 2003; 91: 133.
7. Taylor KS, Counsell CE, Gordon JC, Harris CE. Screening for undiagnosed parkinsonism among older people in general practice. *Age Ageing.* 2005; 34: 501-504.

8. Dick FD, De Palma G, Ahmadi A, Scott NW, Prescott GJ, Bennett J, et al. Environmental risk factors for Parkinson's disease and parkinsonism: the Geoparkinson study. *Occup Environ Med.* 2007; 64: 666-672.
9. Allam MF, Campbell MJ, Hofman A, Del Castillo AS, Fernández-Crehuet Navajas R. Smoking and Parkinson's disease: systematic review of prospective studies. *Mov Disord.* 2004; 19: 614-621.
10. Hernán MA, Zhang SM, Rueda-deCastro AM, Colditz GA, Speizer FE, Ascherio A. Cigarette smoking and the incidence of Parkinson's disease in two prospective studies. *Ann Neurol.* 2001; 50: 780-786.
11. Ascherio A, Weisskopf MG, O'Reilly EJ, McCullough ML, Calle EE, Rodriguez C, et al. Coffee consumption, gender, and Parkinson's disease mortality in the cancer prevention study II cohort: the modifying effects of estrogen. *Am J Epidemiol.* 2004; 160: 977-984.
12. Ascherio A, Chen H, Schwarzschild MA, Zhang SM, Colditz GA, Speizer FE. Caffeine, postmenopausal estrogen, and risk of Parkinson's disease. *Neurology.* 2003; 60: 790-795.
13. Evans A, Lawrence A, Potts J, MacGregor L, Katzenschlager R, Shaw K, et al. Relationship between impulsive sensation seeking traits, smoking, alcohol and caffeine intake, and Parkinson's disease. *J Neurol Neurosurg Psychiatry.* 2006; 77: 317-321.
14. Quik M, Jeyarasasingam G. Nicotinic receptors and Parkinson's disease. *Eur J Pharmacol.* 2000; 393: 223-230.
15. Elbaz A, Moisan F. Update in the epidemiology of Parkinson's disease. *Curr Opin Neurol.* 2008; 21: 454-460.
16. Fowler JS, Volkow ND, Wang GJ, Pappas N, Logan J, Shea C, et al. Brain monoamine oxidase A inhibition in cigarette smokers. *Proc Natl Acad Sci U S A.* 1996; 93: 14065-14069.
17. Liu B, Gao HM, Hong JS. Parkinson's disease and exposure to infectious agents and pesticides and the occurrence of brain injuries: role of neuroinflammation. *Environ Health Perspect.* 2003; 111: 1065-1073.
18. Ogata SA, Beaman BL. Site-specific growth of *Nocardia asteroides* in the murine brain. *Infect Immun.* 1992; 60: 3262-3267.
19. Hubble JP, Cao T, Kjelstrom JA, Koller WC, Beaman BL. *Nocardia* species as an etiologic agent in Parkinson's disease: serological testing in a case-control study. *J Clin Microbiol.* 1995; 33: 2768-2769.
20. Yassin A, Rainey F, Steiner U. *Nocardia cyriacigeorgici* sp. nov. *International journal of systematic and evolutionary microbiology.* 2001; 51: 1419-1423.
21. Martinot B. Separating and characterizing products generated by *Nocardia cyriacigeorgica*. *Biological.* 2009.
22. Caldwell KA, Tucci ML, Armagost J, Hodges TW, Chen J, Memon SB, et al. Investigating bacterial sources of toxicity as an environmental contributor to dopaminergic neurodegeneration. *PLoS One.* 2009; 4: e7227.
23. Damier P, Hirsch EC, Agid Y, Graybiel AM. The substantia nigra of the human brain. II. Patterns of loss of dopamine-containing neurons in Parkinson's disease. *Brain.* 1999; 122: 1437-1448.
24. Ince P CB, Holton J, Revesz T, Wharton SB. Disorders of movement and systems degenerations. 2008: 889-981.
25. Greffard S, Verry M, Bonnet A-M, Seilhean D, Hauw J-J, Duyckaerts C. A stable proportion of Lewy body bearing neurons in the substantia nigra suggests a model in which the Lewy body causes neuronal death. *Neurobiology of aging.* 2010; 31: 99-103.
26. Bower JH, Maraganore DM, McDonnell SK, Rocca WA. Incidence and distribution of parkinsonism in Olmsted County, Minnesota, 1976-1990. *Neurology.* 1999; 52: 1214-1220.
27. de Rijk MC, Breteler MM, Graveland GA, Ott A, Grobbee DE, van der Meché FG, et al. Prevalence of Parkinson's disease in the elderly: the Rotterdam Study. *Neurology.* 1995; 45: 2143-2146.