World Journal of Pharmaceutical and Life Sciences
WJPLS
www.wjpls.org
SJIF Impact Factor: 5.088

CIGARETTE SMOKING IS THE LOWER RISK OF PARKINSON DISEASE

Suresh Undati*, Madhuri Boompally, Saikumar Manneboina, Srikanth Kotari

Nicotine From Cigarette May Increases The Levels of Dopiminergic Systems.

***Corresponding Author: Suresh Undati**

Nicotine From Cigarette May Increases The Levels of Dopiminergic Systems.

Article Received on 11/07/2018 **Article Revised on 02/08/2018** **Article Accepted on 23/08/2018**

ABSTRACT

Parkinson disease is the one of the most common neurologic disorder, affecting approximately 1% of individuals over than 60 years causing progressive disability that can be slowed, but not halted, by treatment. As a major component of tobacco smoke, nicotine has been proposed to be asubstance for preventing against Parkinson disease risk. With a key role in regulating straital activity and behaviours mediated through the dopiminergic system. Animal studies also showed that nicotine could modulate dopamine transmission and reduce levadopa-enduced dysakinesias. However, previous clinical trials yield contaversial results regarding nicotine treatment. In this review, we updated epidemiological, preclinical and clinical data, and studies on nicotine from diet.

KEYWORDS: Nicotine, Parkinson disease, Cigerette smoking, Dopamine, Gene.

INTRODUCTION

Parkinson disease is an age- related neurodegenerative disorder, with a prevalence of 1-2% among adults aged 55 years and older.[1] It is charecterised by a progressive de generation of dopaminergic neurons in the substantia nigra pars compacta that results in tremor, rigidity, bradykinesia, and possibly dementia.[2] The current evidence realated to the pathogenesis of parakinson disease includes defective handling of proteins mitochondrial dysfunction, oxidative stress, and inflammation.[3-6] There is no cure for the disease and only sympathomatic relief is available. Especially in the erly stages of the disease, dopamine replacement therapies provide effective control of the motor symptoms with L- dopa as the gold standard. However, chronic L- dopa use does not adequately manage the non-motor deficits and additionally induces a variety of motor and psychiatric side effects that limits its effectiveness.[7,8] These shortcomings strengthen the importance of identifying alternate treatment stratagies that delay or halt disease progression, or ideally restore function in parkinson disease. Previous reviews reported the association between nicotine and Parkinson disease risk, but the sources were limited to cigarette smoking and medical use. We thus updated epidemiological, preclinical and clinical data, and studies on nicotine from other sources. We also reviewed interactions between genetic factors and cigarette smoking to broaden the understanding of the potential protective nicotine on Parkinson disease.

EPIDEMIOLOGY

Physicians working on parkinson diasease have found nicotine of particular interest since it has been shown that there was less Parkinson disease among smokers. It was firstly noticed 1959,[9] and more than 50 epidemiological studies have confirmed this obesrevation.[10,11] Some suggested that this association could be give either to a selective mortality of smokers.[12] (as parkinsons disease affects more middle-aged and elderly people), to the disease affecting smoking habits or to a reporting bias. However, a case-control study,[13] with 350 parkinsons disease patients and 350 matched contros showed a relative risk of 0.52,with an intermediate risk for people who had quit. Further more, huge prospective cohort studies.[14,15] brought even more credibility to the hypothesis of a protective role of smoking in Parkinson disease. A pooled analysis confirmed the negative association between Parkinson disease and smoking that was more pronounced for current smokers than former ones;[16] it also showed that there was a dose-response, with the ratio decreasing when the pack-years increase, and that other forms of tobacco also had this effect. Even passive smoking seems to be less frequent in parkinson disease than in controls.[17] Durat of smoking also seems to be more important than intensity.[18] A study in twin pairs in which at least one twin had Parkinson disease found the same negative correlation: the twins without Parkinson disease smoked significantly more than their brothers, even when smoking dose was calculated only until 10 years before Parkinson disease onset;[19] this argued against a genetic and behavioral hypothesis, as a link between genitical propensity to smoke and

susceptibility to Parkinson disesase. A recent review on nicotine addiction suggested a potential interaction between multiple small-effect alleles and the environment.[20]

Table 1: Gene-smoking interactions for Parkinson Disease.

First author, year of publication	Study participants	Gene studied	Results
Greenbaum, 2013.[21]	667 Italian PD patients	CHRNA3, CHRNA4, and CHRNA5	A significant interaction between the CHRNA5 (rs588765) and smoking status (never smokers vs. ever-smokers)
Hancock, 2006.[22]	466 singleton and 286 multiplex families in the United States	NOS2A	Significant interactions of NOS2A (rs2255929 and rs1060826) with smoking in a subset of the families
Miyake, 2012.[24]	229 PD patients and 357 controls in Japan	SNCA	Significant interactions between SNCA (rs356219 and rs356220) and smoking with respect to sporadic PD
McCulloch, 2008.[23]	932 PD patients and 664 controls in the United States	MAPT, SNCA, UCHL1, and APOE	A significant interaction between SNCA REP1 and smoking
Palma, 2010.[25]	767 PD patients and 1989 controls in Europe	CYP1B1, CYP2D6, GSTM1, GSTM3, GSTT1, GSTP1, NQ01, SOD2, EPHX, NAT2, MAOA, MAOB, DAT1, and DRD2	Significant interactions between smoking and GSTM1 Pos/Null, GSTP1 haplotype, and NAT2 Fast/slow

Except positing that nicotine is neuroprotective, inherent aversion to nicotine-containing products could be another potential explanation. To date, several genetic studies have been conducted to explore the potential interaction between smoking and genetic factors on PD risk (Table 1). For example, a study including 677 PD patients reported a significant interaction between the nicotinic cholinergic receptor gene cluster CHRNA5 (rs588765) and smoking on age of onset of PD.[21] In an early study that included 466 singletons and 286 multiplex families, significant interactions between smoking and two SNPs (rs2255929 and rs1060826) of inducible nitric oxide synthase (iNOS) gene (NOS2A) in relation to PD risk were observed.[22] One possible interpretation for this observed interaction is that cigarette smoking condensates could attenuate inflammatory induction of iNOS, and reduce its cytotoxic effects.[22] doi: 10.1016/j.parAlthough earlier results were inconsistent, recent studies showed that a dinucleotide repeat polymorphism (REP1) in the promoter region of the α-synuclein gene (SNCA) was associated with the risk of common late-onset PD.[24] For example, a case-control study with 932 PD cases showed an interaction between SNCA REP1 and smoking on the risk of PD.[23] Another recent case-control study including 229 PD cases reported that smoking significantly modified the association between SNCA polymorphisms (rs356220 and rs356219) and PD risk.[24] In addition, many other genetic polymorphisms have been implicated in tobacco-PD relation, such as SLC2A13, GSTM1, NAT2, and GSTP1, etc., but the findings are inconsistent.[25] kreldis.2012.07.007.

ACKNOWLEDGEMENTS

Not applicable.

CONCLUSION

Despite of extensive evidence from epidemiological and basic research studies suggesting that nicotine may represent an effective agent with potential for prevention and alleviation PD, clinical data vary to a major extent between individuals. Discrepancies of those controversial results can be partly explained by differences of clinical stages of PD participants and research methodologies, and may also be due to the underlying gene-environment interactions. In addition, poor tolerability and high drop-out rate are inevitable concerns in such clinical trials. As a small amount of nicotine can saturate a substantial portion of nicotine receptors in the brain, nicotine from other sources, such as diet, could be a promising therapeutic substance for protection against PD.

REFERENCES

1. Bezard E, Brotchie JM, Gross CE. Pathophysiology of levadopa-induced dyskinesia: potential for new therapies. Nat Rev Neurosci, 2001; 2: 577-588. Doi: 10.1038 by 35086062.
2. Olanow CW, Tatton WG. Etiology and pathogenisis of Parkinson's disease. Annu Rev Neurosci, 1999; 22: 123-144. Doi: 10.1146/annurev.neuro.22.1.123.
3. Bueler H. Mitochodrial dynamics, cell death and the pathogenesis of Parkinson's disease. Apoptosis, 2010; 15: 1336-1353. doiL: 10.1007/s10495-010-0465-0.
4. Zuo L, Mother well MS. The impact of reactive oxygen species and genetic, mitochondrial mutations in parkinson's disease. Gene, 2013; 532: 18-23. Doi: 10.1016/j.gene.2013.07.085.
5. Camilleri A, v assallo N. The centrality of mitochondria in the pathogenesis and treatment of parkinsons disease. CNS Neurosci Ther, 2014; 20: 591-602. Doi: 10.1111/cns.12264.
6. Celardo I, Martins LM, Gandhi S. Unravelling mitochondrial pathways to parkinson's disease. Br J Pharmacol, 2014; 171: 1943-1957. Doi: 10.1111/bph.12433.
7. Connolly BS, Lang AE. Pharmacological treatment of Parkinson disease: a review. JAMA, 2014; 311: 1670-1683. Doi: 10.1001/jama.2014.3654.
8. Huot P, Johnston TH, Koprich JB FOX SH, Brotchie JM. The pharmacology of L-DOPA-induced dyskinesia in parkinson's disease. Pharmacol Rev., 2013; 65: 171-222. Doi: 10.1124/pr.111.005678.
9. Elbaz AMF. (Parkinson disease:Is there a strong environmental contribution?) Rev. Neurol. (Paris), 2010; 166; 757-763.
10. Quick M. Smoking,nicotine and parkinsons disease. Trends Neurosci, 2004; 27: 561-568.
11. Quick M, Huang LZ, Parameswaran N, Bordia T, Campos C, Perez XA. Multiple roles for nicotine parkinsons disease. Biochem. Pharmacol. Exellent and up-to-date coverage of the literature about experimental results with nicotine, 2009; 78: 677-685.
12. Quick M, O' Leary K, Tanner CM. Nicotine and parkinsons disease: implications for therapy. Mov. Tisord, 2008; 23: 1641-1652.
13. Kas A, Bottlaender M, Gallezot JD et al. decrease of nicotinic receptors n the nigrostriatal system in Parkinson disease. j. Cerev. Blood Flow Metav, 2009; 29: 1601-1608.
14. Dorn HF. Tobacco consumption and mortality from cancer and other diseases. Public Health Rep. 74,581-593(1959).
15. Baumann RJ, Jameson HD, McKean HE, Haack DG, Weisberg LM. Cigerette smoking and parkinsons disease: 1.comparison of cases with matched neighbors. Neurology, 1980; 30: 839-843.
16. Godwin-Austen RP, Lee PN, Marmot MG, Stern GM. Smoking and parkinsons disease. J. Neurol. Neurosurg. Psychiatry, 1982; 45: 577-581.
17. Grandinetti A, Morens DM, Reed D, MacEachern D.Prospective study of cigarette smoking and the risk of developing idiopathic parkinsons disease. Am. J. Epidemiolm, 1994; 139: 1129-1138.
18. Hernan MA, Zhang SM, Rueda-de castro AM, Colditz GA, Speizer FE, Ascherio A. Cigerette smoking and the incidence of Parkinson disease in to prospective studies. Ann. Neurol, 2001; 50: 780-786.
19. Ritz B, Ascherio A, Checkoway H et al.Pooled analysis of tobacco use and risk of Parkinson disease. Arch. Neurol, 2007; 64: 990-997.
20. Mellick GD, Gartner CE, Silburn PA Battistutta T. Passive smoking and Parkinson disease. Neurology, 2006; 67: 179-180.
21. Greenbaum L, Rigbi A, Lipshtat N, et al. Association of nicotine dependence susceptibility gene, CHRNA5, with Parkinson's disease age at onset: gene and smoking status interaction. Parkinsonism Relat Disord, 2013; 19: 72–76. doi: 10.1016/j.parkreldis.2012.07.007.
22. Hancock DB, Martin ER, Fujiwara K, et al. NOS2A and the modulating effect of cigarette smoking in Parkinson's disease. Ann Neurol, 2006; 60: 366–373. doi: 10.1002/ana.20915.
23. McCulloch CC, Kay DM, Factor SA, et al. Exploring gene-environment interactions in Parkinson's disease. Hum Genet, 2008; 123: 257–265. doi: 10.1007/s00439-008-0466-z.
24. Miyake Y, Tanaka K, Fukushima W, et al. SNCA polymorphisms, smoking, and sporadic Parkinson's disease in Japanese. Parkinsonism Relat Disord, 2012; 18: 557–561. doi: 10.1016/j.parkreldis. 2012.02.016.
25. De Palma G, Dick FD, Calzetti S, et al. A case-control study of Parkinson's disease and tobacco use: gene-tobacco interactions. Mov Disord, 2010; 25: 912–919. doi: 10.1002/mds.22980.