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Reply to "Risk reduction of Parkinson's disease by caffeinated beverage consumption"

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To the Editor,

We thank Dr. Kawada for his interest in our paper entitled "Relationship between risk and protective factors and clinical features of Parkinson's disease" [1], in which we observed that coffee consumption prior to Parkinson's disease (PD) development can predict older age at onset and milder motor symptom severity as tested by the International Parkinson and Movement Disorder Society-sponsored revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS) part III. Conversely, we found no associations between coffee consumption and non-motor symptoms, motor complications, motor subtype, or dopaminergic treatment dosage.

In his letter, Dr. Kawada pointed out the potential protective dose-dependent effect of tea consumption on PD development and reported previous observations on gender and regional differences regarding the effects of tea consumption on incident PD risk. In our paper, we did not include tea consumption because our analysis was based on nine factors that were found to be independently associated with PD after a simultaneous assessment of 31 potential risk and protective factors, which did not include tea consumption [2]. In addition, coffee is the most consumed caffeinated beverage in the Italian population, thus representing the most representative factor from an epidemiological point of view. Finally, the relationship between coffee consumption and PD is supported by a huge body of evidence, whereas a relatively limited number of authors have investigated the potential protective role of tea consumption in PD development and most papers on this topic examined only tea consumption alone or in association with a few other factors [3].

However, we accept with great interest the point raised by Dr. Kawada and we share the opinion that our novel finding that risk and protective factors of PD development can predict PD clinical expression may guide future investigations assessing the possible relationship between PD motor and non-motor symptom severity and other factors, including tea consumption. In particular, we agree with the author that tea has the biological plausibility to represent a possible long-term moderator of PD clinical expression and could be an interesting option for possible future preventive strategies aimed at mitigating PD clinical manifestations and their progression. Animal models suggest that black and green tea bioactive components, including theanine, caffeine, and polyphenols, may prevent neurodegenerative mechanisms [4] and that tea polyphenols can improve alpha-synuclein aggregation, cerebral alpha-synuclein aggregation, and motor impairment in nonhuman primates [5]. It is therefore recommendable to perform further prospective studies to: i) verify the independence of the association between tea and PD development by evaluating a large number of potential risk and protective factors simultaneously; ii) investigate possible differences between black and green tea since animal models suggest that polyphenols, mainly contained in green tea, may be neuroprotective against PD [4] whereas epidemiological investigations suggest that the protective power of black tea to prevent PD development is higher than that of green tea [3]; iii) explore the potential role of tea consumption preceding PD development in mitigating motor and non-motor PD manifestations; and iv) clarify whether tea may represent a novel target for PD progression preventive strategies.

To conclude, we thank Dr. Kawada for this point of reflection that stresses the complexity of risk and protective factors acting on upstream PD pathogenetic mechanisms and their possible influence on the variability that characterizes PD clinical expression and progression.

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Declaration of competing interest

The authors have no conflicts of interest to declare.

References

[1] D. Belvisi, R. Pellicciari, A. Fabbrini, M. Costanzo, G. Ressa, S. Pietracupa, M. De Lucia, N. Modugno, F. Magrinelli, C. Dallocchio, T. Ercoli, A. Nicoletti, M. Zappia, P. Solla, M. Bologna, G. Fabbrini, M. Tinazzi, A. Conte, A. Berardelli, G. Defazio, Relationship between risk and protective factors and clinical features of Parkinson's disease, Park. Relat. Disord. 98 (2022) 80–85, https://doi.org/10.1016/j.parkreldis.2022.04.017.

[2] D. Belvisi, R. Pellicciari, A. Fabbrini, M. Costanzo, S. Pietracupa, M. De Lucia, N. Modugno, F. Magrinelli, C. Dallocchio, T. Ercoli, C. Terravecchia, A. Nicoletti, P. Solla, G. Fabbrini, M. Tinazzi, A. Berardelli, G. Defazio, Risk factors of Parkinson

https://doi.org/10.1016/j.parkreldis.2022.08.010
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disease: simultaneous assessment, interactions, and etiologic subtypes, Neurology 95 (2020) e2500–e2508, https://doi.org/10.1016/S1474-4422(16)30230-7.
[3] A. Ascherio, M.A. Schwarzschild, The epidemiology of Parkinson's disease: risk factors and prevention, Lancet Neurol. 15 (2016) 1257–1272, https://doi.org/10.1016/S1474-4422(16)30230-7.
[4] S.Q. Chen, Z.S. Wang, Y.X. Ma, W. Zhang, J.L. Lu, Y.R. Liang, X.Q. Zheng, Neuroprotective effects and mechanisms of tea bioactive components in neurodegenerative diseases, Molecules 25 (2018) 512, https://doi.org/10.3390/molecules23030512.
[5] M. Chen, T. Wang, F. Yue, X. Li, P. Wang, Y. Li, P. Chan, S. Yu, Tea polyphenols alleviate motor impairments, dopaminergic neuronal injury, and cerebral α-synuclein aggregation in MPTP-intoxicated parkinsonian monkeys, Neuroscience 12 (286) (2015) 383–392, https://doi.org/10.1016/j.neuroscience.2014.12.003.

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