COMMENT: FRONTAL LOBES, EXECUTIVE DYSFUNCTION, GAIT, AND THE FALLACY OF PSEUDO-TRANSITIVITY

Massimo Filippi, Federica Agosta, Milan, Italy; Vladimir Kostić, Belgrade, Serbia; on behalf of all authors of “Pattern of brain tissue loss associated with freezing of gait in Parkinson disease”: We thank Dr. Montgomery for his stimulating comments on our article. Our study showed that a specific pattern of brain network damage involving frontal and parietal cortices occurs in patients with Parkinson disease (PD) and freezing of gait (FOG). Although voxel-based morphometry may not be sensitive enough to depict damage to subcortical and brainstem nuclei, this finding suggests that impairment of the frontoparietal network may play a role in the development of FOG in PD. Furthermore, we suggested that the occurrence of FOG in patients with PD may be related to cognitive frontal dysfunction. This is based not only on the commonality of frontal lobe pathology between the 2 clinical manifestations but also on the evidence that patients with PD with FOG have frontal executive deficits compared with patients with PD without FOG. In addition, FOG severity correlated with the degree of frontal executive dysfunction. Gait is a complex movement with multiple contributions from many brain regions. Although the mechanisms responsible for FOG are unclear, our findings suggest a role for frontoparietal damage and executive dysfunction, yet still unobserved pathologies may be directly responsible for FOG.

Author Response: Erwin B. Montgomery, Jr., Birmingham, AL: The response of Filippi et al. to my Comment accompanying their article reiterates the original arguments. Their inferences are appropriate as a hypothesis and may be proven correct, as I previously stated. However, their hypothesis has no greater epistemic standing than any number of alternatives which they fail to consider. Failure to genuinely discuss alternatives may further a false sense of validity which is not offset by use of qualifiers such as “may” or “suggests” in their own inferences. They fail to differentiate correlation from cause and effect. Their reasoning is insufficient validation as it relies on the logical fallacy of pseudotransitivity which is of the form: if a implies c and b implies c, then a implies b. This fallacy is repeated throughout their attempted causal explanation from executive dysfunction (a) implying attention or perceptual problems (c) and PD (b) being associated with attention or perceptual problems (c). The fallacy is very seductive because it is such a powerful—and some would argue—necessary route to hypothesis generation but it is not a method of validation. Great confusions are created when the fallacy is taken as validation, as they seemed to have done.

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MUTATION IN THE CHAC GENE IN A FAMILY OF AUTOSOMAL DOMINANT CHOREA–ACANTHOCYTOSIS

Ruth H. Walker, Bronx, NY; Antonio Velayos-Baeza, Oxford, UK; Benedikt Bader, Adrian Danek, Munich, Germany: In 2003, Saiki et al. reported an autosomal dominant transmission of
chorea-acanthocytosis (ChAc) that constituted a significant challenge to the generally accepted recessive inheritance of this disorder. This, and other articles concerning the same family, led to a debate in the research community.

Tomiyasu et al. noted that the proband reported by Saiki et al. is indeed compound heterozygous for mutations in the \textit{VPS13A} (\textit{CHAC}) gene and, therefore, a typical recessive ChAc case. Subsequent to the publication of this report, we found the Correction only via PubMed but missed the original Correction in \textit{Neurology}.1 In their Correction, Saiki et al. mention with regret that “an error in sequencing occurred and the inheritance pattern should have been reported as autosomal recessive,” with no reference to the recent publication of the full details of the mutations in this family. This Correction effectively changes the impact of the original article and, even more so, its title message. We feel that it would be appropriate to retract the 2003 paper in order to reduce further confusion in the literature.

Alternatively, a more compelling communication from the original authors about the significance of the new data could be offered in a manner readily accessible to the scientific community.

Author Response: Shinji Saiki, Ishikawa, Japan:
We regret the error published in our 2003 article. Concerning a pedigree with chorea-acanthocytosis in our previous reports, we would like to correct genetics materials associated with our pedigree already revised by Tomiyasu et al. In addition to a heterozygous splice site mutation (8035G>A, transcript variant A [GenBank NM_033305.2]), which we had reported as 8295G>A (using a different accession number) in the \textit{VPS13A} genes of the patients (III-2 and III-3), we identified a novel nonsense mutation (1305G>A, W435X) in the genes of the unaffected mother (II-4) and patients III-2 and III-3. According to them, the inheritance pattern of the pedigree is actually autosomal recessive (pseudodominant).

We apologize for the mistake and deeply regret any inconvenience this publication has caused for others.

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CORRECTION
Pilomotor seizure: When paroxysmal gooseflesh heralds brain tumor
In the Video NeuroImage “Pilomotor seizure: When paroxysmal gooseflesh heralds brain tumor” by L. Fisch et al. (\textit{Neurology}® 2012;78:1189), there is an error in the author list. The third author’s name should read S. Badoud. The publisher regrets the error.
**Mutation in the CHAC gene in a family of autosomal dominant chorea—acanthocytosis**


*Neurology* 2012;79;198-199

DOI 10.1212/01.wnl.0000416389.94466.01

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