## Guest Editorial

# Caffeine Intake and Dementia Risk-A Health Benefit From One of Life's Simple Pleasures? 

## Martha Clare Morris

Rush University Medical Center, Chicago, Illinois.<br>Address correspondence to Martha Clare Morris, ScD, Rush University Medical Center, 1645W. Jackson, Ste 675, Chicago, IL 60612. E-mail: martha_c_morris@rush.edu

Received August 19, 2016; Accepted August 19, 2016
Decision editor: Stephen Kritchevsky, PhD

In this issue of the Journal of Gerontology, Driscoll and colleagues (1) report on the potential beneficial effects of caffeine consumption on dementia incidence in the Women's Health Initiative Memory Study (WHIMS). This topic is of great interest to the field of dementia, through better understanding of the underlying biologic mechanisms, as well as to the general public, as a simple dietary prevention for this common and dreaded disease. The failure of the field to identify drugs that cure or effectively treat dementia places great emphasis on diet and other modifiable risk factors that may prevent the disease from developing. In this prospective analyses of 6,467 women aged 65 years and older who participated in the Women's Health Initiative randomized trial of hormone therapy, self-reported caffeine consumption $>261 \mathrm{mg}$ per day was associated with a $36 \%$ reduction in the risk of incident dementia ( $\mathrm{HR}=0.74, \mathrm{CI}[0.56,0.99]$ ) over 10 years of follow-up. The equivalent in terms of U.S. consumption patterns would be 2 to 38 -oz cups of coffee per day, 5 to 68 -oz cups of black tea, or 7 to $812-\mathrm{oz}$ cans of cola.

The study of dietary factors is very complex and there are many issues to consider in the interpretation of the study findings. As reported in a number of studies, caffeine consumption among the women was associated with many, mostly protective risk factors for dementia including younger age, higher education, lower body mass index, alcohol consumption, and lower occurrence of cardiovascular-related diseases and conditions. This raises concern that the reported findings are due to uncontrolled confounding by other preventive factors. This concern is mitigated in the WHIMS study by the fact that the estimated hazard ratio and confidence interval were virtually unchanged when these factors were adjusted in the analyses, indicating that there was little evidence of residual confounding by these measured factors. However, as is the case with the majority of the studies on caffeine and dementia, nutritional factors other than alcohol consumption (eg, vitamin E, fat composition) were not controlled in the analyses, leaving open the question of confounding by related diet factors. In addition, coffee consumption, the major contributor to caffeine intake in adults, declines substantially with older age beginning in the 40 s (2). Therefore, it is possible that unmeasured health conditions that cause some individuals
to decrease their caffeine intake during middle age are driving the findings of protective associations in studies of late-onset dementia. This phenomenon would be very difficult to investigate in epidemiological studies of older populations. Driscoll and colleagues partly addressed this issue by conducting analyses that adjusted for number of coffee cups consumed per day and found even stronger estimates of effect; the hazard ratio for probable dementia was $0.64,95 \%$ CI $[0.46,0.90]$. More convincing evidence would be provided by positive studies of caffeine intake in nonconsumers of coffee. The caffeine intake in this type of study would be largely due to tea and cola consumption, the other primary food sources of caffeine, that do not decline during middle age.

Consistency in findings across studies is an important consideration in the assessment of causality. There are a limited number of prospective studies of caffeine and dementia with most reporting protective associations. One would expect that the level of caffeine associated with a protective benefit would also be consistent across studies if there is indeed a true relation. However, this is not the case. The range is quite broad; from as low as 16.5 up to 371 mg per day. By comparison, the WHIMS study reported the level of benefit at greater than 261 mg per day.

Many features of the Driscoll et al. study reflect the best in epidemiological studies of dementia including the large sample size, long follow-up, comprehensive assessment of major risk factors, and validated methods of identifying cases of dementia. This study is an important contribution to the literature but more studies are required that address these important issues before making public health recommendations on caffeine consumption.

## References

1. Driscoll I, Shumaker S, Snively B, et al. Relationships between caffeine intake and risk for probable dementia or global cognitive impairment: the Women's Health Initiative Memory Study (WHIMS). J Gerontol A Biol Sci Med Sci. 2016;00:1-7. doi:10.1093/Gerona/glw078
2. von Boxtel MPJJAJ, Bosma H, Jolles J. The effects of habitual caffeine use on cognitive change: a longitudinal perspective. Pharmacol Biochem Behav. 2003;75:921-927.
