respectively. Using a randomized double-blind crossover design, cerebral vasomotor reactivity was measured by transcranial Doppler ultrasound and calculated using breath-hold index (BHI).

Twenty-four fasted, healthy volunteers on no regular medication (12 female, 12 male, mean age 23.2 years [SD 3.29]) attended twice, each study visit at least 24 hours apart. Chocolate caused a significant change in BHI by −0.06 units 90 minutes after chocolate ingestion (BHI pre 1.3 [SD 0.16]; BHI post 1.24 [SD 0.14]; $p = 0.015$, n = 48). Dark chocolate caused a significant reduction in BHI from baseline by −0.07 units (SD 0.17; $p = 0.05$, n = 24), though the change in BHI between dark and milk chocolate was not significant (BHI dark −0.07 [SD 0.17]; BHI milk −0.04 [SD 0.13]; $p = 0.431$, n = 24). No differences in blood sugar, heart rate, or blood pressure were apparent between groups.

Acute ingestion of chocolate was associated with a measurable change in cerebral vasomotor reactivity. Regular consumption of cocoa polyphenols has been shown to reduce the risk of stroke, and antioxidant, antiplatelet, and anti-inflammatory effects, together with effects on lipid profile, have all been proposed as potential mediators of the effect.[2]

Our data suggest that chocolate consumption is associated with an acute change in cerebral vasomotor reactivity, independent of metabolic and hemodynamic parameters. This acute effect may contribute to the observed relationship between long-term chocolate consumption and stroke risk, and is worthy of further investigation.

1. Larsson SC, Virtamo J, Wolk A. Chocolate consumption and risk of stroke: a prospective cohort of men and meta-analysis. Neurology 2012;79:1223–1229.
2. Corti R, Flammer AJ, Hollenberg NK, Luscher TF. Cocoa and cardiovascular health. Circulation 2009;119:1433–1441.

CORRECTION

Novel brain expression of ClC-1 chloride channels and enrichment of *CLCN1* variants in epilepsy

In the article "Novel brain expression of ClC-1 chloride channels and enrichment of *CLCN1* variants in epilepsy" by T.T. Chen et al., which was published ahead of print on February 13, 2013, reference 8 should not have been listed, as it was retracted in 2009. A corrected version of the article, with this reference removed, is published in *Neurology*® 2013;80:1078–1085. The authors regret the error.

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