A novel anti-inflammatory protective action of ethanol ingestion

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In this issue of the American Journal of Physiology-Heart and Circulatory Physiology, the study entitled “Role of calcitonin gene-related peptide in the postischemic anti-inflammatory effects of antecedent ethanol ingestion,” by Kamada et al. (1), shows that ingestion of ethanol 24 h before ischemia-reperfusion (I/R) of the small intestine conditions the expression of a mechanism that virtually prevents the I/R insult from increasing the number of rolling and adherent leukocytes in postcapillary venules. This protective mechanism involves calcitonin gene-related peptide (CGRP) because its expression was offset by treatment with CGRP-8–37, an antagonist of CGRP receptors, or by 4-day pretreatment with capsaicin, which decreases the amount of CGRP stored in sensory nerves. Moreover, exogenous CGRP, like antecedent ethanol ingestion, was effective in reducing I/R-induced leukocyte rolling and adhesion in postcapillary venules of the small intestine.

That antecedent ethanol ingestion prevents postischemic leukocyte rolling and adherence in the small intestine subjected to I/R is a salubrious effect of ethanol, attributable to activation of a CGRP-mediated, anti-inflammatory mechanism. Can the same ethanol-initiated process minimize the injury produced by I/R in other organs? Could the concept that ethanol promotes an anti-inflammatory mechanism by CGRP have a bearing on folkloric notions that moderate consumption of alcoholic beverages helps the digestive process and even helps fight colds? Although not very scientific, it may explain why people throughout the world thought that it helped their digestion when they drank small amounts of wine with their meals in the early years of our civilization. Also, whiskey was a much-used medication in the early days of the United States, especially for flu and colds. I grew up in west Texas, and when we had our first winter colds, our mother made us drink a hot toddy, which she made with hot water, some whiskey, and lemon juice, if we were lucky. A question posed by this outstanding paper is what amount of ethanol consumption in humans is necessary to reproduce the anti-inflammatory effects observed by Kamada et al. (1). One may wish to consider effects of ethanol that seem to correspond with the idea that good food, good company, and good wine are good for both the body and soul! The study by Kamada et al. is superb and gives us new data to rethink the concept of the mechanisms associated with I/R damage in the intestine and new ways to oppose this occurrence.

REFERENCES