Vascular Effects of Alcoholic Beverages
Is It Only Alcohol That Matters?

Flávio D. Fuchs

Contrasting effects of alcohol and some components of alcoholic beverages, such as antioxidant polyphenols, could eventually explain the paradoxical effects of alcohol and alcoholic beverages, particularly wine, on cardiovascular regulation. In this issue of Hypertension, Zilkens et al present the results of an experiment addressing this possibility. Twenty-eight healthy male individuals, regular daily drinkers, were assigned to 4 periods of a 4-week open-label crossover study. The 4 interventions included abstention from all alcohol and grape products (control period), 375 mL daily of red wine (39 g alcohol), 375 mL of the same red wine dealcoholized, and 1125 mL daily (41 g alcohol) of beer. Ambulatory daily systolic blood pressure was 1.9 and 2.9 mm Hg higher at the end of the beer and wine periods, respectively, compared with the abstinence period. Blood pressure at the end of the dealcoholized red wine period did not differ from the abstinence period. None of the interventions had an effect on flow and glyceryl trinitrate mediated dilatation of the brachial artery. Urinary endothelin-1 (ET-1) excretion was higher in the alcohol (beer or wine) periods than in the nonalcohol (abstinence or dealcoholized wine) periods.

The results of this experiment add 2 more pieces to the whole picture of effects of alcohol and alcoholic beverages on the cardiovascular system. First, it confirms that the most active component of alcoholic beverages is alcohol itself. Blood pressure increased with the intake of 39 g daily of ethanol carried on indistinctly by beer or wine. Second, it shows that other components of red wine have negligible, if any, sustained effect on cardiovascular parameters of human beings. Four weeks of daily intake of 375 mL of wine without alcohol, with ~700 mg of polyphenols, did not produce any detectable effect on blood pressure, heart rate, endothelial function, or endothelin secretion. In this respect, these findings contradict the results of other experiments, which have shown that grape juice or dealcoholized wine improved endothelium-dependent vasodilation.8–10 Zilkens et al attributed these contradictory results to the noncontrolled design of 2 experiments8,9 but did offer an explanation for the differences with the results of the experiment of Agewall et al.10 These differences may be attributable to chance or the evaluation of endothelial function just after the ingestion of wine or dealcoholized wine by Agewall et al. The results of the experiment of Zilkens et al, with a controlled design and adequate statistical power, suggest that the effects of alcohol or other components of wine on potential mediators of their beneficial effects on cardiovascular system may in fact be null or pharmacologically irrelevant. In this regard, these findings strengthen the interpretation that drinking moderately may be a surrogate marker of a healthier behavior.

The opinions expressed in this editorial commentary are not necessarily those of the editors or of the American Heart Association.

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Hypertension. 2005;45:851-852

Hypertension is available at http://www.hypertensionaha.org
DOI: 10.1161/01.HYP.0000164627.01274.ee
responsible for the lower incidence of coronary artery disease.\textsuperscript{1}

Zilkens et al suggested that the vasopressor effect of alcohol was caused, at least in part, via an alcohol-induced increase in ET-1, which was higher in the 24-hour urine collections at the end of wine and beer ingestion periods. Corder et al showed that polyphenols from red wine decreased ET-1 synthesis in cultured bovine aortic endothelial cells.\textsuperscript{11} If this effect happens in vivo, it may be overridden by a subsequent increase in endothelin synthesis, secondary to a dual effect of ethanol on blood pressure regulation. Epidemiological\textsuperscript{12} and experimental\textsuperscript{13,14} studies have demonstrated that the ingestion of alcohol is associated with an immediate decreasing of blood pressure (an effect typical of vasodilators), which is followed by a rebounding elevation of blood pressure. Heart rate increases in response to vasodilation and decreases thereafter. The rebound effects of acute alcohol ingestion on blood pressure and heart rate may be mediated by sympathetic modulation. Structural adaptation of the resistance vessels exposed to the recurrent cardiovascular effects of ethanol would lead to chronic hypertension.

Regardless of source, alcohol consumption, above a certain threshold level, raises blood pressure, and this effect does not appear to be counteracted by other components of wine. In view of the large risk for cardiovascular disease attributable to blood pressure $> 115/75$ mm Hg,\textsuperscript{15} physicians should caution their patients against excess alcohol consumption.

References


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*Hypertension*. 2005;45:851-852; originally published online April 18, 2005;
doi: 10.1161/01.HYP.0000164627.01274.ec

*Hypertension* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/45/5/851

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