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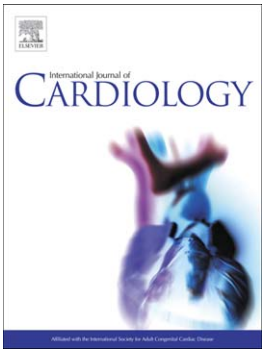

At the heart of the problem - A person-centred, developmental perspective on the link between alcohol consumption and cardio-vascular events

Valéria Lima Passos, Sven Klijn, Kevin van Zandvoort, Latifa Abidi, Paul Lemmens

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At the heart of the problem -

A person-centred, developmental perspective on the link between alcohol consumption and cardio-vascular events

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1. Introduction

Epidemiologic research assessing the cardio-vascular risks associated with alcohol consumption (AC) has produced a variety of shapes for risk curves across major cardio-vascular entities [1]. Among them, the J-shaped function has been consistently used to convey this association, mainly when the endpoint is coronary heart disease (CHD) [2-8]. The curve's lower point of inflection, corresponding to light-to-moderate AC is mapped to the smallest CHD risk estimate. Conversely, both the lower and upper-ends of the J-curve, representing abstention and heavy AC levels, respectively, are linked to higher CHD risks. The harmful effects of chronic heavy alcohol exposure and associated cardiomyopathy are well documented [9-11]. It is the beneficial effect of AC, in particular, that has aroused controversy [12-15], even though research on biological mechanisms underlying the cardio-protection is available. Several pathways have been proposed, and the current consensus is that different mechanisms may be operative. Alcohol increases high-density lipoprotein (HDL) concentration, decreasing endothelial damage, and reduced blood platelet aggregation (reducing the risk of occlusion). It has also been suggested that it affects, among others, glucose metabolism, insulin sensitivity, and lipid action, and has an anti-inflammatory effect [13, 16-18]. However, these beneficial effects have often been challenged, largely on methodological grounds.

Methodological fallacies suggested are: [5, 19-22] bias in self-reported AC measurement [23], publication bias [19], confounding bias, AC being irrefutably linked to certain socio-economic and lifestyle characteristics known to affect cardio-vascular events (CVE) [21, 24];

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