Resveratrol Causes Antiatherogenic Effects in an Animal Model of Atherosclerosis

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Dear Editor,

We read with great interest the article by Matos et al.[1] and would like to comment on its protocol and results.

Studies with dietary formulations require the description of all components included in the diet, because they vary considerably in commercial formulations (especially antioxidants) and might jeopardize the results and reproducibility of the study[2].

The absence of a group of animals treated with a standard diet with no lipid supplementation hinders the assessment of the predisposition of the animal model to develop atherosclerosis. That is a fundamental aspect considering the differential susceptibility to atherosclerosis of the strain of rabbits used[3].

The control animals had significantly increased baseline levels of total cholesterol and triglycerides as compared with those of the resveratrol-treated group (58% and 72%, respectively). Combining such results with the lack of statistical significance of the biochemical parameters at the end of the experiment, it seems unlikely that resveratrol can inhibit the formation of atherosclerotic lesions without changing the serum lipid parameters of the animals treated. In addition, the morphometric analysis performed does not represent a good indicator of the evolution of atherosclerotic processes, because the number and total area of the lesions are more reliable parameters[4].

Thus, we suggest that the results are difficult to interpret based on the limitations regarding the characterization of the animal model used, nutritional characteristics of the diet and methodology selected to assess atherosclerotic lesions.

Keywords:

Atherosclerosis; models; animal; antioxidants; dyslipidemias.

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Response letter,

We would like to thank the interest in our article and the pertinent questions raised. We shared those same doubts. That study is part of our research line in an animal model (rabbits) submitted to a hypercholesterolemic diet and then treated with functional food (linseed and resveratrol)[1]. Within our research line we also work with medications and arterial lesions in hypercholesterolemic rabbits[2,3]. All such models used rabbits, whose hypercholesterolemia had been caused by either the Sigma Aldrich product or lyophilized eggs. In other studies, we had a control group receiving only a normal diet for the species with no cholesterol added, but that group was not included in the present study.

The question about the fact that the baseline measures of cholesterol, HDL cholesterol, LDL cholesterol and triglycerides were higher in the control group had already been answered to the reviewers. The animals were selected at the age of one month, being then randomized to receive resveratrol or to the control group. We had no control on the diet of those animals before the experiment and infer that it might have been the normal diet for the species. Although the initial measurements of total cholesterol and triglycerides differed between the groups (the possible influence of laboratory measuring techniques was not excluded, and the collection might have been made in different days at the beginning of the experiment), we believe that those initial differences have not interfered with the final results of the study. Regarding the morphometric analysis performed, that has been our standard procedure and has been accepted in the following international publications and in the *Thrombosis Journal*[4].

Sincerely,

Liz Andréa V. Baroncini

Dalton Bertolim Précoma

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