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## EFFECT OF ARONIA MELANOCARPA FRUIT JUICE ON THE ACTIVITY OF ANTIOXIDANT ENZYMES IN A RAT MODEL OF AMIODARONE-INDUCED PNEUMOTOXICITY

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### Summary

The effect of *Aronia melanocarpa* fruit juice (AMFJ) on the activity of antioxidant enzymes in a model of amiodarone (AD)-induced pneumotoxicity in rats was studied. AD was instilled intratracheally on days 0 and 2 (6.25 mg/kg as a 3.125 mg/mL water solution). AMFJ (5 mL/kg and 10 mL/kg) was given orally from day 1 to days 2, 4 and 9. The activities of catalase (CAT), glutathione peroxidase (GPx) and superoxide dismutase (SOD) in lung tissue were measured on days 3, 5 and 10, respectively. AD decreased significantly CAT activity on days 3, 5 and 10. It caused a decrease of GPx activity which was significant on day 3. It decreased SOD activity but not significantly. AMFJ antagonized the effects of AD to such an extent that the enzyme activities at all time points did not differ significantly from the control values. The effect of AMFJ is probably due to its polyphenolic ingredients which serve as powerful radical scavengers. AMFJ probably decreased of the oxidative damage of cells by AD-induced overproduction of reactive oxygen species thus preserving the capacity of cells to produce antioxidant enzymes which, in turn, could further reduce oxidative stress.

**Key words:** *Aronia melanocarpa* fruit juice, amiodarone, pneumotoxicity, antioxidant enzymes

### Introduction

Amiodarone is a benzofuran derivative with highly effective class III antidysrhythmic activity. However, its use is associated with many side effects involving many different organ systems. The most serious side effect of amiodarone is pulmonary fibrosis. It has been postulated that the cause of AD-induced pneumotoxicity is complex and multifactorial, possibly involving several mechanisms [1]. Free radical formation, direct cytotoxicity, development of lysosomal phospholipidosis and membrane destabilization are the documented possible cellular mechanisms of toxicity, but it is known that the predominant molecular mechanism of AD-induced cell death is the oxidative damage [2]. The oxidative stress is normally due either to increased production of reactive oxygen species (ROS) or decreased antioxidant capacity of cells. Oxidative stress induced by AD has been demonstrated by the

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