

Acute High Dietary Phosphate Consumption Impairs Macrovascular, But Not Microvascular Function in Young, Healthy Men

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ABSTRACT

Chronic consumption of inorganic phosphate (P_i) is associated with an increased risk of cardiovascular disease in the general population. Importantly, with the growing abundance of processed foods, dietary intake of P_i in the United States has doubled the daily-recommended amount. However, the influence of acute consumption of high P_i on the cardiovascular system is not clearly understood, with equivocal results reported. Shuto *et al.* demonstrated that acute consumption of high P_i results in an impairment of endothelial function in healthy subjects, whereas a recent study by Levac *et al.* has refuted this finding. Moreover, these studies have only investigated conduit artery endothelial function, however microvascular responses, assessed by reactive hyperemia, have not been previously examined. **PURPOSE:** We tested the hypothesis that acute high P_i consumption impairs both macrovascular and microvascular function in young, healthy men. **METHODS:** On separate days, subjects ingested either Monosodium Phosphate (NaP_i) containing 2,000 mg of phosphorus and 1,520 mg of sodium ($N=13$; 23 ± 1 yrs; mean \pm SEM) or Sodium Chloride ($NaCl$) control containing 1,520 mg of sodium ($N=5$; 23 ± 2 yrs). Blood samples were collected at baseline, 60 min and 120 min post- NaP_i and $NaCl$ consumption to measure serum phosphate. Flow-mediated dilation (FMD) was used to assess macrovascular function, and reactive hyperemia was used to assess microvascular function at baseline, and 60 min post. **RESULTS:** Serum phosphate was significantly elevated at 60 min and remained elevated at 120 min post- NaP_i consumption, but did not change with $NaCl$. Importantly, FMD was significantly attenuated at 60 min post- NaP_i consumption, (pre, $5.4 \pm 0.6\%$; post, $3.1 \pm 0.5\%$; $P<0.01$) and did not change post- $NaCl$ consumption ($P=0.97$). In contrast, reactive hyperemia remained unaffected by both NaP_i (pre, 7633.26 ± 657.12 AU; post, 7357.31 ± 958.61 AU; $P=0.69$) and $NaCl$ conditions, ($P=0.96$) indicating preserved microvascular function. **CONCLUSION:** Our findings indicate that acute high NaP_i impairs peripheral macrovascular, but not microvascular, function in young healthy men. These data suggest that conduit artery endothelial function may be more susceptible than the microvasculature to acute consumption of high NaP_i .