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**Effects of potassium citrate or potassium chloride in patients with combined glucose intolerance: A placebo-controlled pilot study.**

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BACKGROUND:

Experimental K(+) depletion reversibly inhibits insulin secretion, while chronic metabolic acidosis decreases insulin sensitivity. We aimed to investigate the effects of potassium supplementation and alkali supplementation in non-acidotic, normokalemic humans with combined glucose intolerance.

STUDY DESIGN AND RESULTS:

In this double-blind, placebo-controlled study in 11 subjects (7 male, 4 female, ages 47-63 years), 90meqs of oral KCl or Kcitrate per day for 2weeks each increased insulin production as measured by homeostasis model assessment Beta [KCl=86 (CI 81-91), Kcitrate=88 (82-94), placebo=78 (73-83)%, $p<0.04$], but only Kcitrate attenuated insulin resistance as assessed by HOMA-IR (insulin resistance, Kcitrate=2.8 (2.5-3.1), placebo=3.2 (2.9-3.5), $p<0.03$) and only Kcitrate increased quantitative insulin sensitivity check index (Quicki, Kcitrate=0.355 (0.305-0.405), placebo=0.320 (0.265-0.375) $p<0.04$). These results were confirmed by independent measurements, i.e. HOMA C-peptide and whole body insulin sensitivity index measured during oral glucose tolerance testing. Kcitrate significantly decreased systolic and diastolic 24-hour ambulatory blood pressures (-4.0 (-3 to -5) and -2.7 (-1.9 to -3.5), respectively as compared to placebo, $p<0.02$) while KCl was without a significant effect.

CONCLUSIONS:

K(+) supplementation in the absence of overt K(+) depletion improves beta-cell function in subjects with combined glucose intolerance. The insulin-sensitizing and hypotensive effect, however, depend on citrate as the accompanying anion.