People given beta blockers were 10 to 24 times more like to dropout of studies due to fatigue.
People given beta blockers were 5 times more like to dropout of studies due to sexual dysfunction.
Beta blockers only prevent one stroke per year out of every 1400 patients given these drugs.
Beta blockers only prevent one heart attack per year out of every 1400 patients given these drugs.
Beta blockers only prevent one death per year out of every 2500 patients given these drugs.
For every one heart attack or stroke that is prevented by these drugs...
3 people are made impotent...
8 people have so much fatigue that they stop taking the drug.
“... hardly an acceptable risk/benefit ratio for a completely asymptomatic disease such as mild essential hypertension.”

— Franz Messerli, MD

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“In the case of hypertension in patients older than 60 years, such benefits [of beta blockers] have not been documented.”

— Franz Messerli, MD
In other words...
... beta blockers have not been shown to benefit people over the age of 60.
Hi, this is
Larry Hobbs @ FatNews.com
Beta blockers include drugs like atenolol (Tenormin) and propranolol (Inderal).
To the Editor: In their review article, Dr Ko and colleagues found no significant increased risk of depressive symptoms and only small increased risk of fatigue and sexual dysfunction associated with β-blocker therapy. They concluded that concerns about depression, fatigue, and sexual dysfunction should not deter physicians from initiating β-blocker therapy. I would like to raise the following caveats.

First, in addition to varying degrees of lipid solubility and generation, there are other pharmacological differences among β-blockers. Several β-blockers, such as pindolol, have antagonistic activity at somatodendritic 5-HT₁A autoreceptors, and thereby increase serotonin release. This action may lead to an improvement in depression. Rasänen et al reported that treatment with pindolol was associated with a slightly but significantly lower rate of antidepressant use, suggesting that pindolol may be beneficial in the treatment of depression.

To the Editor: We disagree with the conclusions of Ko et al that the conventional wisdom of β-blocker therapy being associated with depressive symptoms, fatigue, and sexual dysfunction is not supported by data from clinical trials. Their data show that the withdrawal rate of β-blockers because of fatigue was more than 2 times higher, and that due to sexual dysfunction almost 5 times higher, than in patients receiving placebo. In the Medical Research Council studies, the withdrawal rate for patients taking β-blockers because of fatigue was between 10 and 24 times that for those receiving placebo and also significantly higher than that for those taking diuretics, which are known to have a well-documented adverse effects profile. However, in contrast to β-blockers, diuretics have been clearly shown to reduce morbidity and mortality in hypertension.

Withdrawal rates provide more reliable information than “reported symptoms,” which, according to the principle “don’t ask, don’t tell,” are often neither solicited nor volunteered.
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Franz Messerli, MD disagrees with the Yale researchers who claimed that beta blockers do not increase fatigue, depression and sexual dysfunction.

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found no significant increased risk of depressive symptoms and only small increased risk of fatigue and sexual dysfunction as β-blocker therapy. They concluded that concerns about depression, fatigue, and sexual dysfunction should accompany β-blocker therapy. I would /H9252:

First, in addition to varying degrees of lipid solubility and generation, there are other pharmacological differences among β-blockers, such as pindolol, which antagonizes α1-autoreceptors, and /H9252-blockers, have actions that may lead to an association with depressive symptoms, fatigue, and sexual dysfunction. In the Medical Research Council studies,\(^2,3\) the withdrawal rate for patients taking /H9252-blockers because of fatigue was more than 2 times higher, and that due to sexual dysfunction almost 5 times higher, than in patients receiving placebo. In the withdrawal rate for those taking diuretics, which are known to have a well-documented adverse effects profile, however, in contrast to /H9252-blockers, diuretics have been clearly shown to reduce morbidity and mortality in hypertension.\(^4\)

He notes that even the data that the Yale researchers presented showed dropouts due to fatigue were 2 times higher and dropouts due to sexual dysfunction were almost 5 times higher. However, in contrast to β-blockers, diuretics have been clearly shown to reduce morbidity and mortality in hypertension.
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clearly argues against the use of β-blockers for the treatment of this disease.

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In Reply: Dr Terao cautions that factors other than lipid solubility and generation may influence the risks of adverse effects generally assessed in a standardized fashion in both treatment groups. We believe that both withdrawals and reported symptoms should be examined, as we did in our meta-analysis.

Finally, Messerli and Grossman suggest that diuretics (as opposed to β-blockers) are a better choice of therapy for elderly patients with mild hypertension. Our aim was not to compare the efficacy of different classes of drugs in specified conditions, but rather to quantify the risks of adverse symptoms assessed in patients randomized to receive β-blockers as compared with placebo for the treatment of myocardial infarction, heart failure, and hypertension. We believe that this information will be helpful to clinicians in placing the adverse effects of β-blockers within the context of their documented benefits.

Dennis T. Ko, MD
Patricia R. Hebert, PhD
Jeptha P. Curtis, MD
Harlan M. Krumholz, MD
Department of Medicine
Artyom Sedrakyan, MD
Department of Epidemiology and Public Health
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I believe that potassium bicarbonate is vastly superior to beta blockers...
and the other blood pressure medicines...
... for improving health.
I’ve been taking 1000 mg of potassium twice a day (2000 mg per day) in the form of potassium bicarbonate since 2000.
My blood pressure dropped from roughly 140/80 mm Hg to 124/73 mm Hg (taken last night).
124 73
WARNING: Only take potassium under a doctor’s supervision. Too much potassium can kill you.
Theoretically, we humans should be better adapted physiologically to the diet our ancestors were exposed to during millions of years of hominid evolution than to the diet we have been eating since the agricultural revolution a mere 10,000 years ago, and since industrialization only 200 years ago. Among the many health problems resulting from this mismatch between our genetically determined nutritional requirements and our current diet, some might be a consequence in part of the deficiency of potassium alkali salts (K-base), which are amply present in the plant foods that our ancestors ate in abundance, and the exchange of those salts for sodium chloride (NaCl), which has been incorporated copiously into the contemporary diet, which at the same time is meager in K-base-rich plant foods.

Deficiency of K-base in the diet increases the net systemic acid load imposed by the diet. We know that clinically-recognized chronic metabolic acidosis has deleterious effects on the body, including growth retardation in children, decreased muscle and bone mass in adults, and kidney stone formation, and that correction of acidosis can ameliorate those conditions. Is it possible that a lifetime of eating diets that deliver evolutionarily superphysiologic loads of acid to the body contribute to the decrease in bone and muscle mass, and growth hormone secretion, which occur normally with age? That is, are contemporary humans suffering from the consequences of chronic, diet-induced low-grade systemic metabolic acidosis?

Our group has shown that contemporary net acid-producing diets do indeed characteristically produce a low-grade systemic metabolic acidosis in otherwise healthy adult subjects, and that the degree of acidosis increases with age, in relation to the normally occurring age-related decline in renal functional capacity. We also found that neutralization of the diet net acid load with dietary supplements of potassium bicarbonate (KHCO₃) improved calcium and phosphorus balances, reduced bone resorption rates, improved nitrogen balance, and mitigated the normally occurring age-related decline in growth hormone secretion— all without restricting dietary NaCl. Moreover, we found that co-administration of an alkalinizing salt of potassium (potassium citrate) with NaCl prevented NaCl from increasing urinary calcium excretion and bone resorption, as occurred with NaCl administration alone.

Earlier studies estimated dietary acid load from the amount of animal protein in the diet, inasmuch as protein metabolism yields sulfuric acid as an end-product. In cross-cultural epidemiologic studies, Abelow [1] found that hip fracture incidence in older women correlated with animal protein intake, and they suggested a causal relation to the acid load from protein. Those studies did not consider the effect of potential sources of base in the diet. We considered that estimating the net acid load of the diet (i.e., acid minus base) would require considering also the intake of plant foods, many of which are rich sources of K-base, or more precisely base precursors, substances like organic anions that the body metabolizes to bicarbonate. In following up the findings of Abelow et al., we found that plant food intake tended to be protective against hip fracture, and that hip fracture incidence among countries correlated inversely with the ratio of plant-to-animal food intake. These findings were confirmed in a more homogeneous population of white elderly women residents of the U.S.

These findings support affirming...
LONG-TERM POTASSIUM SUPPLEMENTATION LOWERS BLOOD PRESSURE IN ELDERLY HYPERTENSIVE SUBJECTS

MD FOTHERBY MD, MRCP, JF POTTER DM, FRCP, University Department of Medicine for the Elderly, The Glenfield Hospital, Leicester

SUMMARY Following a randomised cross-over trial of the effect of a four-week 60 mmol/day potassium supplement versus placebo on blood pressure (BP), eight of the original 18 hypertensive subjects continued with a 48 mmol daily potassium supplement for four months. For these eight subjects 24-h potassium excretion during placebo, one month of 60 mmol and four months of 48 mmol daily potassium supplementation phases was 56 ± 23, 102 ± 28 and 90 ± 35 mmol/24 hours, respectively, and mean 24-h BP following each phase was 160 ± 16/89 ± 11, 147 ± 13/83 ± 12 and 145 ± 14/81 ± 9 mmHg respectively, a significant fall in mean 24-h SBP between four months of potassium supplement and placebo period of 15 ± 13 mmHg (95% CI: 4, 26 mmHg, p=0.02), although the fall in 24-h DBP was not significant (8 ± 11 mmHg, 95% CI: 0, 17 mmHg, p=0.08). Modest increases in dietary potassium intake could have significant effects on lowering BP in the large proportion of elderly subjects with hypertension. (Int J Clin Pract 1997; 51(4): 219-222)
LONG-TERM POTASSIUM SUPPLEMENTATION LOWERS BLOOD PRESSURE IN ELDERLY HYPERTENSIVE

Potassium chloride reduced blood pressure in older people from 160/89 to 145/81 mm Hg.

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Why not try potassium (bicarbonate) first?