

PH 12 Kre- Alkalyn™	No	0%	0%	0%	0%	0%	Never
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Jaffe Reaction

I. Metabolic Toxicity of Excess Creatinine

In the presence of normal kidney function (and the absence of artificial ingestion), the level of serum creatinine generated is closely monitored via a series of physiological regulators. The normal amounts of the toxic waste creatinine, are kept very low.

But, when creatinine is ingested from the conversion of creatine to creatinine (instantaneous conversion when activated with a fluid or ingested), concomitant metabolic abnormalities can be observed in-vitro, and in-vivo in animals and in humans. This ingestion of pure creatinine is toxic to humans and animals.

Lis and Bijan (1970) observed a sedating or stupefying property of creatinine when it was injected into mice. Giovanetti (et al., 1969) demonstrated that dogs could be chronically intoxicated by injecting them with creatinine. In addition to their observations of the animal's aberrant behavior, creatinine was also responsible for a significant decrease in the animal's erythrocyte survival time. The addition of creatinine to normal human blood initiated a significant increase in spontaneous hemolysis. This same red cell lysing pattern was observed in normal human volunteers whom had ingested creatinine. Golini and Golini (2004) also observed a sedating effect when creatinine was given orally to mice. Mice were unable to run on wheels and showed the same stupefying property reported by Lis and Bijan (1970).

Balestri's research in 1970 gave us further evidence of this potential membrane-associated molecular blockade by showing that creatinine was able to effectively inhibit glucose utilization by erythrocytes.

II. Mutagenicity and Carcinogenicity of Creatinine

It has been postulated that exposure to dietary creatinine may play a very significant part in the initiation of cancer (most probably bowel and bladder cancer) in man. (Doll and Peto, 1981; Felton, et al., 1994). Creatinine's ability to cause chromosomal damage and mutations in cultured cells has been confirmed, as has their carcinogenicity in mice and rats (Aeschbacher, 1991) and in one instance, in monkeys (Adamson, et al., Felton et al. 1994)

III. Creatinine: Friend or Foe?

Fact:

- 1). *Creatinine*- A waste product in the blood that results from the normal breakdown of muscle. (Medline)
- 2). *Creatinine*- A component of urine and the final product of creatine catabolism; formed by the nonenzymatic dephosphorylative cyclization of phosphocreatine to form the internal anhydride of creatine. (PDR Medical Dictionary)
- 3). *Creatinine*- Creatinine (CRN) is a by-product of the creatine production. The ingested creatinine is waste for the body and is excreted by the kidney. (Degus Germany)
- 4). Creatinine is excreted by the kidneys. (Greys, Anatomy & Physiology)

5). Normal Creatinine Clearance levels in healthy humans:

Men .97 to 1.37 mg/dl (97-137 ml/min)

Women .88 to 1.28 mg/dl (88-128 ml/min)

(St. Vincent Healthcare, Department of Pathology, Billings, MT)

(American Kidney Foundation)

- 6). Creatinine clearance levels above 1.37 mg/dl (for men) and 1.28 mg/ml (for women) is dangerous to healthy kidneys.

(St. Vincent Healthcare, Department of
Pathology, Billings, MT)
(American Kidney Foundation)

- 7). Increased creatinine levels will damage kidneys
- 8). Damaged Kidneys are non reversible.

Do you want to be ingesting grams of creatinine?

- 7). **NO**

I think by now it is very obvious that creatinine that is ingested from the conversion of creatine-creatinine is toxic, dangerous and even life threatening.

Creatinine is thought to be the major cause (possible the sole cause) of renal failure. Studies (such as Rooney, et al., 2002) using animals and humans have reported renal damage from the ingesting of creatinine. By taking creatine (that has converted to creatinine) these healthy human volunteers were shown to have induced abnormalities in pancreatic insulin secretion and changes in glucose homeostasis, resulting in renal failure.

Edmund's (et al.,2001) studies, using a rat model of cystic kidney disease, reported that when regular creatine monohydrate supplementation was given, larger and faster cyst growth and exacerbation of disease progression was observed. This was due to the conversion of creatine to creatinine.

Various types of glomerulonephritis, diabetic glomerulosclerosis, and glomerular amyloidosis in humans, a significant correlation exists between the severity of fibrosis of the renal cortical interstitium and tubular atrophy resulting from chronic interstitial inflammation and serum creatinine concentrations (Bohle et al., 1986)

Today, more than 20 million people in the United States have some form of renal disease, and more than 350,000, the US alone, require regular dialysis in order to stay alive. Renal failure with the exception of toxic drug over dose or injury related acute failure) is a stepwise deterioration process that is cumulative. This means it shows up gradually, and only after a substantial percentage of the organ has suffered damage. This stepwise progression stems from more than one primary 'insult' to a specific facet of the organ's filtration system. Creatinine poisoning, un-stable creatine, & residual manufacturing contaminants in creatine play a big part in this failure. (Benzi 2001: and 2000)