



## POTASSIUM DEFICIENCY AS A CAUSE OF RHEUMATOID ARTHRITIS

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(A more complete discussion of the role of potassium in arthritis may be found at Weber's homepage [http://members.tripod.com/~charles\\_W/arthritis.html](http://members.tripod.com/~charles_W/arthritis.html).)

This discussion of potassium is presented in the hope that one of its readers will consider performing an experiment establishing the effect of potassium on rheumatoid arthritis. There is no report in the literature going back to 1914 of such an experiment.

Every essential nutrient should have been explored before this. In view of the way hormones which are regulated by or regulate potassium, such as cortisol and deoxycorticosterone (DOC) are involved with rheumatoid arthritis (RA), and the low whole body potassium content in Rheumatoid Arthritis (RA), potassium especially should have been investigated before now.

### INTRODUCTION

Since the most serious aspect of the diarrheas is wasting potassium, cortisol has acquired the attribute of conserving potassium by moving it into the cells when cortisol declines. Cortisol (but not corticosterone) is reduced during a potassium deficiency, and this reduction accounts for many of the symptoms of RA.

Cortisol shuts down most of the copper enzymes when it declines so that excretion of copper is increased and Lysyl oxidase inhibited. These last two attributes are proposed to account for most of the mortality from aneurysms and infections during rheumatoid arthritis (RA).

Thus the urgent necessity to survive during virulent diarrheas has set people up in the course of evolution for some of the worst symptoms of rheumatoid arthritis.

### DISCUSSION

Judging by the drastic decline of mortality in babies suffering from a virulent strain of diarrhea by potassium supplements,<sup>1</sup> potassium loss in those diseases which force cyclic AMP to excrete water into the intestines<sup>2</sup> must be the most serious effect of the diarrheas. I suggest that this is the reason why cortisol has acquired the attribute of moving potassium out of cells<sup>3</sup> and therefore into the cells upon declining. It is also undoubtedly the reason why the adrenal's cortisol secretion is inhibited by low serum potassium in vitro (in the test tube) but not corticosterone.<sup>4</sup> The body thus has a way of signaling for a decrease in cortisol secretion during a serious intestinal disease independently of ACTH. Thus the body inversely mobilizes defenses.

Endotoxin bacterial diseases force the body to secrete cortisol by increasing ACTH<sup>5</sup> probably an adaptation by the bacteria to force the body to inhibit the immune system. Glucocorticoid response modifying factor (GRMF) secreted by T- cells then prevents the cortisol from having full effect on white cells other than suppressor cells<sup>6</sup> and thus raises the set point, as does interleukin-1.<sup>6</sup> Interleukin-1 also stimulates cortisol secretion,<sup>7</sup> as does cachectin (tumor necrosis factor).<sup>8</sup> I suspect that this is an adaptation to provide some

cortisol maintenance<sup>9</sup> when normal ACTH production is later cut off during endotoxin attack.<sup>10</sup>

In other words, the immune system takes over its own regulation but at a higher set point. The role of GRMF has not yet been demonstrated for physiological processes. GRMF will probably prove to inhibit cortisol for most of those processes as well, surely at least for cortisol's various affects on potassium.

One of the most important of the cortisol controlled immune defenses is the mobilization of the availability of copper to the white cells, an attribute which probably arose because copper is crucial to an adequate immune defense.<sup>11</sup> The primary way cortisol does this is by, inversely to its concentration, shutting down production of copper-containing enzymes such as Lysyl oxidase and superoxide dismutase.<sup>12</sup> Lysyl oxidase catalyzes the formation of cross links in all connecting tissue including elastin.<sup>13</sup> Since elastin makes up the main strength of normal blood vessels<sup>12</sup> and has a rapid turnover, this is the most serious problem in arthritis. Ruptured aneurysms along with poor resistance to infection and heart disease are the chief terminal events in arthritis.<sup>14</sup>

The body uses ceruloplasmin to carry copper to the immune system during infection.<sup>12</sup> Probably the main reason for this development is that the copper is not in equilibrium with the serum and so is not available to pathogens. However, ceruloplasmin is also used to carry copper to the bile for excretion.<sup>15</sup> Therefore I submit that the rise in serum ceruloplasmin in RA<sup>16</sup> causes an increased excretion in members of a society who, even before this, were receiving less than the minimum daily requirement.

### CONCLUSIONS

Evidence can be provided for this proposal in several ways. Arthritic people should have a lower whole body potassium content than normal people. This has been proved.<sup>17</sup> Red blood cells have a higher potassium content than normal during RA.<sup>18</sup> This should not be taken as counter evidence because I suspect that this is an adaptation to help avoid circulatory collapse when dehydration reduces the blood volume during diarrhea. There should be a lower incidence of RA among people on potassium supplementation or who eat Morton's Lite Salt (TM) or Stirling's Half and Half (TM). I know of no epidemiologic study showing this. However, people who work in potash mines have a 25% lower incidence of heart disease than the surrounding population<sup>19</sup> and heart disease is prevalent in RA. There should be a healing of RA upon starting potassium supplements. No controlled experiment has been reported which would indicate this. However there is a case history of a single arthritic brought up to 3.5 grams per day in order to explore the effects of various steroid hormones on the body's mineral balance.<sup>20</sup> A total of 3.5 grams is about the amount an adult would obtain from unprocessed food. The subject showed consistent improvement throughout the experiment even though potassium was the only consistent change. His total body potassium consistently rose. There should be a negative correlation between potassium-caused muscle spasms and RA, but I have no supporting data. Neither do I know of a positive correlation with eating licorice or potassium losing diuretics, both of which increase potassium loss. There should be a negative correlation between eating acids which have an indigestible anion and RA since the hydrogen ion interferes with potassium excretion.<sup>21</sup> I know of no good experiment or epidemiologic study.

However, it has been suggested from folk custom that eating vinegar<sup>22</sup> or cherries is efficacious. The vinegar seems doubtful since it is my understanding that acetate can be metabolized by the body. However, it is conceivable that people on a diet high in calories do not utilize all the acetate. RA should not be present in people who eat predominantly vegetables instead of grains.

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An experiment has been performed in which RA was healed in a group of people by switching to a vegetable diet.<sup>23a</sup>

I suspect that people with rheumatoid arthritis tend to have a poorer ability to conserve potassium than other people because of damage to their kidneys by a poison such as bromine gas or long term poisons in plant foods (such as solenaceous vegetables) or by poisons excreted by pathogenic bacteria. Screening some common poisons currently in use in food might be enlightening. Since GRMFs inhibit cortisol, it is possible that a discordance in the immune response or some infection types may accentuate RA. (See *Pain Foods!* <http://www.arthritistrust.org>.)

If animals are used for experiments, it is futile to use rats or mice because they rely primarily on corticosterone to regulate the immune response, not cortisol. I suspect that this developed because they have a factor in their intestinal fluid which counteracts cholera toxin.<sup>23</sup> They also have the ability to absorb water under cyclic AMP stimulation in part of their colon<sup>24</sup> instead of excretion, unlike other animals.

Since the disturbance in copper metabolism is proposed as the most serious aspect of RA, evidence for copper's effect should be possible. Supplementing with copper should remove some of the symptoms of RA. I know of no such experiment. (See "The Use of Ionic Copper in the Treatment of Arthritis," <http://www.arthritistrust.org>.)

However, it is known that Finnish men who work in copper mines have little arthritis or susceptibility to infection.<sup>25</sup> The high milk diet along with frequent saunas may be two reasons why other Finns have one of the highest rates of arthritis in the world,<sup>26</sup> since milk is the poorest source of copper<sup>27, p.92</sup> and perspiration loses potassium.<sup>28</sup> Milk has been shown to have a high statistical correlation with cardiovascular disease, said to be as great a risk as smoking,<sup>29</sup> which disease in turn is correlated with RA. Laplanders on a meat diet have a lower rate of RA not much further north.<sup>26</sup> The Massai of Africa have a higher rate of RA than the surrounding tribes.<sup>30, p.768</sup> The Masai also use a lot of milk as well as very few vegetables, which vegetables would have increased potassium intake. Men who work in copper mines must have stronger tissues than other miners because the percentage of injuries which result in lost time is significantly lower<sup>31</sup> even though injuries like eye damage and burns which are not affected by strength are part of the data. Eating a lot of shellfish or liver should reduce those symptoms related to copper deficiency since they are the richest sources, but I know of no study. The same is true of drinking acid water out of copper plumbing.

I believe that it is unwise to give cortisol to any class of people whose immune system is weak, such as arthritic people. If it is felt that cortisol should be raised in the body, why not use something relatively safe, like potassium supplements? If potassium supplements are used, be certain that vitamin B<sub>1</sub> is adequate because the "wet" heart disease of Beri-Beri can not materialize when potassium is deficient.<sup>32</sup> Obviously the reverse is also true for vitamin B<sub>1</sub> supplementation. For this reason, if the patient has heart trouble, it is very important to determine whether it is caused by vitamin B<sub>1</sub> or potassium. If potassium chloride is dissolved in fruit juice it tastes good and avoids the danger to the intestines that even slow release enteric tablets may present.

The chloride is the most efficacious form.<sup>33</sup> It would be better and safer yet to provide potassium from food high in potassium such as celery or bamboo shoots as Effinger proposed.<sup>34</sup> Unboiled, unfrozen, uncanned vegetables low in starch are the richest sources.<sup>35</sup> However, removing a deficiency will be slower since the potassium is not associated with chloride and would take a few months longer.

A deficiency can arise from diarrhea, processed food, reliance on grain or fatty foods,<sup>35</sup> psychic stress stimulation of aldosterone<sup>36, p.209</sup> (which is the main regulator of potassium)<sup>37</sup>, stress stimulation of cortisol (as in an operation, for instance,<sup>38</sup>) diuretics, licorice<sup>39</sup> as well as probably grapefruit,<sup>39a</sup> profuse perspiration,<sup>28</sup> excessive vomiting,<sup>40</sup> eating sodium bicarbonate,<sup>41</sup> hyperventilating,<sup>42</sup> laxatives,<sup>43</sup> enemas,<sup>44</sup> shock from burns or injury,<sup>45</sup> hostile or fearful emotions,<sup>36</sup> and very high or very low sodium intake.<sup>46</sup> All of these increase excretion or decrease intake of potassium.

A chronic potassium deficiency must surely cause a degenerative disease. I believe it materializes in some people as RA. If not, then what is the name of the degenerative disease which attends a potassium deficiency? It is not hypokalemia. This is only a word which describes low serum potassium, a marker.

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