



Polio Biology XI

The Biology of Fatigue

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William Shakespeare, Sonnet XXVII: 'Weary with toil, I haste me to my bed.'

Everyone has experienced fatigue, even Shakespeare. It is a reaction the body has evolved to maintain a healthy steady-state. It is a countermeasure to overuse and acts to avoid damage to muscles and nerves. If our bodies, sculpted through millions of years of evolution, had no way to tell us that it is time to shut down, we wouldn't have gotten this far. So my first point in this essay is to emphasize that fatigue is not just the province of people living with post-polio syndrome. Everyone in the human family has experienced fatigue.

One area of the biomedical literature which has examined fatigue, and its components, is the discipline of sports medicine. The 2000 Sydney Olympics is on the docket and you can be sure that trainers and sport's physicians are studying every aspect of fatigue that can effect their athletes. Why? Fatigue degrades performance. An understanding of all of its manifestations allows athletes to take steps to avoid and minimize the likelihood of a poor performance.

Of course we with post-polio syndrome live with fatigue. It is a much more common state for us and does not abate easily and normally as it does for an athlete. We have to be especially careful about it and also knowledgeable about it if we are to improve our lives.

Elite athletes know about fatigue to win medals, we must know about fatigue to improve our lives. Who knows more about the biology of fatigue, the elite athlete or the post-polio survivor? I hope this essay can help improve our general knowledge, from the biological perspective, and allow us to intelligently use this information to improve our lives.

Biology of Fatigue.

Throughout this series we have mentioned the motor unit. This is the nerve/voluntary muscle unit of contraction. The nerve depolarizes (that means the charge on the outside of the cell membrane (+) rushes inside (-) resulting in the charge on either side canceling). This depolarization moves along the nerve away from the Central Nervous System eventually to a voluntary muscle. When an activity requiring muscle contraction begins (running is a good example) the rate of depolarization and re-polarization (which requires energy) is rapid in the motor unit. The nerve cell depolarization triggers depolarization in the muscle cell membrane. The muscle cell depolarizes and the muscle fiber contracts (shortens) due to

the activity of proteins inside the muscle fiber. The muscle and nerve quickly recover and re-polarize. This uses nutrient energy. Then they fire again. If this rapid firing is not interrupted, either by other motor units taking over (which we lack- see [Polio Biology X](#)), or because of maximum intensity exercise over a long period, the nerve, nerve/muscle junction, and/or the muscle fiber itself will fatigue.

Inside the nerve the energy used to re-polarize the membrane is derived from specialized molecules, nutrients, and enzymes. As they become depleted waste products accumulate. If the waste products interfere with normal energy production and utilization we have metabolic fatigue inside the cell. The same can happen in the muscle fiber where chemicals like lactic acid, ammonia, and creatine build up faster than they can be excreted. All of this biochemistry, which may manifest as a feeling of burning inside a muscle, cramping, weakness, pain, or creepy crawly feelings are the result of fatigue, specifically **localized fatigue**. Any fatigue in the nerve, muscle fiber, or entire motor unit (muscle and nerve) constitutes localized fatigue.

If fatigue occurs in any nerves above the motor unit it is called **Central Fatigue**. It too is a common protective mechanism for the muscles and nerves below it and athletes experience it mostly as a consequence of over training. I get the sense that many polio survivors think that Central Fatigue is reserved for them and is a unique and disturbing symptom not known to others. This is not so.

There is a way to measure the magnitude of subtypes of Central fatigue. If you use a small electric shock to stimulate a muscle and measure the maximum force of contraction you can use this as a baseline of strength of contraction. If you then prompt a person to contract the same muscle as vigorously as possible you can measure the difference between tetanic contraction (of the unit with the electric pulse) and the maximum **voluntary** force possible. This is one measure of one subtype of Central Fatigue. We don't know a lot about Central Fatigue at the molecular level but it manifests itself, often, as a loss of drive or motivation to perform. Again, one can appreciate the role of Central Fatigue in protecting the nerves and muscles by inhibiting their over work at the level of the Central Nervous System.

There is a part of the human brain you may have heard about: **The Reticular Formation**. Nerve cell firing there keeps us awake. Some say, or have said, that polio damage has affected the reticular formation and that this is why some of us sleep so much and cannot easily rouse. I think this would be a very difficult idea to prove because of the complexity of the arousal system and the role of Central Fatigue in overwork, blood gases, and other factors. If you experience a blow to the head the neurons of the Reticular Formation can all depolarize and you will be rendered unconscious. If re-polarization occurs (hopefully in a short while) you will awaken. Anyway, this depolarization and re-polarization is how all neurons work.

Let's get back to Central Fatigue. Any negative effect on muscular performance, which is generated above the motor unit, is Central Fatigue. This includes inhibitory processes arising in the brain. We know less about this but we do know that it occurs in athletes as well as in polio survivors. The Central Nervous System is involved to a large extent in the regulation of muscular activity. (Bompa 1990)/[\[1\]](#). Elements within the Central Nervous System result in a number of psychological factors, and emotions, which can degrade muscle performance and result in a loss of drive due to Central Fatigue. (McComas 1996)/[\[3\]](#).

During prolonged exercise in an athlete, or during any activity which exceeds the fatigue point for a polio survivor, the blood chemistry changes. Amino acids, which make up proteins, change with respect to their relative abundances. Those amino acids with branched chains decrease and fatty acids increase. Examples of branched chain amino acids are leucine, isoleucine, and valine. Tryptophan is an amino acid but is not a branched chain amino acid. The ratio of Tryptophan/BCAA rises during fatigue, which results in a biochemical alteration of tryptophan to 5-hydroxytryptamine (5-HT) in the brain. You may know (5-HT) by its more commonly used name of serotonin.

To make a very complicated story short serotonin can act on serotonin receptors in the brain to cause sleep and also increase the mental effort (motivation) needed to maintain muscular activity. It's all rather complicated chemically, and this is but one theoretical biochemical pathway of many that helps explain Central Fatigue.

We've also heard of dysfunction of the hypothalamus and pituitary to explain Central Fatigue in post-polio syndrome. As is to be expected, these areas are implicated in Central Fatigue for elite athletes too. Possible indicators of Central Fatigue, cited by Lehman et.al. (1993)[2] include an impaired neurotransmitter metabolism, with increased cerebral 5-hydroxytryptamine (5-HT) concentration, partial hypothalamic and pituitary dysfunction, with resultant dysfunction of the neuro-endocrine axes.

It has been observed that over-training in athletes (which happens often for our normal activities) results in faulty prolactin metabolism as well as adrenocortical insufficiency. Well, enough of the biochemistry.

Suffice it to say that fatigue is a complicated phenomenon which involves motor units and the CNS and regulates the body by preventing overuse on many levels. There is not much difference in the CNS aspects of fatigue comparing elite athletes and polio survivors in my opinion. We just must do more with less and get to fatigue sooner.

I've never heard of anyone trying Branched Chain Amino Acids to help with Central Fatigue but I believe they are available at health food stores. To do any good (and they may not) you probably need 10 to 20 grams. Ask a physician before you try this. The fatigue is there for a purpose. If you reduce it without addressing the root of it you could hurt yourself.

References.

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