PEMF and Heat Shock Protein (hsp70)

It is known that myocardial protection can be accomplished by induction of the stress protein hsp70 through the use of elevated temperature (heat shock) [1] Studies on myocardial function have shown that hsp70, stimulated by an increase in temperature, leads to improved survival following ischemia reperfusion. Otherwise heat stress pre-treatment (hyperthermia) is of limited clinical utility since it requires a temperature elevation to 42°C, a level impractical for clinical use or to achieve sufficient hsp70 increases. It also has been shown that ELF-EMF induced stress responses protect chick embryo myocardium form anoxia damage [2]. And it was also revealed that an EMF of 80 nT at 16 Hz protected against coronary artery occlusion [3]. PEMF exposure (75 Hz, 3 mT) for 18 hours significantly reduced necrotic area in rats subjected to acute myocardial infarction [4] .

Low frequency electromagnetic fields (EMF) also induce the stress protein hsp70, but without elevating temperature. It has been shown previously that **60 Hz electromagnetic fields** upregulate the heat shock gene, HSP70 and induce elevated levels of hsp70 protein in the absence of elevated temperature [5,6,7]. Therefore PEMF exposure offers clinical advantage over thermal procedures because hsp70 does not turn off baseline protein synthesis, in contrast to elevated temperature [8]. A significant increase in hsp70 stress protein is induced within **five minutes** at 14 orders of magnitude lower energy input than thermal stress. Additionally, unlike thermal stress, the induced protection can be re-stimulated even after the stress is already present, and re-stimulation with even higher hsp70 levels can be induced by a different field strength, higher (800 µT) or lower (80 µT) [9,10].

As is known intracellular Ca2+ overload by chronic hypoxia alters Ca2+ homeostasis, whereas ameliorating calcium homeostasis is believed to be responsible for cardioprotection. A recent study hypothesizes that cardioprotection by PEMF exposure may restore C2+ homeostasis altered by hypoxia insults [11]. But notification submitted by Eduard David [12] suggests no treatment **for no longer than 4 hours** because it could come up an calcium overload (**not study-based**). In any case PEMF exposure may be on of the tools to manipulate the Ca2+, handling under pathological conditions.

QRS does not have any own studies about hsp70.

PEMF, Heat Shock Factors and Senescence

There are a few signs in present research that transcription factor **HSF-1**, which regulates the heat-shock response, also influences aging. Reducing hsf-1 activity accelerates tissue aging and shortens life-span, and it has been shown that its overexpression extends life-span [13].. Aging is characterized by increased oxidative stress and the accumulation of abnormal (malfolded) proteins, and these stresses induce Hsp gene expression through the transcription factor HSF. When proteins misfold, they can acquire alternative proteotoxic states that seed a cascade of deleterious molecular events resulting in cellular dysfunction. The Hsps counteract the toxicity of abnormal proteins by facilitating protein refolding and turnover, and through other mechanisms including inhibition of apoptosis [14]. These studies suggest the promise of new therapeutic strategies that harness existing cellular mechanisms

to prevent the widespread disruption of protein homeostasis. It can be assumed that **HSF-1 activity promotes longevity**

Referred study is based on a previous investigation ("Repetitive electromagnetic field shock **REMFS**) from the same authors [15]. In that case REMFS increased HSF-1 phosphorylation, enhanced HSF1-DNA binding, and improved Hsp70 expression relative no non-REMFS-treated cells. These results show that non-thermal REMFS activates an ant-aging effect as well as reduces cell mortality during lethal stress. **But:** REMFS has a frequency of **50 MHz**) and **cannot always be simply extrapolated to PEMF.**

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