

# **Biointiative Report:**

## **A Rationale for a Biologically-based Public Exposure Standard for Electromagnetic Fields (ELF and RF)**

### **Myocardial Function Improved by Electromagnetic Field Induction of Stress Protein hsp70**

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**Reba:** This paper provides a basis for understanding how applying just the right EMF exposure to the heart at just the right time can reduce heart muscle damage. We think it does so by reducing the heart's demand for oxygen in response to this EMF signal. Needing less oxygen at a time when the heart is getting less (during or after a heart attack, for example) means that heart function is protected. EMF is becoming recognized as a medical treatment of great promise.

#### **Abstract**

Cardiovascular disease is the leading cause of morbidity and mortality in the United States, accounting for 70-80% of deaths in men and women over the age of 65. Furthermore, congestive heart failure is the most common cause of hospitalization of the elderly, and its incidence continues to increase (Schneider, 1999). In open and percutaneous revascularization procedures (coronary artery bypass surgery and percutaneous coronary interventions (PCIs)) and in the treatment of myocardial infarction and heart failure, it is essential to protect cardiomyocytes from the effects of hypoxia and ischemia (Bolli et al., 2004). Currently, myocardial protection can be accomplished by induction of the stress protein hsp70 through the use of elevated temperature (heat shock) (Currie et al., 1993; Udelsman et al., 1993; Nitta et al., 1994; Plumier and Currie, 1996). Induction of stress proteins by heat to prevent stroke and myocardial infarction during reperfusion has been shown to partially protect the myocardium under ischemic stress in a variety of models (Hinds et al., 1995; Mestril et al., 1996; Plumier and Currie, 1996; Benjamin and McMillan, 1998; Chong et al., 1998; Cornelussen et al., 1998). The use of heat stress pre-treatment leads to moderate increases in hsp70 levels, but does not improve ischemia tolerance in isolated hearts (Cornelussen et al., 1998). Moreover, 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. We have shown previously that 60 Hz electromagnetic fields (EMFs) upregulate the heat shock gene, HSP70 and induce elevated levels of hsp70 protein in the absence of elevated temperature (Goodman et al., 1994; Goodman and Blank, 1998; Han et al., 1998; Lin et al., 1998, 1999, 2001; Carmody et al., 2000). Of particular relevance, we previously elevated hsp70 levels in cultured rodent cardiomyocytes using EMF pre-treatment (Goodman and Blank, 2002). Additionally, studies from Di Carlo et al. (1999) and Shallom et al. (2002) confirmed that cardiomyocytes were protected from anoxic damage in EMF exposed chick embryos. The induction of increased levels of hsp70 protein by low frequency EMF exposures offers multiple clinical advantages over thermal, chemical or gene-transfer methods of induction for both patient and clinician. EMF stimulation of cytoprotective proteins is a non-invasive procedure easily administered to the patient. EMF-induced hsp70 does not turn off baseline protein synthesis, in contrast to elevated temperature (Goodman et al., 1989). A significant increase in hsp70 stress protein is induced within 5 min at 14 orders of magnitude lower energy input than thermal stress. Additionally, unlike thermal stress, the induced protection can be restimulated even after the stress is already present, and restimulation with even higher hsp70 levels can be induced by a different field strength, higher (800 mG) or lower (8 mG; Blank et al., 1994; Lin et al., 1997).