

Assignment example

[Science](#), [Biology](#)



HSP60 and HSP70 and Their Confusing Roles in Aging Based on the first ment, “ Starvation causes a cellular response...that encodes...hsp70 and hsp60 family members,” and the second statement, “ Starvation can diminish some of the signs of aging,” it seems that there is a link between the encoding of hsp70 and hsp60 and the aging process, and that this relationship seems inhibitory. However, based on ideas gleaned from several reputable references, it remains inconclusive whether hsp70 and hsp60 indeed inhibit the aging process.

Hsp70 and hsp60 are molecular chaperones, or “ abundant, well-conserved proteins responsible for the maintenance of the conformational homeostasis of cellular proteins and RNAs” and are believed to be usually induced by environmental stress (Soti & Csermely, “ Molecular Chaperones” 227). In short, as molecular chaperones are often associated with aging tissues and aging in general, these proteins are especially found in such situations. There is, however, a debate whether their presence in the aging tissue is merely induced or it is this presence that induces aging. Hsp60 is involved in protein folding and hsp70 has a role in the regulating the heat shock response as well as membrane translocation. While hsp60 are found in the mitochondria, hsp70 is found in the eukaryotic cytosol, ER and mitochondria. Both families of proteins actually function for protein translocation and transport as well as folding and assembly (“ Molecular Chaperones”). In aging and diseased tissues, high amounts of hsp60 are found in people with atherosclerosis, acute coronary syndromes and angina, while hsp70 is found in those with peripheral as well as renal vascular diseases (Frostegard & Pockley 201-202).

A number of experts and experiments in the field of molecular biology are pointing out that chaperones are the ones that bring about the aging process at the molecular level. Soti and Csermely, in their study entitled “Chaperones and aging: role in neurodegeneration and in other civilizational diseases, underlined the possibility that HSPs “ might contribute to the onset of...atherosclerosis, cancer, diabetes and several neurodegenerative diseases” (384). In an actual experimental study in Korea, it was confirmed that the HSPs “ might be involved in tumorigenesis” as it did in patients with hepatocellular carcinomas (Lim et al. 2077). This is so because, as proven by rodent experiments, “ HSPs are known to be essential for the survival of cancer cells in different cancers” (Lim et al. 2077). Moreover, one particular HSP, hsp90, is believed to “[maintain] the functional quality of proteins involved in the progression of cancer” (Lim et al. 2077). Another HSP, hsp27, is also involved in promoting “ accelerated growth and senescence” in bovine endothelial cells (Morrow & Tanguay 209). These aforementioned statements somehow suggest that HSPs, presumably including all types and hsp60 and hsp70, may actually induce diseases, which may translate as a role in promoting cellular destruction and aging, instead of inhibiting these events. This conclusion, however, does not take into consideration the possibility that hsp70 and hsp60 may have other functions aside from possibly inducing aging and senescence.

On the other hand, other findings suggest that HSPs are produced by aged cells only as a form of an “ adaptive response” or as a way to “ prevent protein aggregation and to assist in refolding, or degradation,” which are actually cellular reactions to the aging process (Soti & Csermely, “ Molecular

Chaperones" 227). Studies of stressed cardiac muscle leading to an increased production of hsp70 somehow also demonstrate that such an increase is linked to "myocardial protection" (Broome et al. 85). Moreover, it is possible that "hsp could function to slow the rate of aging" particularly for hsp70 because of a reduction of its production in aged rats (Morrow & Tanguay 209). This means that since there is less hsp70 in aging tissue, then aging is taking place at a regular or fast rate, while since there is more hsp70 in young tissue, then aging is taking place at a slower rate. Still, in another study, "exercise has been shown to induce the release of hsp70 into the peripheral circulation of normal individuals" (Frostegard & Pockley 201). Since exercise is associated with cellular repair and youth, then its subsequent production of hsp70 must also be associated with the same events. Aging is caused by alterations in pH, hypoxia and oxidative stress, and there is a corresponding increase in HSPs in cells, particularly hsp70 (Broome et al. 85).

Despite the information which states that hsp60 and hsp70 may either induce aging or slow it down, there is still much to know about what exactly their roles in aging are. Although experimental data have established the possibility of such a role, it must nevertheless still be defined. Moreover, current data mostly account for the general function of HSPs as well as the specific functions of hsp70, but very little on hsp60. Hence, until everything has been cleared by further experiment and research, nothing much can be concluded regarding the roles of hsp60 and hsp70 in aging.

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