

**HEPATIC CELLULAR RESPONSE IN GILTHEAD SEA BREAM  
INDUCED BY SUDDEN WATER TEMPERATURE CHANGES**

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**EXTENDED ABSTRACT ONLY- DO NOT CITE**

The gilthead seabream *Sparus aurata* reared in the Mediterranean sea, may suffer a metabolic disorder that happens after sudden water temperature changes, especially when at temperatures below 12°C. This syndrome, called winter syndrome (WS) often causes fish mortality. Other factors have an influence in WS, like bacterial infection, especially *Pseudomonas anguilliseptica*. Previous work on this disease has focused on a general organism viewpoint, including endocrine and immunological assays. In the present work, we intend to face a cellular point of view and hence we have designed an experiment to test the effect of the WS on the hepatic cell regulation.

**Methods**

Four groups of fish were used, three of these were acclimated for one month under laboratory conditions (17°C). The last one was obtained from the farm while WS was occurring (February at 11°C). In the first three subgroups, one was the control group (17°C) and the others were subjected to a thermal shock (down to either 12°C or 6°C) with or without bacterial challenge (*P. anguilliseptica*). Blood samples were taken from all animals to perform cortisol and glucose assays and liver was removed in order to determine heat shock protein (Hsp70) expression and DNA apoptosis.

## **Results and Discussion**

A relationship between cortisol released and intracellular HSP has been previously described as playing a role in the recovery from metabolic imbalances. From our results, we have found a negative correlation between temperature, and both cortisol and HSP70 expression exists in all groups assayed. However the HSP values were only significant in fish exposed to thermal shock plus bacterial injection and in WS group. Although HSP production is known to inhibit the expression of many proteins, those involved in the hepatic gluconeogenesis pathway appear to remain functional, as glucose levels found in serum (also following cortisol increase) remained at normal or high values. However, the high levels of cortisol found in the WS fish (mean of 208.31 ng/mL , n= 15) and the high expression of HSP70, resulted in a drop in serum glucose levels compared with the other groups. These findings together with DNA assays showing apoptosis, confirm that temperature and bacterial infection are relevant factors in the generation of WS. The syndrome outbreaks, detected at metabolic level by the high levels of cortisol, would be related to an increase of HSP expression and to DNA apoptosis at the cellular level and perhaps following an initial drop of glucose.