IN VITRO STUDIES OF SATELLITE CELL BEHAVIOUR IN FISH

Fauconneau B.[1], Paboeuf G., Lebail P.Y., Weil C., Houlihan D.F.[2], Smith R., Guttierez J.[3], Castillo J.
[1] Fish Growth and Quality INRA 35 042 Rennes France, [2] Zoology Department Univ Aberdeen, Aberdeen AB9 2TN Scotland, [3] Physiology Dept., Fac.Biologia, 08028 Barcelona, Spain

The rainbow trout satellite cells express a complete *in vitro* myogenesis program characterised by an active proliferation step and by a differentiation step up to large myotubes. The proliferation step not observed for other fish species require require adhesion on components of the extracellular matrix. The pattern of differentiation of satellite cells suggest that these cells share some characteristics of myoblasts. These characteristics have been studied depending on *in vivo* previous environment and *in vitro* exposure to various factors including xenobiotics.

Intrinsic factors such as ageing and genetic origin would affect either number or proliferation capacities of satellite cells. Factors involved in these intrinsic differences in proliferation capacities have been analysed. The response of satellite cells to different hormones and growth factors (IGFs) is generally low. The origin of such a low response have been search in the characteristics of IGFs receptors.

Amongst the extrinsic factors it is feeding which affect deeply the state of satellite cells and especially their initial proliferation capacities. The specific effect of some micro-nutrients (Vitamin C) on satellite cells status have also been studied but only toxic effects have been measured. The effect of extrinsic factors suggest that there are different populations of satellite cells which would contribute to hyperplasic and hypertrophic growth of muscle.

The sensitivity of satellite cells to Xenobiotics *in vitro* exposure is high. Negative effects of pesticides and heavy metals (copper) on proliferation rate and protein synthesis capacities have been demonstrated. The response to nonyl-phenol polyethoxylate compounds known for their detergent action is different and used as additives in the preparation of pesticides. At low concentration, these compounds stimulate proliferation rate and at high concentration they induce removal of the cells from the substrate. These specific effects could be related to the inhibition of cells adhesion which altered the balance between proliferation and differentiation.

The behaviour of satellite cells *in vitro* help thus to understand the sensitivity of satellite cells to their *in vivo* environment and to analyse the effect of physiological and sub-physiological alteration of this environment.

OSMOTIC REGULATION OF EPITHELIUM-DERIVED RELAXING FACTOR (EPDRF) RELEASE IN AIRWAY EPITHELIUM

Fedan J.S., Johnston R.A., Dortch-Carnes J., Rengasamy A. and Van Scott M.R.
Health Effects Laboratory Division, National Institute for Occupational Safety & Health, Morgantown, WV, and Department of Physiology, East Carolina University, Greenville, NC, USA

The respiratory epithelium modulates the reactivity of the underlying smooth muscle to bronchoconstrictors *via* the release of epithelium-derived relaxing factor (EpDRF). The osmolarity of airway surface liquid increases during exercise, leading to bronchoconstriction in a subpopulation of asthmatics (exercise-induced asthma). To understand the consequences of elevated osmolarity in the airways we examined the effects of hyperosmolar solutions on the epithelium and its modulatory relationship with the smooth muscle using the guinea-pig isolated, perfused trachea preparation. This preparation allows mechanical responses of the smooth muscle and bioelectric responses of the epithelium to be measured in response to agents applied to the mucosal (apical) or serosal (basolateral) surface of the airway. Elevation of mucosal or serosal osmolarity with NaCl, KCl, D-mannitol, urea, N-methyl-D-glucamine chloride or Na gluconate induced an osmolar concentration- and epithelium-dependent relaxation of methacholine-contracted tracheas. These responses were not mediated by nitric oxide or prostanoids. Relaxation responses, whether initiated *via* mucosal or serosal application, were preceded by transepithelial depolarization. Elevation of mucosal osmolarity decreased reactivity to mucosally- and serosally-applied methacholine, and inhibited contractile responses of the smooth muscle in response to transmural stimulation of parasympathetic, cholinergic neurons. The relaxation was inhibited by amiloride and DIDS, but not by bumetanide or ouabain. To determine whether the role of EpDRF is altered in pulmonary disease, mechanical and bioelectric responses to hypertonicity were examined in animals 18 hr after treatment with lipopolysaccharide (4 mg/kg; i.p.). After this treatment relaxation responses to hyperosmolar NaCl were potentiated, the transepithelial potential difference was hyperpolarized, and depolarization responses to hyperosmolar NaCl solution were potentiated. These observations demonstrate that the airway epithelium is an osmotic sensor which transduces alterations in mucosal osmolarity into changes in smooth muscle tone, and this property may be altered in lung disease.

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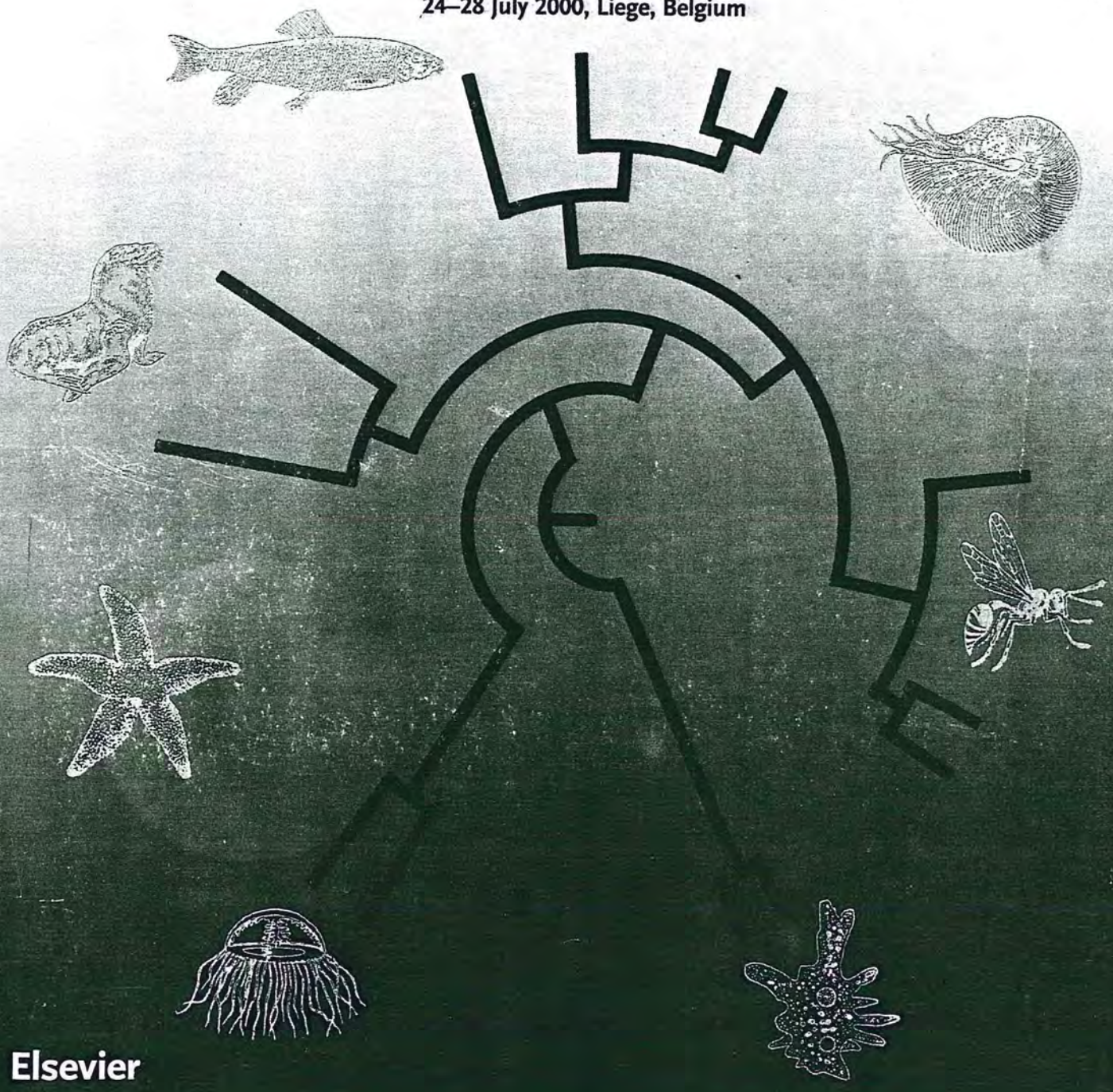

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