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Muc1 expression during rat embryonic and postnatal development

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Introduction: MUC1/Muc1 mucin (MUC1 in humans and Muc1 in others species) is a transmembrane glycoprotein expressed at the apical surface of most epithelia. Studies on mouse and human embryos have established that Muc1/MUC1 expression coincides with the onset of epithelial sheet and glandular formation.

Objective: Objective is to analyze temporal and spatial expression of Muc1 at different stages of rat development.

Method: A total of 8 embryos of each gestational stage, 8 neonates and 8 adults were included. Embryonic organs studied were esophagus, stomach, small intestine, salivary gland, liver, pancreas, trachea, lung and kidney as well as embryonic and adult mammary gland.

Results: By immunohistochemistry (IHC), Muc1 epithelial cell expression was observed in the stomach, lung and kidney at the gestational age of 13 days (13 D). In embryonic pancreas, Muc1 expression appeared at 14 D stage. In small intestine, a strong reaction was observed from 15 D while esophagus and trachea showed expression from 18 D. In the liver, a positive reaction was detected from 16 D while the ducts of the salivary glands showed reaction from 18D while at 20 D when mucous and serous acini were distinguished; Furthermore, Muc1 was observed in lactating mammary gland, post-lactational involution and old mammary gland. Lactating mammary gland showed an intense staining with a high percentage of reaction. In post-lactational involution and old mammary gland, a moderate reaction with a lower percentage of expression was detected. IHC results were validated by RT-PCR.

Conclusion: Results suggested a possible role of Muc1 during epithelia formation, differentiation and glandular morphogenesis during mammary gland postnatal development.

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Targeting RANK/RANKL/OPG as a novel treatment for muscular dystrophy

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Receptor-activator of nuclear factor κB (RANK), its ligand RANKL and the soluble decoy receptor osteoprotegerin (OPG) are the key regulators of osteoclast differentiation and bone. Although there is a strong association between osteoporosis and skeletal muscle atrophy/dysfunction, the functional relevance of a particular biological pathway that regulates synchronously bone and skeletal muscle physiopathology is still elusive. One key determinant of muscle contractility is the Ca2+ pump called sarco/endoplasmic reticulum Ca^{2+} ATPase (SERCA) which transfers Ca^{2+} from the cytosol into the lumen of the SR, making Ca^{2+} available for the next contraction. Here we showed that daily injections of OPG-Fc for 10 days; the inhibitor of RANKL/RANK interactions, greatly increased force of dystrophic mice relative to PBS-treated mdx mice and prevented the loss of SERCA activity. To understand more precisely the contribution of muscle RANK in muscular dystrophy, we treated dystrophic mice with an anti-RANKL antibody or generated a RANK/dystrophin double-deficient mouse. These results showed that the genetic deletion of RANK or pharmacological blockade of RANKL preserved muscle force and reduced significantly muscle damage in dystrophic mice. RANK/RANKL/OPG triad is thus a new and key factor in muscular dystrophy and its inhibition represents a new therapeutic avenue for possibly several forms of muscular dystrophy.

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