SMOOS — Result In Brief

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New targets in osteoarthritis

*European researchers put the pathology of osteoarthritis (OA) under the microscope to identify new therapeutic targets. The outcome of the study will hopefully improve OA diagnosis, treatment and prognosis.*

OA constitutes the most common chronic inflammatory condition of the joints. During normal joint development and homeostasis, SMOC2 emerges as a novel secreted protein that can interact with bone morphogenetic protein and Wnt signalling pathways. Expression is upregulated in OA joints, but its precise role in disease remains unknown.

The scope of the EU-funded ‘SMOC2 in osteoarthritis’ (SMOOS) study was to provide mechanistic information regarding the SMOC2 mode of action in health and disease. The project focused on how SMOC2 affects chondrogenesis and osteogenesis using a translational approach that combined both experimental and clinical data.

SMOC2 was found to exert an inhibitory effect on bone calcification through its calcium-binding domain. This piece of data led to a patent application for transferring a SMOC2-related protocol as a therapeutic intervention for medical conditions associated with vascular calcification.

SMOOS researchers additionally evaluated the role of SMOC2 in mouse models of inducible OA. They found that OA lesions were significantly reduced in animals expressing lower levels of SMOC2 harbouring only one smoc2 gene. In line with the in vitro results that support a role for SMOC2 in chondrogenesis, these data underscore the importance of SMOC2 interplay in key processes of joint formation.

Although it remains to be verified at the clinical level, SMOC2 emerges as a potential target that could be exploited therapeutically. Furthermore, the SMOOS in vitro approaches could serve as a platform for future experimentation on deciphering the role of other molecules in joint diseases.

Related information

| Report Summary | Final Report Summary - SMOOS (SMOC2 in osteoarthritis) |

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