of primary MSC characteristics, these MSC lines are expected to be valuable tools for the research on Regenerative Medicine for OA as part of Tissue Engineering in vitro models.

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AB0103 A NOVEL PROTECTIVE ROLE OF GPNMB/OSTEOACTIVIN IN POST-TRAUMATIC OSTEOARTHRITIS

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Background: Osteoarthritis (OA) is a chronic joint disease causes irreversible damage to the articular cartilage resulting in loss of joint function and subchondral bone remodeling. There is no cure for OA and currently the available treatment like pain management, and joint replacement surgery is used to treat this disease. GPNMB/Osteoactivin is a type-I transmembrane protein plays a vital role in osteogenesis and bone remodeling.

Objectives: However, the function of GPNMB/Osteoactivin in chondrogenesis and cartilage repair is unknown. Hence, in this study, we examined the role of GPNMB/Osteoactivin using post-traumatic osteoarthritis model and ex vivo chondrocytes cell culture and assessing the effects of GPNMB/Osteoactivin as anti-inflammatory factor in chondrocytes.

Methods: We first examined the expression GPNMB/Osteoactivin in normal cartilage. Next, we assessed the effects of recombinant GPNMB/Osteoactivin on mouse primary chondrocytes proliferation/viability and extracellular matrix markers.

Results: We found that GPNMB/Osteoactivin treatment did not have any effect on cell proliferation/viability, however, there was an increased in Collagen Type II and aggrecan expression in a dose- and time-dependent manner. These data suggest that GPNMB/Osteoactivin plays a role in cartilage maintenance. Next we determined whether GPNMB/Osteoactivin contributes to the OA disease progression. Human osteoarthritic damaged and undamaged cartilage harvested following knee replacement were used to examine GPNMB/Osteoactivin expression and found that GPNMB/Osteoactivin mRNA was 6-7-fold increased in damaged compared to undamaged cartilage. Similar results were obtained from mouse primary chondrocytes treated with IL-1 β and showed a significant increase (4-fold) in GPNMB/Osteoactivin mRNA compared to untreated control. Next, we evaluated the effects of GPNMB/Osteoactivin deficiency on the OA progression using the DMM model. GPNMB/Osteoactivin mutant (DBA/2J) mice showed significant increased in OARS1 scores compared to WT mice.

Conclusion: Taken together, these data are the first to report a role for GPNMB/Osteoactivin in cartilage maintenance and protection against the progression of OA.

REFERENCES

- [1] The surgical destabilization of the medial meniscus (DMM) model of osteoarthritis in the 129/SvEv mouse. *Methods in Molecular Biology: Osteoporosis and Osteoarthritis*. Loeser RF, Goldring SR, Scanzello CR, Goldring MB. Osteoarthritis: A disease of the joint as an organ. *Arthritis and Rheumatism*. 2012. pp. 1697–1707.
- [2] Zhou, L., Zhuo, H., Ouyang, H., Liu, Y., Yuan, F., Sun, L., ... & Liu, H. (2017). Glycoprotein non-metastatic melanoma protein b (Gpnmb) is highly expressed in macrophages of acute injured kidney and promotes M2 macrophages polarization. *Cellular Immunology*, 316, 53-60.
- [3] Ripoll, V.M., et al., Gpnmb is induced in macrophages by IFN-gamma and lipopolysaccharide and acts as a feedback regulator of proinflammatory responses. *J Immunol*, 2007. 178(10): p. 655766.
- [4] Karlsson C, Dehne T, Lindahl A, Brittberg M, Pruss A, Sittinger M, Ringe J. Genome-wide expression profiling reveals new candidate genes associated with osteoarthritis. *Osteoarthritis and Cartilage*. 2010 Apr 1;18(4):581-92.

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AB0104 ARE POLLUTANTS A NEW RISK FACTOR FOR OSTEOARTHRITIS? RESULTS FROM A SYSTEMATIC LITERATURE REVIEW

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Background: Prevalence of knee osteoarthritis (OA) has doubled since the mid-20th century. Interestingly, this increase cannot be explained solely by longer life expectancy and the obesity epidemics (1). Therefore, other environmental factors known to have increased in the recent years could be part of the explanation.

Objectives: We performed a systematic literature review (SLR) to summarize the existing knowledge about associations between OA and pollutants.

Methods: Pubmed database was used to identify studies reporting data on OA and pollutants in humans (examples of MeSH terms: "Polychlorinated Biphenyls (PCB)" or "Lead"). Abstracts from international congresses were reviewed for the past 2 years, combined with manual curation. Studies were classified in epidemiological clinical studies, pollutants assessments in *ex vivo* OA joint tissues, and *in vitro* effect on human chondrocyte.

Results: As of January 15, 2019, 193 potentially relevant articles were screened. Among them, 14 were selected. After manual curation, a total of 21 full text articles were analyzed. Ten articles reported epidemiological clinical studies. Four articles underlined the link between PCB and OA: in the 3 articles reporting association with past exposure to PCB, the most robust one showed that risk of OA was significantly increased, at least in men (OR 4.1 [95%CI: 1.8-11.2] for men and 1.3 [95%CI: 0.8-2.3] for women). In the 4th study, the highest serum concentration of PCB was associated with a higher risk of arthritis: maximal adjusted OR (aOR) was 3.2 [95%CI: 1.6-6.7], $p < 0.01$. However, sensitivity analysis of OA patients did not find any significant association in this study. Two articles focused on serum levels of perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS): aOR to have OA in the PFOA highest serum level quartile was 1.42 [95%CI: 1.26-1.59], $p = 0.00001$ and 1.55 [95%CI: 0.99-2.43]. Data were conflicting for PFOS: aOR to have OA in the PFOS highest serum level quartile was 1.77 [95%CI: 1.05-2.96], $p < 0.05$ and 0.76 [95%CI: 0.68-0.85], $p = 0.00001$. Two articles showed an association between serum lead levels and knee OA in the general population. In Korea, the risk of radiographic OA was increased in the highest quartile of lead serum level compared to the lowest one (aOR 1.90 [95%CI: 1.09-3.32] for men and 1.81 [95%CI: 1.17-2.77] for women). In the United States, risk knee radiographic OA correlates with increased lead serum levels. In the last study, household air pollution induced by the cooking heat source was examined: compared to electricity, the risk of OA was increased with the use of liquids (kerosene/paraffin: aOR 1.73 [95%CI 1.33-2.25]) or solid (coal, wood: aOR 1.73 [1.32-2.27]), (agriculture/crop: aOR 2.0 [1.47-2.72]). Lead and zinc accumulation was reported in 7 articles: 4 of them analyzed the concentration in OA joint obtained during arthroplasty and showed highest concentration in cartilage versus bone. In another, lead serum levels were associated with biomarkers of joint tissue metabolism. Two studies with X-ray fluorescence on cadavers found a high accumulation of lead and zinc in the articular cartilage tidemark. Four studies assessed the *in vitro* effect of pollutants on human chondrocytes: the viability of chondrocytes was reduced in presence of PCB, gold, silver, fluvic acid and bisphenol A.

Conclusion: This SLR suggests a possible link between OA and pollutants but the designs of the studies, the populations investigated and the type of pollutants were still too heterogeneous and limited to conclude definitively that pollutants represent a new environmental risk factor for OA. However, this SLR highlights a critical need for novel epidemiological, clinical and basic research studies in order to identify other potential environmental factors in OA.

REFERENCES

- [1] Wallace, PNAS 114(35), 2017

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AB0105 THE EFFECT OF TROMBOSIT RICH PLASMA APPLICATION ON DEGENERATED CARTILAGE: IN VITRO EXPERIMENTAL STUDY

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Background: Osteoarthritis is the most common problem among musculoskeletal system disorders. Platelet-rich plasma (PRP) has been suggested to be