



<b>Title</b>	<b>Osteopontin in rheumatoid arthritis and osteoarthritis</b>
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### **G-RI-3**

#### **Validity and Reliability of a Chinese Version of the Arthritis Impact Measurement Scales 2 (CAIMS2)**

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**Purpose:** Although the Arthritis Impact Measurement Scales 2 (AIMS2) had been validated among English speaking arthritis patients, it has not been validated into Chinese. The aim of this study was to evaluate the validity, reliability and cultural relevance of Chinese Arthritis Impact Measurement Scales 2 (CAIMS2) as a healthy assessment tool for Chinese speaking patients with rheumatic disease.

**Method:** AIMS2 was translated into Chinese (CAIMS2). An expert panel evaluated the cultural relevancy and content validity of CAIMS2. Reliability of scales, test-retest reliability and construct validity were tested on 166 subjects [RA=68, OA=21, Healthy=77]. Erythrocyte sedimentation rate, c-reactive protein, complete blood count, global pain score, visual analogue scale (VAS) for pain, total tender and joint counts as well as grip strength of RA patients were compared to CAIMS2 scores. 125 subjects were re-tested within 2 weeks.

**Results:** 3 items were modified and 2 items were added after expert review. Inter-item reliability was satisfactory [ICC: 0.8552 to 0.9594]. Test-retest reliability of the CAIMS2 sub-scales was 0.77 to 0.95. Significant score differences observed in mobility [ $p=0.000$ ], walking and bending [ $p=0.000$ ], hand and finger function [ $p=0.000$ ], arm function [ $p=0.000$ ], self-care ability [ $p=0.028$ ], social activities [ $p=0.000$ ], arthritis pain [ $p=0.000$ ], work [ $p=0.000$ ] and mood [ $p=0.00$ ] between arthritis patients and healthy subjects supported discriminant validity. In RA patients, AIMS sub-scales scores correlated with indicators of disease activities as well as results of functional assessments that further support construct validity [e.g. mobility vs ESR:  $r=0.456$ ,  $p=0.000$ ; walking and bending vs no. of total tender joints:  $r=0.371$ ,  $p=0.002$ ; VAS -pain vs arthritis pain:  $r=0.518$ ,  $p=0.000$ ; duration of morning stiffness vs arthritis pain:  $r=0.413$ ,  $p=0.001$ ].

**Conclusion:** Empirical data supported the reliability and validity of AIMS2 (Chinese) as a health status measure for Chinese speaking RA patients.

### **G-RI-4**

#### **Osteopontin in Rheumatoid Arthritis and Osteoarthritis**

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**Objectives:** Osteopontin (OPN) is a sialic acid-rich, adhesive, extracellular matrix (ECM) protein with Arg-Gly-Asp cell-binding sequence that interacts with several integrins, including  $\alpha$ -v- $\beta$ 3. OPN plays important roles in immunity, infection, inflammation and cancer. We sought to investigate the expression of OPN in rheumatoid arthritis (RA) and OA synovial tissue.

**Methods:** The expression of OPN mRNA and protein in synovia from 10 RA and 15 OA patients was examined by in situ hybridization and immunohistochemistry, flow cytometry, ELISA and RT-PCR. Regulation of OPN expression was investigated by treatment of cultured fibroblasts with IL-1 $\beta$ , IL-10 and TNF- $\alpha$ .

**Results:** In all RA and OA patients studied, we observed an expression of OPN mRNA and protein. OPN was present in synovial lining and sublining layer. Double labeling revealed that the majority of OPN expressing cells in RA synovial tissue were CD4+ lymphocytes. In contrast, the majority of OPN expressing cells in OA synovial tissue were fibroblasts. Expression of OPN by epithelial, endothelial, smooth muscle cells and fibroblasts was observed in both RA and OA patients. Interestingly, OPN was not detectable in cultured fibroblasts from RA or OA but it could be induced by IL-1 $\beta$  and TNF- $\alpha$  in a dose- and time-dependent manner. Furthermore, the increased OPN expression was specifically inhibited by p38 mitogen-activated protein (MAP) inhibitor SB203580.

**Conclusion:** These results demonstrated for the first time that OPN was produced by various cells in OA as well as RA patients. The TNF- $\alpha$  and IL-1 $\beta$  induced upregulation of OPN is possibly mediated through the p38 MAP kinase pathway.