Not all juvenile arthritis is the same

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Systemic juvenile idiopathic arthritis (SJIA) is currently classified as a subtype of juvenile idiopathic arthritis but with the addition of systemic inflammation often resulting in fever, rash and serositis. New research published in BioMed Central's open access journal *BMC Medicine* shows that the arthritic and systemic components of SJIA are related, but that the inflammatory pathways involved in SJIA are different from those in the more common polyarticular juvenile idiopathic arthritis (POLY). Of particular interest, distinct pathways involved in the arthritis of early and established SJIA raise the possibility that the immune system alters its behaviour over the course of this disease.

At diagnosis SJIA can resemble other diseases such as a viral infection or Kawasaki disease, but persistent SJIA can lead to chronic arthritis. The lack of autoantibodies (antibodies the body produces against itself rather than to fight an infection) and other rheumatoid factors has led people to suggest, unlike other forms of juvenile arthritis, SJIA should be classified as autoinflammatory rather than autoimmune.

In a collaboration between the Stanford University School of Medicine and Celera a team led by Prof Elizabeth Mellins looked at the genes switched on in the blood of children with either SJIA or POLY. When these genes were grouped into biological pathways such as IL-signalling,CD40 signalling, or communication between immune cells it became apparent that in SJIA the pathways involved in elevated erythrocyte sedimentation rate (ESR), which is used as a marker for disease flare-up, were also linked to joint arthritis.

Prof Mellins explained, "In our study we identified molecular pathways involved in both the systemic and arthritic components of SJIA. We discovered that the set of pathways involved in SJIA inflammation were different from those in POLY, perhaps explaining the differences in affected organs. This was especially true for the genes involved in increased ESR. For example, glucocorticoid signalling was more heavily involved in inflammation associated with SJIA than POLY, which may explain why non-glucocorticoid treatment is less effective for children with SJIA."

Even within the SJIA group different pathways were involved in different stages of the disease. Knowledge like this should help us refine treatment plans for these children and help to control their disease.

More information: Correlation analyses of clinical and molecular findings identify candidate biological pathways in systemic juvenile idiopathic arthritis Xuefeng B Ling, Claudia Macaubas, Heather C Alexander, Qiaojun Wen, Edward Chen, Sihua Peng, Yue Sun, Chetan Deshpande, Kuang-Hung Pan, Richard Lin, Chih-Jian Lih, Sheng-Yung P Chang, Chang Lee, Christy Sandborg, Ann B Begovich, Stanley N Cohen and Elizabeth D Mellins *BMC Medicine* (in press)

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