Commentary: Juvenile idiopathic arthritis—issues of definition and causation

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Juvenile inflammatory arthritis (JIA) in children comprises a number of different conditions—some of which have adult equivalents and others do not. The term juvenile rheumatoid arthritis (JRA) has different meanings on either side of the Atlantic. In the USA it is a collective term for all types of inflammatory arthritis in childhood. In Europe, at the time of the reported study, the term was confined to children with a positive rheumatoid factor test, i.e., the childhood equivalent of adult rheumatoid arthritis. True JRA is rare in childhood—and when it does occur, it predominantly affects adolescent females. By far, the most common form of inflammatory arthritis in children <7 years is oligoarticular JIA, a condition without an adult equivalent. Unfortunately, the diverse types of JIA were not reflected in ICD9. All forms of JIA should be coded 714.3. Technically, no children should receive the code 714 since the rubrics other than 714.3 refer to adult disease. Some cases of juvenile arthritis have features of ankylosing spondylitis and may have been under an entirely separate rubric.

Thus, it is rather surprising to find in this Finnish study that 31 cases were found classified as juvenile arthritis (714.3) and 44 as ‘adult RA’. On the assumption that all these children had some form of inflammatory arthritis, it is best to focus on the whole group of 75. It is in this whole group that we see the risk of maternal smoking on the subsequent development of arthritis—but predominantly in girls. These findings are of interest since it is only relatively recently that smoking has been recognized as a risk factor for adult RA—in particular rheumatoid factor positive RA. In contrast to the finding in the study of Jaakola and Gissler, the risk of smoking has been found to be higher in adult males than females. In adults the risk of smoking is thought to be mediated via rheumatoid factor production. Smokers without arthritis are much more likely to be rheumatoid factor positive than non-smokers. Rheumatoid factor positivity frequently precedes the development of adult RA by many years. However, this is unlikely to be the mechanism in JIA since, for the reasons outlined above, few of these young children with JIA will have had positive rheumatoid factor tests.

So how might smoking influence the subsequent development of JIA in the child? First it might induce some permanent immunological abnormality in the child, which later leads to arthritis. Second the role of maternal smoking could be via low birth weight and small-for-gestational age. This in turn might lead to an increased susceptibility to childhood infections, which might trigger arthritis. Jaakola and Gissler considered this possibility but felt it could not explain the size of the observed effect. Third the maternal smoking might be a marker for what is actually the true risk factor. For example, if a mother smokes during pregnancy she is also likely to smoke after pregnancy—and it may be the exposure to environmental smoke as an infant that is the true culprit. Smoking is an important contributor to autoimmunity.


have abnormalities in T-lymphocyte function, a reduction in the number of natural killer cells and abnormalities in humoral and cellular immunity. This might be conveyed to the child in utero or might be acquired by passive smoking. Finally, an earlier study showed that the risk of JIA was 40% lower in breast-fed than in non-breast-fed children. It is possible that mothers who smoke are less likely to breast-feed and so this might be the link between smoking and arthritis onset.

In conclusion further studies are needed to confirm these results in other populations and to try and understand the mechanism underlying the association.

References


