

Rather than indicating causality, an association between volume and better outcome might be due to a common underlying factor, such as a hospital's longer history, better associated services (such as intensive care), its ability to attract and retain skilled staff, or its ability to attract more patients because of its reputation. None of these factors would necessarily be obtained by, say, merging the caseloads of two centres. It is also important not to extrapolate beyond the available data; further increases in the case volume in larger centres may even lead to poorer outcomes, if communication in the hospital were to start to decline. Finally, it is possible that the concordance between centres might have increased since 1995, because experience with operations such as the arterial switch has been gained.

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## Relative importance of genetic effects in rheumatoid arthritis: historical cohort study of Danish nationwide twin population

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### Abstract

**Objective** To determine the relative importance of environmental and genetic effects in the development of rheumatoid arthritis.

**Design** Historical cohort study with record linkage between a twin registry and the Danish discharge registry as well as the Danish national registry of deaths used to estimate completeness.

**Setting** Two population based nationwide twin birth cohorts.

**Participants** 37 338 twins were sent a questionnaire about rheumatic diseases. Self reported rheumatoid arthritis was verified by clinical examination and from medical records.

**Main outcome measures** The probandwise concordance rate of rheumatoid arthritis in monozygotic and dizygotic twins.

**Results** The response rate was 84.7%. Rheumatoid arthritis was verified in 13 monozygotic and 36 dizygotic twins. There were no concordant monozygotic twin pairs and two concordant dizygotic twin pairs. Based on capture-recapture methods the probability of ascertainment was 78.3%. The probandwise concordance rate was 0 (95% confidence interval 0 to 24.7) in monozygotic twins and 8.8 (1.9 to 23.7) in dizygotic twins.

**Conclusion** Genes are of minor importance in the development of rheumatoid arthritis.

### Introduction

Rheumatoid arthritis is a systemic inflammatory autoimmune disease of unknown cause. Environmental and genetic risk factors have been identified, but no single risk factor has emerged as necessary or sufficient to cause the disease.

Twin studies represent one of the simplest ways to unravel the relative importance of genetic and environmental effects. In studies of specific diseases or traits in twins who volunteer to take part, monozygotic, concordant, and female twins tend to be over-represented.<sup>1,2</sup> Hence, much of the available literature on rheumatoid arthritis in twins overestimates the contribution of genetic factors.<sup>3-5</sup> Only two previous studies were population based, but confirmation of the diagnosis according to validated classification criteria was not performed.<sup>1,6</sup>

We undertook a nationwide study among twins in Denmark to estimate the importance of genetic effects in the development of rheumatoid arthritis.

### Methods

**Ascertainment of twins**—The study comprised two nationwide twin populations. The older birth cohort comprised 1631 same sex pairs of twins born 1921-40 in which both twins were alive in 1994.<sup>7</sup> The younger birth cohort comprised 34 076 surviving twins from same and opposite sex pairs of twins born 1953-82.<sup>7</sup> To

estimate a possible selection bias introduced by the requirement that both twins had to be alive we also sent questionnaires to 990 surviving individuals from same sex twin pairs born 1921-30.

**Ascertainment and verification of rheumatoid arthritis**—In 1994 the twins were asked by questionnaire if they had ever suffered from rheumatoid arthritis. Twins who reported that they had rheumatoid arthritis subsequently received a clinical profile questionnaire followed by a telephone interview. If rheumatoid arthritis could not be ruled out they were asked for permission to approach their non-affected cotwin. Both twins were invited to have a clinical examination. They underwent a structured interview and clinical examination, and blood samples were drawn for measurement of rheumatoid factor, HLA typing, and determination of zygosity by blood group analysis. We used the modified 1987 revised criteria of the American Rheumatism Association to confirm the diagnosis.<sup>8</sup> Time of onset was defined as the time when the diagnosis was established for the first time. Time of discordance was defined as the time from onset in one twin until onset in the second twin or the end of observation.

**Validation of completeness**—We used record linkage with the Danish discharge registry to find any twins with rheumatoid arthritis that we had not already identified. We estimated the completeness of the study with capture-recapture.<sup>9</sup>

We linked records of the twin registry and the Danish national registry of deaths. We sent a questionnaire to individual twins who would have been eligible for participation except for the death of their cotwin. The surviving twin was asked whether he or she or the deceased cotwin had ever had rheumatoid arthritis.

**Ethics**—The study was approved by all the regional scientific ethics committees in Denmark and the Danish data protection board.

**Analysis**—We used the probandwise concordance rate.<sup>10,11</sup> In our study a proband was a twin who independently of his or her cotwin reported rheumatoid arthritis and who fulfilled our classification criteria for rheumatoid arthritis. A secondary case was a twin ascertained through a cotwin and who fulfilled our classification criteria for rheumatoid arthritis. We excluded pairs of twins in which both twins had rheumatoid arthritis and neither twin fulfilled the proband criteria.

The calculation of concordance rates is given in more detail in the full version on the *BMJ's* website [bmj.com](http://bmj.com).

## Results

The overall response rate after one reminder was 75% (2445/3262) in older twins and 86% (29 433/34 076) in younger twins. Forty nine twins satisfied the modified criteria of the American Rheumatism Association.

There were no significant differences between monozygotic and dizygotic twins regarding sex, age, age at onset, mean discordance time, presence of rheumatoid factor, bony erosions, or shared epitope (table). Nodules were present more often in monozygotic twins than in dizygotic twins.

There was no difference between monozygotic and dizygotic twin pairs in concordance rates. The probandwise concordance rate was 0 (95% confidence interval 0 to 24.7) in monozygotic twins and 8.8 (1.9 to 23.7) in dizygotic twins, and the pairwise concordance rate was 0 (0 to 24.7) and 5.9 (0.7 to 19.7), respectively.

The record linkage study identified 46 twins with possible rheumatoid arthritis, but in 23 cases the diagnosis could not be verified. Among the remaining 23 twins, we identified five solely through the Danish discharge registry. On the basis of the capture-recapture model our questionnaire had an estimated probability of ascertainment of 78%, and hence nine cases (95% confidence interval 3 to 16) may have been missed. We found no concordant monozygotic pairs through the Danish discharge registry.

The response rate from twins whose cotwin had died was 74%. There was no difference in response rate between monozygotic and dizygotic twins nor between men and women. There was no difference in concordance for rheumatoid arthritis between monozygotic and dizygotic twin pairs.

From the record linkage study with the registry of deaths we identified six twins from six different twin pairs. In three cases the cotwins of the dead twins who had had rheumatoid arthritis were still alive and responded to the questionnaire. In one case a dizygotic twin pair was concordant for rheumatoid arthritis according to self report. We found no monozygotic concordant pairs.

## Discussion

In this large twin study monozygotic twins were no more likely to be concordant for rheumatoid arthritis than dizygotic twins. Our results do not support a genetic contribution to the development of rheumatoid arthritis. This is the first study to combine the recruitment of twins with rheumatoid arthritis through a questionnaire sent to a population based random sample with a subsequent clinically based validation of the diagnosis.

Cross sectional studies cannot show the lifetime risk for cotwins, but this does not imply any bias as long as the mean and distribution of discordance time does not differ between monozygotic and dizygotic twins, as in our study. None of the previously published twin studies on rheumatoid arthritis have dealt with this potential bias. The association between rheumatoid

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Distribution of demographic and clinical data in cases of twins with rheumatoid arthritis according to zygosity. Figures are numbers (percentage) of individuals unless stated otherwise

Characteristic	Monozygotic (n=13)	Dizygotic (n=36)	95% CI for difference
Women	9 (69.2)	25 (69.4)	-29.5% to 29.0%
Mean (SD) age (years):			
Older cohort (born 1921-40)	68.0 (2.7)	67.2 (3.3)	-1.9 to 3.5
Younger cohort (born 1953-82)	31.2 (5.8)	35.1 (5.1)	-10.0 to 2.2
Mean (SD) age at onset (years)	40.5 (20.7)	44.1 (15.1)	-14.4 to 7.4
Mean (SD) discordance time (years)	13.3 (13.4)	13.7 (10.87)	-8.0 to 7.3
Ever positive for rheumatoid factor	12 (92.3)	29 (80.6)	-7.7% to 31.2%
Ever had erosions	9 (69.2)	24 (66.7)	-26.9% to 32.0%
Ever had nodules	10 (76.9)	15 (41.7)	7.3% to 63.3%
Positive for shared epitope*	9 (69.2)	26 (74.3)	-34.0% to 23.9%

\*Known for 35 dizygotic twins as one dizygotic twin pair refused to give blood samples.

### What is already known on this topic

Rheumatoid arthritis is a multifactorial disease determined by both genetic and environmental factors

Previous twin studies have shown a higher concordance for rheumatoid arthritis in monozygotic than in dizygotic twins, but the results have been biased in favour of genetic effects

### What this paper adds

As concordance for rheumatoid arthritis in this study was no more common in monozygotic twins than in dizygotic twins environmental effects may be more important than genetic effects in the development of rheumatoid arthritis

arthritis and HLA markers is related to age at onset and sex.<sup>12,13</sup> We did not find any difference in age at onset and sex between monozygotic and dizygotic probands, though rheumatic nodules, an indicator of more severe disease, were more common in monozygotic twins. The UK rheumatoid arthritis twin study is the only other such study that can reject this potential bias.<sup>5</sup> Even though our study was population based, twins with rheumatoid arthritis did not differ from twins recruited from clinical settings with regard to the traditional measures of severity, including HLA associated antigens.

The lack of concordant pairs could be due to observer bias, but this is unlikely as the cotwins in pairs discordant for disease did not show any signs of arthritis, and in most cases disease classification was based on medical records and blood samples, both of which are resistant to observer bias. We identified only a small number of twins with rheumatoid arthritis because participants were mostly young, with 90% of the twins born in 1952-83.

Our study had an estimated probability of ascertainment of 78%. As there is always bias in ascertainment towards monozygotic twin pairs and twin pairs concordant for specific diseases and traits, complete ascertainment would not change our estimate of concordance for rheumatoid arthritis.

We acknowledge that our sample was relatively small, but we consider our results to be the most unbi-

ased estimate of the genetic contribution to rheumatoid arthritis and support the observation of a weak familial aggregation.<sup>14,15</sup> Genetic makeup seems of minor importance in the development of rheumatoid arthritis.

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## Commentary: Do genes or environment influence development of rheumatoid arthritis?

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The aetiology of rheumatoid arthritis, like several other chronic disorders, is widely accepted to be "multifactorial." This term suggests that the disease results from one or more environmental influences acting on a genetically susceptible background. Although the relative contributions of genetic and environmental influences are the source of several investigations, in

the strictest sense, as with most other diseases, neither is enough on its own to explain onset of the disease.

The size of a genetic effect is typically estimated from the familial risk of recurrence, defined as the increased risk in first degree relatives of affected individuals compared with the background occurrence in the population. With this approach the genetic con-

tribution to rheumatoid arthritis is indeed small. The risk of recurrence in a sibling is possibly not much greater than 2,<sup>1</sup> substantially lower than that seen, for example, with other autoimmune disorders such as multiple sclerosis and thyroid disease.<sup>2</sup> An extension to that approach is to consider the comparative risk between identical and non-identical cotwins of affected individuals. The underlying hypothesis is that identical (monozygotic) and (same sex) non-identical (dizygotic) twins are similar in their sharing of the environment and hence the magnitude of any excess risk of disease in the monozygotic twins quantifies the genetic effect.

Two nationwide twin studies, one from Finland,<sup>3</sup> the other from the United Kingdom,<sup>4</sup> yielded similar concordance rates in monozygotic twins of around 15%, four times greater than that seen in dizygotic twins. Indeed, from these two studies it was estimated that shared genetic factors in the monozygotic twins explained about 60% of the incidence of rheumatoid arthritis.<sup>5</sup> The assumption about similarity in environment between the two types of twins might not be true,<sup>6</sup> and hence this proportion represents the upper limit.

Against this background the results of Svendsen et al seem somewhat surprising. In this carefully conducted investigation with a national twin register they found no concordant monozygotic pairs and two concordant dizygotic pairs. The authors conclude that their data argue against a major genetic influence. There are some limitations in their interpretation. Small numbers and consequently wide confidence intervals mean that their results are not inconsistent with those from previously published studies. Also, the authors relied on recalled diagnosis that might have underestimated the true occurrence of the disease.

There is an undoubted genetic contribution to rheumatoid arthritis which, at least in part, is explained by a susceptibility allele at the HLA-DRB1 locus.<sup>7</sup> Indeed, possession of susceptibility alleles at this locus explains why some monozygotic twins are, and others

are not, concordant for rheumatoid arthritis.<sup>8</sup> HLA, however, may explain only about half of the genetic contribution to rheumatoid arthritis,<sup>9</sup> although it has been a difficult task to show genetic susceptibility factors other than HLA consistently among studies.<sup>10 11</sup>

In summary the study by Svendsen et al cannot disprove a genetic component in susceptibility to rheumatoid arthritis. However, their results emphasise that the genetic effects are weak compared with environmental ones in explaining differences in occurrence of the disease. This makes the task more difficult for those attempting large scale linkage studies aimed at revealing the genetic basis for rheumatoid arthritis.

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## A nose is a nose is a nose

As soon as I hit the asphalt, I knew what had happened. Another Smith's fracture, my third in 10 years. I was in the public car park next to one of my two offices, running to my next meeting when I was tripped up by a pebble. The car park attendant was extremely kind: he left his post and took me in his own car the couple of miles to the local accident and emergency department. The queue was horrendous, but I eventually reached the front, only to be told that the wait to be seen was 1 hour and 10 minutes and there was nowhere to sit down.

I would never normally have done what I did next, but—with a broken arm, a blackening eye, and a bruised face—it seemed the only thing to do. With many silent apologies to the rest of the waiting room, I pulled the only string I could and declared my identity as a medical practitioner. Suddenly a seat was found, the consultant on take was called from his lunch, I was given a telephone so that I could call my husband, and I was sent off to radiology. When I came back the consultant looked at the films, confirmed my diagnosis, and explained that he would add me to that afternoon's operating list and put in a plate.

"And while you're under the anaesthetic I'll sort out your nose," he said.

"My nose?" My nose is one of which Julius Caesar himself would have been proud and was currently the only pain-free feature on my face.

"Your nose," he repeated. "It's broken. Have you looked in the mirror?"

I looked in the mirror. My nose was its usual Roman self. "My nose is fine," I said.

"Fine? You can't go about looking like that. I've got to do something about it."

"But it's fine," I repeated, "I always look like this."

"You can't do," he shrieked.

We argued back and forth like this for several minutes and were almost coming to blows when, through the open door, I spotted my husband wandering down the corridor looking for me.

"Robert," I shouted, "Come and settle this."

Introductions were swiftly made, and the consultant got in first. "Tell me," he said, "Does this lady look like your wife?"

Robert surveyed me carefully. "Well she's a bit battered," he said, "but, yes, that's her."

The consultant subsequently made an excellent job of my arm but left my nose strictly alone. The accident and emergency department, however, has since had a massive facelift, thereby improving the service beyond recognition.

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