

EXTENDED REPORTS

Low prevalence of rheumatoid arthritis in Black-Caribbeans compared with Whites in inner city Manchester

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Abstract

Objective—To compare the prevalence of rheumatoid arthritis (RA) in Black-Caribbeans and Whites living in the same urban area.

Methods—Cases of inflammatory joint disease were ascertained initially from a postal screening survey of 1851 Black and 1829 age and sex-matched non-Blacks identified from general practice age-sex registers of seven general practices in the Moss Side and Hulme districts of Manchester. The ethnicity of respondents was confirmed using data from a postal screening questionnaire. Those reporting joint swelling or a history of arthritis were reviewed by a rheumatologist at surgeries held in each practice. The clinical records of the questionnaire non-responders and questionnaire-positive non-attenders at surgery were reviewed.

Results—In an adjusted denominator population of 1046 Black-Caribbeans and 997 Whites, the cumulative prevalence of RA was 2.9/1000 in Black-Caribbeans and 8/1000 in Whites, representing a prevalence in Black-Caribbeans of 0.36 times that found in Whites (95% confidence interval 0.1–1.3).

Conclusions—Rheumatoid arthritis occurs less commonly in Black-Caribbeans than in Whites. The findings are consistent with published studies showing a low RA prevalence in rural African Black populations.

(*Ann Rheum Dis* 1994; 53: 293–297)

The lowest prevalence estimates for rheumatoid arthritis (RA) worldwide have been recorded in certain rural African Black populations where the disease appears to be exceptionally rare.^{1–4} RA has also been reported to be mild in a number of studies of Blacks in Africa and the Caribbean,^{5–7} though reports are conflicting.^{8,9}

The apparent low prevalence of RA in Blacks may be due either to the low frequency of RA susceptibility genes^{4,10,11} or to environmental factors affecting disease incidence or survival. Interestingly, with respect to environmental risk factors, urban-dwelling South African Blacks have an RA prevalence closer to the 1%

figure found in European Whites.¹² Similarly, in temperate regions of Africa,⁸ the Caribbean¹³ and the United States of America,¹⁴ RA occurs in Blacks with a frequency close to that reported in Whites.

It is difficult, however, to compare prevalence results between populations when different methodological approaches have been used. The present study aimed to investigate the prevalence of RA in Blacks of Caribbean origin in a United Kingdom (UK) urban environment and compare the results with those in a White population living in the same area.

Methods

The study was conducted between 1990–92 in the Moss Side and Hulme districts of Manchester. These areas have an estimated population of 60 000 and have a high proportion of individuals of Black-Caribbean origin.

SAMPLING

On the basis of an expected prevalence of RA in Whites of 8/1000, a sample size of approximately 1300 Blacks and 1300 Whites was required to distinguish a prevalence in Blacks a third of that figure, with 80% power and a significance of 5%.

Complete patient lists comprising individuals over 18 years were compiled for seven general practices in the area, known to have a high proportion of Black-Caribbeans. The lists were obtained from the practice's age-sex register in four, and from the Family Health Service Authority (FHSA) listing in the remaining three.

Data on ethnicity are not recorded routinely in the United Kingdom by general practitioner (GP) or any other population register. A pilot study had, however, shown that the practice reception staff were able to identify correctly the ethnic origin of patients from the names listed on the practice register. In the main study we therefore asked the receptionists to identify the names of Blacks on the practice lists that had been compiled. For each Black, the next consecutive age and sex-matched non-Black was selected to form the comparison group. As a validation of the receptionists' assignment, the questionnaire used to detect joint disease

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Accepted for publication
12 January 1994

included a question on ethnic status using categories recommended by the Commission for Racial Equality (White, Black-African, Black-Caribbean, Asian, Oriental and Other).

SCREENING FOR INFLAMMATORY JOINT DISEASE

A postal screening questionnaire aimed at detecting any individual with current or past peripheral joint pain or swelling and those with a previous diagnosis of 'arthritis' was sent to all Blacks identified on the practice lists and to the corresponding comparison group. Individuals were asked directly to report (a) any past occurrence of joint swelling lasting more than four continuous weeks; (b) any joint pain lasting more than four continuous weeks; (c) current joint pain or swelling (they were asked to indicate the distribution on a mannequin); (d) current morning stiffness lasting more than 30 minutes; (e) whether they had been told in the past that they had arthritis; (f) whether they had been seen by a doctor in the past for any joint problem.

The postal screening aimed to be maximally sensitive to detecting all possible cases of RA in the denominator population. Its sensitivity to detect both current and previously diagnosed RA had been shown to be 100% in a pilot study of 20 individuals with RA (confirmed by a physician) that had been participating in a different population-based study.

The addresses of those who failed to respond to the initial screening questionnaire were checked with the electoral register. This has been shown to be an efficient method of identifying those actually resident at the address given on the FHSA list.¹⁵ Those whose names matched with information on the register were sent a second questionnaire within a period of four weeks. The GP records of all those not responding to the second questionnaire were reviewed for evidence of the following: (a) joint swelling ever detected by GP or hospital physician; (b) RA ever diagnosed by a GP or hospital physician; (c) results of previous radiological investigations; (d) results of previous serological test for rheumatoid factor.

CLINICAL EVALUATION

Responders who answered positively to a history of joint swelling of two or more joints were invited to attend for a clinical assessment by a rheumatologist (AJM or JMWH) in a special clinic held at the GPs surgery. Both observers saw an approximately equal

proportion of Black and non-Black individuals. Additionally, those who reported a diagnosis of 'arthritis' which was confirmed by the GP records were also invited to attend. Individuals who were unable to attend the clinic were visited at home. Radiographs of the hands and feet were arranged and blood was taken for rheumatoid factor determination (tube latex agglutination test) in all those in whom there was a clinical suspicion of RA or inflammatory polyarthritis (IP). The GP records of those invited who failed to attend for clinical evaluation were reviewed in a similar manner to the non-responders to the screening questionnaire.

Disease status was classified using the 1987 criteria, modified for use in population studies.^{16 17} The modification incorporates evidence of past and inactive disease and is a sensitive measurement of RA lifetime cumulative prevalence.

Those who had an episode of swelling in two or more joints which was present on clinical assessment or had been documented in the past by a physician, and for whom no other specific rheumatic disease diagnosis was appropriate, were classified as having IP.

The proportion of individuals with RA and IP was estimated for each ethnic group. The prevalence ratio and its 95% confidence interval was also evaluated.

Results

RESPONSE

From an initial 1851 Black and 1829 non-Black individuals identified from the practice lists 688 Blacks and 784 non-Blacks were subsequently excluded because: (a) individuals were not listed on the electoral register as being present at the address to which the questionnaire had been mailed; (b) the Post Office returned the questionnaire; (c) recorded delivery letters were unclaimed or (d) subsequent enquiry showed them to have moved off the GPs list. The age and sex matched structure of the sample was not significantly altered by the exclusion of these individuals (table 1).

The overall response to the screening questionnaire was 77.1%. The response was higher amongst females (79.1%) than males (74.7%) and in older than in younger age groups (50 and over: 83.7% less than 50: 72.7%). The response varied between practices, ranging from 73% to 88%. The response was lower in the Black (74%) than in the non-Black (81%) group.

In all, 20% of both the Black-Caribbean and White responders (based on self-defined ethnicity) reported a history of swelling of two or more joints. An additional 11% of the Black-Caribbeans and 9% of the Whites reported a history of 'arthritis' in the absence of swelling. In total, these yielded 344 individuals eligible to attend for clinical evaluation. After review of the notes of those reporting a diagnosis of 'arthritis', four Black Caribbeans, five Whites and one for whom ethnicity was unconfirmed were also invited to attend. The attendance

Table 1 Frequency distribution by age and sex in the 1163 'Blacks' and 1045 'non-Blacks' remaining after exclusion of those whose addresses could not be confirmed

Age	Males		Non-Black		Females		Non-Black	
	Black		n	%	Black		n	%
	n	%	n	%	n	%	n	%
<40	263	22.6%	227	21.7%	356	30.6%	296	28.3%
40-60	132	11.3%	130	12.4%	201	17.3%	190	18.2%
≥60	118	10.1%	109	10.4%	93	8.0%	93	8.9%

Percentages are expressed as a proportion of the total number of Blacks and non-Blacks respectively.

was 71% for Black-Caribbeans and 59% for Whites. The general practice records of both questionnaire non-responders and the questionnaire positive non-attenders were also reviewed, as indicated above. All the required records were available for examination.

CASES

RA was identified in 11 individuals: three Black-Caribbeans and eight Whites. Seven cases were identified following clinical evaluation and the remaining four from review of the records of the non-responders and the non-attenders at clinic. One of the non-attenders, a White female, was under the care of a rheumatology outpatient clinic with a diagnosis of RA but had declined radiographic examinations. She had, however, documented evidence of symmetrical inflammatory and destructive changes affecting the hands and feet, was receiving disease modifying drugs and would have satisfied the ARA criteria classification for RA, even in the absence of erosive changes in the joints. The ethnic origin of all three non-attenders with RA was confirmed in the clinical records. All three cases of RA in Black-Caribbeans occurred in women born in Jamaica whose disease onset occurred after they had emigrated to the United Kingdom. The clinical features of RA were similar in the Black-Caribbean and White patients but small numbers preclude a detailed comparison (table 2).

Current or past non-RA inflammatory polyarthritis (IP) was identified in 10 Black-Caribbeans. No case was identified in the Whites. In the Black-Caribbeans with IP, synovitis was found at current examination in seven and had been documented in the past in three, one of whom had histologically confirmed synovitis on biopsy. In all cases the disease had been confined to the wrists and small joints of the hands. None of these had other features of autoimmune rheumatic disease. All but one were seronegative for rheumatoid factor and none was antinuclear antibody positive. Although three White patients reported non-current non-specific joint pain or swelling, none had synovitis that had been confirmed at a clinical examination. No case of IP was identified from review of the clinical records of the non-responders and non-attenders.

Table 2 Clinical features of cases with rheumatoid arthritis

Patient	Age	Sex	RF	Erosions
Black-Caribbean:				
A	44	f	pos	present
B	60	f	neg	present
*C	66	f	pos	absent
White:				
D	45	f	neg	present
*E	47	f	pos	present
F	50	f	pos	present
G	53	f	pos	absent
*H	54	f	neg	-
I	61	f	pos	present
J	62	f	neg	absent
*K	65	m	pos	present

*Details obtained from notes review only.

ETHNICITY ASSIGNMENT

The estimated numbers of Black-Caribbeans and Whites in the study population were calculated based on the receptionists' original allocation and the answer of those responding to the ethnicity question. In all, 816 (96%) of the 'Black' (based on receptionists allocation) respondents answered the ethnicity question of whom 673 (82.3%) reported Black-Caribbean ethnicity, 52 (6.4%) White ethnicity and 93 (11.4%) reported belonging to other ethnic groups. By contrast, 835 (99%) of the 'non-Black' respondents answered the ethnicity question of whom 739 (88.5%) reported White ethnicity, 90 (10.8%) Black-Caribbean ethnicity and six (0.7%) other ethnicity. These rates were applied to the numbers of 'Blacks' and 'non-Blacks' who were either non-responders to the survey or who had not answered the ethnicity question. As a consequence, 1046 of the population were estimated to be Black-Caribbean, 997 to be White and 163 belonging to other ethnic groups. This latter group was excluded from further analysis.

PREVALENCE OF RA AND IP

The cumulative prevalence of RA was 2.9/1000 for Black-Caribbeans and 8.0/1000 for Whites. This represents a lower prevalence in Black-Caribbeans of 0.36 times (95% confidence interval 0.1-1.3). The prevalence of IP in the Black-Caribbeans was 9.6/1000. The difference could not be explained by age and sex, given the matched design of the study.

Discussion

This study has shown that the prevalence of RA in Black-Caribbeans is lower in comparison with that of Whites living in the same urban area. We were unable to demonstrate a difference in the disease manifestations of RA between Black-Caribbeans and Whites. However, non-specific IP in the absence of other evidence of autoimmune disease was detected only in the Black-Caribbeans. A number of inflammatory joint disorders including systemic lupus erythematosus (SLE)¹⁸ and sarcoidosis¹⁹ occur more commonly in Blacks and may account for the excess of IP seen in this group. None, however, had recognised clinical features which would have permitted their classification as a specific inflammatory disorder. An alternative explanation is that these were cases of mild or early RA.

It is difficult to compare our prevalence figures for RA with those of published studies. Several different classification schemes have been developed for RA over the last 40 years and no single method of disease definition has been used consistently. Furthermore, whilst most studies report point prevalence estimates, the extent to which these measures include information on individuals' past disease experience is often unclear. Failure to account for evidence of past disease may significantly underestimate RA prevalence.²⁰

Comparison with the results of studies of RA in African populations encounters additional problems. Adequate sample sizes are difficult to attain, random population sampling may be difficult, and the size of the denominator population may be poorly enumerated.²¹ Disease definition criteria are difficult to apply in their published form¹² and their characteristics may be altered by a higher background prevalence of rheumatoid factor seropositivity or joint erosions.¹⁻³ For example, in the only previous substantive study of RA in Caribbeans, conducted by Lawrence amongst agricultural workers in Jamaica, a high frequency of radiological erosions affecting the metatarsophalangeal joints alone was reported.¹³ These may have occurred as a result of mechanical trauma. Furthermore, rheumatoid factor seropositivity (as measured by the bentonite flocculation test) was common. That study reported a prevalence of RA of 1.5% in men and 2.2% in women from a sample size of 536.

In the present study we have measured lifetime cumulative prevalence of RA by using disease definition criteria that can accommodate the occurrence of past and inactive disease. The records of all those participating in the study were systematically examined for documented evidence of past inflammatory arthritis and seropositivity. Furthermore, we have measured disease prevalence in the Black-Caribbeans and Whites in the same area using the same sampling and evaluation techniques. This has enabled a valid comparison of RA prevalence between the two groups.

The approach used in the present study, however, faced a number of problems. The study was centred in an area of multiple deprivation and high crime levels. The sampling frame was restricted to individuals in primary care registered with their GP. The ultimate sample size fell short of the target size as a high proportion of individuals could not be traced to their listed address.

Complete enumeration of all the population in the area to include those not listed with their GPs would, however, only have been possible by a door-to-door census. This would have been impractical in these districts of Manchester. We have no reason to suspect that there might have been selective differences in registration between Blacks and non-Blacks which would have influenced the representativeness of the initial sample. Further, no important selective differences in the distribution of Black-Caribbeans and Whites were present in the final sample after exclusion of individuals whose GP-listed addresses could not be confirmed (table 1). It is unlikely that the comparison of prevalence in the two groups would have been influenced selectively by the loss of these individuals.

A high non-response rate was expected both to the screening questionnaire and also, for those positive on screening, to the motivation to attend for clinical assessment. As a result of non-response, we had to rely heavily on review of clinical records for evaluation of disease occurrence. The concern over the dependence

on review of records is that documentation of transient or limited disease in the past may be incomplete, hence lifetime disease occurrence may be underestimated. This is likely to be less important for established cases of RA than for cases of inflammatory synovitis. In this respect, it is interesting that while three of the 11 cases of RA were ascertained from the GP notes, no case of IP was detected in this way. As the level of non-response was similar in the Black and non-Black groups, it is unlikely that our reliance on GP notes could have accounted for the differential disease occurrence in Black-Caribbeans and Whites.

A further problem specific to this study was the characterisation of ethnicity. We relied on receptionists' recognition of names on a patient list to define the target population of Blacks and non-Blacks. The number of Black-Caribbeans and Whites in the final sample was estimated by adjusting the numbers in which ethnic status was not subsequently confirmed by using practice-specific misclassification rates. As a result, we were unable to define the denominator population precisely. This method was a practical solution to the problem of defining ethnicity where no independent source for this information exists. The method is likely to be broadly accurate and selective differences introduced in the estimate of population size between Black-Caribbeans and Whites arising through misclassification were minimal.

Our findings support the results of a number of published studies that have found a lower RA prevalence in Blacks.¹⁻⁴ The results, however, contrast with reports from South Africa, which have implicated the urban environment as a risk factor for RA in the Bantu people. In two studies, Solomon *et al*¹² and Beighton *et al*¹ found that the prevalence of RA in Bantu living in urban Johannesburg was similar to that found in Whites in the West but higher than that found in the same people living in the rural Western Transvaal.

In the present study, no adequate comparison group is available to allow us to comment directly on the influence of urbanisation in Black-Caribbeans. Furthermore, the only cases of RA identified in Black-Caribbeans in this study occurred in native Jamaicans after migration. As the peak of emigration from the Caribbean to Moss Side and Hulme occurred in the 1950s and 1960s²² it may be, as yet, too soon to evaluate the influence of the change to an urban environment in this population.

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