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Patterns of Peripheral Joint Involvement in Psoriatic Arthritis - Symmetric, Ray and/or Row?

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Abstract

Objective—We sought to examine whether joint involvement in psoriatic arthritis (PsA) follows a symmetric, ray, and/or row pattern using longitudinal data.

Methods—Data on activity and clinical damage of the joints of the hands and feet were obtained from a PsA cohort. For each analysis (symmetry, ray or row) for each outcome (joint damage and activity) expected values for table cells under the null hypothesis that joints progress independently to damage or activity were calculated based on a logistic regression model with patient level random effects for the probability of involvement developing between clinic visits. To determine the consistency of observed with expected values, goodness-of-fit tests were performed.

Results—Data from 704 patients were available. The 511 (552) patients with no hand (foot) damage at clinic entry were used for analyses of hand (foot) damage. When considering joint damage, there was strong evidence against independence of joint involvement based on evident symmetric patterns. There was little suggestion of ray patterns of joint damage. There was considerable evidence for row pattern of involvement of joints. When considering joint activity, symmetric patterns were also evident but, unlike joint damage, there was evidence of ray patterns, most notably in the hands. There was also evidence for row pattern involvement.

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Conclusion—Patterns of peripheral joint involvement seen over time in PsA patients, demonstrate consistency with expected ray patterns of disease activity, especially in the hands, but there is also considerable evidence for symmetric and row patterns for both joint damage and activity.

Keywords

Psoriatic arthritis; patterns; symmetry; ray pattern; damage; inflammation

Introduction

Psoriatic arthritis (PsA) is an inflammatory musculoskeletal disease that may involve the peripheral and axial joints as well as the entheses.[1] Clinically, peripheral arthritis is the most common manifestation of PsA.[2] There are no diagnostic markers for PsA. Therefore, clinical diagnosis of PsA is based on recognizing patterns of inflammatory joint involvement.[3]

Moll and Wright recognized five subsets of PsA.[4] Of these 5 subsets, the two most common are asymmetric oligoarthritis and symmetric polyarthritis.[5,6] A “ray” pattern involving all three joints of an affected digits has been described for the distribution of affected joints with other digits of the hand being less involved.[2,7] Asymmetric arthritis and the ray distribution are clinical features often used to distinguish PsA from rheumatoid arthritis (RA). However, Helliwell *et al.* examined damage patterns seen over follow-up and demonstrated that the perceived difference in symmetry between PsA and RA is due to the higher number of joints generally being involved in RA.[8] Moreover, using data from patients registered with the Norfolk Arthritis Register, Bukhari *et al.* demonstrated that inflammatory polyarthritis is a symmetrical disease irrespective of rheumatoid factor status.[9] The authors challenged the use of symmetry as an important feature in identifying subgroups of patients with inflammatory arthritis, such as RA and PsA. It is also possible that joint involvement in PsA can be described as a “row” pattern where all joints of a particular type (e.g. MCP) are involved. However, apart from the attempt by Helliwell *et al.*, as far as we know there has not been an attempt to formally investigate whether the pattern of joint involvement in PsA is appropriately described as symmetric, ray, and/or row.[8]

Thus, the long term issue of patterns in PsA has not been resolved and there is a need for additional clinical evidence for the presumed patterns including symmetry and ray distribution.

We therefore sought to examine whether joint involvement (damage as well as activity) in PsA follows a symmetric (joints on both sides are involved), ray (all joints within a particular digit involved) or row (joints at the same level are involved) pattern using longitudinal data on joint involvement in patients diagnosed with PsA from the University of Toronto PsA clinic. Although an explicit comparison with RA is not made, we believe that characterizing the patterns actually observed in a large cohort of well characterized PsA patients with extended follow-up allows one to examine these observed patterns in light of current presumptions and therefore provide information that would be of interest to clinicians.

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Methods

Patient and assessments

The University of Toronto PsA clinic is the source of an observational cohort of PsA patients followed prospectively since 1978.[10] Patients are followed at approximately 6–12-month intervals according to a standard protocol. Most patients satisfy the CASPAR classification criteria for PsA.[11] The clinical evaluation includes assessment of disease activity and clinical damage of 68 individual joints. A total of 704 patients registered in the clinic between 1978 and 2006 and observed for at least two clinic visits were included in the current study. While all 704 patients were used for the activity analyses, hand (foot) damage analyses were based on the 511 (552) patients without hand (foot) damage at clinic entry. Four patients were excluded from some foot activity analyses due to incomplete data. The study was approved by the University Health Network Research Ethics Board.

Assessment of activity and clinical damage

Disease involvement in an individual joint in PsA may be described in terms of clinical activity and clinical damage. An actively inflamed joint is described as either only tender (presence of stress pain and/or joint line tenderness) or swollen (joint swelling), where a swollen joint may or may not be tender. Clinical damage is determined by the presence of a limitation of range of movement of more than 20% of the range not related to the presence of joint effusion, the presence of joint deformities, subluxation, flail joints or ankylosis. The reliability of these measures has been demonstrated.[12–15] The advantage of using data on clinical over radiographic damage, particularly for the study of individual joints, is that clinical joint damage is measured at each clinic visit, whereas radiographs are undertaken, usually, once every 2 years and provide relatively sparse data. In addition, in contrast to only 42 joints being assessed using radiographs (by the modified Steinbrocker method), clinical joint damage can be assessed in all 68 joints thus allowing for the assessment of ray pattern in the feet.[5,16] We have previously demonstrated that there is a relationship between clinical and radiographic damage.[17]

Assessment of symmetry in joint damage and activity

Statistical modelling and tabulations—In order to examine whether joint involvement in PsA follows a symmetric pattern or not, tabulations of data on joint damage at last clinic visit, and activity in a joint at any time, were examined for evidence of symmetry; i.e. cross-tabulations of the number of hand joints involved in symmetric pairs and the number of hand joints involved ‘singularly’ were performed. Details of the statistical methodology are described elsewhere [18], and more technical detail on the methodology is provided in a supplementary statistical appendix.

Briefly, expected values for table cells under the null hypothesis that joints progress independently to damage or activity were calculated. These were based on estimation of a logistic regression model with patient level random effects (to account for the expected correlation due to joints in the same patient "behaving" more similarly than joints in different patients) for the probability of involvement (separate models for damage and for activity), at each joint location, developing between clinic visits. The model allowed for

differential rates of involvement by joint location and for the time between visits, thus accounting for varying follow-up times. These expectations were conditional on a patient having at least one involved joint as patients with no damage or activity are not informative about symmetry. Because the modelling is done at the joint level and the tables of interest are at the patient level, these expectations were estimated by simulating joint involvement for each patient based on the estimated logistic model. The simulated joint involvement was used to determine which cell of the table of interest the patient would enter in order to produce a simulated table for all patients. One thousand tables of this nature were simulated and, for each cell, the average number of patients observed in the cell, over these 1000 samples, was calculated. These average values comprise the expectations reported in each table.

Goodness-of-fit Method: A goodness-of-fit test to determine the consistency of observed values with these expected values were performed. Departures from expectations, if present, were then examined for evidence of symmetry. A Pearson-type test statistic, G, of the form $\Sigma(O-E)^2/E$, was used. The summation is over all cells in the table as is done for the well-known chi-squared based goodness-of-fit tests. However, the chi-squared methodology assumes that the “counts” and “expectations”, under the null hypothesis, derive from a simple multinomial distribution. Here, these derive from a more complex model where the probability of being in a cell varies from individual to individual. Therefore, it is necessary to estimate the distribution of G, under the null hypothesis, by an additional simulation. This was done through the generation of 1000 bootstrap samples [19]. For each sample, the appropriate table is determined and the counts in the table, along with the expectations previously calculated, are used to calculate a G statistic. The simulated sample of 1000 G values are then ordered to determine the critical values for the significance test. For example, the 950^{th} highest value corresponds to the 95^{th} percentile of the distribution and the 5% critical value for a test of the null hypothesis of independence of joint involvement. For presentation purposes, the 95^{th} and 99^{th} percentiles, the latter corresponding to a 1% critical test value, of the simulated distributions are provided with each table, along with the maximum value observed in the 1000 samples. An observed value of the G which is bigger than this maximum would generate a significance level for the test of the null hypothesis of $p<0.001$.

Unlike the method of Helliwell et al. for assessing symmetric damage, this approach, while based on the same type of data, does not assume that all joints are equally likely to develop damage, and is not restricted to use of a binary definition of symmetry.[8] In contrast with the method of Bukhari et al, this methodology accounts for differences in patients’ susceptibility to damage, and allows evaluation of symmetry at the patient or joint group (e.g. hand) level in addition to that seen in individual joints.[9] This method also accounts for the varying times between clinic visits and the number of visits.

We examined symmetry of joint damage and activity in the hands including the metacarpophalangeal (MCP), proximal interphalangeal (PIP) and distal interphalangeal (DIP) and wrist joints (15 joints in each hand). We also evaluated the symmetry of damage and activity in joints of the feet (ankles, metatarsophalangeal (MTP) and interphalangeal (IP- the two IPs of toes 2-5 combined into one for a total of 11 joints in the feet).

Assessment of ray patterns of damage and activity

To examine ray patterns, cross-tabulations were produced of the number of joints involved in matching left and right digits for patients with joint damage at their last observation. If a ray pattern is present, the observed number of pairs with one side fully damaged (or active) but the other not should be greater than expected. The tabulations were compared with that expected if involvement of the joints was independent, similar to the analyses done for assessment of symmetry. However, expectations for these analyses were not conditioned on having at least one involved joint in the pair of digits since more than expected digits with no joints damaged could also be seen as consistent with a ray pattern.

Assessment of row patterns of damage and activity

Row patterns were examined through tabulations of the number of involved joints of each type in the right and left hand and foot (e.g. the number of MCP joints involved in each hand). Again, expected values for the table cells were calculated under an assumption of independence of joint involvement allowing for differences between patients, and joint locations, in terms of their susceptibility to damage/activity. These expectations were conditional on having at least one joint involved at the joint location of interest.

Results

The demographics and disease characteristics at first clinic visit of the patients included in the study are provided in Table 1.

Assessment of symmetry

Table 2 provides the cross-tabulation of the number of hand joints damaged in symmetric pairs and the number of hand joints damaged ‘singularly’, together with the expected number for each cell of the table under the null hypothesis of independence of joint damage. As indicated in the methods, these expectations are conditional on damage being observed in a patient so that the 341 of the 511 patients who do not develop damage over the course of follow-up do not contribute to the test statistic. Also given for each cell is the contribution of that cell to the value of the observed test statistic. For example, the second cell in the first row, corresponding to 2-6 joints being damaged in symmetric pairs and none asymmetrically, contributes a value of $(22\text{-}1.19)^2/1.19=364.31$ based on the larger than expected value of 22 patients in this cell, consistent with a symmetric pattern being observed. The first cell in the second row, corresponding to no observations of symmetric joint involvement but 1-2 joints being damaged asymmetrically, contributes a value $(64\text{-}99.25)^2/99.25=12.52$ based on the lower than expected value of 64 patients in this cell, consistent with less asymmetric joint involvement than would be expected under the null hypothesis.

A table footnote gives the simulated maximum value of the test statistic along with the 95% and 99% percentiles of the null distribution of the test statistic. Comparison of observed and expected counts leads to a test statistic G for the observed data of 4176.03. This value far exceeds the maximum of the G values for the 1000 simulated data sets (536.31) so the corresponding significance level is $p<0.001$. The departure from expectations remains if

columns 3-4 and 5-7 are pooled. This provides strong evidence against the null hypothesis of independence of joint progression to damage. It is consistent with the presence of symmetric joint damage in PsA as the dominating contribution that accounts for over 72% of the observed test statistic value and corresponds to more observations of 2-12 hand joints damaged in symmetric pairs with no joints damaged singularly. Thus, there are more observations of purely symmetrical damage than expected.

Similar analysis for joint damage in the feet (Supplementary Table A1) also showed departures of the observed from expected, consistent with symmetric damage ($p<0.001$). As for the hands, the main contributions to the highly significant test statistic value corresponded to more observations of purely symmetrical foot damage than expected.

Table 3 provides a similar tabulation with respect to joints that have been active (tender or swollen) at any assessment. The evidence against independence of joint activity is strong ($p<0.001$) and the results are consistent with symmetric activity patterns as typically more joints are involved symmetrically than individually. However, in contrast with hand damage, the results are not so dominated by the occurrence of purely symmetric activity. When we examined evidence of symmetric disease activity in the joints of the feet (Supplementary Table A2), the results were dominated by the occurrence of purely symmetric activity ($p<0.001$). The test statistic had the value 269.72 while the maximum simulated value was 51.97.

Assessment of ray pattern

Table 4 provides the cross-tabulation of the number of right and left hand (excluding the thumbs since that digit has only 2 joints) joints damaged, together with the expected number for each cell, if joint involvement is independent, along with the corresponding contribution to the value of the observed test statistic. The observed goodness-of-fit statistics G for this table is 107.8 greater than the maximum simulated value of 49.1 thus providing evidence against the null hypothesis of independence of joint damage ($p<0.001$). However, the six cells of Table 4 that would be expected to have the largest deviations from expected if a ray pattern was present (the first three entries of the 4^{th} row and 4^{th} column of the body of the table which correspond to one finger being fully damaged and the matching finger not) only contribute 7% to G. Furthermore, the values are smaller than expected for a number of these cells. Thus there seems to be little evidence for ray pattern. The large value of the test statistic is influenced considerably by entries along the diagonal, in particular the cell representing 28 finger pairs with both fingers fully damaged. In light of what is seen in the off-diagonal cells, these departures from expectation seem most likely to be linked to symmetry in damage patterns. A comparable table for the examination of ray damage patterns in the feet has a very similar structure (supplementary Table A3).

Analogous assessment for joint activity in the hands (supplementary Table A4) also provided evidence against independence of joint activity ($p<0.001$) but the largest contributions to the test statistic from this tabulation arose from the two cells corresponding to one finger having activity in all joints and none in the other. These departures are consistent with activity in the hands displaying a ray pattern. A comparable tabulation for activity in the feet (supplementary Table A5) displayed smaller excesses in the cells

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corresponding to one toe being fully active and the other not displaying activity and had a much greater excess in the cell corresponding to both toes being fully active, a result that could derive from symmetric patterns of activity.

Assessment of row pattern

In the course of investigations of symmetric and ray patterns, the data suggested that a large number of hands and feet developed damage and activity (considered separately) in all joints of a particular type (e.g. MCP). We therefore formally investigated evidence for these 'row patterns'. Table 5 presents the number of MCP joints damaged in the right hand excluding the thumb. The expected values were calculated conditional on the number of patients with MCP damage in the right hand. The observed value for G (46.30) is much larger than the maximum value, 16.9 obtained from the simulated datasets demonstrating that the distribution of damage within the MCP joints is significantly different to that expected if joints progressed independently to damage ($p<0.001$). The departures are consistent with row patterns with the value of the test statistic dominated by more observations than expected of damage in all of the MCP joints.

Similar analyses (supplementary Table A6) revealed that there also was evidence for row pattern of joint damage in the left MCP ($p<0.001$). All other joints also demonstrated evidence of row damage patterns ($P<0.001$ for all but right PIP for which $0.01<p<0.05$). See supplementary Tables A7-A14.

Similar analyses of joint activity in the hands and feet were all consistent with the occurrence of row patterns (supplementary Tables A15-A24.).

Discussion

Asymmetric arthritis and 'ray' pattern of involvement have been used to clinically distinguish PsA from RA.[2] In the original description of PsA, Moll and Wright identified symmetric arthritis in a small proportion of their patients. However, subsequent cohort studies demonstrated that polyarticular disease is more common, and at least half the patients had symmetric distribution. We therefore sought to investigate whether there indeed was evidence of occurrence of asymmetric joint involvement as well as ray (involving all three joints of an affected digit, other digits of the hand being less involved) or row (where all joints of a particular type are involved) patterns in by analyzing longitudinally observed data on joint involvement in our large well-defined cohort of patients with PsA.

We demonstrate that when considering joint damage, there was strong evidence against independent damage processes in the joints based on the presence of symmetric patterns in both hand and feet joints. There was little evidence for the presence of ray pattern of joint damage in the hands or feet. There was considerable evidence for a row pattern of involvement in the joints of the hands as well as the feet. When considering joint activity (tender or swollen), there was also strong evidence of a symmetric pattern but, unlike the situation for damage, there was evidence of ray patterns in the hands and feet, most notable in the hands. There was also evidence for row pattern involvement of the joints of the hands and feet. A summary of our findings is:

1. Damage: Symmetry present in hands and feet, ray absent in hands and feet, row present in hands and feet
2. Activity: Symmetry present in hands and feet, ray present in hands and feet, row present in hands and feet

These results build on similar work done by Helliwell *et al.* and Bukhari *et al.* and indicate that pattern of peripheral joint involvement, especially in the hands, may not be a reliable way to distinguish PsA from other forms of chronic inflammatory arthritis.[8,9]

Helliwell *et al.* had previously shown that symmetry is largely a function of the total number of joints involved and that in terms of joint pattern, differences between RA and PsA are more quantitative than qualitative, and both disorders have high absolute values of symmetry, particularly in the joints of the wrist and hand.[8] Bukhari *et al.* investigated symmetry in erosions comparing rheumatoid factor positive to negative patients from a primary care based cohort of patients with inflammatory arthritis and found no evidence of more symmetry in the rheumatoid factor positive group. In their analysis, symmetry for each joint location (e.g. wrist) was assessed separately via contingency tables, and the cell frequencies in such tables were interpreted using log-linear modelling. Here using a more general statistical method than those of Helliwell *et al.* and Bukhari *et al.*, their findings with respective to symmetric patterns in PsA have been confirmed. The application of the methods of Helliwell *et al.* (after a minor correction) and Bukhari *et al.* to our data have been reported elsewhere and also demonstrated evidence for a symmetric joint involvement in PsA.[8,9,18] The discrepant finding with regard to ray damage/activity compared to findings for the symmetry and row patterns is intriguing. The smaller number of patients/joints in the damage analyses does not explain this finding. One explanation may be that ray pattern of activity may not lead to ray pattern of damage. This was not formally investigated.

The strengths of our study, in addition to the use of statistical methodology that accounts for variation in joint involvement by location, length of follow-up and patients' varying susceptibility to joint involvement, is that the dataset used to conduct the analyses was collected prospectively using a standardised protocol over a relatively long duration of follow up.[10] The methods used to collect data on disease activity and joint damage have been previously shown to be reliable [12–15] We were also able to evaluate the feet in detail and have shown some differences in pattern especially with regard to ray involvement. Although damage is often assessed by plain radiographs, clinical assessment of joint damage is reliable and closely parallels radiographic damage.[12,17] The observation points, with regard to frequency as well as the number of joints assessed, are much larger, providing additional power for statistical analyses. The detailed collection and electronic tracking of data also allowed us to comprehensively investigate the hands and feet (right and left) separately. Weaknesses include the inability to compare the results found with other inflammatory arthritis such as RA since we did not have access to a dataset similar to the one we have for PsA. Such a comparison would help us determine the clinical utility of the symmetry, ray and row patterns in distinguishing PsA from other arthritides especially RA in early as well as late disease. Although an explicit comparison with RA is not made, we believe that characterizing the patterns actually observed in a large cohort of well characterized PsA patients with extended follow-up allows one to examine these observed

patterns in light of current presumptions and therefore provide information that would be of interest to clinicians. Moreover, we did not investigate sub-phenotypes of PsA, such as arthritis mutilans. It is possible that joints that have severe damage (such as subluxation, flail joints or ankylosis) that may indicate arthritis mutilans may have asymmetric involvement as well as ray involvement. Future studies are needed to address this severe phenotype.

Conclusion

In conclusion, our analyses argue against the presence of asymmetry in joint involvement of the hands in PsA. There is also no evidence of a ray pattern for damage in the hand or feet joints but some for activity. Row pattern of involvement predominates in the hands and feet.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be published. Dr. Gladman had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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Table 1
Characteristics of the patient population at clinic entry

Patient's characteristics	Proportion or median (range) All patients	Proportion or median (range) Patients without hand damage at entry	Proportion or median (range) Patients without foot damage at entry
Number of patients	704	511	552
Female/Male	313/391	225/286	242/310
Age, years	43 (14,86)	41 (14,86)	41 (14,79)
Duration of arthritis, years	4.3 (0,47.7)	2.9 (0,47.3)	3.2 (0, 47.3)
Number of clinically damaged joints*	0 (0,53)	0 (0,20)	0 (0,19)
Number of swollen joints*	2 (0,33)	2 (0,33)	2 (0,33)
Number of active joints*	8 (0,55)	6 (0,55)	7 (0, 48)
*Median (range)			

Table 2

Damage in hand joints: cross-tabulation of 511 patients by the number of hand joints damaged in symmetric pairs and the number of hand joints damaged 'singularly' at last observation

	No. symmetrically damaged joints					
No. singularly damaged joints	0	2-6	8-12	14-18	20-24	26-30
0	341*	22	2	2	0	4
	341†	1.19	0	0	0.01	0.8
	(0)‡	(364.31)	(1197.17)	(2398.34)	(0.01)	(12.84)
1-2	64	21	4	2	1	2
	99.25	5.2	0.18	0.09	0.7	1.06
	(12.52)	(48.06)	(79.94)	(43.19)	(0.13)	(0.84)
3-4	16	10	3	1	0	
	19.44	7.84	1.27	0.67	1.91	
	(0.61)	(0.59)	(2.34)	(0.16)	(1.91)	
5-6	2	7	1	2	0	
	6.46	7.9	2.97	1.01	0.22	
	(3.07)	(0.10)	(1.31)	(0.96)	(0.22)	
>6	3	1	0	0		
	2.65	6.40	2.61	0.18		
	(0.05)	(4.56)	(2.61)	(0.18)		

*Observed value

†Expected value

‡Contribution to the test statistic

Observed data: G=4176.03; Simulated samples: max. G=536.31, 95% <32.09, 99% <125.38.

Table 3

Activity in hand joints: cross-tabulation of 704 patients by the number of hand joints active in symmetric pairs and the number of hand joints active 'singularly' at last observation

	No. symmetrically active joints					
No. singularly active joints	0	2-6	8-12	14-18	20-24	26-30
0	47*	8	1	2	4	23
	47†	2.43	0.14	0.22	2.16	3.58
	(0) ‡	(12.81)	(5.15)	(14.07)	(1.57)	(0.01)
1-2	62	42	7	14	26	37
	69.30	23.75	3.57	2.48	13.58	32.55
	(0.77)	(14.02)	(3.29)	(53.56)	(11.36)	(0.61)
3-4	39	49	34	22	41	
	49.2	46.40	14.37	15.94	40.67	
	(2.12)	(0.15)	(26.84)	(2.3)	(0.003)	
5-6	27	56	36	35	5	
	31.16	57.88	32.80	40.84	8.88	
	(0.56)	(0.06)	(0.31)	(0.83)	(1.7)	
>6	17	25	39	6		
	20.36	63.49	47.57	13.69		
	(0.55)	(23.34)	(1.54)	(4.32)		

*Observed value

†Expected value

‡Contribution to the test statistic

Observed data: G=181.83; Simulated samples: max. G=44.63, 95% <28.60, 99% <35.84.

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Table 4

Damage in hand joints: Cross-tabulation of 532 finger pairs (from 133 patients with finger damage) by number of joints damaged in left and right fingers excluding the thumbs at last observation

	Left finger			
Right finger	0	1	2	3
	229	59	19	2
0	263.45	78.31	9.22	0.81
	(4.50)	(4.76)	(10.38)	(1.73)
	70	57	8	2
1	78.56	32.20	12.24	2.30
	(0.93)	(17.06)	(1.47)	(0.04)
	12	14	24	3
2	9.29	12.31	10.43	4.94
	(0.79)	(0.23)	(17.67)	(0.76)
	2	2	1	28
3	0.82	2.25	5.12	8.74
	(1.68)	(0.03)	(3.32)	(42.46)

*Observed value; † Expected value; ‡Contribution to the test statistic
Observed data: G=107.82; Simulated samples: max. G=49.14, 95% <23.24, 99% <28.75.

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Table 5

An example of a row pattern: tabulation of 511 patients by number of MCP joints damaged in right hand at last observation.

	No. of MCP joints damaged in the right hand (excluding thumbs)				
	0	**1**	**2**	**3**	**4**
Observed	486	10	6	1	8
Expected	486	17.80	4.27	1.80	1.13
Test statistic contribution	0	3.42	0.700	0.35	41.83
Observed data: G=46.30;					
Simulated data: max. G=16.90, 95%<6.03, 99%<8.62					