Clin Rheumatol (2013) 32:659–663
DOI 10.1007/s10067-013-2165-y

ORIGINAL ARTICLE

# Association of cigarette smoking with Chinese ankylosing spondylitis patients in Taiwan: a poor disease outcome in systemic inflammation, functional ability, and physical mobility

**Chun-Hsiung Chen · Hung-An Chen · Chin-Li Lu · Hsien-Tzung Liao · Chin-Hsiu Liu · Chang-Youh Tsai · Chung-Tei Chou**

Received: 17 April 2012 / Revised: 30 December 2012 / Accepted: 3 January 2013 / Published online: 18 January 2013


**Abstract** We investigated the association between smoking and the disease activity, functional ability, physical mobility, and systemic inflammation in Chinese ankylosing spondylitis (AS) patients. Seventy five male Chinese AS patients in Taiwan were enrolled in the cross-sectional study. These patients fulfilled the 1984 modified New York criteria. Patients completed the questionnaires, containing the demographic data, disease activity, functional ability (BASFI), and patient's global assessment. Meanwhile, physical examinations were performed to determine the patient's physical mobility. Acute-phase reactants, erythrocyte sedimentation rate (ESR), and C-reactive protein levels were also measured in the AS patients. Smoking habits with smoking duration and smoking intensity (pack–years of smoking) were recorded. Among these physical mobility parameters, modified Schober's index ($p<0.001$), cervical rotation ($p=0.034$), later lumbar flexion ($p=0.002$), chest expansion ($p=0.016$), and occiput-to-wall distances ($p=0.003$) were significantly impaired in smoking AS patients ($n=35$) as compared to non-smoking ($n=40$). Systemic inflammation parameter, ESR was significantly higher in smoking AS patients than non-smoking ($p=0.03$). The odds ratio of advanced modified Schober's index, lateral lumbar flexion, fingertip-to-floor distance, chest expansion, and occiput-to-wall were significantly elevated in smoking AS patients as compared to non-smoking. Moreover, the smoking intensity correlated significantly with BASFI ($r=0.481$, $p=0.005$), cervical rotation ($r=-0.401$, $p=0.031$), fingertip-to-floor distance ($r=0.485$, $p=0.004$), and occiput-to-wall distance ($r=0.473$, $p=0.005$) in the 35 smoking AS patients. The cigarette smokers in the Chinese AS patients have increased systemic inflammation and poor physical mobility. In addition, the higher smoking intensity in the AS smokers is associated with poor disease outcome, including functional ability and physical mobility. Thus, it is quite important for the physician to emphasize the association of smoking with poor disease prognosis in AS, and patients should be strongly recommended to avoid smoking cigarette.

**Keywords** Ankylosing spondylitis · Functional ability · Physical mobility · Smoking inflammation

C.-H. Chen · C.-H. Liu
Buddhist Tzu Chi General Hospital, Taipei Branch, Taipei, Taiwan

C.-H. Chen · C.-Y. Tsai · C.-T. Chou
Yang-Ming University and Taipei Veterans General Hospital, Taipei, Taiwan

H.-A. Chen · C.-L. Lu
Chi Mei Medical Center, Liouying, Tainan, Taiwan

H.-T. Liao
Taipei Medial University–Municipal Wan Fang Hospital, Taipei, Taiwan

C.-T. Chou (✉)
Division of Allergy–Immunology–Rheumatology, Taipei Veterans General Hospital, No. 201, Section.2, ShiPai Road, Taipei 112, Taiwan
e-mail: ctchou@vghtpe.gov.tw

## Introduction

Spondyloarthritis is a family of chronic arthritis diseases, characterized by inflammatory back pain, peripheral arthritis,

and enthesitis [1]. Ankylosing spondylitis (AS) is the typical disease among the family of spondyloarthritis, and it predominantly involves the axial joints and bilateral sacroiliac joints. It is a potentially debilitating disease, which may lead to progressive limitation of the spinal mobility, loss of the functional ability, and reduced quality of life. Inflammatory rheumatic diseases are considered to be due to a complex interaction between environment and genetic factors, which may lead to immune reactions and cause different rheumatic disorders [2]. The environmental factor might be quite important in the development of chronic rheumatic and immune disease.

Cigarette smoking, one of the most serious health problems, has been identified as one of the major environmental risk factor of rheumatic diseases, including rheumatoid arthritis (RA) [3–6] and systemic lupus erythematosus (SLE) [7, 8]. Previous studies also showed that smoking was associated with rapid disease progression with poor functional outcome of AS [9–12]. The present data about the association of cigarette smoking with AS disease entity in Chinese population is absent. The association of smoking intensity with the AS smokers has rarely been reported [13]. The aim of this study was to assess the differences between the smoking and non-smoking Chinese AS patients, including the systemic inflammation, disease activity, functional ability, and physical mobility, and also evaluate its association with smoking intensity.

## Patients and methods

We consecutively collected 75 male AS patients who fulfilled the 1984 modified New York criteria [14] and visited the Outpatient Department of Division of Allergy–Immunology–Rheumatology, Taipei Veterans General Hospital. These patients were asked to fill out a questionnaire which contains the demographic data, disease activity, functional ability, and patient's global assessment. Disease activity, functional ability, and patient's global assessment in the AS patients were assessed by using the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) [15], Bath Ankylosing Spondylitis Functional Index (BASFI) [16], and Bath Ankylosing Spondylitis Patient Global Score (BAS-G) [17], with visual analogue scale. Meanwhile, physical examinations were performed to determine the patient's physical mobility, including tragus-to-wall distance, lumbar flexion (modified Schober's index), intermalleolar distance, cervical rotation (CROT), lateral lumbar flexion, fingertip-to-floor distance (FFD), chest expansion, and occiput-to-wall distance (OWD). The BASDAI, BASFI, or BAS-G scores had a final range from 0 to 10. Acute-phase reactants, including erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels were also measured in these AS patients. History of peripheral joint involvement was obtained through the questionnaire inquiry.

Smoking habit was evaluated by face-to-face interview: 1. □ Smoker (A. □ Current smoker: Pack per day, duration years (age started smoking); B. □ Past smoker: Pack per day, duration years (age started smoking, number of years since quitting); 2. □ Non-smoker. We calculated the smoking duration and smoking intensity (pack–years of smoking, the product of years of smoking and packs of cigarette per day).

### Statistical analysis

Statistical analyses were carried out using the SPSS statistical package. The Mann–Whitney *U* test or Fisher's exact test was used to analyze group differences. Correlations between variables were determined by the Spearman's rank correlation test. Multivariate logistic regression analysis was performed to calculate the odds ratios (OR) of different clinical parameters, and the positive cutoff values of each clinical parameter were obtained by receiver operating characteristic (ROC) curve analysis. *P* values were regarded as being significant if they were less than 0.05.

## Results

Table 1 shows the demographics and clinical characteristics of the 75 male AS patients. These AS patients were divided into two subgroups with smoking (including current smoker

**Table 1** Demographic and disease characteristics of the 75 male AS patients

| Characteristic | Total AS patients ($n$=75) |
|---|---|
| Age (years) | 33.55 (10.67) |
| Disease duration (years) | 10.71 (9.59) |
| HLA B27 (+) % | 71/75 (94.67 %) |
| Peripheral joint involvement (+) % | 44/75 (58.67 %) |
| ESR (mm/h) | 24.60 (17.91) |
| CRP (mg/dl) | 1.81 (1.82) |
| BASDAI | 4.05 (2.02) |
| BASFI | 2.28 (2.23) |
| BAS-G | 4.80 (2.67) |
| Tragus-to-wall distance (cm) | 13.82 (4.68) |
| Modified Schober's index (cm) | 3.57 (1.78) |
| Intermalleolar distance (cm) | 115.30 (19.79) |
| Cervical rotation (degree) | 49.10 (16.66) |
| Lateral lumbar flexion (cm) | 11.79 (6.27) |
| Fingertip-to-floor distance (cm) | 25.85 (29.92) |
| Chest expansion (cm) | 4.47 (1.92) |
| Occiput-to-wall distance (cm) | 3.61 (6.19) |

Values are shown as mean (SD)

and past smoker) and non-smoking. Clinical parameters were compared between the two subgroups (Table 2). There was no statistically significant difference between smoking AS patients and non-smoking in age ($p$=0.068), onset age ($p$=0.56), and disease duration ($p$=0.261). There was also no significant difference of peripheral joint involvement rate between smoking AS patients and non-smoking ($p$=0.251).

Among the physical mobility parameters (Table 2), it is of interest that modified Schober's index ($p$<0.001), cervical rotation ($p$=0.034), later lumbar flexion ($p$=0.002), and chest expansion ($p$=0.016) were significantly reduced in smoking AS patients as compared to those with non-smoking. In addition, occiput-to-wall distances were significantly increased in smoking patients than those with non-smoking ($p$=0.003). Taken together, smoking AS patients showed relatively poor physical mobility than those with non-smoking. ESR was significantly higher in smoking AS patients than those with non-smoking [mean (SD), 29.63 (17.97) vs. 20.63 (17.05) mm/h, $p$=0.03]. The values of BASDAI ($p$=0.283), BASFI ($p$=0.240), BAS-G ($p$=0.305), and CRP ($p$=0.171) levels were higher in smoking AS patients than those with non-smoking, but these difference did not show statistical significance.

Furthermore, among the 35 smoking AS patients, the smoking intensity correlated significantly with BASFI ($r$=0.481, $p$=0.005), cervical rotation ($r$=−0.401, $p$=0.031), fingertip-to-floor distance ($r$=0.485, $p$=0.004), and occiput-to-wall distance ($r$=0.473, $p$=0.005) (Table 3). The smoking duration also correlated significantly with BASFI ($r$=0.409, $p$=0.018), fingertip-to-floor distance ($r$=0.482, $p$=0.004), and occiput-to-wall distance ($r$=0.402, $p$=0.021).

Using multivariate logistic regression analysis, the odds ratio for the relationship between smoking status and different clinical parameters has also been calculated (Table 4). The smoking AS patients showed significantly elevated odds ratio (OR, 95 % confidence interval) than those with non-smoking in advanced ESR (3.34, 1.08 to 10.34), CRP (6.06, 1.23 to 29.78), modified Schober's index (12.17, 2.60 to 56.98), lateral lumbar flexion (4.61, 1.51 to 14.08), fingertip-to-floor distance (4.06, 1.14 to 14.43), chest expansion (4.44, 1.05 to 18.78), and occiput-to-wall (4.61, 1.36 to 15.6).

## Discussion

Recent studies demonstrated that cigarette smoking could be a trigger of autoimmune disease, such as RA and SLE and related to the presence of rheumatoid factor, anti-citrulline antibody, and anti-dsDNA antibodies [3, 18]. Smoking could be an important environmental factor in the disease process of inflammatory rheumatic disease, including AS. Our data showed that smoking AS patients suffered from poor physical mobility compared with those with non-smoking. The length of modified Schober's index, cervical rotation, later lumbar flexion, and chest expansion were significantly decreased in smoking AS patients as compared to those with non-smoking. In addition, distances of occiput-to-wall significantly increased in smoking AS patients. Moreover, multivariate logistic regression analysis revealed that the odds ratio of advanced modified Schober's index, lateral lumbar flexion, fingertip-to-floor distance, chest expansion, and occiput-to-wall were

**Table 2** Comparison of the clinical parameters between smoking AS patients (current and past smokers) and non-smoking

| Clinical parameters | Smoking ($n$=35) | Non-smoking ($n$=40) | $P$ value |
|---|---|---|---|
| Age (years) | 35.03 (9.48) | 32.25 (11.57) | 0.068 |
| Onset age (years) | 23.43 (8.42) | 22.13 (8.17) | 0.56 |
| Disease duration (years) | 11.50 (9.27) | 10.13 (9.90) | 0.261 |
| Peripheral joint involvement (+) % | 18/35 (51.43 %) | 26/40 (65 %) | 0.251 |
| ESR (mm/h) | 29.63 (17.97) | 20.63 (17.05) | 0.03* |
| CRP (mg/dl) | 2.23 (2.02) | 1.46 (1.58) | 0.171 |
| BASDAI | 4.30 (1.91) | 3.84 (2.11) | 0.283 |
| BASFI | 2.54 (2.30) | 2.05 (2.16) | 0.240 |
| BAS-G | 5.20 (2.62) | 4.46 (2.69) | 0.305 |
| Tragus-to-wall distance (cm) | 14.61 (5.44) | 13.13 (3.84) | 0.148 |
| Modified Schober's index (cm) | 2.75 (1.77) | 4.30 (1.47) | <0.001* |
| Intermalleolar distance (cm) | 118.30 (20.35) | 112.83 (19.23) | 0.199 |
| Cervical rotation (degree) | 44.56 (17.01) | 52.91 (15.57) | 0.034* |
| Lateral lumbar flexion (cm) | 9.30 (6.02) | 13.97 (5.71) | 0.002* |
| Fingertip-to-floor distance (cm) | 29.87 (28.03) | 22.24 (23.64) | 0.121 |
| Chest expansion (cm) | 3.89 (1.81) | 5.0 (1.88) | 0.016* |
| Occiput-to-wall distance (cm) | 5.71 (7.21) | 1.76 (4.47) | 0.003* |

Values are shown as mean (SD). $P$ values are determined by Mann–Whitney test or Fisher's exact test

*$P$ values <0.05 are significant

**Table 3** Correlation between smoking status and clinical parameters in the 35 smoking AS patients

| Clinical parameters | Smoking intensity[a] | | Smoking duration | |
|---|---|---|---|---|
| | *r* | *P* value | *r* | *P* value |
| Age | 0.725 | <0.001* | 0.764 | <0.001* |
| Disease duration | 0.588 | 0.001* | 0.60 | 0.001* |
| BASDAI | 0.288 | 0.103 | 0.152 | 0.398 |
| BASFI | 0.481 | 0.005* | 0.409 | 0.018* |
| BAS-G | 0.176 | 0.328 | 0.016 | 0.931 |
| ESR | 0.191 | 0.331 | 0.092 | 0.641 |
| CRP | 0.255 | 0.199 | 0.190 | 0.342 |
| Tragus-to-wall distance | 0.319 | 0.07 | 0.256 | 0.151 |
| Modified Schober's index | −0.332 | 0.059 | −0.212 | 0.236 |
| Intermalleolar distance | −0.219 | 0.244 | −0.304 | 0.103 |
| Cervical rotation | −0.401 | 0.031* | −0.331 | 0.079 |
| Lateral lumbar flexion | −0.242 | 0.174 | −0.193 | 0.282 |
| Fingertip-to-floor distance | 0.485 | 0.004* | 0.482 | 0.004* |
| Chest expansion | −0.220 | 0.218 | −0.133 | 0.460 |
| Occiput-to-wall distance | 0.473 | 0.005* | 0.402 | 0.021* |

*r* is determined by Spearman's rank correlation test

**P* values <0.05 are significant

[a]Pack–years of smoking (product of years of smoking and packs of cigarette per day)

significantly elevated in smoking AS patients compared to those with non-smoking. In a UK cohort of 53 AS patients, Averns HL et al. demonstrated that smoking patients have poor outcome of finger–floor distance, Schober's test, total spinal movement, occiput–wall distance, functional index, stiffness, and spine X-ray scores [12]. One study of 48 AS patients in Turkey showed that there were significantly decreased lumbar modified Schober's test and chest expansion, and increased hand–ground distance in smokers than non-smokers [11]. Ward MM et al. in the USA showed that smoking was associated with more rapid progression of functional ability and a poor prognostic factor for radiographic severity in the AS patients [10, 19]. Previous studies also showed that cigarette smoking is associated with poor functional status outcome in AS [20] and radiographic spinal progression in early axial spondyloarthritis [21]. Bodur H et al. also showed that smoking negatively affected the quality of life in AS [22].

In our study, ESR was significantly higher in smoking AS patients than those with non-smoking. In addition, BASDAI, BASFI, BAS-G, and CRP levels had trend to be higher in smoking AS patients than those with non-smoking, but these differences did not show statistical significance. Multivariate logistic regression analysis showed that the odds ratio of higher ESR, and CRP were significantly elevated in smoking AS patients compared to those with non-smoking. Previous reports ever showed that smoking AS patients had significantly higher BASDAI and BASFI scores [9, 11]. However, significantly higher BASDAI and BASFI scores were not observed in our results, which may be due to our

**Table 4** Variables associated with smoking in the logistic regression analysis

| Clinical parameters (positive cutoff values[a]) | OR[b] (95 % C.I.) | *P* value |
|---|---|---|
| ESR (≥21.5 vs. <21.5 mm/h) | 3.34 (1.08 to 10.34) | 0.036* |
| CRP (≥2.99 vs. <2.99 mg/dl) | 6.06 (1.23 to 29.78) | 0.027* |
| BASDAI (≥3 vs. <3) | 2.905 (0.922 to 9.155) | 0.069 |
| BASFI (≥1.5 vs. <1.5) | 1.661 (0.622 to 4.431) | 0.311 |
| BAS-G (≥4.93 vs. <4.93) | 1.87 (0.68 to 5.15) | 0.227 |
| Tragus-to-wall distance (≥16.25 vs. <16.25 cm) | 2.51 (0.70 to 9.01) | 0.159 |
| Modified Schober's index (≤2.25 vs. >2.25 cm) | 12.17 (2.60 to 56.98) | 0.002* |
| Intermalleolar distance (≥140.75 vs. <140.75 cm) | 3.29 (0.53 to 20.52) | 0.202 |
| Cervical rotation (≤61.25 vs. >61.25 degree) | 3.32 (0.90 to 12.18) | 0.071 |
| Lateral lumbar flexion (≤11.25 vs. >11.25 cm) | 4.61 (1.51 to 14.08) | 0.007* |
| Fingertip-to-floor distance (≥3.6 vs. <3.6 cm) | 4.06 (1.14 to 14.43) | 0.030* |
| Chest expansion (≤2.9 vs. >3.6 cm) | 4.44 (1.05 to 18.78) | 0.043* |
| Occiput-to-wall (≥4 vs. <4 cm) | 4.61 (1.36 to 15.6) | 0.014* |

**P* values <0.05 are significant

[a]Positive cutoff values obtained by ROC curve analysis

[b]The ratio of smoking odds (smoking versus non-smoking) between two clinical parameter groups; adjusted for onset age, disease duration, and peripheral joint involvement

small patient number or possible confounders. In the previous cohort of 48 AS patients, ESR ($p$=0.094) and CRP levels ($p$=0.082) were higher in the smokers than non-smokers but did not reach statistical difference [9]. In another recent cohort of 647 early axial spondyloarthritis patients, smoking was independently associated with higher disease activity, increased axial inflammation and structural damage on magnetic resonance imaging (MRI), poorer functional status, and poorer quality of life [23]. Smoking in AS patients could be related with chronic inflammation, which lead to progressive joint damage and new bone formation, resulting in ankylosing of adjacent vertebral body and poor spinal mobility. Furthermore, our study demonstrated that the AS smokers with higher smoking intensity have poorer disease outcome, such as functional ability (BASFI), cervical movement (CROT, OWD), and spinal mobility (FFD). Our results were compatible with a recent study which showed that smoking has a dose-dependent relationship with measures of disease severity in AS, including decreased function and poor quality of life [13].

In conclusion, cigarette smoking, one of the most concerned public health problems, is associated with higher systemic inflammation and poor physical mobility in the Chinese AS patients. Also, smoking intensity has a relationship with disease severity in AS patients, including poor functional ability and physical mobility. Smoking in AS patients may be associated with increase systemic inflammation, poor functional, and physical status; thus, quitting smoking is an important recommendation for the AS patients to improve their long-term disease outcome. However, the effect of smoking on AS disease process cannot be established on such a cross-sectional study only. To assess the association between the smoking and AS disease outcome, a larger-scale study with longitudinal follow-up in disease activity, functional ability, and physical mobility is necessary.

**Disclosure** None.

## References


1. Zochling J, Brandt J, Braun J (2005) The current concept of spondyloarthritis with special emphasis on undifferentiated spondyloarthritis. Rheumatology (Oxford) 44:1483–91
2. Aho K, Heliovaara M (2004) Risk factors for rheumatoid arthritis. Ann Med 36:242–251
3. Stolt P, Bengtsson C, Nordmark B, Lindblad S, Lundberg I, Klareskog L et al (2003) Quantification of the influence of cigarette smoking on rheumatoid arthritis: results from a population based case–control study, using incident cases. Ann Rheum Dis 62:835–841
4. Costenbader KH, Feskanich D, Mandl LA, Karlson EW (2006) Smoking intensity, duration and cessation and the risk of rheumatoid arthritis in women. Am J Med 119:503–511
5. Klareskog L, Stolt P, Lundberg K, Källberg H, Bengtsson C, Grunewald J et al (2006) A new model for an etiology of rheumatoid arthritis: smoking may trigger HLA-DR (shared epitope)-restricted immune reactions to autoantigens modified by citrullination. Arthritis Rheum 54:38–46
6. Criswell LA, Merlino LA, Cerhan JR, Mikuls TR, Mudano AS, Burma M et al (2002) Cigarette smoking and the risk of rheumatoid arthritis among postmenopausal women: results from the Iowa Women's Health Study. Am J Med 112:465–471
7. Costenbader KH, Kim DJ, Peerzada J, Lockman S, Nobles-Knight D, Petri M et al (2004) Cigarette smoking and the risk of systemic lupus erythematosus: a meta-analysis. Arthritis Rheum 50:849–857
8. Freemer MM, King TE Jr, Criswell LA (2006) Association of smoking with dsDNA autoantibody production in systemic lupus erythematosus. Ann Rheum Dis 65:581–584
9. Ward MM, Weisman MH, Davis JC Jr, Reveille JD (2005) Risk factors for functional limitations in patients with long-standing ankylosing spondylitis. Arthritis Rheum 53:710–717
10. Ward MM (2002) Predictors of the progression of functional disability in patients with ankylosing spondylitis. J Rheumatol 29:1420–1425
11. Kaan U, Ferda O (2005) Evaluation of clinical activity and functional impairment in smokers with ankylosing spondylitis. Rheumatol Int 25:357–360
12. Averns HL, Oxtoby J, Taylor HG, Jones PW, Dziedzic K, Dawes PT (1996) Smoking and outcome in ankylosing spondylitis. Scand J Rheumatol 25:138–142
13. Mattey DL, Dawson SR, Healey EL, Packham JC (2011) Relationship between smoking and patient-reported measures of disease outcome in ankylosing spondylitis. J Rheumatol 38:2608–2615
14. Van der Linden S, Valkenburg HA, Cats A (1984) Evaluation of diagnostic criteria for ankylosing spondylitis: a proposal for modification of the New York criteria. Arthritis Rheum 27:361–368
15. Garrett S, Jenkinson T, Kennedy LG, Whitelock H, Gaisford P, Calin A (1994) A new approach to defining disease status in ankylosing spondylitis: the Bath Ankylosing Spondylitis Disease Activity Index. J Rheumatol 21:2286–2291
16. Calin A, Garrett S, Whitelock H, Kennedy LG, O'Hea J, Mallorie P et al (1994) A new approach to defining functional ability in ankylosing spondylitis: the development of the Bath Ankylosing Spondylitis Functional Index. J Rheumatol 21:2281–2285
17. Jones SD, Steiner A, Garrett SL, Calin A (1996) The Bath Ankylosing Spondylitis Patient Global Score (BAS-G). Br J Rheumatol 35:66–71
18. Klareskog L, Padyukov L, Alfredsson L (2007) Smoking as a trigger for inflammatory rheumatic diseases. Curr Opin Rheumatol 19:49–54
19. Ward MM, Hendrey MR, Malley JD, Learch TJ, Davis JC Jr, Reveille JD et al (2009) Clinical and immunogenetic prognostic factors for radiographic severity in ankylosing spondylitis. Arthritis Rheum 61:859–866
20. Doran MF, Brophy S, MacKay K, Taylor G, Calin A (2003) Predictors of longterm outcome in ankylosing spondylitis. J Rheumatol 30:316–320
21. Poddubnyy D, Haibel H, Listing J, Märker-Hermann E, Zeidler H, Braun J et al (2012) Baseline radiographic damage, elevated acute phase reactants and cigarette smoking status predict radiographic progression in the spine in early axial spondyloarthritis. Arthritis Rheum 64:1388–1398
22. Bodur H, Ataman S, Rezvani A, Buğdaycı DS, Cevik R, Birtane M et al (2011) Quality of life and related variables in patients with ankylosing spondylitis. Qual Life Res 20:543–549
23. Chung HY, Machado P, van der Heijde D, D'Agostino MA, Dougados M (2011) Smokers in early axial spondyloarthritis have earlier disease onset, more disease activity, inflammation and damage, and poorer function and health-related quality of life: results from the DESIR cohort. Ann Rheum Dis 71(6):809–16