A Personality Characteristic, Somatic Absorption, and the Perception of Somatic Symptoms in Rheumatoid Arthritis Patients

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ABSTRACT. ***Objective.*** This study tested the hypothesis that a personality trait, somatic absorption, is correlated with symptom severity in patients with rheumatoid arthritis (RA).

Methods. Patients completed self-report questionnaires assessing intensity of their RA symptoms, somatic absorption, and psychiatric distress. Disease activity and severity were measured through erythrocyte sedimentation rate, joint examination, and aggressiveness of medication regimen. We examined the cross-sectional association between somatic absorption and RA symptoms using multivariable regression analyses.

Results. Somatic absorption was significantly ($p < 0.05$) associated with an overall measure of RA symptoms, and this association persisted after taking into account demographic data, disease severity, and extent of psychological distress. Somatic absorption was more closely associated with constitutional symptoms than with localized, articular symptoms of arthritis. Somatic symptoms were also independently associated with psychiatric distress ($p < 0.001$). Psychiatric distress was a more powerful predictor of extraarticular or constitutional symptoms than were measures of arthritis activity and severity.

Conclusion. Our findings suggest that there may be a role for psychological intervention in the management of extraarticular symptoms of RA as these symptoms are relatively more influenced by a personality characteristic than the localized articular symptoms of the disease. (First Release Mar 1 2008; J Rheumatol 2008;35:782–9)

Key Indexing Terms:
RHEUMATOID ARTHRITIS PERSONALITY PSYCHOLOGY SOMATIC ABSORPTION

The symptoms of rheumatoid arthritis (RA) guide diagnosis and treatment, prompt utilization of medical services, and profoundly affect the quality of life and productivity of patients. A common clinical observation and well documented research finding is that patients with similar RA by objective measures (such as joint swelling, radiographic appearance of structural damage, extraarticular manifestations, performance-based tests of impairment, and acute-phase reactants) differ widely in the severity of their symptoms and level of function[1,2]. It is likely that symptoms derive from a complex interplay between soma and psyche and that psychological factors contribute to an arthritis patient's symptomatology and disability[1-3].

A number of psychological influences on RA symptoms have been examined, including psychiatric distress, coping and cognitive style, social support, and life stress. Increased pain is associated with depression and anxiety, even after taking RA severity into account[1-4]. Coping and cognitive style have been conceptualized as moderators between disease severity and the resulting pain and role impairment; similarly, greater pain is associated with passive coping, helplessness, and catastrophizing[5-14]. Social support moderates the influence of disease, and greater social support is associated with more favorable reports of pain and health status[15,16]. Finally, pain is correlated with perceived life stress, even after controlling for disease activity[17-19].

Our study, nested in a randomized, controlled intervention trial, tested the hypothesis that a positive association exists between one specific personality characteristic, somatic absorption (SA), and arthritis symptoms. SA refers to the perceptual tendency to attend to a single sensory stim-

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ulus and to "gate out" other competing stimuli[20,21]. We hypothesized that individuals with higher SA would be more aware of and focused on their symptoms, and would therefore report more severe RA symptoms, even after arthritis severity was taken into account. As a secondary goal, we investigated whether certain symptoms are more closely associated with SA than others; that is, whether certain arthritis symptoms reflect the patient's state of mind to a greater degree. This distinction is important clinically, since RA symptoms more closely associated with psychological factors might benefit from cognitive and behavioral interventions, and RA symptoms closely associated with disease severity would presumably be more responsive to antirheumatic medication.

MATERIALS AND METHODS

Study design. We conducted a randomized, controlled clinical trial of 2 behavioral interventions to improve the symptoms and role impairment of patients with RA. Brigham and Women's Hospital Human Research Committee approved the study. Potential subjects were telephoned after their physicians granted us permission to make contact. The investigators described the study and sought informed consent. Patients who consented and met eligibility criteria completed a baseline interview to measure the variables of interest.

Participants completed questionnaires and underwent a standardized joint examination by a rheumatologist who was blinded to their questionnaire responses. Blood was obtained for laboratory testing. The participants were then randomized to one of 3 treatment arms. This report presents data gathered at the baseline assessment only.

Subjects. Subjects were included if they were 18 to 75 years old; met American College of Rheumatology criteria for RA[22]; and were literate in English. The 3 exclusion criteria were: fibromyalgia; serious medical comorbidity likely to progress substantially or cause death in the ensuing 12 months; and current participation in psychosocial treatment for RA.

Subjects were recruited in 2 ways. The majority were identified through the hospital's HIPPA-compliant computerized patient registry that was queried for the names of all patients diagnosed with RA in the previous year. A smaller number of subjects volunteered in response to public announcements and advertisements. Although subsequent analyses found that there were some significant differences between these 2 cohorts (i.e., the volunteers were younger, had been diagnosed for a shorter period of time, were more likely to be single, and were more likely to be employed), cohort membership did not modify the effect of the primary relationship of interest between symptom reports and SA. Therefore, only a simple binary indicator of cohort membership was needed in our regression analyses in order to control for potential confounding.

RA symptom assessment. The primary outcome was RA symptoms, assessed with the Rheumatoid Arthritis Symptom Questionnaire (RASQ). This 14-item, face-valid, self-report questionnaire was designed to approximate questions directed to patients in the course of typical clinical practice. It includes items on pain, stiffness, swelling, restriction of movement, fatigue, appetite, difficulty sleeping, and generalized malaise. Each symptom is rated on a 10-cm visual analog scale from "no" distress (0) to the "worst possible" distress (10). Visual analog scales have been in long-standing use in the measurement of arthritis symptoms such as pain and have demonstrated adequate reliability and validity[2,23,24].

Disease severity and disease activity. There is no single best method for assessing overall disease activity and disease severity in RA, and a variety of measures have been used alone or in combination. These include laboratory findings, physical findings, and symptoms. We used the erythrocyte sedimentation rate (ESR; Westergren method) and standardized physician ratings of joint swelling to assess disease severity. ESR is considered a useful measure of RA activity, especially in circumstances when the physical examination is indeterminate or when active disease is suspected and the joint examination reveals little or no abnormality[25]. It has demonstrated superior sensitivity as compared to C-reactive protein[26]. Since it was crucial in this study to distinguish subjective symptoms from objective, demonstrable pathology, we purposely did not rely on measures that include subjective symptom reports, for instance, the Disease Activity Score (DAS).

Joint swelling was rated with a standardized, 28-joint physical examination by a rheumatologist[27-31]. Each joint was rated (0 = no swelling, 1 = detectable synovial thickening without loss of bony contours, 2 = loss of distinctness of bony contours, 3 = bulging synovial proliferation with cystic characteristics), and a total joint swelling score was obtained. This method yields reproducible results that are associated with ESR and immunochemical determinations[27-31]. Joint swelling has been found to be a good index of overall disease activity[32].

Somatic absorption. Absorption is a dimension of personality defined as the capacity for deep involvement in sensory and imaginal events during which there is an imperviousness to distracting stimuli[20,21,33]. Psychologists consider absorption to be one facet of the trait "openness to experience," a basic personality trait measured by the NEO Personality Inventory[34]. Absorption is the most frequently studied correlate of hypnotizability[35], and has been associated with bodily distress, somatic symptom-reporting[36,37], and anticipatory nausea and vomiting during chemotherapy[38,39].

Absorption has been assessed with the Tellegen Absorption Scale[20,40]. This self-report questionnaire was revised by Watson to specifically assess somatic and visceral sensation. Respondents are asked to what degree each of 32 bodily sensations or experiences is true or false on a 5-point Likert scale. Examples are, "I could imagine that my arm is so heavy that I could not move it," "I often notice how clothes feel against my skin," "The taste of food stays in my mouth for a long time," and "When I'm lying in bed at night, I often become aware of my body," "When I watch TV or a movie, I am very aware of my bodily reactions." This SA scale has satisfactory test-retest reliability (r = 0.78 over 1 month and r = 0.65 over 2 months) and internal consistency (coefficient alpha = 0.82–0.86) (Watson D, unpublished data). Factor analysis has revealed a single factor and no discrete subscales. A low, although significant, correlation with neuroticism has been found, suggesting some degree of discriminant validity (Watson D, unpublished data).

Psychiatric symptoms. We assessed psychiatric symptoms rather than diagnosable psychiatric disorders. Psychiatric symptoms are closely associated with more numerous and more intense somatic symptoms. Depression in particular is common among patients with RA and has been shown to affect somatic symptoms, role impairment, and disability[8,14,41]. Psychiatric symptoms were measured with the Rand Mental Health Inventory (MHI), a standard, widely used self-report questionnaire. It is composed of common symptoms associated with the more prevalent mental disorders, and has a high degree of internal consistency and external validity[42-45]. Importantly for our purposes, it does not include physical or psychosomatic symptoms. We employed the 18-item version, and report the summary score of all items.

Medications. During the baseline interview, patients enumerated their current RA medications, including analgesics, nonsteroidal antiinflammatory agents, cyclooxygenase-2 inhibitors, salicylates, disease modifying agents, biologic response modifiers, steroids, and antidepressants prescribed for pain. These medications were categorized hierarchically according to aggressiveness of treatment and degree of risk associated with use. These categories included symptomatic drugs, steroids, disease modifying antirheumatic drugs (DMARD), and biological response modifiers.

Statistical analyses. The primary outcome variable was the mean RA symptom score for each subject, calculated as an average across the 14 individual items of the RASQ questionnaire. Symptom scores could range from 0 (no distress) to 10 (worst possible distress).

We carried out factor analysis with varimax rotation to identify any clusters of RA symptoms that were highly intercorrelated. This disclosed 3 factors with eigenvalues > 1, dividing the 14-item scale into 3 coherent subscales. The first subscale was composed of 6 items (joint pain, limb pain, joint stiffness, joint swelling, movement limitations, and joint deformities) and was interpreted to represent localized, articular symptoms. It explained 28% of the variability in RASQ symptoms. The second subscale was composed of 6 items (fatigue, feverishness, generalized aching, "sickness all over," sleeping difficulties, and back pain) and was interpreted to represent constitutional, extraarticular symptoms. It explained 22% of the variability in RASQ symptoms. The third subscale focused on gastrointestinal (GI) symptoms and included 2 items (poor appetite and weight loss) that explained 15% of the variability in RASQ symptoms. We examined the relationship between each of these 3 subscales and somatic absorption.

For the average of all RASQ symptoms and for each subscale, we constructed a series of regression models, examining their relationship with the SA score. Demographic variables were selected from Table 1 for inclusion in the model if they were related at a level of $p \leq 0.06$ either to the initial outcome, the overall RASQ score, or to the primary predictor, SA. The objective measures of disease severity in Table 1, the measure of psychological distress, and a binary marker for recruitment source (volunteer vs clinic registry) were included in the model regardless of p values. In this way, we had good assurance of identifying the variables that could potentially be confounders. For each series of regression models, the initial model was unadjusted for any covariates except recruitment source. We then added adjustments for the basic demographic factors of age, sex, race (dichotomized to White versus non-White because of the low number of minority patients in our study), receipt of disability benefits, and marital status (married vs not married). Then we adjusted for the demographic and design factors plus 6 objective measures of disease severity (ESR; number of swollen joints; and the use of symptomatic drugs, steroids, DMARD, and biological response modifiers). Finally, we adjusted for all the preceding factors plus the Rand MHI score. Through this staged approach, we hoped to isolate the distinct contributions of somatic absorption, disease severity, and psychiatric symptoms. To allow comparisons of the relative importance of each of these domains, the results of the regressions were summarized through partial r-squared values for each predictor.

RESULTS

A total of 168 subjects were enrolled (144 Brigham and Women's Hospital patients and 24 volunteers); Table 1 shows their demographic and disease characteristics. The cohort was predominantly female, highly educated, and employed.

There was considerable variability among patients on each of the 14 RASQ scale items. Individual patient scores ranged from 0 to > 9 for each item. On average, the most distressing symptoms were joint stiffness, joint pain, and fatigue. The least distressing symptoms were weight loss, poor appetite, and feverishness.

SA was more pronounced in younger subjects (r = –0.20, p = 0.008) and in subjects with more severe psychiatric symptoms (r = 0.19, p = 0.012). Hispanic patients (p = 0.007), Black patients (p = 0.004), and patients receiving disability benefits (p = 0.03) also exhibited higher levels of SA. SA was not related to time since diagnosis, symptoms, education, sex, marital status, or employment status. We found no relationship between SA and either measures of disease severity or number of medications. These results suggest that RA severity and SA are distinct domains. The individual RASQ symptoms that correlated most closely with SA scores were pain in limbs (r = 0.20, p = 0.009) and back (r = 0.18; p = 0.024); fatigue (r = 0.26, p = < 0.001); generalized aching (r = 0.23, p = 0.003); and feeling sick all over (r = 0.20, p = 0.009).

To test the hypothesized association between absorption and arthritis symptoms, we used multivariable regression with the overall RASQ symptom scale (Table 2) and RASQ subscales (Tables 3–5) as dependent variables, and SA as the independent variable. Since there were a number of potential confounding factors, we conducted these regressions in a serial fashion, successively adding covariates and examining the resulting change in the association between absorption and symptoms. In Table 2, the first column shows that

Table 1. Patient demographics and disease characteristics (n = 168). Data are percentage (n), unless indicated otherwise.

Characteristic	Data
Age, yrs; mean (SD)/median (range)	53.4 (13.0)/55.5 (24, 75)
Sex	
Female	87 (146)
Male	13 (22)
Education	
< High school	1 (2)
High school	14 (23)
Some college	20 (34)
College	25 (42)
Graduate school	40 (67)
Race	
White	80 (134)
Black	13 (21)
Asian	5 (9)
Other	2 (4)
Marital status	
Single	22 (37)
Married	49 (82)
Widowed, divorced, separated	29 (49)
Employment (% currently working)	
No	43 (73)
Yes	57 (95)
Disability (% receiving disability)	
No	74 (125)
Yes	26 (43)
Medications	
Symptomatic drugs	80 (134)
Steroids	29 (48)
DMARD	60 (101)
Biologic response modifiers	44 (74)
Time since initial diagnosis, mo; mean (SD)/median (range)	160 (140)/130 (8, 600)
Somatic Absorption Scale (32–160); mean (SD)/median (range)	3.3 (0.44)/3.3 (1.9, 4.5)
Psychiatric symptoms (Rand Mental Health Inventory; 18–108); mean (SD)/median (range)	43.3 (13.6)/41.0 (18, 85)
Total swollen joints; mean (SD)/median (range)	4.0 (4.5)/3.0 (0, 32)
Westergren ESR, mm/h; mean (SD)/median (range)	23.3 (21.1)/18.0 (0, 112)

Table 2. Multivariable predictors of overall symptom (Rheumatoid Arthritis Symptom Questionnaire) score. The importance of each predictor is depicted through adjusted partial r-squared and p value. All models are adjusted for whether patients were found through hospital registries or as volunteers.

Variables	Unadjusted Effect of Somatic Absorption, n = 167	Model Adjusted for Demographics, n = 167	Model Adjusted for Demographics and Disease Severity*, n = 147	Model Adjusted for Demographics, Disease Severity, and Psychological Distress†, n = 147
Full model	r^2 = 5%	r^2 = 8%	r^2 = 19%	r^2 = 26%
Somatic absorption	r^2 = 5% (p = 0.005)	Partial r^2 = 4% (p = 0.016)	Partial r^2 = 5% (p = 0.008)	Partial r^2 = 3% (p = 0.045)
Age	NA	Partial r^2 < 1% (p = 0.93)	Partial r^2 < 1% (p = 0.83)	Partial r^2 < 1% (p = 0.58)
Sex	NA	Partial r^2 < 1% (p = 0.92)	Partial r^2 < 1% (p = 0.95)	Partial r^2 < 1% (p = 0.70)
Race, White vs non-white	NA	Partial r^2 < 1% (p = 0.59)	Partial r^2 < 1% (p = 0.19)	Partial r^2 < 1% (p = 0.09)
Marital status, married vs unmarried	NA	Partial r^2 = 3% (p = 0.044)	Partial r^2 < 1% (p = 0.18)	Partial r^2 < 1% (p = 0.55)
Receiving disability benefits	NA	Partial r^2 < 1% (p = 0.92)	Partial r^2 < 1% (p = 0.48)	Partial r^2 < 1% (p = 0.40)
Swollen joints	NA	NA	Partial r^2 = 8% (p = 0.001)	Partial r^2 = 8% (p < 0.001)
ESR	NA	NA	Partial r^2 < 1% (p = 0.42)	Partial r^2 < 1% (p = 0.51)
Symptomatic drugs	NA	NA	Partial r^2 < 1% (p = 0.32)	Partial r^2 = 2% (p = 0.12)
Steroids	NA	NA	Partial r^2 < 1% (p = 0.39)	Partial r^2 < 1% (p = 0.28)
DMARD	NA	NA	Partial r^2 < 1% (p = 0.75)	Partial r^2 < 1% (p = 0.88)
Biologic response modifiers	NA	NA	Partial r^2 < 1% (p = 0.70)	Partial r^2 < 1% (p = 0.71)
Psychiatric symptoms	NA	NA	NA	Partial r^2 = 8% (p < 0.001)

* ESR, number of medications, joint examination. † Rand Mental Health Inventory. NA: not applicable; ESR: erythrocyte sedimentation rate; DMARD: disease modifying antirheumatic drug.

SA is significantly associated with arthritis symptom severity, explaining 5% of the variability in the severity scores. As more covariates are added to the model in each successive column of Table 2, the overall variance explained increases to 26%, and the independent contribution of SA falls to 3%, although the association remained significant (p = 0.045). In the final model, in the last column of Table 2, we see that the most important independent predictors of symptom severity are disease severity (measured by swollen joints) and psychiatric morbidity (measured by the Rand MHI), each explaining 8% of the variability in symptom severity. Therefore, each of these 3 domains — somatic absorption, disease severity, and psychiatric morbidity — has a distinct and independent influence on symptom severity.

Tables 3 and 4 depict similar findings. SA is significantly associated with both the extraarticular RA symptom and GI symptom subscales. SA initially explains 8% of the variability in systemic symptom scores and 4% of the variability in GI symptom scores. In the case of the systemic symptom subscale, its independent effect attenuated to 3% after accounting for psychiatric morbidity (Rand MHI: partial r^2 = 10%) and disease severity (swollen joints; partial r^2 = 3%). Each of these domains (SA, psychiatric distress, and disease severity) remained independently and significantly associated with systemic, extraarticular symptom severity in the final, fully adjusted regression model. In the case of the GI symptom subscale, the independent effect of SA remained 4%, even after psychiatric distress and disease severity were taken into account.

By contrast, Table 5 shows that SA is associated to only a small degree with the variability in localized articular symptoms. In the unadjusted model, SA demonstrates no significant association with joint-specific symptoms. In the fully adjusted model, it accounts for only 1% of symptom variance.

DISCUSSION

Although clinicians tend to assume a fixed, one-to-one relationship between demonstrable pathology and symptom reporting, psychological factors play an important role in the

Table 3. Multivariable predictors of systemic, extraarticular symptom subscale of Rheumatoid Arthritis Symptom Questionnaire scores. The importance of each predictor is depicted through adjusted partial r-squared and p value. All models are adjusted for whether patients were found through hospital registries or as volunteers.

Variables	Unadjusted Effect of Somatic Absorption, n = 167	Model Adjusted for Demographics, n = 167	Model Adjusted for Demographics and Disease Severity*, n = 147	Model Adjusted for Demographics, Disease Severity, and Psychological Distress†, n = 147
Full model	r^2 = 8%	r^2 = 10%	r^2 = 15%	r^2 = 23%
Somatic absorption	r^2 = 7% ($p < 0.001$)	Partial r^2 = 5% ($p = 0.006$)	Partial r^2 = 6% ($p = 0.005$)	Partial r^2 = 3% ($p = 0.036$)
Age	NA	Partial r^2 < 1% ($p = 0.68$)	Partial r^2 < 1% ($p = 0.63$)	Partial r^2 < 1% ($p = 0.90$)
Sex	NA	Partial r^2 < 1% ($p = 0.92$)	Partial r^2 < 1% ($p = 0.75$)	Partial r^2 < 1% ($p = 0.40$)
Race, Caucasian vs non-Caucasian	NA	Partial r^2 < 1% ($p = 0.60$)	Partial r^2 = 1% ($p = 0.17$)	Partial r^2 = 2% ($p = 0.07$)
Marital status, married vs unmarried	NA	Partial r^2 = 2% ($p = 0.12$)	Partial r^2 = 1% ($p = 0.35$)	Partial r^2 < 1% ($p = 0.92$)
Receiving disability benefits	NA	Partial r^2 < 1% ($p = 0.38$)	Partial r^2 < 1% ($p = 0.80$)	Partial r^2 < 1% ($p = 0.89$)
Swollen joints	NA	NA	Partial r^2 = 3% ($p = 0.052$)	Partial r^2 = 3% ($p < 0.041$)
ESR	NA	NA	Partial r^2 < 1% ($p = 0.94$)	Partial r^2 < 1% ($p = 0.91$)
Symptomatic drugs	NA	NA	Partial r^2 < 1% ($p = 0.48$)	Partial r^2 = 1% ($p = 0.19$)
Steroids	NA	NA	Partial r^2 < 1% ($p = 0.51$)	Partial r^2 < 1% ($p = 0.37$)
DMARD	NA	NA	Partial r^2 < 1% ($p = 0.99$)	Partial r^2 < 1% ($p = 0.86$)
Biologic response modifiers	NA	NA	Partial r^2 < 1% ($p = 0.89$)	Partial r^2 < 1% ($p = 0.86$)
Psychiatric symptoms	NA	NA	NA	Partial r^2 = 10% ($p < 0.001$)

* ESR, number of medications, joint examination. † Rand Mental Health Inventory. NA: not applicable; ESR: erythrocyte sedimentation rate; DMARD: disease modifying antirheumatic drug.

patient's symptom reports and subjective experience of disease. Our results indicate that the personality characteristic, somatic absorption, is independently and significantly associated with symptom intensity, even taking into account the severity of the disease process, medication regimen, and other confounding variables. Although the magnitude of this association is modest, symptoms are extremely complex and multi-determined phenomena with multiple biological, psychological, and sociocultural causes. It is notable that this single personality characteristic predicts as much of the variance in symptoms as it does. Our results also confirm previous studies in finding an association between psychiatric distress and somatic symptom reporting[2,5]. Indeed, in the case of the constitutional, extraarticular symptoms, psychiatric distress was a more powerful correlate of symptoms than was joint swelling, accounting for 9% of the variance. It is important to note that SA is not simply a mediator of psychiatric distress. First, although it is significantly associated with psychiatric symptoms ($r = 0.19$, $p = 0.012$), this correlation is relatively modest. Second, SA remains a significant predictor of RA symptoms, while taking psychiatric distress into account in the multivariable regression models.

The relationship between SA and RA symptoms is most powerful for the constitutional and GI symptoms of the disease: fatigue, aching, "feeling sick all over," insomnia, weight loss, and poor appetite. In contrast, it is not a significant predictor of articular symptoms such as pain, stiffness, or swelling. This discrepancy between generalized and GI symptoms on the one hand and localized symptoms on the other is interesting; the nonarticular symptoms of RA appear to be more closely linked to psychological factors than their localized, articular counterparts.

Our study has several limitations. First, assessment of arthritic symptoms was accomplished through a face-valid questionnaire designed to approximate the questions posed to patients in clinical practice. The psychometric properties of the items were not established in advance. Second, the cross-sectional design prevented clear separation of cause and effect between SA and RA symptoms. Third, the generalizability of our findings is uncertain. Our study population

Table 4. Multivariable predictors of gastrointestinal symptom subscale of Rheumatoid Arthritis Symptom Questionnaire scores. The importance of each predictor is depicted through adjusted partial r-squared and p value. All models are adjusted for whether patients were found through hospital registries or as volunteers.

Variables	Unadjusted Effect of Somatic Absorption, n = 167	Model Adjusted for Demographics, n = 167	Model Adjusted for Demographics and Disease Severity*, n = 147	Model Adjusted for Demographics, Disease Severity, and Psychological Distress†, n = 147
Full model	r^2 = 4%	r^2 = 4%	r^2 = 17%	r^2 = 20%
Somatic absorption	r^2 = 4% (p = 0.012)	Partial r^2 = 3% (p = 0.023)	Partial r^2 = 5% (p = 0.008)	Partial r^2 = 4% (p = 0.025)
Age	NA	Partial r^2 < 1% (p = 0.77)	Partial r^2 < 1% (p = 0.82)	Partial r^2 < 1% (p = 0.68)
Sex	NA	Partial r^2 < 1% (p = 0.63)	Partial r^2 < 1% (p = 0.87)	Partial r^2 < 1% (p = 0.94)
Race, Caucasian vs non-Caucasian	NA	Partial r^2 < 1% (p = 0.95)	Partial r^2 < 1% (p = 0.77)	Partial r^2 < 1% (p = 0.63)
Marital status, married vs unmarried	NA	Partial r^2 < 1% (p = 0.33)	Partial r^2 < 1% (p = 0.85)	Partial r^2 < 1% (p = 0.81)
Receiving disability benefits	NA	Partial r^2 < 1% (p = 0.96)	Partial r^2 < 1% (p = 0.83)	Partial r^2 < 1% (p = 0.78)
Swollen joints	NA	NA	Partial r^2 = 7% (p = 0.002)	Partial r^2 = 7% (p < 0.002)
ESR	NA	NA	Partial r^2 = 3% (p = 0.041)	Partial r^2 = 3% (p = 0.049)
Symptomatic drugs	NA	NA	Partial r^2 = 2% (p = 0.15)	Partial r^2 = 2% (p = 0.08)
Steroids	NA	NA	Partial r^2 = 1% (p = 0.24)	Partial r^2 < 1% (p = 0.28)
DMARD	NA	NA	Partial r^2 = 1% (p = 0.18)	Partial r^2 = 2% (p = 0.15)
Biologic response modifiers	NA	NA	Partial r^2 < 1% (p = 0.46)	Partial r^2 < 1% (p = 0.47)
Psychiatric symptoms	NA	NA	NA	Partial r^2 = 3% (p < 0.063)

* ESR, number of medications, joint examination. † Rand Mental Health Inventory. NA: not applicable; ESR: erythrocyte sedimentation rate; DMARD: disease modifying antirheumatic drug.

was notable for its high level of education, which is typical of the patients seen at our center, but requires confirmation in other populations. Further, because the subjects volunteered for a behavioral intervention study, they may have been more predisposed than the general population to the psychosocial aspects of their disease. Fourth, our measures of disease severity did not include radiographs to document structural damage. However, ESR has been used by itself as an objective measure of RA activity and clinical examination of hands and feet has been shown to correlate significantly with radiographic assessment (r = 0.79 and r = 0.66, respectively)[46,47]. Fifth, although swollen joints were significantly associated with symptoms, the ESR was not uniformly so. This may have been due to technical difficulties connected with laboratory test performance. Finally, it should be noted that the number of patients varies somewhat across analyses because of missing data.

These findings require replication, but suggest that there may be a role for psychological intervention in the management of extraarticular symptoms, and that it is possible clinicians should titrate antirheumatic medications more against articular symptoms (pain, stiffness, and swelling) than against the constitutional, extraarticular symptoms (fatigue, aching, malaise, and insomnia) that may be more influenced by nondisease factors such as SA and psychiatric distress. Further inquiry into the possible differential treatment effects of antirheumatic medications and psychological interventions for RA is in order.

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Table 5. Multivariable predictors of localized, articular symptom subscale of Rheumatoid Arthritis Symptom Questionnaire scores. The importance of each predictor is depicted through adjusted partial r-squared and p value. All models are adjusted for whether patients were found through hospital registries or as volunteers.

Variables	Unadjusted Effect of Somatic Absorption, n = 167	Model Adjusted for Demographics, n = 167	Model Adjusted for Demographics and Disease Severity*, n = 147	Model Adjusted for Demographics, Disease Severity, and Psychological Distress†, n = 147
Full model	r^2 = 2%	r^2 = 5%	r^2 = 21%	r^2 = 24%
Somatic absorption	r^2 = 2% (p = 0.09)	Partial r^2 = 1% (p = 0.13)	Partial r^2 = 3% (p = 0.065)	Partial r^2 = 1% (p = 0.18)
Age	NA	Partial r^2 < 1% (p = 0.76)	Partial r^2 < 1% (p = 0.41)	Partial r^2 < 1% (p = 0.28)
Sex	NA	Partial r^2 < 1% (p = 0.86)	Partial r^2 < 1% (p = 0.70)	Partial r^2 < 1% (p = 0.94)
Race, Caucasian vs non-Caucasian	NA	Partial r^2 < 1% (p = 0.58)	Partial r^2 = 1% (p = 0.24)	Partial r^2 = 2% (p = 0.15)
Marital status, married vs unmarried	NA	Partial r^2 = 3% (p = 0.029)	Partial r^2 = 2% (p = 0.11)	Partial r^2 < 1% (p = 0.30)
Receiving disability benefits	NA	Partial r^2 < 1% (p = 0.32)	Partial r^2 = 2% (p = 0.12)	Partial r^2 = 2% (p = 0.10)
Swollen joints	NA	NA	Partial r^2 = 10% (p < 0.001)	Partial r^2 = 10% (p < 0.001)
ESR	NA	NA	Partial r^2 < 1% (p = 0.31)	Partial r^2 < 1% (p = 0.37)
Symptomatic drugs	NA	NA	Partial r^2 < 1% (p = 0.36)	Partial r^2 = 1% (p = 0.20)
Steroids	NA	NA	Partial r^2 = 1% (p = 0.20)	Partial r^2 = 2% (p = 0.15)
DMARD	NA	NA	Partial r^2 < 1% (p = 0.38)	Partial r^2 < 1% (p = 0.44)
Biologic response modifiers	NA	NA	Partial r^2 < 1% (p = 0.48)	Partial r^2 < 1% (p = 0.48)
Psychiatric symptoms	NA	NA	NA	Partial r^2 = 4% (p < 0.015)

* ESR, number of medications, joint examination. † Rand Mental Health Inventory. NA: not applicable; ESR: erythrocyte sedimentation rate; DMARD: disease modifying antirheumatic drug.

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