INSTRUCTIONS FOR LETTERS TO THE EDITOR

Editorial comment in the form of a Letter to the Editor is invited; however, it should not exceed 800 words, with a maximum of 10 references and no more than 2 figures (submitted as camera ready hard copy per Journal Guidelines) or tables and no subdivision for an Abstract, Methods, or Results. Letters should have no more than 4 authors. Full name(s) and address of the author(s) should accompany the letter as well as the telephone number, fax number, or E-mail address.

Contact. **The Managing Editor, The Journal of Rheumatology, 365 Bloor Street East, Suite 901, Toronto, ON CANADA M4W 3L4. Tel: 416-967-5155; Fax: 416-967-7556; E-mail:jrheum@jrheum.com Financial associations or other possible conflicts of interest should always be disclosed.**

Effect of Ethnicity on Disease Activity in Systemic Lupus Erythematosus

To the Editor:

We read the recent article by Ghaussy and colleagues, comparing disease activity in patients with systemic lupus erythematosus (SLE) from 2 different ethnic groups, Hispanics and Caucasians from New Mexico[1]. Our observations about it are the result of our combined experience with Hispanic patients with SLE from Texas (mainly of Mexican ancestry), from Puerto Rico, and from Spain, as well as from our interpretation of the literature[2].

A major concern in our view is that no information is provided about the ethnic background of the Hispanic patient subset studied. As we know, the Hispanic population in the US is heterogeneous (although language and culture are shared)[3]. There are, however, fundamental differences among various Hispanic subgroups; this variability is best expressed as differences in the estimated admixture proportions [Spaniard (European), Amerindian, and African] among the Hispanic subgroups[4].

Second, important differences in the clinical characteristics of SLE and its outcome among various Hispanic subgroups have been reported by our group[5] and by investigators from GLADEL, a Latin American consortium for the study of lupus, which is prospectively recruiting and following SLE patients from different South and Central American countries[2]. Moreover, we have prospectively compared a group of Spaniard patients with SLE and our Hispanic (Mexican ancestry) LUMINA patients, and found them to have a milder disease with lower levels of disease activity over time, suggesting that the larger the Amerindian genetic pool, the greater the severity of the disease among Hispanic patients[6]. These data taken together suggest that it is critical to properly define the Hispanic subgroup studied before reaching conclusions that may not be valid beyond the patients in which the data were generated. Indeed, we suspect that the negative findings from the study may relate to the higher Spanish (European) background in the Hispanic patients those authors have studied than in our Hispanic LUMINA patients.

A third issue of concern relates to the relatively long disease duration of the patients studied; although disease duration was comparable in their Hispanic and Caucasian patients (7.60 vs 9.25 years) and it was adjusted for in the analyses, it is known that disease activity in lupus generally wears off over time, the first few years showing more active disease, when the large majority of severe clinical manifestations occur. Thus, studying patients later in their course may have precluded these investigators detecting differences in disease activity that may have been present earlier in the disease course; further, it is well known that disease activity may portend a poor prognosis, as we and others have shown[7,8], so these investigators could have assessed damage as indirect evidence that disease activity was in fact comparable earlier, but this was not done.

Fourth, although the authors refer to having studied a representative sample of their patients, the sampling frame is not provided. Moreover, by design they have excluded patients with disease severe enough to cause death shortly after onset, and numbers of deaths may have been unbalanced in the 2 groups; however, that cannot be concluded from the data presented.

Finally, while we agree that smoking is a toxic habit that may affect SLE, it cannot be categorized as abnormal illness behavior. The construct of abnormal illness-related behaviors as measured by the Illness Behavior Questionnaire[9] refers to how patients cope with an illness and not to unhealthy behaviors such as smoking or drinking; indeed, our group has recently reported that abnormal illness-related behaviors may negatively influence disease activity in SLE[10].

In short, it seems to us that the authors have reached conclusions that may not be applicable to the large number of Hispanic US patients with SLE.

JAIME CALVO-ALÉN, MD, The University of Alabama at Birmingham Birmingham, Alabama; LUIS M. VILÁ, MD, The University of Puerto Rico Medical Sciences Campus, San Juan, Puerto Rico; JOHN D. REVEILLE, MD, The University of Texas-Houston Health Sciences Center, Houston, Texas; GRACIELA S. ALARCÓN, MD, MPH, The University of Alabama at Birmingham, Birmingham, Alabama, USA.

REFERENCES

1. Ghaussy NO, Sibbitt WL Jr, Bankhurst AD, Qualls CR. The effect of race on disease activity in systemic lupus erythematosus. J Rheumatol 2004;31:915-9.
2. Pons-Estel BA, Catoggio LJ, Cardiel MH, et al. The GLADEL multinational Latin American prospective inception cohort of 1,214 patients with systemic lupus erythematosus: ethnic and disease heterogeneity among "Hispanics". Medicine 2004;83:1-17.
3. Borak J, Fiellin M, Chemerynski S. Who is Hispanic? Implications for epidemiologic research in the United States. Epidemiology 2004;15:240-4.
4. Reveille JD, Beasley TM, Tan FK, et al. Admixture as a measure of genomic control. Data from a multiethnic lupus cohort [abstract]. Arthritis Rheum 2003;48 Suppl:S225.
5. Vilá LM, Alarcón GS, McGwin G Jr, et al. Early clinical manifestations, disease activity and damage of systemic lupus erythematosus among two distinct US Hispanic subpopulations. Rheumatology Oxford 2004;43:358-63.
6. Calvo-Alén J, Reveille JD, Rodríguez-Valverde V, et al. Clinical, immunogenetic and outcome features of Hispanic systemic lupus erythematosus patients of different ethnic ancestry. Lupus 2003;12:377-85.
7. Alarcón GS, McGwin G Jr, Bartolucci AA, et al. Systemic lupus erythematosus in three ethnic groups. IX. Differences in damage accrual. Arthritis Rheum 2001;44:2797-806.
8. Stoll T, Stucki G, Malik J, Pyke S, Isenberg DA. Association of the Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index with measures of disease activity and health status in patients with systemic lupus erythematosus. J Rheumatol 1997;24:309-13.
9. Pilowsky L, Spence ND. Illness behavior questionnaire. In: Corcoran K, Fischer J, editors. Measures for clinical practice: a source book. New York: The Free Press; 1996:182-5.
10. Calvo-Alén J, Uribe A, McGwin G Jr, Reveille JD, Alarcón GS. Persistently active disease early in the course of systemic lupus erythematosus is associated with sociodemographic factors and portends a poor prognosis (damage accrual). Proceedings of the VII

International Congress of Systemic Lupus Erythematosus and Related Conditions, New York, NY. May 9-13, 2004.

Dr. Ghaussy, *et al,* reply

To the Editor:

We thank Dr. Calvo-Alén and colleagues for their interest in our study. We defined ethnicity as 4 grandparents of the same ethnic background, as defined in other studies[1]. After our study had been completed, several reports were published suggesting that there are important differences in the clinical characteristics of systemic lupus erythematosus (SLE) among various Hispanic subgroups[2-4]. As we stated in our discussion, this was one possible explanation why we did not find a significant difference in overall disease activity between Hispanics and Caucasians, as the majority of our Hispanic population had a Spanish background rather than a strictly Mexican ancestry. We believe our study gives additional support for the findings of Dr. Calvo-Alén, *et al*, that there are fundamental differences among various Hispanic subgroups; and we agree that future studies regarding Hispanics should provide information about the ethnic background of the Hispanic patient subset. The following 2 paragraphs are part of a discussion addressed to a reviewer in August 2003 regarding the complex history of New Mexican Hispanics.

As to the Hispanic ethnic makeup of New Mexico, it is somewhat complex. The earliest Spaniards in New Mexico came as soldiers without women; thus, the children of these soldiers were of mixed Amerindian and Spanish blood. However, the next generation saw a large influx of Spanish settlers including women; thus, there are many communities where there is largely Spanish (Mediterranean-European) blood with very little Amerindian blood. These are the people who call themselves "Spanish" in New Mexico. However, this is complicated by the fact that the Spanish settlers were accompanied by "Spanish" soldiers and laborers, many of whom were actually Indians recruited from the villages around Mexico City; thus, certain of the communities in New Mexico had considerable Indian blood. Finally, in the last 150 years there has been considerable Mexican immigration to New Mexico, particularly to the southern part of New Mexico.

Cinco de Mayo, Mexican Independence Day, is a big celebration in southern New Mexico, but is unimportant in northern New Mexico because northern New Mexicans have no historical connection with the Mexican Revolution of Juarez. Thus, the northern New Mexicans actually are more "Spanish" than the southern New Mexicans, who tend to be more Mexican culturally and genetically. There are also language differences that reflect this; northern New Mexico speaks a dialect very close to 16th and 17th century Spanish rather than the more or less contemporary Mexican Spanish spoken in the south. Thus our population in this study was predominately northern New Mexican and was roughly 75% European-Mediterranean blood and 25% Amerindian blood.

Our study design, a case controlled study, has certain limitations and potentials for bias that are well known. Although not presented in this study, we did not find any significant difference in overall cumulative organ damage as measured by the Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index among the New Mexico Hispanic and Caucasian population with SLE.

It is noted that the Illness Behavior Questionnaire does not consider smoking as an abnormal illness-related behavior. Cigarette smoking has been shown to be associated with increased disease activity in SLE, even after adjusting for ethnicity, as well as various aspects of SLE activity and outcome[5-9]. We believe a detailed smoking history should be included and adjusted for when comparing disease activity and outcomes in SLE.

We appreciate the valuable insights of Dr. Calvo-Alén and colleagues and their significant contributions to the scientific literature.

NAJEEB GHAUSSY, MD; WILMER L. SIBBITT Jr, MD; ARTHUR D. BANKHURST, MD, Departments of Internal Medicine, Rheumatology, Neurology, Mathematics and Statistics, and Epidemiology, and the Clinical and Magnetic Research Center, Albuquerque, New Mexico, USA.

REFERENCES

1. Reveille JD, Moulds JM, Ahn C, et al. Systemic lupus erythematosus in three ethnic groups. I. The effects of HLA Class II, C4, and CR1 alleles, socioeconomic factors, and ethnicity at disease onset. Arthritis Rheum 1998;41:1161-72.
2. Pons-Estel BA, Catoggio LJ, Cardiel MH, et al. The GLADEL multinational Latin American prospective inception cohort of 1,214 patients with systemic lupus erythematosus: ethnic and disease heterogeneity among "Hispanics". Medicine 2004;83:1-17.
3. Vila LM, Alarcon GS, McGwin G Jr, et al. Early clinical manifestations, disease activity and damage of systemic lupus erythematosus among two distinct US Hispanic subpopulations. Rheumatology Oxford 2004;43:358-63.
4. Calvo-Alén J, Reveille JD, Rodriguez-Valverde V, et al. Clinical, immunogenetic and outcome features of Hispanic systemic lupus erythematosus patients of different ethnic ancestry. Lupus 2003;12:377-85.
5. Ghaussy NO, Sibbitt WL Jr, Bankhurst AD, Qualls CR. Cigarette smoking and disease activity in systemic lupus erythematosus. J Rheumatol 2003;30:1215-21.
6. Petri M. Smoking is a risk factor for musculoskeletal, pulmonary, and cardiac disease in systemic lupus erythematosus [abstract]. Arthritis Rheum 1997;40 Suppl:S118.
7. Rahman P, Gladman DD, Urowitz MB. Smoking interferes with efficacy of antimalarial therapy in cutaneous lupus. J Rheumatol 1998;25:1716-9.
8. Brown K, Petri M, Goldman D. Cutaneous manifestations of systemic lupus erythematosus: associations with other manifestations of SLE and with smoking [abstract]. Arthritis Rheum 1995;38 Suppl:R27.
9. Ward MM, Studenski S. Clinical prognostic factors in lupus nephritis. Arch Intern Med 1992;152:2082-8.

Altitude and Giant Cell Arteritis

To the Editor:

Major interest has been directed to establishing etiological factors in primary systemic vasculitides. Interestingly, Mittal, *et al* recently described the potential influence of altitude in the disease development and relapses in a patient with Churg-Strauss syndrome[1].

Giant cell arteritis (GCA) is the most common primary systemic vasculitis in Europe and North America in people older than 50 years of age[2]. Since genetic influences cannot explain the great variation in the incidence of GCA in different parts of the world[2], we investigated the potential role of altitude as a geographical factor that might be implicated in the incidence of GCA in the Lugo region of Northwest Spain[3]. However, when we assessed the altitude of the site of residence in 210 patients from that region, we could find no differences in disease incidence related to this variable[3]. Also, in a reappraisal of relapse of the disease, we confirmed what had been reported[4,5]. We observed that relapse in Lugo patients occurred mainly when prednisone dose was less than 10 mg/day, and it generally presented as polymyalgia rheumatica or was associated with headache or asthenia. We could find no implication of the altitude of the site of residence in the incidence of relapse of GCA. Nevertheless, since the range of altitude in the Lugo region is not very broad, additional studies in regions with broader ranges of altitude would be required to definitively exclude the role of this factor in the development of GCA.

JAVIER LLORCA, MD, PhD; MARIA BRINGAS-BOLLADA, MD, Division of Preventive Medicine, Universidad de Cantabria, Santander; CARLOS GARCIA-PORRUA, MD, PhD; MIGUEL A. GONZALEZ-GAY, MD, PhD, Rheumatology Division, Hospital Xeral-Calde, Lugo, Spain.

REFERENCES

1. Mittal G, Hasso N, Maddison P. Primary vasculitis at high altitude.

J Rheumatol 2004;31:1450-1.
2. Gonzalez-Gay MA, Garcia-Porrua C. Epidemiology of the vasculitides. Rheum Dis Clin North Am 2001;27:729-49.
3. Llorca J, Bringas-Bollada M, Amor-Dorado JC, Garcia-Porrua C, Gonzalez-Gay MA. Lack of association between altitude and incidence of giant cell arteritis in Northwest Spain [letter]. Clin Exp Rheumatol 2004;22:270.
4. Gonzalez-Gay MA, Alonso MD, Aguero JJ, Bal M, Fernandez-Camblor B, Sanchez-Andrade A. Temporal arteritis in a northwestern area of Spain: study of 57 biopsy proven patients. J Rheumatol 1992;19:277-80.
5. Gonzalez-Gay MA, Garcia-Porrua C, Vazquez-Caruncho M, Dababneh A, Hajeer A, Ollier WE. The spectrum of polymyalgia rheumatica in northwestern Spain: incidence and analysis of variables associated with relapse in a 10 year study. J Rheumatol 1999;26:1326-32.

Drs. Mittal and Maddison reply

To the Editor:

We read with interest the letter from Llorca, et al, in which they quoted their studies that showed no apparent influence of altitude on the incidence of giant cell arteritis in Northwest Spain.

We presume that the subjects they studied were physiologically acclimatized to altitude. This may explain the lack of effect. By contrast, we felt that it was the rapid ascent to high altitude that was a major factor in exacerbating the effects of underlying vasculitis in the case that we described, as has also been reported in people with ischemic heart disease.

GAYATRI MITTAL, MD; PETER MADDISON, MD, FRCP, Department of Rheumatology, Ysbyty Gwynedd, Bangor, Wales, UK.

Reactivity of IgG Antibody with Cyclic Citrullinated Peptide Occurs Through the F(ab)′$_2$ Part of the Ig Molecule

To the Editor:

Recently, a test was developed for the detection of antibodies reacting with cyclic citrullinated peptide (CCP), which is useful for the diagnosis of rheumatoid arthritis (RA)[1-4]. The anti-CCP assay has gained interest in the rheumatology community because it is reported to show a higher specificity for RA than IgM rheumatoid factor, and this can be determined early in the course of disease. Although numerous studies on anti-CCP have been published, as far as we can determine no documentation exists that the reaction of serum IgG anti-CCP occurs through engagement of the F(ab) antigen-binding portion of the immunoglobulin molecule and not some other site. Thus, we examined this question directly.

Whole IgG and F(ab)'$_2$ and Fc fragments were prepared from 3 sera positive by ELISA for anti-CCP and 2 sera from normal blood donors. Whole IgG was isolated using protein A from ammonium sulfate precipitates of serum. F(ab)'$_2$ and Fc fragments were prepared with ImmunoPur® kits from Pierce (Rockford, IL, USA) according to the manufacturer's instructions, and further purified over an anti-Fc or anti-F(ab)'$_2$ affinity column, respectively. Fc-specific horseradish peroxidase-conjugated goat anti-human IgG and F(ab)'$_2$-specific rabbit anti-human Ig (both from Jackson Immunoresearch, West Grove, PA, USA) were balanced for reactivity against each other using different dilutions of each conjugate to test a number of positive sera. Samples were tested by ELISA on a CCP-coated plate to measure specific binding, and an RNP-coated plate to measure nonspecific binding, as well as an Fc capture plate made with goat anti-human IgG, Fc-specific (Jackson). Standard procedures were used for all ELISA. All sera were diluted 1:100, while all Fc and F(ab)'$_2$ fragments were diluted 1:10 for ELISA.

Table 1. Optical density ELISA anti-CCP reactivity.

Sample	Type	Conjugate Specificity F(ab)′$_2$	Conjugate Specificity Fc-specific
Blank	None	0.03	0.03
CCP1	IgG	2.33	2.37
	F(ab)′$_2$	0.60	0.04
	Fc	0.04	0.08
CCP2	IgG	2.44	2.56
	F(ab)′$_2$	0.65	0.07
	Fc	0.06	0.05
CCP3	IgG	2.67	2.41
	F(ab)′$_2$	0.86	0.04
	Fc	0.05	0.04
Normal 1	IgG	0.04	0.05
	F(ab)′$_2$	0.03	0.03
	Fc	0.03	0.03
Normal 2	IgG	0.04	0.06
	F(ab)′$_2$	0.03	0.03
	Fc	0.03	0.03

The F(ab)'$_2$ fragments from the 3 anti-CCP positive sera were positive for CCP reactivity on ELISA when detected by the F(ab)-specific conjugate, but not with the Fc-specific conjugate (Table 1). The whole IgG from these samples were positive with both types of conjugates. These results showed that F(ab)'$_2$ fragments — and not contaminating whole IgG — were responsible for the observed reactivity of the F(ab)'$_2$ preparations. The whole IgG, Fc, and F(ab)'$_2$ fragments from all samples were negative on the ELISA plates coated with RNP, indicating that reactivity observed with CCP was not due to nonspecific binding.

To ensure that Fc fragments were present, we did 2 tests. Polyacrylamide gel electrophoresis of the Fc fragments yielded a light broad band on the stained gel, showing the presence of protein in a wide molecular weight range. For a more specific test, we found a good reagent pair to develop an Fc capture ELISA. With the reagent blank, the optical density of the Fc capture plate was 0.009, while with the Fc fragments the optical densities ranged from 0.92 to 2.19. Thus, our Fc preparations did contain immunoreactive fragments.

These observations confirm that the F(ab) portion of IgG is responsible for the positive anti-CCP ELISA reactivities of these sera. This result helps solidify ongoing interpretations of basic research into the cause of anti-CCP in RA patients as well as the clinical relevance to rheumatologic applications of this assay.

RUFUS W. BURLINGAME, PhD, Inova Diagnostics, Inc., 9900 Old Grove Road, San Diego, California 92131-1638; RALPH C. WILLIAMS Jr, MD, Division of Rheumatology, Department of Medicine, ACC5, University of New Mexico Medical Center, 2211 Lomas Boulevard, Albuquerque, New Mexico 87131, USA.

REFERENCES

1. van Gaalen FA, Linn-Rasker SP, van Venrooij WJ, et al. Autoantibodies to cyclic citrullinated peptides predict progression to rheumatoid arthritis in patients with undifferentiated arthritis: a

prospective cohort study. Arthritis Rheum 2004;50:709-15.
2. Lee DM, Schur PH. Clinical utility of the anti-CCP assay in patients with rheumatic diseases. Ann Rheum Dis 2003;62:870-4.
3. Jansen LM, van Schaardenburg D, van der Horst-Bruinsma I, et al. The predictive value of anti-cyclic citrullinated peptide antibodies in early arthritis. J Rheumatol 2003;30:1691-5.
4. Schellekens GA, Visser H, de Jong BA, et al. The diagnostic properties of rheumatoid arthritis antibodies recognizing a cyclic citrullinated peptide. Arthritis Rheum 2000;43:155-63.

Association Between Systemic Lupus Erythematosus and *Helicobacter Pylori*

To the Editor:

I read with interest the recent report of *Helicobacter pylori* (*H. pylori*) seroprevalence in patients with systemic lupus erythematosus (SLE) by Sawalha and colleagues[1]. Using a large number of samples taken from SLE patients and controls, they suggested the possible role of *H. pylori* infection in delaying onset of SLE in women in some ethnic groups. They indicated possible roles of a polymorphism in the human interferon-γ receptor gene and the presence of *H. pylori*-specific CD4+ $CD25^{high}$ regulatory T cells.

Both phenomena are important in assessing influence of *H. pylori* on the immune system of infected patients; however, I would like to add at least 4 other mechanisms that involve the immune system of the infected person:

1. *H. pylori* upregulates secretion of cytokines such as tumor necrosis factor-α (TNF-α); Zentilin, *et al* reported beneficial effect of *H. pylori* eradication on disease activity in rheumatoid arthritis (RA)[2]. In addition, Gomez and colleagues reported a protective effect of TNF-α on induction of SLE[3].
2. It is now widely accepted that in *H. pylori* infected patients there is a shift to a Thl cytokine response, although this finding remains controversial. SLE cannot easily be classified into definite types of Th response, and disease activity in SLE may alter the shift[4,5].
3. *H. pylori* can produce human stress proteins such as heat shock protein 60 (hsp60) and mycobacterial hsp65. Kalabay, *et al* showed that *H. pylori* infection is associated with high levels of antibodies to mycobacterial hsp65 but not to human hsp60 in patients with connective tissue disorders including SLE[6]. Mycobacterial hsp65 have reportedly been associated with RA[7] and can activate the complement system through the classical pathway to exacerbate disease activity of RA[8].
4. Presence of *H. pylori* colonization in the stomach results in continuous ingestion of *H. pylori* constituents to establish oral tolerance against some shared antigens including mycobacterial hsp65. Oral administration of mycobacterial hsp65 has been reported to ameliorate adjuvant-induced arthritis in rats[9]. A successful eradication of *H. pylori* can result in annihilation of such oral tolerance to exacerbate disease activity of a *H. pylori* infected patient with RA[10].

Further studies are required to evaluate roles of *H. pylori* infection in establishing, exacerbating, and ameliorating rheumatic diseases, using *H. pylori* eradication in a prospective fashion.

YOSHIHIRO MATSUKAWA, MD, Department of Medicine, Division of Hematology and Rheumatolgy, Nihola University School of Medicine, Oyaguchi-Kamimachi, Itabashi, Tokyo,173-8610, Japan. E-mail:m-2000@mbk.ocn.ne.jp

REFERENCES

1. Sawalha AH, Schmid WR, Binder SR, Bacino DK, Harley JB. Association between systemic lupus erythematosus and Helicobacter pylori seronegativity. J Rheumatol 2004;31:1546-50.
2. Zentilin P, Seriolo B, Dulbecco P, et al. Eradication of Helicobacter pylori may reduce disease activity in rheumatoid arthritis. Aliment Pharmacol Ther 2002;16:1291-9.
3. Gomez D, Correa PA, Gomez LM, Cadena J, Molina JF, Anaya JM. Thl/Th2 cytokines in patients with systemic lupus erythematosus: is tumor necrosis factor alpha protective? Semin Arthritis Rheum 2003;33:404-13.
4. Amit M, Mor A, Weissgarten J, et al. Inactive systemic lupus erythematosus is associated with a normal stimulated Th(1)/Th(2) cytokine secretory pattern. Cytokine 2000;12:405-8.
5. Chen S, Hu D, Shi X, Shen N, Gu Y, Bao C. The relationship between Thl/Tb2-type cells and disease activity in patients with systemic lupus erythematosus. Chin Med J 2000;113:877-80.
6. Kalabay L, Fekete B, Czirjak L, et al. Helicobacter pylori infection in connective tissue disorders is associated with high levels of antibodies to Mycobacterial hsp65 but not to human hsp60. Helicobacter 2002;7:250-6.
7. Lai NS, Lan JL, Yu CL, et al. A dominant human leucocyte antigen DR4-Dwl 5 restricted mycobacterial 65kDa antigen-specific T-cell immunity in Chinese patients with rheumatoid arthritis. Br J Rheumatol 1995;34:498-506.
8. Prohaszka Z, Duba J, Lakos G, et al. Antibodies against human heat-shock protein (hsp) 60 and mycobacterial hsp65 differ in their antigen specificity and complement-activating ability. Int Immunol 1999;11:1363-70.
9. Cobelens PM, Heijnen CJ, Nieuwenhuis EE, et al. Treatment of adjuvant-induced arthritis by oral administration of mycobacterial Hsp65 during disease. Arthritis Rheum 2000;43:2694-702.
10. Matsukawa Y, Asai Y, Kitamura N, Sawada S, Kurosaka H. Exacerbation of rheumatoid arthritis following Helicobacter pylori eradication: disruption of established oral tolerance against heat shock protein? Med Hypotheses 2005;64:41-3.

Book reviews

Practical Rheumatology, 3rd Edition.

Marc C. Hochberg, Alan J. Silman, Josef S. Smolen, Michael E. Weinblatt, and Michael H. Weisman, editors. Edinburgh, London, New York, Oxford, St. Louis, Sydney, Toronto: Mosby of Philadelphia, 2004, 643 pages, Price: $74.95.

This well illustrated text is light-weight and filled with practical information for the generalist, as well as the rheumatologist. With regard to disease management, it is generally up to date. It comprises 13 sections, with 46 chapters covering rheumatic disease. As well as providing an overall approach to arthritis, it details the most common problems in rheumatology. In the first section, the 2 chapters are succinct: the first reviews chemokines and cytokines and their roles in the rheumatic diseases. The second on inflammation is nicely detailed. Throughout the text one finds tables and illustrations, as well as a very concise introduction.

Section II and the sections thereafter are much easier reading. The chapter on steroid injections even mentions adrenal suppression, adrenal autoantibodies and acute adrenal crises as the result of steroid intraarticular injections. In the chapter on nonsteroidal antiinflammatory drugs, it is mentioned that Indocid is more active as a COX-1 inhibitor than even aspirin. The section on COX-2 inhibitors also briefly discusses the rarely mentioned antilipocortin antibodies.

In section IV, the clinical spectrum of injury under the area of sports medicine is detailed. In subsequent chapters the most common varieties of rheumatic disease are mentioned, but I could not find mention of relapsing polychondritis. The chapter on vasculitis is too brief. Only polymyalgia and temporal arteritis are dealt with in detail. Similarly, the chapters on osteoarthritis are too brief, although this is the most common articular disorder. There is an excellent section on osteoporosis, but no other metabolic bone diseases are mentioned.

The text concludes with an appendix of patient self-help pages that

would be very helpful for both beginning and more experienced rheumatologists; included is information on where to find more information on arthritis, in the UK, the USA and Canada. This text would be recommended for the beginning rheumatologist, as well as the medical resident, as it is light-weight and it is well illustrated and filled with practical details.

Janet J. Markland, BSC, HONS., M.D, FRCPC, Assistant Professor of Medicine, University of Saskatchewan, Saskatoon, Saskatchewan, S7K 0H6, Canada.

Systemic Lupus Erythematosus, 4th Edition,

Robert G. Lahita, Editor. St. Louis, MO: Elsevier Academic Press, 2004, 1343 pages, price $199.95 (US).

Spanning over 1300 pages, the 4th edition of *Systemic Lupus Erythematosus* is a comprehensive compendium of a fascinating and complex disease. Edited by Robert Lahita with a foreword by Murray Urowitz, the list of contributing authors is a "who's who" of the lupus field. Significant revisions have been made to many chapters to include the most up-to-date research. Contributions from new authors cover psychiatric, nervous system, and dermatologic manifestations. New chapters detail fibromyalgia and osteoporosis in SLE.

Overall the contents are broadly appealing and logically ordered, progressing from basic immunology to clinical presentations and treatment. The discussions are detailed, in-depth, and inspiring. References are cited throughout the text and the bibliography at the end of each chapter is exhaustive, often containing several hundred references. The illustrations are informative and add interest and variety. Both as a reference book and as a textbook, this publication has something to offer everyone. *Systemic Lupus Erythematosus* is a worthwhile investment for student, clinician, and scientist alike.

Mandana Nikpour, MBBS, FRACP, Clinical Research Fellow, University of Toronto Lupus Clinic, Toronto Western Hospital. Toronto, ON, Canada.

Correction

Pincus T, Wang X, Chung C, Sokka T, Koch GG. Patient preference in a crossover clinical trial of patients with osteoarthritis of the knee or hip: face validity of self-report questionnaire ratings. J Rheumatol 2005;32:533-9. Professional affiliation for Dr. T. Sokka should include both Jyväskylä Central Hospital, Jyväskylä, Finland, and Vanderbilt University Medical School, Nashville, Tennessee, USA. We regret the error.

Correction

J Rheumatol 2005; volume 32, number 3, March issue (US edition only). The Table of Contents, as continued on page iv, was incorrect, and should have appeared as shown below. We regret the error.

What WOMAC Pain Score Should Make a Patient Eligible for a Trial in Knee OA?
J. Goggins, K. Baker, D. Felson 540

Self-Management in OA of Hip or Knee: A Randomized Clinical Trial in a Primary Healthcare Setting
P.H.T.G. Heuts, R. de Bie, M. Drietelaar, et al 543

Fracture Prevalence and Treatment with Bone-Sparing Agents: Are There Urban-Rural Differences? A Population Based Study in Ontario, Canada
S.M. Cadarette, S.B. Jaglal, G.A. Hawker 550

Pediatric Rheumatology

Evaluation of Revised ILAR Classification Criteria for JIA in Spanish Children (Edmonton 2001)
R. Merino, J. De Inocencio, J. García-Consuegra 559

Case Reports

Two Cases of ANA Negative Lupus Showing Increased Proportion of B Cells Lacking RP105
S. Koarada, M. Ide, Y. Haruta, et al 562

Multicentric Reticulohistiocytosis Responding to TNF-α Inhibition in a Renal Transplant Patient
S.E. Shannon, H.R. Schumacher, S. Self, A.N. Brown 565

Correspondence

Trial of Tramadol/Acetaminophen Tablets for OA Pain in Subjects Receiving a COX-2 NSAID
M.H. Ellman, J. Curran 568

Reply *R. Emkey* 568

Book Review

Low Back and Neck Pain: Comprehensive Diagnosis and Management 569

Correction

Chronic Back Pain: Searching for Causes and Cures
J.H. Atkinson 569

Meetings in Rheumatology xiv