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 3 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, 920 Yonge Street, Suite 115, Toronto, Ontario M6J 3G7, CANADA. 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.

Simple Analgesics versus NSAID

To the Editor:

Dr. Moskowitz criticizes[1] our recent paper that failed to demonstrate a relationship between the severity of joint pain and magnitude of improvement in patients with knee osteoarthritis (OA) treated with acetaminophen or with the nonsteroidal antiinflammatory drug (NSAID) ibuprofen[2]. He concludes that our subjects with a high level of baseline pain did, in fact, respond better to the NSAID and that only "...after correction using additional statistical tests" was this difference between treatments obliterated. He considers the adjustment for multiple comparisons we made in subgroup analyses as use of "new statistical testing methods."

Adjustment for multiple comparisons is neither "new" nor arcane. Because failure to adjust for multiple comparisons frequently leads to a conclusion that treatment differences exist when they do not (Type I error), it has been unacceptable in the medical literature for at least 2 decades. To put our results into perspective, without adjustment, 9 tests between pairs of means (3 pain instruments × 3 levels of baseline pain severity) comparing the antiinflammatory dose of NSAID with acetaminophen yielded only a single nominally significant p value ($p = 0.047$). Further, this solitary hint of superiority of the NSAID was evident only for rest pain and was not seen for pain with walking or overall pain. We mentioned the nominal p value only in the interest of completeness. Taken in its entirety, our study did not indicate superiority of an antiinflammatory dose of ibuprofen in comparison with either a low, analgesic, dose of ibuprofen or with acetaminophen.

Dr. Moskowitz contends that recent studies describe "...superior efficacy of NSAID... compared to acetaminophen." In support of that statement he cites 3 abstracts. Those citations require examination.

The data in the abstract by Wolfe, *et al* are presented in greater detail in a peer reviewed manuscript[3]. Patients with OA who had taken both acetaminophen and NSAID responded to a survey asking them to compare the effectiveness of, and their level of satisfaction with, the two therapies. About 45% indicated that acetaminophen was about the same as, or better than, their NSAID. Thus, although a majority of respondents preferred NSAID, the margin of difference was rather minimal. Further, preferences for acetaminophen increased with age.

The second reference[4] describes a 6 day clinical trial, the results of which have not been reported in a full peer reviewed manuscript, but which indicated superiority of an analgesic dose of ibuprofen, relative to acetaminophen, among subjects with more severe knee pain. However, in a study of only 6 days' duration results may be influenced heavily by differences from patient to patient in pharmacokinetics and pharmacodynamics of the agents being tested and may tell us little about the comparative efficacy of the drugs in treatment of chronic OA pain.

The third study had been reported only as an abstract when the editorial was published, but has since been published as a full-length article[5]. Improvements in joint pain, function, and quality of life were significantly greater after treatment with diclofenac/misoprostol than with acetaminophen. Further, subjects who had more severe joint pain at baseline showed greater improvement with the NSAID than with acetaminophen. However, an important finding is presented in the full article that was omitted from the abstract — nearly 50% of subjects and investigators reported that acetaminophen was about as good as, or better than, the NSAID.

Dr. Moskowitz concludes that the safety profile of coxibs permits their consideration as initial therapy for OA. However, there are important considerations: In the CLASS study, among subjects who were concomitantly taking low dose aspirin, no statistically significant difference in the incidence of gastrointestinal ulcers or ulcer complications was apparent between those taking celecoxib and those taking diclofenac or ibuprofen[6]. While only 23% of subjects in that study were taking low dose aspirin, in practice as many as 60% of subjects 65 years or older may do so (Brandt KD, unpublished observations). On the other hand, in the VIGOR study, which excluded subjects taking low dose aspirin, treatment with rofecoxib significantly reduced the incidence of clinically important gastropathy, relative to naproxen. However, the incidence of myocardial infarction was 4-fold greater in the rofecoxib group than among subjects treated with the comparator[7]. Thrombotic events have been reported also in patients with predisposing risk factors (e.g., antiphospholipid antibodies, Raynaud's phenomenon) who were treated with celecoxib[8].

Dr. Moskowitz's statement, "Caveats remain in the use of any of these agents, whether it be liver toxicity with acetaminophen, or renal toxicity/hypertension concerns with traditional NSAID or the COX-2 selective agents," does not provide a fair comparison of the relative risks. Although massive overdoses of acetaminophen may cause liver failure, acetaminophen hepatotoxicity is exceedingly uncommon with therapeutic doses. Further, administration of acetaminophen to subjects with chronic liver disease did not result in an increase in liver damage[9]. On the other hand, development of renal toxicity/hypertension with a therapeutic dose of traditional NSAID or COX-2 selective agent is not uncommon and limits the use of these agents in patients with OA[10].

What is important is not whether Dr. Moskowitz and we agree, but that clinicians have all the evidence, that expert opinion is differentiated from data, and that the quality of the data is assessed. Dr. Moskowitz frequently treats OA patients with acetaminophen[1]; we treat many with NSAID. We agree that some patients experience insufficient benefit from acetaminophen. However, data do not exist to support the contention that NSAID are more efficacious than acetaminophen as initial therapy in OA patients, even in those with clinical signs of inflammation or more severe joint pain[11].

We recognize the limitations of *post hoc* analyses and the need for prospective studies to determine whether the severity of joint pain can guide treatment recommendations for OA. It would be reasonable now for the manufacturers of acetaminophen and NSAID or federal funding agencies to sponsor clinical trials to determine: "Which OA patient will do better with an NSAID than with acetaminophen?" Until results of such studies are available, the clinician must recognize that the evidence is not adequate to provide a definitive answer and treatment decisions must be based on considerations of safety, efficacy, and cost.

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To continue

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Dr. Moskowitz replies

To the Editor:

I am pleased to have the opportunity to respond. I might note, however, that this invited response to his letter, in response to my invited editorial[1], in response to his article in *The Journal*[2] represents a point:counterpoint that I hope will soon end! My responses are as follows.

Dr. Brandt's rebuttal regarding my statement that he and his colleagues were using new statistical testing methods that were neither "new" nor arcane represents a misinterpretation of my statement. I apologize if the statement was confusing. By the term "new" statistical methods, I refer to the fact that Dr. Brandt and his colleagues were now utilizing different statistical methods in their retrospective reanalysis of the data, rather than to a statistical testing method that was new per se. I can understand the misinterpretation, but stand by my statement that retrospective statistical pursuit of data utilizing post hoc analysis with multiple methodologies different from those originally utilized may lead to supportive but not necessarily valid conclusions.

Dr. Brandt's statement in the first line of the second paragraph is not clear to me. He states that "because failure to adjust from multiple comparisons frequently leads to a conclusion that treatment differences exist when they do not (Type I error), it has been unacceptable in the medical literature for at least two decades." What has been unacceptable?

In his commentary on the studies by Wolfe, *et al*[3], he states that "about 45% indicated that acetaminophen was about the same as, or better than, NSAID. Thus, although a majority of respondents preferred NSAID, the margin of difference was rather minimal." The data can be viewed in a different fashion, which would not support his conclusion that differences between the efficacy of NSAID and acetaminophen are minimal. Specifically, of the patients with osteoarthritis (OA) treated with acetaminophen, only 14.3% of individuals found acetaminophen to be more effective or much more effective; contrariwise, 56% of individuals treated with acetaminophen found it to be somewhat less effective or much less effective!

Similarly, in the study by Pincus, *et al*[4], Dr. Brandt states that nearly 50% of subjects and investigators reported that acetaminophen was about as good as, or better than, the NSAID. Dr. Brandt once again combines as good as, or better than in these analyses. When one looks at the data objectively in this latter article, diclofenac plus misoprostol was rated as better or much better by 57% of the 174 patients who provided such ratings for both treatment periods in this crossover trial; acetaminophen was rated as better or much better for only 20% of these patients. It is of further interest that in this study[4] differences favoring diclofenac plus misoprostol over acetaminophen were greater in patients with more severe OA according to baseline pain scores, radiographs, or number of involved joints. With respect to whether the efficacy of NSAID exceeds that for acetaminophen, I would refer the reader to the excellent editorial by Dr. David Felson[5]. As he notes, "the safety advantage that motivated the original American College of Rheumatology Committee recommendation of acetaminophen is dissolving with the introduction of cyclooxygenase 2 inhibitors and with the recognition that some gastroprotective drugs can substantially reduce the risk of NSAID-induced and bleeding ulceration."

Relative to whether hepatic disease may be of concern in patients being administered acetaminophen, I stated that caveats remain in the use of any agent, whether it be liver toxicity with acetaminophen, or renal toxicity/hypertension concerns with traditional NSAID, or the cyclooxygenase 2 selective agents. I agree with Dr. Brandt that in most patients hepatic toxicity related to acetaminophen is not a significant concern; however, it remains a clinical consideration in individuals with hepatic disease or in those who have a high ethanol intake[6].

Dr. Brandt states that data do not exist to support the contention that NSAID are more efficacious than acetaminophen as initial therapy in patients with OA, even in those with clinical signs of inflammation or more severe joint pain. In his letter, Dr. Brandt refers to 3 studies[3,4,7] that indeed support the conclusion that NSAID are more efficacious than acetaminophen in therapy in OA patients, whether used as initial therapy or therapy at some later time. Dr. Brandt asks the question, "Which OA patients will do better with an NSAID than with acetaminophen?" As shown in the studies by Pincus, *et al*[4], patients shown to do better with NSAID are those with more severe pain and, perhaps, those with hip OA[5].

As I stated in my editorial[1], acetaminophen has a meaningful role in the management of OA, either as initial therapy or as continuing/adjunctive therapy. Based on current new knowledge, however, it is no longer appropriate to state that acetaminophen is the drug of choice in the initial therapy of all patients with OA. I respect Dr. Brandt's tenaciousness and sincerity — at this point it appears we will have to agree to disagree.

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Autoimmune Disease Among Teachers

To the Editor:

I have just read the abstract of the study entitled "Excess Autoimmune Disease Mortality Among School Teachers" by Walsh and DeChello[1] with great interest. My career as a speech pathologist in preschool settings was recently ended and I am now disabled at the age of 46 with neurological problems. I would like to raise 2 points that are worthy of further medical scrutiny.

First, school settings are indeed hotbeds of infectious agents. Most teachers spend their first few years of teaching taking many sick days, frequenting their doctors' offices with greater regularity than at any other point in their lives, and taking many courses of antibiotics for ailments such as respiratory tract/sinus infections, conjunctivitis, ringworm, scabies, bacterial gastrointestinal infections, etc. We are exposed to each child's home environment, as well as the school environment, in what is carried to us daily by these youngsters. Vaccinations, now including novel immunizations like hepatitis and yearly PPD tests, are also mandatory or recommended and carry their own inherent set of risks.

However, one cannot ignore the contribution of environmental factors that take a huge toll upon us. My neurological problems were precipitated by a 6 month long course of pyrethroid (neurotoxic) pesticide spraying in the educational setting where I worked as a supervisor and therapist. Sudden onset of symptoms and signs of neurotoxicity, along with previously demonstrated sensitivities to pesticides, permitted the connection to be made between the damage seen on a variety of tests and these toxins. Mold damage is also rife amidst school buildings, making recovery from respiratory/sinus complaints difficult and inciting asthma attacks. Poorly ventilated areas gather fumes from pesticides, cleaning materials, air fresheners, newly laid flooring materials and furnishings, fresh paint, science experiments, personal fragrances/cosmetics, printed materials, drymarkers, etc. Ingredients in all of the above have been noted to be health hazards by OSHA/NIOSH.

Overcrowded classrooms and recirculated air in rooms with sealed windows or lacking windows all have great impact upon the health of the students and teaching staff. Let's remember to examine the environmental factors at work here in combination with infectious agents. There is usually an interaction among them that can cause the poor health of so many educators and that is regularly recognized and discussed among us.

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REFERENCE

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Prof. Walsh and Ms DeChello reply

To the Editor:

Ms Rubin's letter indeed raises points that deserve additional scientific scrutiny. Following publication of our article we received much correspondence that, like this letter, suggests a wider spectrum of occupational exposures among teachers than we considered and that points to the occurrence of additional, nonautoimmune, occupation related illness in this profession. It seems that many teachers suspect that something about the teaching profession affects their health in unusual ways, but that, prior to publication of our paper, no scientific evidence was available to support their suspicions.

Our decision to focus on infectious agents as a possible explanation of excess autoimmune disease mortality among teachers was motivated by 2 factors. First, each of the individual autoimmune diseases that exhibited significant excess mortality in teachers was associated with a possible bacterial or viral trigger in earlier research studies. Second, there is documentation supporting a role for schools in facilitating transmission of each of the implicated infectious agents. The *hypothesis* that greater exposure to infections *could* lead to greater autoimmune disease incidence among teachers was not meant to exclude other plausible explanations of our findings. However, we are not aware of any other class of environmental exposures that, based on published evidence, appears to constitute both a risk factor for multiple autoimmune diseases and an exposure associated with the school environment.

We agree that concentrations of molds, pesticides, or other environmental toxins in school buildings carry the potential to affect health. The prevalence of such exposures in schools has yet to be systematically documented, and associations between those exposures and autoimmune disease incidence have yet to be reported. In this regard it is important to recognize that the findings presented in our paper relate only to autoimmune diseases. The relevance of our findings to frequent reports of allergies, asthma, or other syndromes among students and teachers is not clear.

We hope that our study broadly motivates further research regarding exposures present in the school environment and the degree to which those exposures affect morbidity of all kinds in students and teachers. More specifically, we hope the study reveals the opportunity that schools and the teaching profession provide for identifying factors that trigger autoimmune pathogenesis.

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Breast Implants and Fibromyalgia

To the Editor:

S. Lori Brown[1] responded to my letter[1] regarding the article on silicone breast implants and fibromyalgia (FM)[2], but missed the point. As the symptoms of FM cannot be verified except by the very unreliable tender points, and FM itself is a catalog of symptoms of social mimicry, a "mass somatisation model"[3], the responses to history questions and examination can be programmed — and often are programmed by lawyers and so-called support groups. Thus, though all of the patients in the series reported had implants, regardless of rupture status, knowing whether they came to attention through lawyers, advocacy groups, and physicians who cooperate with the above is all important. As it is, the statistical significance disappears on reanalysis[1], and no verifiable laboratory abnormalities, such as serological markers, identify any of these patients[4].

Since the witch scares of the middle ages[5], and probably before, somatization syndromes have been attributed to some malign cause. This is even more true here, where profit can result from the attribution. And the ill advised decision by the commissioner of the US Food and Drug Administration (FDA) to place draconian restrictions on silicone breast

implants[6], prompted at least in part by the infamous Connie Chung interview, helped only the trial lawyers. In my years as Chairman of the Arthritis Advisory Committee and later as a member of the Council of Chairs of the FDA, I never found anyone who was willing to comment who agreed with this decision.

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The Death of Mozart

To the Editor:

The media have recently made much of a pathologist's conclusion that Wolfgang Amadeus Mozart died as a result of contracting trichinosis. A letter he had written to his wife, who was vacationing in the nearby spa, Baden, several weeks before his death mentioned how much he had enjoyed eating pork cutlets. Now, Wiener schnitzel, the likely meat in question, can be made with veal or pork, and in the late 18th century, pork might well have been the preferred choice (as unbreaded *Schweineschnitzel*). But pigs in Vienna were usually grain-fed and therefore unlikely to have harbored the larvae of *Trichinella spiralis*, so this conjecture may seem newsworthy but assuredly is wrong.

On the occasion of the 200th anniversary of Mozart's death (1991), I was asked to discuss his health problems at a conference in Halifax, Nova Scotia[1]. I had previously delivered a paper on his terminal illness at the XVI International Congress of Rheumatology in Sydney, Australia, in 1985, after perusing his extant medical records and biographical essays and books, and this led to the above invitation. Delving into his medical records, I discovered that he had developed hectic fevers accompanied by severe joint pains in his seventh year, which caused him to remain in bed, unable to perform, for many months. He ultimately recovered, but in his final year of life, became increasingly edematous and he died with anasarca. The most likely diagnosis, despite the many that have been bruited about, is secondary amyloidosis, assuming the earlier disease was Still's disease, yet to be named and understood.

In continental Europe and the United Kingdom, amyloidosis is (or was) a frequent sequel of juvenile polyarthritis, and Mozart's medical records, still extant and complete, fit this pattern better than any other. Though some have claimed to have identified his skull, removed from the common grave to which his body was consigned because of the then existent regulations governing funerals during a time of contagious epidemics, the skull in question is unlikely to be his and thus offers no further evidence for a cause of death or of lifetime diseases[2].

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Book reviews

Kelley's Textbook of Rheumatology, 6th edition, CD-ROM.

Shaun Ruddy, MD, Edward D. Harris Jr, MD, Clement B. Sledge, MD, editors, Ralph C. Budd, MD, John S. Sergent, MD, associate editors. Philadelphia: W.B. Saunders (Harcourt Health Sciences), 2001, price $299.00 US

This is the CD-ROM version (Windows and MacIntosh) of the most recent (6th) edition of *Kelley's Textbook of Rheumatology,* which has been eloquently reviewed by Prof. Watson Buchanan (J Rheumatol 2001; 28: 1742–3). In addition to the entire contents of the 2 volume textbook, the CD-ROM version has 61 additional color figures and two 35-minute video loops, which include a lecture of differential diagnoses of rheumatoid arthritis (RA) and a synthesis of the pathophysiology of RA (on a single CD).

The question is whether to buy the textbook or CD-ROM version of this excellent publication. One issue is portability. Because of the size and weight of current textbooks, they can no longer be considered portable, even with the multiple volume versions. It is certainly much easier to put a single CD into your bag or pocket to take it home or to the office so long as you have access to a suitable computer.

Is the CD-ROM version user friendly? I had no difficulty finding my way around and accessing specific information. With the computer version there are inevitable delays (response time). For example, it takes my computer (Compaq deskpro) just over a minute to download the text. You can browse and select your topic of interest either using the table of contents or more specific subject index. A major advantage over the textbook version is the presence of an advanced search engine that enables you to carry out more complex searches (for example, the association between scleroderma and malignancy). Facilities are available to add notes of your own at the end of each section.

Unlike a textbook, with the CD-ROM version you cannot open a chapter on a particular topic and browse. Each chapter is divided into multiple sections that need to be opened individually through menus. The convenience is in looking up more specific information. At least you won't risk a hernia lifting the textbook off your shelf. The cost of the textbook and CD-ROM version are identical.

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Rheumatoid Arthritis—Epidemiology, Pathogenesis and Treatment

Larry W. Moreland, editor, London: Re Medica Publishing, 2001, 122 pages, price $30.00.

This monograph is intended to provide rheumatologists with "a concise and up-to-date overview of current concepts in the etiopathogenesis and treatment options for RA." There are 6 chapters with an introduction and sections dealing with the epidemiology, pathophysiology and etiology, current therapeutic options (non-biologic agents), cytokine therapies, and emerging therapies in rheumatoid arthritis (RA). The contributors are all experts in their field.

The book is certainly concise, with each chapter averaging 20 to 30 small pages. Good use is made of tables to summarize information in a manner that can be readily assimilated. Each chapter has been extensively referenced.

In a fast moving area, particularly with the recent advances in treatment of RA, such a concise but comprehensive "state of the art" review is just what the busy practicing rheumatologist (and rheumatology trainee) needs to keep abreast of the field. The editor has done an excellent job, as the publication is easy to read and the chapters are of uniform quality. One would hope that such a publication will be updated frequently to incorporate important new information and treatment issues that are rapidly accumulating. Examples are recent concerns regarding COX-2 inhibitors and risk of tuberculous reactivation with the TNF-α inhibitors.

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Correction

Falsetti, P, Frediani B, Storri L, et al. Evidence for synovitis in active polymyalgia rheumatica: Sonographic study in a large series of patients. J Rheumatol 2002;29:123-30. The names of authors of this article should have been published as follows: Bruno Frediani, Paolo Falsetti, Lara Storri, Stefania Bisogno, Fabio Baldi, Valeria Campanella, Caterina Acciai, Georgios Filippou, Francesca Chellini, Roberto Cosentino, and Roberto Marcolongo. We regret the error.