mine the biologic basis and clinical significance of the altered collagen turnover that we observed in patients with fibromyalgia.

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HLA–B27 and the spondylarthropathies in Lebanon: comment on the article by Awada et al

To the Editor:

I work at an overseas American institution, and there are extended delays in receipt of journals by our medical library. So, it was only a few days ago that I saw the article entitled "Weak Association between HLA–B27 and the Spondylarthropies in Lebanon," which was published in the February 1997 issue of *Arthritis & Rheumatism* (1).

I would like to draw the attention of the Lebanese authors of that article to 2 points. First, the tabulated results in their Table 1 are misleading with respect to their reference 6, an article published by us (2). We published 2 tables in that article. Awada et al failed to mention that the frequencies of A9, A10, B12, B15, B16, B17, B21, B22, B40, and B47 were reported in our Table 1. Moreover, they erroneously report that the data for A23 and A24 (splits of A9), A25 and A26 (splits of A10), B51 and B52 (splits of B5), B44 and B45 (splits of B12), and B49 and B50 (splits of B21) were not available in our article. Our Table 2 clearly showed the ratios for each of these splits.

Second, Awada et al failed to acknowledge the fact that it was first suggested and reported in 1985 that B27 may not be associated with ankylosing spondylitis in Lebanese patients (3).

Two important statements we made in that article, one in the abstract and the other in the discussion section, were as follows. "The frequency of B27 in the Lebanese group studied was 1%. This may suggest that the established strong association between B27 and ankylosing spondylitis in Caucasians may not be applicable to Lebanese." "It is also noteworthy that the B27 frequency in this group was similar to that in blacks. If this frequency remains stable, then the strong association between B27 and ankylosing spondylitis in Caucasians may not be applicable to Lebanese, since it has already been mentioned that such a strong association does not exist in blacks."

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1. Awada H, Baddoura R, Naman R, Klayme S, Mansour I, Tamouza R et al. Weak association between HLA–B27 and the spondylarthropathies in Lebanon. Arthritis Rheum 1997;40:388–9.
2. Abdelnoor M, Abdelnoor A. Comparative study of HLA-A, B, and C frequencies in Christians and Moslems in Lebanon. Leb Sci Bull 1993;6:67–71.
3. Abdelnoor AM, Heneine W. HLA-A, B, and C typing of a selected group of Lebanese: a preliminary report. Leb Med J 1985;35:246–50.

Reply

To the Editor:

In response to Dr. Abdelnoor's comments, we would like to raise the following points. First, in our article published in the February issue of *Arthritis & Rheumatism,* the footnote to Table 1 does specify that "some rare phenotypes found in the general population but not in our patients are not shown." Hence, the frequencies of A9, A10, B12, B15, B16, B17, B21, B22, B40, and B47, which were not found in our patients, were not taken from either Dr. Abdelnoor's work or other works referred to in our Table 1.

Second, in the Abdelnoor and Abdelnoor article published in the *Lebanese Science Bulletin* in 1993 (1), Table 2 did not show the prevalences of A23, A24, A25, A26, B51, B52, B44, B45, B49, and B50, but rather, only the ratio between these splits.

Third, the statement made by Abdelnoor and Heneine in their article published in the *Lebanese Medical Journal* in 1985 (2), suggesting that HLA–B27 may not be associated with ankylosing spondylitis (AS) was only speculative and was not based on a case–control study. The 1% prevalence of HLA–B27 found in a sample of 129 individuals from the general Lebanese population could not allow such a conclusion. Indeed, as we specify in our article, "in other populations in which the prevalence of B27 is low, such as Japan (1%) and other countries of the Middle East (3% in Israel and Iraq), the B27 prevalence remains rather high among AS patients (80% in Japan and 60% in Israel)" (see ref. 3). Moreover, we would like to point out that a low prevalence of B27 in the Lebanese population (1.4%) was reported earlier in an article published in *Tissue Antigens* by Serre et al (4).

We acknowledge that the low association of AS with HLA–B27 in the Lebanese population was suggested by Dr. Abdelnoor in his article published later, and that our work confirms and extends this hypothesis.

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1. Abdelnoor M, Abdelnoor A. Comparative study of HLA-A, B, and C frequencies in Christians and Moslems in Lebanon. Leb Sci Bull 1993;6:67–71.
2. Abdelnoor AM, Heneine W. HLA-A, B, and C typing of a selected group of Lebanese: a preliminary report. Leb Med J 1985;35:246–50.
3. Khan MA. HLA-B27 and its subtypes in world populations. Curr Opin Rheumatol 1995;7:263–9.
4. Serre JL, Lefranc G, Loiselet J, Jacquart A. HLA markers in six Lebanese religious subpopulations. Tissue Antigens 1979;14:251–5.

Does knuckle cracking lead to arthritis of the fingers?

To the Editor:

During the author's childhood, various renowned authorities (his mother, several aunts, and, later, his mother-in-

law [personal communication]) informed him that cracking his knuckles would lead to arthritis of the fingers. To test the accuracy of this hypothesis, the following study was undertaken.

For 50 years, the author cracked the knuckles of his left hand at least twice a day, leaving those on the right as a control. Thus, the knuckles on the left were cracked at least 36,500 times, while those on the right cracked rarely and spontaneously. At the end of the 50 years, the hands were compared for the presence of arthritis.

There was no arthritis in either hand, and no apparent differences between the two hands.

Knuckle cracking did not lead to arthritis after a 50-year controlled study by the one participant. While a larger group would be necessary to confirm this result, this preliminary investigation suggests a lack of correlation between knuckle cracking and the development of arthritis of the fingers. A search of the literature revealed only one previous paper on this subject, and the authors came to the same conclusion (Swezey RL, Swezey SE. The consequences of habitual knuckle cracking. West J Med 1973;122:377–9.).

This result calls into question whether other parental beliefs, e.g., the importance of eating spinach, are also flawed. Further investigation is likely warranted.

In conclusion, there is no apparent relationship between knuckle cracking and the subsequent development of arthritis of the fingers.

This study was done entirely at the author's expense, with no grants from any governmental or pharmaceutical source.

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Reply

To the Editor:

I appreciate the opportunity to review Dr. Unger's report. His "self-controlled" study adds considerable credence to our 1973 study findings.

Dr. Unger exercised amazing self control by performing 50 years of knuckle cracking (KC) on his left hand at least twice daily, "while those on the right cracked only rarely and spontaneously." No evidence of arthritis in either hand was found at the end of 50 years.

I have taken the liberty of consulting Dr. John Adams, PhD, at the Rand Corporation, who has generously provided me with the following statistical analysis.

> The basic study designed by Dr. Unger is a two-arm trial without randomization. Although it is not clear, it appears that the study was not blinded. Blinding would only be possible if the investigator didn't know left from right. This is not likely since studies indicate that only 31% of primary care physicians don't know left from right. (The figure is reportedly somewhat higher for most specialists.) The lack of randomization suggests the need for a multivariate analysis to reduce bias. Controlling for knuckle-to-knuckle variation in race, sex, socioeconomic status, initial severity, comorbidities, and Ecuadorian barometric pressure at the time of measurement would be advisable. The sample size appears too small to support accurate inference. Typically, sample sizes of roughly twice the available research budget are required for valid inference. Restrictive eligibility criteria and convenience sampling limit generalization of the results to knuckle-cracking physicians with a lot of time on their hands.

I should note that SES, the co-author of our 1973 investigation, was 12 years old at the time of the study and that the study was stimulated because of his grandmother's concern about the arthritic consequences of his KC. It is now 22 years later and he continues to enjoy frequent KC without manifestations or evidence of arthritis.

Closer scrutiny of the data in both studies raises the question of a possible osteoarthritis preventative therapeutic benefit from the exercise effect on joint lubrication resulting from habitual KC. Clearly, further study should be undertaken, with the caveats as given by Dr. Adams.

The possible utilization of KC by managed care providers as an economic, noninvasive, home preventative treatment for arthritis of the hands should be given further consideration. A clear distinction between hand wringing related to managed care procedures and therapeutic KC will have to be made.

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