

## Where in the World is Oral Triamcinolone?

To the Editor:

Recent reports regarding circadian treatment of rheumatoid arthritis (RA) with a modified form of prednisone indicate this affords significant control of arthritis without affecting the hypothalamic-pituitary axis by maximizing the glucocorticoid's effect in early morning<sup>1</sup>. They support previous reports on the beneficial effects of evening triamcinolone (9 alpha-fluoro-11 beta, 16 alpha, 17 alpha, 21-tetrahydroxy-1, 4 pregnandione-3, 20-dione)<sup>2</sup> presented in a 1991 longitudinal study of early therapy in RA.

Now that the European League Against Rheumatism intends to assess and set guidelines for this use of modified prednisone, the American College of Rheumatology (ACR) and all drug trial groups in the USA should request from the pharmaceutical industry the immediate reintroduction of oral triamcinolone, a vastly superior disease-modifying glucocorticoid.

Although the steroid-sparing concept is the worst and longest-lasting therapeutic tragedy ever suffered on this planet, it still remains publicly at the core of rheumatologic therapeutics. For decades this concept gained support from didactic sectors limited by an almost exclusive exposure to prednisone side effects and the contradiction of prednisone's use in drug trials for the therapy of RA as an irrelevant adjunct to other disease-modifying antirheumatic drugs (DMARD) without a disease-modifying role of its own.

Prednisone is a dysfunctional glucocorticoid, and experience has shown its surreptitious use has failed to affect the index of remission and major improvement in RA significantly. Oral triamcinolone's side effects are minimal compared to prednisone's; it is also more effective in combination with other DMARD or with the now lengthy, confusing list of expensive biologics, most of which are not accessible to our patients for reasons of cost. Oral adjunct triamcinolone opened a therapeutic window of remission and justified its claim as a disease-modifying drug by reducing the incidence of erosions when given alone in RA. We also know that first-line nonsteroidal antiinflammatory drug (NSAID) therapy is less efficient and more damaging in RA and particularly lupus, given its renal, gastrointestinal, and cardiovascular toxicity (this longterm damage is yet to be determined and cannot be easily blamed on the disease itself).

In our study, low-dose (4 mg) adjunct oral triamcinolone used at 7:00 PM daily in RA cut the early morning inflammatory surge by at least 75% without adrenal insufficiency, salt retention, edema, obesity, moonface, hypertension, metabolic syndrome, infections, or mood disorders. Triamcinolone produces weight loss and diuresis — both beneficial to patients with pain, inflammation, and disability, and also easy bruising in some cases. What happens is that the potency of evening oral triamcinolone provides the desired effect without significant side effects. Except for stress relapses, rarely is there a need to increase the adjunct, continual

dose more than 4 mg/day, and only temporarily (Aristocort and Kenacort are that good).

Nevertheless, by 1982, rheumatology had set longterm guidelines on the basis of short-term trials with few patients, little assessment of entry variables on longterm outcome, and the contradiction of saying one thing and doing another with prednisone use. Methotrexate became the "gold standard" in patients with RA nonresponsive to first-line NSAID therapy, (most of them erosive already) — but prednisone on the side would be acceptable.

Sometime after, a strange thing happened in America. The manufacturer of methotrexate and one of the 2 brand triamcinolones sold away the rights of triamcinolone — maybe due to lack of support in our clinical trials, or maybe because triamcinolone became too good for its own good and its sale would decimate sales of other DMARD or its more expensive forms of delivery. Eventually, even generic oral triamcinolone, the most effective of all oral glucocorticoids, was taken off the market here — quietly and without explanation. It is now unavailable in the USA for single oral use or as a DMARD in the combination therapy of early RA, a tragedy. But it is out there. Later, Kenacort, the other brand of oral triamcinolone, also disappeared.

Triamcinolone is the premier oral glucocorticoid; its removal from the market in America is nothing short of inhumane. Now that the true disease-modifying role of the glucocorticoid in rheumatic diseases is no longer in question, the ACR should exert its influence with the pharmaceutical industry and restore oral triamcinolone to its proper place as a stronger alternative to a modified form of prednisone. It remains to be seen whether an expensive modified form of prednisone with its notorious side effects can be as effective as generic triamcinolone.

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Dr. Roger has never had a financial arrangement in his clinical research with any company manufacturing any product mentioned in this article.

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