Letters to the Editor
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Retraction of an Interpretation

WE WRITE TO RETRACT AN INTERPRETATION in our Report, “Contribution of human α-defensin 1, 2, and 3 to the anti–HIV-1 activity of CD8 antiviral factor” (1), wherein we demonstrated that human α-defensin 1, 2, and 3 account for much of the anti–HIV-1 activity that is not attributable to activity of the CD8 antiviral factor (CAF). Although the antiviral activity of human α-defensins has not been called into question, this method of stimulating CD8 T cells had not been repeatable. In particular, we have solidified our experimental findings in our Report (1) are repeatable. In particular, we have solidified the results shown in figs. 3 and 4 of our paper (1). The removal or neutralization of α-defensins by specific antibodies again resulted in the loss of anti–HIV-1 activity in the supernatant of CD8 T cells stimulated by allogeneic feeders. It should be pointed out that there is, in fact, very little residual anti–HIV-1 activity remaining once α-defensins and β-chemokines are eliminated. More importantly, we have also tested human neutrophil-derived α-defensins from independent sources and found their antiviral potency to be equivalent to those previously described (1), irrespective of the viral strain or target cell used in the experiment. Other investigators have confirmed this observation (12, 13). Thus, the anti–HIV-1 activity of α-defensins we have described (1) is not in doubt, and the mechanism of their antiviral effect should be pursued.

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References

Brightening Depression

CONSTANCE HOLDEN’S OVERVIEW “FUTURE—brightening for depression treatments” (Special Issue on Brain Disease, News, 31 Oct., p. 810) explored the current exciting approaches for creating novel antidepressants. Absent from this discussion were two major nonpharmacological, biological antidepressant treatments that have been clearly demonstrated to be highly efficacious and fast.

“Given the psychological suffering that depression inflicts... it is surprising how little notice is taken of these remarkable chronobiological interventions [sleep deprivation and light therapy].”

—WIRZ-JUSTICE ET AL.

A single night of total or partial sleep deprivation—“wake therapy”—induces rapid and dramatic, albeit usually short-lasting, improvement of mood in about 60% of all depressed patients, independent of diagnostic subgroup (1). A positive response to sleep deprivation predicts and hastens the response to antidepressant medication (1). Sleep deprivation can be combined with a variety of drugs to maintain the response attained within hours (2–4)—theoretically, a perfect combination (5).

Light therapy is the only treatment in psychiatry that evolved directly out of neurobiological models of behavior (6, 7). It is the treatment of choice for seasonal affective disorder, or winter depression (6), but is
also efficacious in nonseasonal depression (8–10). Light therapy is characterized by a fast onset of antidepressant action—within days—and it can prevent the depressive relapse after recovery sleep following sleep deprivation (4, 11). Furthermore, light and medication can be combined (8–12).

Sleep deprivation and light therapy cannot be patented, and they will not bring profits to the conventional psychopharmacology industry, but they can help the patient in a shorter time and with fewer side effects than drugs—and can be easily and successfully combined with medication (3, 4, 11, 12). Given the psychological suffering that depression inflicts—including the danger of suicide—and the financial pressures to minimize the duration of hospitalization, it is surprising how little notice is taken of these remarkable chronobiological interventions. We must include them in the therapeutic armamentarium. For light therapy, an American Psychiatric Association task force recently has concluded the same (13).

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The Difficulties of Testing for SARS

We read with interest the article “Search for SARS origins stalls” (M. Enserink, D. Normile, News Focus, 31 Oct., p. 766). The experience of the Canadian National Microbiology Laboratory, stemming from a positive SARS test that was later found to be a false alarm (Sidebar, “Unexplained false alarm may hold lessons,” M. Enserink, p. 767), struck a particularly resonant chord, as it mirrored almost exactly our own experience in Hong Kong, one of the places most affected by SARS.

As the provider of the only private test service for the SARS coronavirus (CoV) in Hong Kong, we had been contracted by a local private hospital to test patient samples from cases of atypical pneumonia. The majority of tests were conducted after Hong Kong had been declared free from SARS. After testing many samples with our enhanced real-time PCR method, which we developed in-house, one sample gave a preliminary positive result with our