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11. We translated Lo's β mortality coefficients (*7*) into an average instantaneous daily mortality rate, M, for each year using her annual data on t_1 and t_2, the age of larvae (in days) at the beginning and end of the sampled larval period, and P_t, the daily production of larvae of age t

$$M = \left[\int_{t_1}^{t_2} (\beta/t)\, P_t\, dt\right] \Big/ \left(\int_{t_1}^{t_2} P_t\, dt\right)$$

12. A. Bakun (Pacific Fisheries Environmental Group, National Marine Fisheries Service, Monterey, CA) provided us with daily wind speed data for 1954 through 1984 (for standard reference height of 10 m). These data were derived from marine observations (archived in National Climatic Center, Tape Data Family 11, Asheville, NC) by use of synoptic wind-pressure analyses.
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15. Histological field data show 35 to 46% mortality per day due to starvation among first-feeding northern anchovy larvae (*19*). Insufficient food in the first 1.5 to 4.5 days of feeding by these larvae can reduce survival substantially (*9*). Because high wind speeds can dissipate food patches, we assumed that calm periods of 4 days are required for good survival through this critical first-feeding period.
16. For example, eight continuous days of low wind speed, followed by one of high wind, were tallied as five overlapping 4-day calm periods, one starting on each of days 1 through 5. This method reflects the uncertain exact date of spawning. If a calm period began in one month but ended in the next month, it was tallied in the month when the calm period began. Although we report only results for analyses based on overlapping calm periods, we did the same analyses based on nonoverlapping periods (the 8-day example above counted as two calm 4-day periods). Because high speed winds were relatively infrequent and clustered in time, these analyses led to identical conclusions in all cases, although significance levels were slightly different. In addition, conclusions did not change when wind speed indices were calculated using two, three, or five consecutive days of low wind speeds as the criterion for a calm period, instead of four.
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22. Although transport is influenced by wind, our wind speed index and the transport variable were not related ($P = 0.94$) because (i) wind direction determines whether transport is offshore or onshore and (ii) our wind speed index is not a summation of daily wind speeds but a tally of days below the 10 m/sec threshold, regardless of wind direction.
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26. We thank A. Bakun, N. Lo, R. Methot, and P. E. Smith who provided data and these colleagues and others for providing advice and comments on a draft: J. Anderson, L. Botsford, P. Fiedler, J. Hunter, R. Lasker, A. MacCall, R. Owen, R. Parrish, J. Simpson, P. Smith, and G. Theilacker. Supported by fellowships to R.M.P. from the National Research Council, Washington, DC, the Scientific Affairs Division of the North Atlantic Treaty Organization, and a grant to R.M.P. from the Natural Sciences and Engineering Research Council of Canada.

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Technical Comments

In Vivo Activation of CD4$^+$ Cells in AIDS

T-CELL ACTIVATION PLAYS A PIVOTAL role in the expression of human immunodeficiency virus (HIV) cultured in CD4$^+$ cells (*1*). Since immunologic activation of HIV-infected CD4$^+$ cells leads to interleukin-2 (IL-2) and interferon-gamma (IFN-γ) production and then to HIV expression and cell death, these processes appear to be closely linked in vitro. Studies of neopterin excretion in risk groups for AIDS suggest the situation is similar in vivo (*2*). Neopterin is a sensitive indicator of T-cell activation, since it is produced from macrophages specifically in response to IFN-γ (*3*), which is secreted from activated T lymphocytes.

However, other studies demonstrate a reduced ability of lymphocytes from patients with AIDS and AIDS-related syndrome (ARC) to react to antigens or to produce IFN-γ in vitro (*4*). It has even been suggested that this failure contributes to the development of opportunistic infections.

While we do not question the validity of the data obtained in vitro, we propose an alternative explanation on the basis of data from studies of patients with systemic lupus erythematosus (*5*). There is an inverse correlation between the concentration of serum IFN and the production of IFN by lymphocytes in culture. The decreased production of IFN-γ in patients with AIDS and ARC appears to be restricted to studies in vitro and thus does not indicate defective IFN-γ production in vivo. Other data also support this view. It has been shown that CD4$^+$ cells from AIDS patients proliferate spontaneously at a higher rate than CD4$^+$ cells from controls (*6*); the blood of AIDS patients contains high levels of thymosin α-1 (*7*), acid-labile IFN-α (*8*), and activated lymphocytes as well as cells expressing the receptor for IL-2 (*9*). These data support the view that the activation of T cells is important as a cofactor for HIV expression not only in vitro (*1*) but also in vivo, and that the decreased production of IFN-γ from cells from AIDS and ARC patients in vitro results from the continuous endogenous exposure to IFN in vivo.

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