same time, the most that can be concluded is that fusion could cause activation, but it is no less likely that there is another cause. In fact, we have shown in *Urechis* that protein isolated from sperm acrosomal granules causes activation of eggs, including the electrical response[8,9]. Thus activation does not require sperm–egg fusion.

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1. Dale, B. *Nature* **325**, 762–763 (1987).
2. Jaffe, L.A. & Gould, M. in *Biology of Fertilization* Vol. 3 (eds Metz, C.B. & Monroy, A.) 223–250 (Academic, New York, 1985).
3. Kline, D. *et al. J. exp. Zool.* **236**, 45 (1985).
4. Shen, S. & Steinhardt, R.A. *Proc. natn. Acad. Sci. U.S.A.* **81**, 1436 (1984).
5. Dale, B. *J. exp. Biol.* **118**, 85 (1985).
6. Longo, F. *et al. Devl biol.* **118**, 148 (1986).
7. Hinkley, R.E. *et al. Devl Biol.* **118**, 148 (1986).
8. Gould, M. *et al. Devl biol.* **117**, 306 (1986).
9. Gould, M. & Stephano, J.L. *Science* **235**, 1654 (1987).

AIDS predictions

SIR—In criticizing our work, May and Anderson[1] not only refer to its presentation at the WHO meeting on the containment of AIDS (acquired immune deficiency syndrome) in Geneva when they mean the WHO meeting in Graz[2] but they also mistakenly characterize our work[2-4] as an exercise in curve fitting. There is no joint work by us based on statistical analyses to fit polynomial or exponential curves to existing data on the incidence of AIDS. On the contrary, like May and Anderson[1], we have used the approximation of exponential growth for the number of human immunodeficiency virus (HIV) carriers to describe the initial stage of the epidemic. Taking into account the incubation time distribution we have studied the resulting incidence of AIDS. We have shown that the long and variable incubation times lead to transient phenomena characterized by an increase of the doubling times in the observable AIDS epidemic, of the incubation times, of the ratio of AIDS cases to HIV carriers, and so forth. As a consequence, the incidence of AIDS cases in the initial stage of the epidemic is nonexponential, notwithstanding the assumed exponential spread of the virus. In all our work we have stressed that the early exponential phase of growth of HIV carriers passes because of the depletion of the susceptible population and other inhibitory factors. We have looked at this depletion to provide upper limits for the duration of the exponential phase in various countries.

May and Anderson have a point in that extrapolation of trends gained by curve-fitting is unsafe, specially if done uncritically over an extended period. One of us (M.G.K.) made precisely this point in Anderson's presence at the Bilthoven meeting, December 1986. There are many statistical analyses of AIDS based on curve fitting procedures with extrapolation over several years, some of them made at influencial institutions (at the US Centers for Disease Control for example). Why, then, do May and Anderson aim their critical remarks only at our joint work, which has nothing to do with curve-fitting, and a very cautious projection for the United Kingdom[5] made at a time when other prognostic analyses were scarce?

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1. May, R.M. & Anderson, R.M. *Nature* **326**, 137–142 (1987).
2. Gonzalez, J.J. & Koch, M.G. *AIDS Forsch* **11**, 621–630 (1986).
3. Gonzalez, J.J. & Koch, M.G. in *Proc. 1st Int. Meeting of AVIS on AIDS* (Solei, Milano, 1986).
4. Gonzalez, J.J. & Koch, M.G. *Am. J. Epid.* (in the press).
5. McEvoy, M. & Tillett, H.E. *Lancet* **ii**, 541–542 (1985).

Screwworm eradication and climate

SIR—I am surprised that Krafsur and his newly acquired transatlantic colleagues[1] can dismiss my analysis[2] of the screwworm data so lightly. In respect of my equations (3) and (4), predicting seasonal numbers of screwworm cases in Texas from winter temperatures and summer temperatures, the regression coefficients for temperature are significant at the $P<0.01$ and $P<0.02$ levels, respectively, and crucially affect the model's predictability (see Fig. 4)[2]. How can they dismiss my conclusions as "an artefact of pooling heterogeneous data", especially when they agree that "exceptional weather can have dramatic effects on screwworm incidence"?

In fact, if my model is applied to their new data for the seven separate climatological divisions in Texas, assuming that autumn cases (A) represent on average half the preceding summer's cases (the actual proportion does not matter), then the number of winter cases (W) in any particular region can be predicted from winter temperature by equation (1) in Table 1. The parameters are clearly significant (see box), and, in fact, winter temperature looms larger in importance than autumn cases in the prediction (R^2 = 0.49, and 0.44, respectively). In the winter to summer model, equation (2), summer temperature is just short of being significant ($P>0.05$), but that is not surprising in such a small data set involving highly mobile fly populations.

Moreover, since publishing my analysis I have stumbled across independent evidence in support of the idea that change in climate rather than the release of sterile males might be responsible for screwworm eradication.

In their study of case incidence in various counties of South Texas in 1975–76, Krafsur and Garcia[3] inadvertently provide a fix on the overwintering temperature threshold for screwworm. In terms of reported cases, overwintering was possible only in the western counties of Webb, Zapata, J. Hogg, Star and Hildago where winter temperatures are about 2.7°C warmer than the average for South Texas as a whole (see graphs and p.691 in ref. 3). The average winter temperature for South Texas in 1975–76 was in fact 14.4°C (see Fig. 2 of ref. 3) so that an overwintering threshold of about 17.0°C is indicated.

This implies that the species would have been unable to overwinter even in southernmost Florida in 1957–58 when the mean winter temperature at Miami, for example, was only 16.7°C, the coldest on record in nearly 100 years. Similarly, in more recent times, the species could not have overwintered at, for example, Brownsville in southern Texas in 1977–78 or 1978–79, when the outbreaks collapsed and when the winters were the second and third coldest on record, nor in the winter of 1976–77 or any of the winters from 1982 to 1985, which were also very cold. More significantly, in relation to the current campaign in Mexico, the species would have experienced difficulty

	estimate	±	s.e.m.	t	P
a (intercept)	−22.94		3.66	6.26	<0.01
b (autumn cases)	1.49		0.34	4.40	<0.01
c (winter temperature)	0.28		0.05	5.40	<0.01

Table 1 Total reported cases of screwworm and average temperature for seven regions in Texas over 22 years[1]

Region	Screwworm cases: Autumn* (A_{n-1})	Winter (W_n)	Summer (S_n)	Temperature (°F): Winter (wt_n)	Summer (st_n)
Low Plains	2,010	2	4,020	43.2	81.4
Trans Pecos	4,635	11	9,270	46.8	80.1
East Texas	1,434	0	2,868	47.6	80.9
Edwards Plateau	11,475	137	22,951	47.8	81.3
South Central	8,631	103	17,263	53.4	82.9
Southern	9,633	830	19,265	55.4	84.6
Lower Valley	1,670	194	3,340	59.7	83.9

*Autumn cases assumed to be ½ summer cases.

$$\ln(W_n+1) = -22.94 + 1.49\ln(A_{n-1}+1) + 0.28\,wt_n, \quad R^2 = 0.93, P<0.001. \quad (1)$$

$$\ln(S_n+1) = 41.16 + 0.45\ln(W_n+1) - 0.41\,st_n, \quad R^2 = 0.68, P<0.05, st_n \text{ not significant}, P>0.05. \quad (2)$$