mother having one of the ten genotypes. The frequency of these offspring groups is simply the frequency of the mother's genotype times the probability that a female of that genotype will be polygamous (1, 1 − g_1, and 1 − g_2 for *mm*, *mM*, and *MM* females, respectively). Each of the remaining 70 offspring groups are produced by a monogamous mating between a female with one of the seven genotypes capable of monogamy and a male having one of the ten genotypes. The frequency of each of these offspring groups equals the product of the frequencies of the two genotypes involved in the mating times the probability that the female is monogamous (g_1 and g_2 for *mM* and *MM* mothers, respectively). Let us number the offspring groups in arbitrary order from 1 to 80, and let f_j be the frequency of offspring groups of the jth type. Let $y_{i,j}$ be the frequency of the ith genotype in the jth offspring group. The $y_{i,j}$ values are derived in the usual way from knowledge of the parents' genotypes and the value of r. Finally, let z_j denote the frequency of altruists in the jth type of offspring group [$z_j = h_1(y_{1,j} + y_{4,j} + y_{8,j}) + h_2(y_{2,j} + y_{5,j} + y_{6,j} + y_{9,j}) + h_3(y_{3,j} + y_{7,j} + y_{10,j})$]. We may now write:

$$w_i = 1 - h_k\gamma + \left(\frac{\sum_{j=1}^{j=80} f_j y_{i,j} z_j}{\sum_{j=1}^{j=80} f_j y_{i,j}}\right)\beta$$

$$= 1 - h_k\gamma + \left(\frac{\sum_{j=1}^{j=80} f_j y_{i,j} z_j}{\bar{u}_i}\right)\beta$$

where k = 1, 2, and 3, respectively, for genotypes with *AA*, *Aa*, and *aa* at the altruism locus.

When m is fixed, the recursions specified by Eq. 2 become a two-dimensional system and the frequency of A at polymorphic equilibria ($\hat{p}_A$) must satisfy:

$$0 = (h_1\hat{p}_A^2 + 2h_2\hat{p}_A\hat{q}_A + h_3\hat{q}_A^2)(2\gamma - \beta)^2 - \beta\gamma(h_1\hat{p}_A + h_3\hat{q}_A) + \beta - 4\gamma \quad (3)$$

where $\hat{q}_A = 1 - \hat{p}_A$. Furthermore, polymorphic equilibria cannot exist unless the following holds:

$$\beta > 2\gamma \quad (4)$$

We can use Eq. 2 to derive linearized recursions for the frequencies of the *Mm* genotypes in the vicinity of an equilibrium at which m is fixed and the altruism locus is polymorphic. In matrix form, the linearized recursions are:

$$\begin{bmatrix} \epsilon_4' \\ \epsilon_5' \\ \epsilon_6' \\ \epsilon_7' \end{bmatrix} = \begin{bmatrix} c_1(1-r)c_2 & rc_2 & 0 \\ c_3(1-r)c_4 & rc_4 & 0 \\ 0 & rc_5 & (1-r)c_5\ c_6 \\ 0 & rc_7 & (1-r)c_7\ c_8 \end{bmatrix} \begin{bmatrix} \epsilon_4 \\ \epsilon_5 \\ \epsilon_6 \\ \epsilon_7 \end{bmatrix} \quad (5)$$

where $\epsilon_i = u_i$ and $\epsilon_i' = u_i'$ in the vicinity of the equilibrium. The c_i values are positive constants that depend on h_1, h_2, h_3, β, γ, and the particular root of Eq. 3 chosen as $\hat{p}_A$.

Let B be the 4 × 4 matrix in Eq. 5. When r = 0, B breaks down into two 2 × 2 submatrices. Under the requirements of inequalities 1 and 4, each of these submatrices may be shown to have its largest eigenvalue greater than unity, so that the full 4 × 4 stability matrix has two eigenvalues greater than unity when r = 0.

Let $d(\lambda)$ represent the characteristic polynomial of B:

$$d(\lambda) = \det[B - \lambda I]$$

where I is a 4 × 4 identity matrix. It is possible to show that $d(1)$ is linear in r, and using this fact, along with a continuity argument, the Perron-Frobenius theorem, and the above results for r = 0, one can demonstrate that B has two distinct and real eigenvalues in excess of unity for r small and positive. Thus, for B to have no eigenvalue in excess of unity for some value of r, $d(1)$ = 0 must hold for at least two values of r. However, this is impossible in light of the linearity of $d(1)$, and thus B has at least one eigenvalue in excess of unity for all r.

11. We made all selections of random numbers in the numerical studies by using a nonlinear additive feedback random number generator. In the first study (invasions by M when the altruism locus is polymorphic) possible values for h_1, h_2, h_3, g_1, g_2, and γ were selected according to a uniform distribution on (0, 1); β was selected according to a uniform distribution on (0, 10), and r according to a uniform distribution on (0, ½). Each set of possible parameter values was tested for satisfaction of inequalities 1 and for satisfaction of 0 < $g_1 \leq g_2$. The parameters were tested further to see whether they allowed for the existence of polymorphic equilibria (that is, Eq. 3 was tested for solutions satisfying 0 < $\hat{p}_A$ < 1). Parameter sets that passed all of these tests were used in the computer trials. The polymorphic equilibria always occur in pairs, and numerical evaluation of the associated eigenvalues suggests that one equilibrium is always stable while the other is unstable. We picked the stable polymorphic equilibrium to initialize the computer trials.

It is relatively simple to show that the frequencies of the first three genotypes at polymorphic equilibria when m is fixed are given by:

$$\hat{u}_1 = \frac{\hat{p}_A[\beta - \hat{q}_A(2\beta - 4\gamma)]}{\beta}$$

$$\hat{u}_2 = \frac{\hat{p}_A\hat{q}_A(4\beta - 8\gamma)}{\beta}$$

$$\hat{u}_3 = \frac{\hat{q}_A[\beta - \hat{p}_A(2\beta - 4\gamma)]}{\beta}$$

These expressions were used to calculate the initial genotype frequencies [$u_i(0)$], which are given by:

$$\begin{aligned} u_1(0) &= 0.998001\hat{u}_1(1 + \Upsilon_1)N \\ u_2(0) &= 0.998001\hat{u}_2(1 + \Upsilon_2)N \\ u_3(0) &= 0.998001\hat{u}_3(1 + \Upsilon_3)N \\ u_4(0) &= 0.001998\hat{u}_1(1 + \Upsilon_4)N \\ u_5(0) &= 0.000999\hat{u}_2(1 + \Upsilon_5)N \\ u_6(0) &= 0.000999\hat{u}_2(1 + \Upsilon_6)N \\ u_7(0) &= 0.001998\hat{u}_3(1 + \Upsilon_7)N \\ u_8(0) &= 0.000001\hat{u}_1(1 + \Upsilon_8)N \\ u_9(0) &= 0.000001\hat{u}_2(1 + \Upsilon_9)N \\ u_{10}(0) &= 0.000001\hat{u}_3(1 + \Upsilon_{10})N \end{aligned}$$

where the Υ_i values are independent random variables chosen anew for each computer trial from a uniform distribution on (0, 10^{-8}), and N is a normalizing factor calculated from the requirement that

$$1 = \sum_{i=1}^{10} u_i(0)$$

The use of these equations to choose the initial genotype frequencies assures that the initial values of p_M will approximate 0.001 and that the initial values of u_1, u_2, and u_3 will be only slightly perturbed from their equilibrium values. Furthermore, the initial values of D, the linkage disequilibrium coefficient, will be small but nonzero ($D = x_1x_4 - x_2x_3$).

In the second numerical study the parameters were chosen in the same manner as in the first study, except that no test for the existence of polymorphic equilibria was required. To assign the initial genotype frequencies [$u_i(0)$], we started by independently choosing two random variables, $p_A(0)$ and $p_M(0)$, from a uniform distribution on (0, 1). The following equations were then used to generate the u_i (0) values:

$$\begin{aligned} u_1(0) &= [p_A(0)q_M(0)]^2(1 + \Upsilon_1)N \\ u_2(0) &= 2p_A(0)q_A(0)\ [q_M(0)]^2(1 + \Upsilon_2)N \\ u_3(0) &= [q_A(0)q_M(0)]^2(1 + \Upsilon_3)N \\ u_4(0) &= 2[p_A(0)]^2p_M(0)q_M(0)(1 + \Upsilon_4)N \\ u_5(0) &= 2p_A(0)q_A(0)p_M(0)q_M(0)(1 + \Upsilon_5)N \\ u_6(0) &= 2p_A(0)q_A(0)p_M(0)q_M(0)(1 + \Upsilon_6)N \\ u_7(0) &= 2[q_A(0)]^2p_M(0)q_M(0)(1 + \Upsilon_7)N \\ u_8(0) &= [p_A(0)p_M(0)]^2(1 + \Upsilon_8)N \\ u_9(0) &= 2p_A(0)q_A(0)[p_M(0)]^2(1 + \Upsilon_9)N \\ u_{10}(0) &= [q_A(0)p_m(0)]^2(1 + \Upsilon_{10})N \end{aligned} \quad (6)$$

where $q_A(0) = 1 - p_A(0)$ and $q_M(0) = 1 - p_M(0)$; N and the Υ_i values are defined as in the first numerical study. This scheme assures that the initial values of D will be small and that the initial values of p_A and p_M will approximate $p_A(0)$ and $p_M(0)$, respectively.

The use of initial genotypic distributions that have small values of D allowed for the production of a simple and concisely communicable set of results in the numerical studies. However, a similar set of results can be obtained even when arbitrarily large initial deviations from the D = 0 surface are allowed. For example, we repeated the first numerical study using the following initial genotype frequencies:

$$\begin{aligned} u_1(0) &= 0.999\hat{u}_1 & u_2(0) &= 0.999\hat{u}_2 \\ u_3(0) &= 0.999\hat{u}_3 & u_4(0) &= \Upsilon_1N \\ u_5(0) &= \Upsilon_2N & u_6(0) &= \Upsilon_3N \\ u_7(0) &= \Upsilon_4N & u_8(0) &= \Upsilon_5N \\ u_9(0) &= \Upsilon_6N & u_{10}(0) &= \Upsilon_7N \end{aligned}$$

In this case the Υ_i values were chosen from a uniform distribution on (0, 1), and N is again a normalizing factor. This scheme assures that the initial values of p_M will not exceed 0.001 but otherwise leaves the starting values of D unconstrained. Although this allowed a variety of outcomes during the early generations of the computer trials, the effects of the initialization tended to "wash out" later on. Thus, although both p_A and p_M increased during the first generation in only 5,854 of the 10,000 trials, by generation 100 these two gene frequencies were increasing in all but 569 cases.

We also repeated the second numerical study using completely arbitrary initial genotype frequencies:

$$\begin{aligned} u_1(0) &= \Upsilon_1N & u_2(0) &= \Upsilon_2N & u_3(0) &= \Upsilon_3N \\ u_4(0) &= \Upsilon_4N & u_5(0) &= \Upsilon_5N & u_6(0) &= \Upsilon_6N \\ u_7(0) &= \Upsilon_7N & u_8(0) &= \Upsilon_8N & u_9(0) &= \Upsilon_9N \\ & & u_{10}(0) &= \Upsilon_{10}N & & \end{aligned}$$

where the Υ_i values are again chosen from a uniform distribution on (0, 1), and N is a normalizing factor. Here also the effects of the initial conditions were generally short-lived and eventually the gene frequencies behaved in a manner analogous to the results obtained when Eqs. 6 were used to assign the initial genotype frequencies. For example, in 8,504 of the trials p_A was increasing by generation 100, and in all but 186 of these trials the same was true of p_M.

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13. We thank G. Barlow, F. Christiansen, S. Frank, K. Holsinger, J. M. Peck, F. Pitelka, M. Slatkin, and M. Uyenoyama for helpful suggestions. Supported by NIH grants GM 28016 and GM 10452.

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Technical Comments

PCB Dechlorination in Hudson River Sediment

The report "Polychlorinated biphenyl dechlorination in aquatic sediments" by John F. Brown *et al.* (*1*) purports to show that polychlorinated biphenyls (PCBs) in Hudson River sediments are undergoing dechlorination and detoxification by anaerobic microbes. The postulation of anaerobic activity is based on the departure of the PCB conge-

ner pattern from a presumed original pattern.

The composition and quantity of PCB discharged to the Hudson River from two General Electric (GE) capacitor factories for approximately 30 years was essentially unmonitored (*2*). Cooling drainage, steam condensation, detergent washing processes, and a network of interconnected diffuse sources supplied PCB from one of the plants to the river (*3*). A significant portion of the PCB derived from the other plant was discharged to the river from a municipal sewage treatment plant (*3*). The discrepancy between the composition of PCB in river sediment and the proportions of Aroclors in GE's incomplete purchase records has been known since the early 1970s (*2*).

Simple quantitative analyses invalidate the presumption of sediment with a PCB composition like Aroclor 1242. A summary of our recent analyses of PCB composition in sediment within 12 km of GE discharges is shown in Table 1. Mono-, di-, hexa-, and heptachlorobiphenyls are enriched relative to Aroclor 1242, whereas tetra- and pentachlorobiphenyls are in proportions similar to that of Aroclor 1242. If Aroclor 1242 is used as a yardstick, the logic of Brown *et al.* would indicate chlorination as well as dechlorination.

When the authors' pattern A is used as a reference, there are substantial differences between patterns A and B that cannot be explained by the proposed dechlorination scheme. J. F. Brown provided detailed data for 18 consecutive sections of the sediment core 18-6. These data have been summarized elsewhere (*6*) and have been used to calculate numbers of halftimes for the dechlorination process (*7*). According to the authors, 2,4,2′,4′-tetrachlorobiphenyl appears to be stable, with a net reduction of approximately 5% occurring over 15 years (*7*).

Using their data we calculated the mole ratio of 2,4,2′,4′-tetrachlorobiphenyl to total PCB. The four samples with pattern A have mole ratios of 2.2 to 2.9%. Mole ratios of the six samples with pattern B ranged from 1.4 to 1.7%. The averages of these ratios imply a 68% enrichment of PCB molecules relative to 2,4,2′,4′-tetrachlorobiphenyl in pattern B. Moreover, the sum of the comparatively stable 2,4,2′,4′-, 2,4,2′,5′-, and 2,5,2′,5′-tetrachlorobiphenyls (*7*) indicates enrichment of 36% in pattern B relative to pattern A. The mole proportion calculations indicate pattern modification with enrichment by selective deposition. The average total PCB concentration in pattern B samples in this core is 1100 μg/g, which is ten times the average of pattern A samples. In the data provided to us, the concentrations of all individual PCB congeners are higher in pattern B samples than in pattern A samples. A sedimentological and PCB transport framework for the proposed dechlorination scheme appears necessary for the interpretation of real and relative differences between PCB congener concentrations in recent and in earlier sediment deposits in the Hudson River. Such a framework is lacking in the three publications by Brown and his colleagues (*1, 6, 7*) regarding anaerobic dechlorination of PCB in the environment.

Table 1. Composition of PCB in Hudson River sediment the Fort Edward Area (*4*) and in Aroclors (*5*).

PCB isomer group	Sediment (%)	PCB Aroclors			
		1221 (%)	1242 (%)	1254 (%)	1260 (%)
Monochlorobiphenyl	6.5	51	1	0.1	
Dichlorobiphenyl	21.7	32	16	0.5	
Trichlorobiphenyl	23.5	4	49	1	
Tetrachlorobiphenyl	29.1	2	25	21	
Pentachlorobiphenyl	10.0	0.5	8	48	12
Hexachlorobiphenyl	4.2		1	23	38
Heptachlorobiphenyl	3.5		0.1	6	41
Octachlorobiphenyl					8

The hypothesis of anaerobic microbial dechlorination of PCB in the Hudson River is perhaps not confirmable by the use of logic that requires an assumption of a prior composition of PCB in river sediment. An alternative hypothesis, that pattern variations could have been caused primarily by a number of physical and chemical processes in the factories, in wastewater treatment, and during the process of river transport and sedimentation, cannot be rejected. We remain unconvinced that microbial PCB dechlorination has occurred in the Hudson River.

MARK P. BROWN
Bureau of Technical Services and Research, New York State Department of Environmental Conservation, Albany, NY 12233-3502
BRIAN BUSH
G. YULL RHEE
LANA SHANE
Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY 12201

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Response: We disagree with the representation by M. P. Brown and his coauthors (*1*) of (i) the extent of the available information on the original composition of the PCBs deposited in the sediments of the Hudson River immediately below Fort Edward, New York; (ii) the nature of the sediment sample subpopulation that was selected for reporting in table 1 of their comment; (iii) our reported data on the stability of 2,4,2′,4′-tetrachlorobiphenyl (24-24 CB) levels in upper Hudson River sediments; and (iv) the compositional alterations that would be expected if deposition occurred by means of their proposed (*2*) sedimentation hypothesis.

Although the middle and lower portions of the Hudson River have received PCBs from many sources, it is generally agreed that most of those in upper Hudson Reaches 8 and 9 (river miles 188.5 to 194.8) came from General Electric capacitor manufacturing plants located just upstream in Hudson Falls and Fort Edward, New York. Releases occurred mainly between 1955 and 1971. In 1971, major control measures were taken that sharply reduced the discharges and changed their pattern to that reported in reference 3 of M. P. Brown's comment (*1*). Monsanto sales records made publicly available in 1982 show that during the period 1955 through 1971 these plants' PCB purchases were 97.4% Aroclor 1242 (50.6×10^6 kg) and 2.6% Aroclor 1254 (1.4×10^6 kg). The composition of the General Electric releases was described by M. P. Brown *et al.* as predominantly Aroclor 1242 as recently as early 1987 (*2*). The overall composition of the minor, non–General Electric contributions to the Reach 8–9 deposits is not known; however, we have seen lightly contaminated sediment and fish specimens, collected both