Correction. In the article "Triene prostaglandins: Prostacyclin and thromboxane biosynthesis and unique biological properties" by Philip Needleman, Amiram Raz, Mark S. Minkes, James A. Ferrendelli, and Howard Sprecher, which appeared in the February 1979 issue of *Proc. Natl. Acad. Sci. USA* (**76,** 944–948), a printer's error resulted in an incorrect sequence of sentences on p. 947. The text of p. 947 should be as follows:

to 18 ± 3) all increase platelet cyclic AMP and inhibit platelet aggregation. PGH_1 has been demonstrated to increase cyclic AMP levels in PRP (11, 23).

Effect of PGH_3 on ADP Release Reaction and Thromboxane Synthesis. Preincubation of PGH_3 with PRP prevented aggregation and suppressed the ADP release induced by exogenous arachidonic acid. Addition of 50 μg of arachidonate to PRP caused complete aggregation and release of 8 μM ADP, and high concentrations of arachidonate caused no further ADP release (data not shown). As evidence that the platelets were not lysed by the arachidonate, subsequent lysis with Triton caused the release of a total of 20 μM ADP. Pretreatment of PRP with PGH_3 (100 ng) inhibited aggregation and ADP release.

Thromboxane A_2 formation was monitored directly by measuring the rabbit aorta contractile activity generated in PRP by exogenous arachidonate or by thrombin. Thrombin was used to liberate endogenous fatty acid from platelet phospholipids. PGH_3 (0.1–2 μM), PGI_2 (0.1–2 μM), PGH_1 (up to 5 μM), and dibutyryl cyclic AMP (up to 5 mM) did not alter the contractile activity formed by exogenous arachidonate (0.7 mM) in PRP (data not shown). However, very low levels of PGH_3 (0.3–0.5 μM) or PGI_2 (0.1 μM) inhibited arachidonate-induced PRP aggregation. In contrast, a 50% decrease in formation of contractile activity was induced by thrombin (10 units) in PRP preincubated with PGH_3 (0.4–0.7 μM), PGI_2 (0.3–0.6 μM), or dibutyryl cyclic AMP (5 mM).

Both [^{14}C]eicosapentaenoate and [^{14}C]arachidonate were readily and similarly acylated into phospholipids when incubated with human platelets (Fig. 4 *A* and *E*). In addition, deacylation by thrombin or an ionophore (A-23187) of separately prelabeled platelets released comparable amounts of [^{14}C]-arachidonate or [^{14}C]eicosapentaenoate, respectively (Fig. 4 *B* and *F*). Pretreatment of the arachidonate-labeled platelets with PGH_3 (Fig. 4*C*), PGI_2 (Fig. 4*D*), or PGH_1 (100 ng) markedly decreased the release of fatty acid and abolished the thromboxane formation. There was no increase in fatty acid release over basal levels when thrombin was added to labeled platelets treated with PGH_3 or PGI_2 (Fig. 4 *C* and *D*).

Competition of Eicosapentaenoate with Arachidonate for Platelet Cyclooxygenase. Unlabeled eicosapentaenoic acid and [^{14}C]arachidonate were mixed in varying ratios and the reaction was initiated by the addition of washed platelets. The eicosapentaenoate effectively competed with arachidonate such that a 1:1 mixture of the fatty acids resulted in a 50% inhibition of formation of thromboxane B_2 and 12-hydroxyeicosatetraenoic and hydroxyheptadecatrienoic acids and block of arachidonic destruction (Fig. 5*A*). On the other hand, the eicosapentaenoate was a much poorer substrate for platelet cyclooxygenase, being converted only one-eighth as efficiently as arachidonate to thromboxane (Fig. 5*B*).

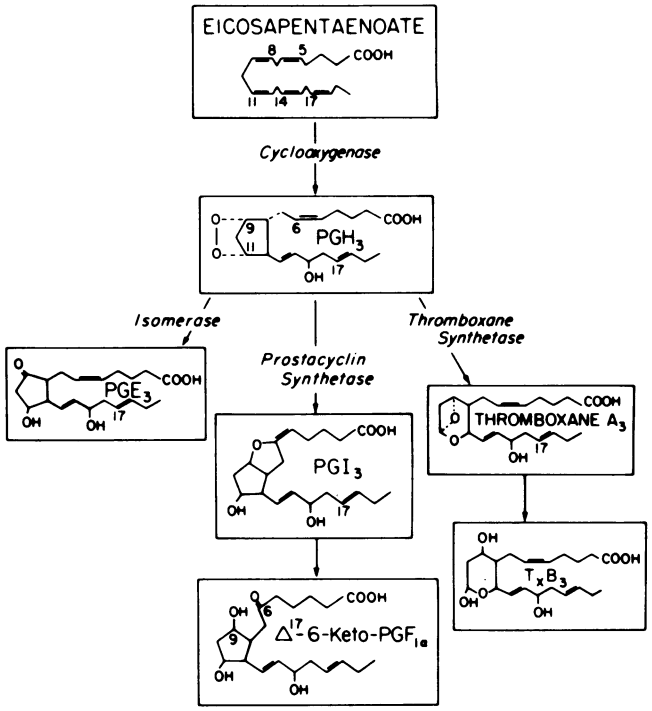

FIG. 6. Eicosapentaenoic acid metabolic pathway.

Correction. In the article "Influence of major histocompatibility haplotype on autoimmune disease varies in different inbred families of chickens" by Larry D. Bacon and Noel R. Rose, which appeared in the March 1979 issue of *Proc. Natl. Acad. Sci. USA* (**76,** 1435–1437), there was a printer's error in Table 1. In this table, the groups of chickens that do not differ significantly in their immunological responses are joined by continuous lines. In the OSC subsets, for birds 6–10 weeks of age, the B^5B^5 group should be joined with the B^5B^{13} and $B^{13}B^{13}$ groups. That is, the OSC B^5B^5 juvenile birds do not differ from those of other *B* haplotypes in development of thyroid autoimmunity.

Correction. In the article "Transformation of DBA/2 mouse fetal liver cells infected *in vitro* by the anemic strain of Friend leukemia virus" by D. W. Golde, N. Bersch, C. Friend, D. Tsuei, and W. Marovitz, which appeared in the February 1979 issue of *Proc. Natl. Acad. Sci. USA* (**76,** 962–966), the authors request that ref. 6 be corrected to read:

6. Pragnell, I. B., McNab, A., Harrison, P. & Ostertag, W. (1978) *Nature (London)* **272,** 456–458.

Correction. In the article "Biosynthesis *in vitro* of immunoreactive 31,000-dalton corticotropin/β-endorphin-like material by bovine hypothalamus" by Anthony S. Liotta, David Gildersleeve, Michael J. Brownstein, and Dorothy T. Krieger, which appeared in the March 1979 issue of *Proc. Natl. Acad. Sci. USA* (**76,** 1448–1452), the authors request that the following correction be noted. In the legend to Fig. 2, the two sentences on lines 9–12 should read "An aliquot of the pooled extract was first allowed to react with the ACTH immunoadsorbent and activity not retained was then allowed to react with the β-endorphin immunoadsorbent. Activity retained on this latter column was eluted and subjected to Sephadex G-50 gel filtration (curve 1)."