MgO, FeO, Mn_2O_3, Cr_2O_3, and most of the SiO_2 remain behind.

Other serpentinization processes apparently lead to the same result as the process we propose. The conclusion is supported by comparison of chemical analyses of bulk rock samples from fresh peridotites and pyroxenites (from California) with analyses made of serpentinites (*10*). The fresh peridotites and pyroxenites (*10*) contain 0.037 to 16.9 percent of CaO by weight, whereas the serpentinites by analysis contain CaO in amounts ranging from "not detectable" to 2.49 percent by weight; the comparison shows a probable removal of CaO upon serpentinization. Twelve analyses of serpentine-group minerals (*11*) show a maximum CaO content of 0.3 percent by weight, indicating that CaO is not readily incorporated into serpentine-group minerals. A study of fresh and serpentinized ultramafic rocks in California and elsewhere (*12*) also has shown that the CaO component is selectively removed upon serpentinization.

In some serpentinization, the transformation of olivine and pyroxene must be accompanied by an increase in rock volume. The increase in volume at depth could result in serpentinite extrusion (*4*). Continued rising due to expansion may also explain why so many ultramafic bodies are topographically high, despite their apparently rapid reduction by slumps, landslides, and stream erosion.

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Virus-Induced Erythropoiesis in Hypertransfused-Polycythemic Mice

Abstract. *Friend, Rauscher, and polycythemic viruses produce a marked erythrocytopoiesis in the spleens of infected mice. The erythrocytopoiesis induced by the Friend and Rauscher viruses can be inhibited in a hypertransfused-polycythemic state. Like erythropoietin, the polycythemic virus can initiate erythropoiesis in this state. However, the mechanism by which the polycythemic virus initiates erythropoiesis in this state is not understood. These studies also show similarity of the Friend and Rauscher viruses in their erythropoietic response.*

Viruses, such as Friend (*1*), Rauscher (*2*, *3*), and the polycythemic virus (*4*), produce marked erythrocytopoiesis in the spleen of mice; for this reason they have been referred to as erythropoietic viruses. This report deals with the erythropoietic responses induced by these three erythropoietic viruses in mice in a hypertransfused-polycythemic state (*5*, *6*).

Swiss male mice (Ha/ICR, 5 to 6 weeks old) were used, and a hypertransfused-polycythemic (HP) state (*5*) was induced by intraperitoneal injections of 0.5 ml of washed, packed, homologous red blood cells on 3 consecutive days and again on day 5. This resulted in hematocrits on the order of 70 to 75 percent. The viruses were then given to each group when the HP state was achieved. This state in virus-infected mice was prolonged by giving red-cell transfusions twice a week to maintain the hematocrits above 70 percent.

The polycythemic virus (*4*), first detected in our laboratory, has been passed since 1961, and it is now in its 96th passage generation. This virus could be a variant of Friend virus or a passenger virus present in filtrates prepared originally from Friend virus-infected spleens of Taconic Swiss mice. The erythropoietic patterns induced by Friend and Rauscher viruses are similar, but the polycythemic virus induces an erythrocythemia and increases in granulocytes and platelets (*4*). The mice in each group were inoculated intraperitoneally with 0.2 ml of a splenic filtrate. The titers (infectious dose, 50 percent effective, ID_{50}) of the respective splenic filtrates (per milliliter) used in this study were: $10^{5.0}$ for the Rauscher virus, $10^{4.2}$ for the Friend virus, and $10^{4.5}$ for the polycythemic virus.

Erythropoietic parameters used were the uptake of Fe^{59} (*7*) and reticulocyte counts. All mice were injected intravenously with 0.5 μc of Fe^{59} on the 13th day of virus infection; the uptake of Fe^{59} in the femur, blood, and spleen was determined 24 hours later (the 14th day of virus infection).

In a HP state, erythropoietic production and morphological evidence of erythropoiesis are drastically depressed (*5*, *6*) (Table 1). So far, erythropoiesis has only been initiated in a HP state by the administration of erythropoietin or substances that influence erythropoietin production (*8*). Consequently, this has been used as a reliable assay for the detection of erythropoietin activity in plasma, urine, and other body fluids (*9*). However, as early as the first week, HP mice infected with the polycythemic virus showed resump-

Table 1. Response of polycythemic, Friend, and Rauscher viruses in hypertransfused-polycythemic (HP) Ha/ICR Swiss mice 14 days after infection. ±, Standard deviation.

Mice and treatment	Response to virus					
	Spleen weight (g)	Average 24-hour Fe^{59} uptake* (%)			Reticulocytes (%)	Hematocrit (%)
		Femur	Blood	Spleen		
Normal	0.15 ± .04	0.49 ± .05	24.8 ± 2.8	1.2 ± .08	0.5 ± .03	42 ± 1.2
HP	.24 ± .02	.09 ± .02	0.41 ± .09	0.07 ± .03	0.0	75 ± 3.4
HP + ESF†	.16 ± .03	.54 ± .09	19.8 ± 3.4	5.8 ± 1.2	3.8 ± 1.0	73 ± 2.8
HP + polycythemic virus	.98 ± .12	.10 ± .02	22.8 ± 1.5	15.3 ± 2.1	3.5 ± 1.2	79 ± 5.4
HP + Friend virus	.18 ± .03	.07 ± .01	0.45 ± .08	0.19 ± .02	0.0	70 ± 5.0
HP + Rauscher virus	.21 ± .02	.17 ± .01	.40 ± .06	.5 ± .07	0.0	73 ± 6.0

* Average from minimum of five mice per group. † One unit of erythropoietin was administered subcutaneously on 3 successive days, and 0.5 μc of Fe^{59} was injected intravenously on the 4th day. Uptake of Fe^{59} was determined on the 5th day.

tion of erythropoiesis as evidenced in the bone marrow, blood, and spleen. Table 1 shows resumption of erythropoiesis at 14 days after infection. Uptake of Fe^{59} by the blood of HP mice infected with the virus was comparable to that observed in HP mice receiving erythropoietin. The erythropoiesis in the spleen was approximately three times greater in the HP mice receiving the virus than in HP mice receiving erythropoietin. The polycythemic virus has a strong affinity for the spleen; removal of the spleen prevents the polycythemic response (*4*). In HP mice receiving the polycythemic virus, depression of erythropoiesis in the bone marrow occurs during the 2nd week. This occurs routinely as the spleen increases in size.

The histological examinations of the spleens in HP mice to which erythropoietin had been administered and in those which had received polycythemic virus were similar in showing marked erythropoiesis. Early erythroid cells and mature red blood cells were evident in the area of the red pulp.

Friend virus, like Rauscher virus, did not initiate erythropoiesis in a HP state. Histological examination of the hemopoietic organs, particularly the spleen, confirmed the inability of these viruses to reestablish erythropoiesis. Mice not in a hypertransfused-polycythemic state, which received the polycythemic, Friend, or Rauscher virus at the same time as the hypertransfused-polycythemic mice, showed signs of the disease usually induced by these viruses. Therefore, the inability of the Friend or Rauscher virus to initiate erythropoiesis in HP state is not attributable to a decreased virus titer at the time of inoculation. Possibly, the HP state does not make available a cell type (or types) that permits replication of the Friend or Rauscher virus.

Like immunological techniques, the HP state has borne out the similarity of the Rauscher and Friend viruses (*3*, *10*). It is significant that, although all three viruses stimulate erythropoiesis in a normal state, only the polycythemic virus can initiate erythropoiesis in a HP state; the exact mechanism by which this is accomplished is not known. Perhaps the polycythemic virus influences erythropoietin-producing cells to release erythropoietin, causing differentiation of stem cells (*11*) into erythroid cells. Another possibility is that the polycythemic virus induces erythroid stimulation independently of erythropoietin. If this is so, then presumably the polycythemic virus in itself, like erythropoietin, is capable of enzyme induction (*11*) in stem cells, causing them to differentiate into early erythroid cells.

Attempts to isolate erythropoietin from concentrated plasma and urine from infected normal and HP mice at various stages after viral infection have not been successful. Thus far, it has also been impossible to determine whether the polycythemia induced by the polycythmic virus is erythropoietin-dependent or independent by use of an antierythropoietin (*12*).

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Attention Shift and Errorless Reversal Learning by the California Sea Lion

Abstract. *Repeated combinations of a previously well-learned size cue preference with the previously negative form cue followed by gradual reduction of the size cue resulted in nearly errorless performance by two sea lions on a series of nine form-discrimination reversals. Systematic insertion of probe trials, during which the size cue was not present, revealed that attention was primarily focused on the size dimension during early stages of training, when size differences were large, and then was gradually shifted to the form dimension as a function of the increasing difficulty of the size discrimination.*

As an outgrowth of research on animal learning, a powerful technique has been developed that facilitates getting an animal to attend to the relevant stimulus dimension of a discrimination task. The relevant dimension is introduced in such a way as to minimize or eliminate errors. The technique relies on establishing a simple discrimination between stimuli within a single dimension, superimposing a new relevant dimension on the previous one, and then gradually eliminating the original dimension until it is no longer present (*1*). Recent work has extended the use of this progressive training procedure to a series of discrimination reversals performed virtually without error by the California sea lion (see *2*).

The critical elements of training were the repeated combining of a previously well-learned size cue preference with the previously negative form cue, followed by the progressive reduction of the size cue. Figure 1 shows a prototype of the progressive training procedure used in a series of reversal tasks. The figure shows that the first form reversal may begin by having the negative form (circle) and the positive size (small) constitute the positive or reinforced compound stimulus and by having the positive form (triangle) and the negative size (large) constitute the negative or nonreinforced compound stimulus. During succeeding training stages the area of the circle is gradually increased and the area of the triangle is gradually decreased until size is eliminated as a cue and only the circle and triangle serve as discriminative stimuli. Training on the next form reversal proceeds in a similar manner except that the small triangle becomes the positive compound stimulus and the large circle becomes the negative compound stimulus.

Within the present context, the most important feature of these experiments