of magnetic reconnection between the solar polar magnetic field and the nearby interstellar magnetic field.

Another observed deviation from the Parker model was reported by Ness *et al.* (*4*), who found no trace of the variation in average magnetic field direction as a function of velocity that would be expected from the equation $\tan \alpha = \Omega R / V_S$. A similar result was found by Neugebauer and Snyder (*6*).

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Phosphate Release and Sorption by Soils and Sediments: Effect of Aerobic and Anaerobic Conditions

Abstract. *Anaerobic soils released more phosphate to soil solutions low in soluble phosphate and sorbed more phosphate from soil solutions high in soluble phosphate than did aerobic soils. The difference in behavior of phosphate under aerobic and anaerobic conditions is attributed to the change brought about in ferric oxyhydroxide by soil reduction. The probably greater surface area of the gel-like reduced ferrous compounds in an anaerobic soil results in more soil phosphate being solubilized where solution phosphate is low and more solution phosphate being sorbed where solution phosphate is high.*

The amount of dissolved inorganic orthophosphate in flooded soils, swamp and marsh sediments, and shallow bodies of water depends on the capacity of the soil or sediment to release orthophosphate-P to a solution low in P and to sorb it from a solution high in P. Soils and sediments thus tend to have a buffering effect on solution P. These reactions help determine whether the P concentration in the interstitial and overlying water is adequate for the nutritional requirements of plants and whether the soils and sediments can remove enough P from solutions high in P to influence eutrophication.

The sorption and release of P is affected by, among other factors, the oxidation-reduction status of the soil or sediment. Mortimer (*1*) showed that the disappearance of dissolved oxygen and the subsequent reduction of the sediment resulted in a severalfold increase of dissolved P in a freshwater lake. Oxygenation of the sediment reversed this condition and decreased the P concentration. A higher amount of solution P in equilibrium with soils and sediments under anaerobic (reducing) conditions compared with aerobic (oxidizing) conditions has also been shown in flooded rice soils (*2*). These cited studies dealt with the equilibrium between P in solution and in the solid phase under conditions where the P concentration was low in both phases. Of equal or greater importance is the capacity of soils and sediments to sorb and release P under conditions where the P concentration is high in one or both phases. Little is known about the effect of anaerobic conditions on P sorption and release in interstitial water and overlying floodwater containing relatively high concentrations (5 μg/ml or more) of dissolved P.

Although the mechanism by which P is removed from solutions by sediments is not clearly understood, it is thought to be a sorption process rather than a precipitation process (*3*). Shukla *et al.* (*4*) and Williams *et al.* (*5*) attributed P sorption to a gel complex consisting largely of hydrated iron oxide. In soils and sediments exposed to free oxygen the active iron is in the Fe^{3+} form, probably as ferric oxyhydroxide (*6*), but under anaerobic conditions most of the active iron is in the Fe^{2+} form, with some occurring as ferrous hydroxide gel complex (*5*). Marked differences have been observed in the nature of Fe in aerobic and anaerobic soils. Anaerobic soils and sediments have much more Fe in solution—approximately 50 to 100 parts per million (ppm) compared to less than 1 ppm in aerobic soils—as well as a much greater amount of Fe adsorbed on the exchange complex (*7*). The oxidation state of the iron compounds apparently affects the phosphate equilibrium between solid and solution. Phosphate coprecipitated or occluded in ferric oxyhydroxide in an aerobic soil does not exchange with solution phosphate as readily as in an anaerobic soil.

The apparent relation of P sorption and release to hydrated iron oxide and the known effect of reducing conditions on ferric oxyhydroxide motivated the experiments reported here. These experiments were designed (i) to determine the effect of oxidizing and reducing soil conditions on P sorption and release in the presence of both low and high concentrations of P in solution and (ii) to determine whether release of P to a solution low in P and sorption of P from a solution higher in P in flooded soils could be related to the reduction of ferric oxyhydroxide to ferrous hydroxide brought about by anaerobic conditions.

Five soils which normally undergo differing conditions of oxygen depletion were used in this study. These were Commerce silt loam, Mhoon silt loam, and Sharkey silty clay loam from the Mississippi River floodplain, Moreland silty clay from the Red River floodplain, and Crowley silt loam from the rice-growing Prairie Terrace area of Louisiana. The first four soils are fertile alluvial soils with a high P-supplying capacity, whereas Crowley soil is low in both total P and P available to plants and requires additions of P for adequate plant growth. The Mhoon, Sharkey, and Moreland soils usually undergo seasonal oxygen depletion, and the Crowley soil is flooded during the summer months for rice production.

In the first of two experiments samples of the Commerce, Sharkey, Moreland, and Crowley soils were incubated for 17 days under aerobic and anaerobic conditions. Each soil was then equilibrated with a solution containing 0.01*M* $CaCl_2$ and P (0 or 100 μg/ml) as $Ca(H_2PO_4)_2$. Care was taken not to expose the anaerobic samples to atmospheric oxygen or to oxygenated solutions. The solution was

Table 1. Soluble P and extractable Fe under aerobic and anaerobic conditions. Values of Fe extracted are in micrograms per gram of soil; values of P are in micrograms per milliliter of solution.

Soil	pH	Fe extracted (μg/g)		Added P (μg/ml)	Soluble P (μg/ml)	
		Aerobic	Anaerobic		Aerobic	Anaerobic
Commerce	5.4	1,590	2,670	0	0.02	2.92
				100	79.2	66.1
Crowley	6.1	3,190	12,620	0	0.002	0.005
				100	40.8	6.7
Moreland	6.8	2,925	5,895	0	0.14	4.48
				100	48.2	13.0
Sharkey	6.5	3,025	3,910	0	0.03	1.17
				100	62.7	14.0

analyzed for P by the stannous chloride–molybdenum blue method (*8*). Samples of the incubated soils were extracted with an oxalate solution to remove amorphous and poorly crystallized Fe oxides, and the extracted Fe was analyzed by the α,α'-dipyridyl method (*9*). The amounts of soluble P and extracted Fe are given in Table 1.

In the second experiment larger samples of Crowley and Mhoon soils were incubated under aerobic and anaerobic conditions, and the distribution of added soluble P between the solution and solid phases was determined. Each sample (300 g) was kept in suspension by use of a magnetic stirrer in 1500 ml of 0.01*M* $CaCl_2$ in a sealed 2-liter flask. Slow streams of air for the aerobic treatments and argon for the anaerobic treatments were continuously bubbled through the suspensions. The samples were incubated for 17 days at 30°C before the incremental additions of P as $Ca(H_2PO_4)_2$ shown in Fig. 1 were begun. Samples of the suspension were removed (without oxygen contamination of the anaerobic ones) 24 hours after each addition of P and filtered through a 0.45-μm Millipore filter, and the filtrate was analyzed for P.

Under anaerobic conditions more P was released from the soil into the solution than under aerobic conditions (Table 1). This was most pronounced for the three floodplain soils, which had more total P than the Crowley soil. Even though the anaerobic soils released more P to a P-free solution, they were also capable of sorbing more P from a high-P solution. Under aerobic conditions the soils sorbed about one-half or less of the solution P, while under anaerobic conditions all the soils except the Commerce removed most of the P from solution. The Crowley soil was especially active in removing P under reducing conditions, with 93 percent of the added P sorbed by the solid phase.

The amount of P remaining in solution after incremental additions of P to oxidized and reduced suspensions of Crowley and Mhoon soils is shown in Fig. 1. For both soils the pattern of release and sorption of P described above was obtained: where no P was added (or the amount added was low) there was more P in solution in the reduced suspensions, but at higher concentrations of added P the reduced suspensions removed much more P from the solution than did the oxidized suspensions. The capacity of the reduced Crowley soil to sorb large amounts of P was especially evident at the highest concentrations of added P, with only 45 μg/ml remaining in solution following addition of 220 μg/ml to the suspension.

The difference between reduced and oxidized soils in release and sorption of P suggests that under reducing conditions there is an increase of the solid material that reacts with P. The well-established role of hydrated iron oxide in at least partially governing P sorption and release in soils and sediments suggests that conversion of ferric oxyhydroxide to the more soluble and highly dispersed ferrous form increases the activity and the surface area of the iron compounds reactive with P. To determine whether reducing conditions increased the amount of reactive iron compounds in the soil, we extracted reduced and oxidized samples of the Commerce, Crowley, Moreland, and Sharkey soils with an oxalate solution which has been shown to attack the poorly crystallized iron oxides and hydrous oxides without having much effect on crystalline iron materials (*10*). Considerably more amorphous iron oxide was released by the oxalate under reducing conditions (Table 1), which indicates that there was more material that would react with P. The correlation coefficient between the amount of Fe extractable with oxalate for both aerobic and anaerobic samples and the amount of P sorbed from the solution with a P concentration of 100 μg/ml was + .929.

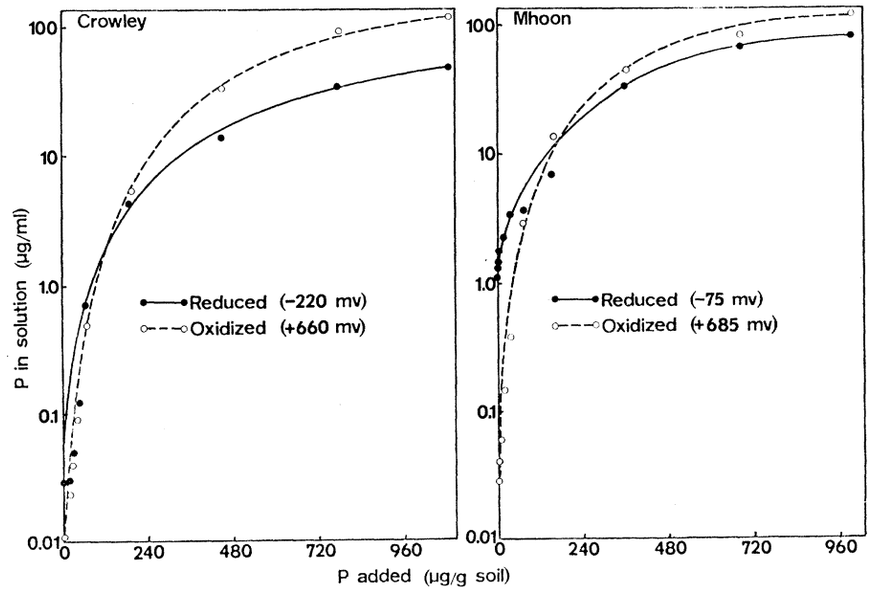

Fig. 1. Amount of P remaining in solution 24 hours after addition to reduced and oxidized soil-water suspensions of Crowley and Mhoon soils. Redox potential measurements showed oxidizing values of +660 and +685 mv for the oxidized soils and reducing values of −220 and −75 mv for the reduced soils.

The difference in sorption and release of P between oxidized and reduced soils and sediments is very likely due to the difference in the capacity of oxidized and reduced forms of iron oxide to sorb and release orthophosphate-P. Ferric oxyhydroxide is apparently capable of binding orthophosphate ions more firmly than the ferrous form, but probably has less surface area exposed to the solution P than the gel-like hydrated ferrous oxide or ferrous hydroxide.

Other factors, such as *p*H and the concentrations of Ca^{2+} and Mg^{2+}, are known to influence P exchange in aerated soil systems. Their effect in anaerobic soils and sediments is not precisely known, but it is likely that they have much less effect than the amount and oxidation state of the iron compounds. Immobilization of P is described here in terms of sorption involving iron oxides instead of precipitation of ferrous phosphate compounds such as vivianite. For most soils and sediments containing appreciable amounts of poorly crystallized hydrated iron oxide, the P distribution between solid and solution is very likely largely dependent on the nature of the iron compounds. Changes in the hydrated iron oxides as a result of oxidation or reduction reactions can be expected to change the concentration of dissolved orthophosphate.

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Serum Globulin in Myasthenia Gravis: Inhibition of α-Bungarotoxin Binding to Acetylcholine Receptors

Abstract. *Serum factors that inhibit the binding of ^{125}I-labeled α-bungarotoxin to the acetylcholine receptor extracted in detergent from denervated rat muscle were detected by a sensitive assay. The serum of at least 5 and possibly 11 out of 15 patients with myasthenia gravis showed inhibitory activity that was localized to the globulin fraction. No controls showed inhibitory activity. The demonstration of inhibitory globulins may help explain the involvement of the immune system in the pathophysiology of the neuromuscular junction in patients with myasthenia gravis.*

Myasthenia gravis is a neuromuscular disease of man characterized by muscle weakness which increases with exertion and improves with rest (*1*). Although numerous electrophysiologic studies have demonstrated involvement of the neuromuscular junction, it is still not clearly resolved whether the primary site of pathology is the nerve terminal or the muscle end plate (*1*). Involvement of the immune system in myasthenia gravis has been suggested by the marked frequency of thymic hyperplasia (*2*), the presence of antibodies directed against muscle structural proteins (*3*), the increased frequency of lymphocytes toxic to muscle tissue (*4*), and the beneficial effects of repeated lymphocyte drainage (*5*). None of these immunologic studies adequately explain the physiologic alterations of the neuromuscular junction, and attempts to demonstrate the presence of antibodies directed against the neuromuscular junction have been negative (*6*).

Two recent studies suggest physiological and immunological involvement of the muscle acetylcholine receptor in myasthenia gravis. Lindstrom and Patrick demonstrated that injection of purified torpedo acetylcholine receptor into rabbits results in a flaccid paralysis with electromyographic and pharmacologic similarity to myasthenia gravis (*7*). Fambrough, Drachman, and Satyamurti demonstrated that myasthenic muscle biopsies had only 11 to 30 percent the binding capacity of normal muscle for α-bungarotoxin, a specific inhibitor of the acetylcholine receptor (*8*). However, as pointed out by the authors, the reduction in the α-bungarotoxin binding and presumably in the number of available acetylcholine receptors may reflect a secondary effect of drugs or of the disease process.

A reasonable approach to both immunologic alterations and neuromuscular pathophysiology would be provided by the demonstration of circulating globulins that interact with the muscle acetylcholine receptor. We have devised a sensitive assay and have used it to demonstrate the presence in myasthenia gravis of circulating globulins with affinity for the muscle acetylcholine receptor. The basis of our assay is the interference of serum factors with the binding of ^{125}I-labeled α-bungarotoxin to the acetylcholine receptor (*9*).

Lyophilized crude venom of *Bungarus multicinctus* (Miami Serpentarium Laboratories) was fractionated by ion-exchange chromatography on carboxymethyl-Sephadex G-25 (*10*). The isolated α-bungarotoxin was desalted on Sephadex G-50, labeled to high specific activity (10^3 c/mole) with ^{125}I (*11*), and separated from free ^{125}I by chromatography on Dowex 1 and Sephadex G-50. The resultant ^{125}I-labeled α-bungarotoxin was homogeneous by both gel permeation chromatography and sodium dodecyl sulfate–polyacrylamide gel electrophoresis. When the labeled α-bungarotoxin (100 n*M*) was applied to a frog sartorius neuromuscular preparation, we noted a rapid and effective elimination of miniature end plate potentials. After both nerve stimulation and the iontophoretic application of acetylcholine, end plate potentials were reduced. The muscle fiber was still excitable by direct stimulation.

The muscle preparation was derived from adult female rats (150 to 200 g) that were unilaterally denervated by removal of a segment of the sciatic nerve 10 days before the animals were killed (*12*). The preparation contained approximately 0.6 to 1.0 pmole of α-bungarotoxin binding units in each 0.1 ml. Serum was fractionated into various globulin subfractions by sodium sulfate precipitation (*11*).

The acetylcholine receptor extracted by Triton X-100 from denervated