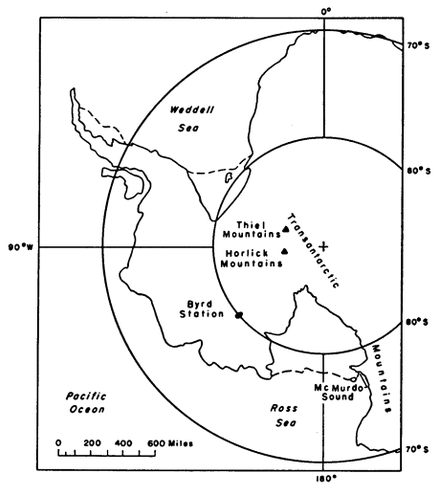

Fig. 1. The Thiel Mountains.

mass has a minimum dimension of 20 miles north-south and 15 miles east-west. On fresh surfaces the rock appears almost even grained, but where weathered the alteration products on feldspars make the strongly porphyritic, nonseriate texture evident. Large and strikingly euhedral insets of purple cordierite are conspicuous at many places. Pleochroic halos around zircons included within cordierite are common in thin sections. Strongly pleochroic hypersthene, generally rimmed by alteration products of talc, chlorite, and green and brown biotite, is the chief mafic constituent. Only rarely does primary biotite occur. At several localities a vague to distinct light and dark layering is present and at two places such layering defines large, open folds. Preliminary petrographic studies show that the layered rocks are mineralogically the same as the more massive rocks. The darker layers, however, have a considerably higher ratio of matrix to phenocrysts than the lighter layers.

A stock of medium-grained biotite granite, intrusive into the quartz monzonite porphyry, is well exposed on a large nunatak on the ice escarpment. Near the contact the quartz and potassium feldspar of the porphyry have been thoroughly recrystallized and have a granoblastic texture. Anhedral hypersthene and poikiloblastic biotite are the chief mafic minerals of the recrystallized porphyry. Very similar granoblastic rocks make up a small nunatak about 20 miles northeast of the massif. Owing to the brownish color of their feldspars and their granoblastic textures, hand specimens of the recrystallized rocks appear to be very similar to charnockites from near Mawson station on the East Antarctic coast.

The quartz monzonite porphyry has also been intruded by a large body of very coarse-grained porphyritic biotite granite. The intrusive relationship is demonstrated by a wide zone of contact breccia containing large blocks of quartz monzonite porphyry that have been contact metamorphosed. Dikes of porphyritic granite cut the quartz monzonite porphyry.

Porphyritic biotite granite forms massive, light-colored cliffs and nunataks in the northwesternmost part of the mountains. It is also exposed on a small nunatak nearly 40 miles away at the southern end of the mountains and therefore may underlie much of the area.

The geologic age of the crystalline rocks is unknown at present. The charnockitic affinities of some of the rocks, however, suggest a Precambrian age by analogy with charnockitic rocks of the East Antarctic shield. More exact dating will be provided by lead-alpha and isotopic studies of zircons in the hypersthene-quartz monzonite porphyry and by isotopic studies of mica and potassium feldspar in biotite granite.

Nearly flat-lying sedimentary and metasedimentary rocks and sill-like bodies of dacite are exposed in several of the peaks in the southeastern end of the mountains. The sediment-derived rocks are chiefly quartzites, argillites, and slaty argillites. Cross bedding is well developed in some quartzites, and the argillites commonly show ripple marks and mud cracks. Locally, near intrusive bodies, spotted hornfelses have formed. At several places these rocks have been involved in high-angle faulting. No recognizable fossils were found, but specimens containing features possibly of organic origin were collected and are awaiting further study.

Although detailed comparisons have not yet been made, these rocks appear to be lithologically different from the Paleozoic sedimentary rocks that lie on plutonic complexes in the Horlick Mountains and elsewhere in the Transantarctic Mountains. Diabase bedrock, accompanied by numerous erratics of white sandstone, is exposed in the southernmost nunatak; therefore Beacon-like rocks may lie beneath ice escarpments seen farther south (*5*).

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6 July 1962

Electron Spin Resonance of Nitric Oxide–Hemoglobin Complexes in Solution

Abstract. The electron spin resonance spectra of solutions of nitric oxide–hemoglobin and nitric oxide–methemoglobin, and whole blood treated at room temperature with nitric oxide, all exhibit resonance with a line width of 83 gauss, a g-value of 2.03, and a spin intensity corresponding to one unpaired electron spin per heme. The minimum detectable concentration of these nitric oxide complexes in solution is $10^{-5}M$. Solutions were stable in a nitrogen atmosphere but when exposed to air in the absence of nitric oxide the spin intensity decreased with a half-life of about 5 hours. A preliminary examination of blood of rats exposed 1 and 9 days to 10 ppm of nitric oxide in air showed no electron spin resonance.

Nitric oxide (NO) is the major component among the oxides of nitrogen in smog, comprising at least half of the total oxides of nitrogen and, at periods during the day, almost the entire amount (*1*). Under appropriate photochemical conditions, it is oxidized slowly to nitrogen dioxide (NO_2) (*2*), which is known to be toxic at relatively low concentrations (*3*), whereas NO injures biological material only in much higher concentrations under experimental conditions (*4*). However, the fact that NO can bind firmly with both hemoglobin (Hb) and methemoglobin (MHb) led us to investigate the possibility that it may enter the blood circulation through the lungs and combine with Hb. Gibson and Roughton (*5*) described the strong complexing of NO, relative to oxygen, with Hb in vitro and demonstrated the slow dissociation of nitric oxide–hemoglobin (HbNO).

The electron spin resonance (ESR) technique was employed to investigate the concentrations of HbNO and nitric oxide–methemoglobin (MHbNO) in

blood because both NO complexes possess one unpaired spin per heme (*6, 7*).

Nitric oxide–hemoglobin was prepared from a 15-percent solution of bovine Hb (*8*) in a solution of either 0.4-percent ammonia or 0.03*M* sodium phosphate buffer at *p*H 10. Five milliliters of the Hb solution were introduced into a 100-ml, three-neck flask, and the entire contents were agitated for 20 minutes while being flushed with prepurified N_2 (*9*). In several experiments carbon monoxide (CO) was substituted for N_2. While the agitation was continued, the flask was flushed with NO for 15 minutes at atmospheric pressure. After the reaction with NO the flask and contents were agitated and flushed out with N_2 for at least 3 minutes and for as long as 30 minutes. (The NO contained about 0.2 percent NO_2 and 0.4 percent CO_2 and was purified by passage over a long column of solid KOH.)

The solution was then introduced, under air-free conditions, into a quartz capillary (0.5 mm inside diameter) which was mounted vertically in the resonance cavity of an ESR spectrometer (Varian V-4500 X band) with a field modulation of 100 kcy/sec, a multipurpose cavity whose Q is approximately 7000, and a modulation amplitude of about 7 gauss. The spin intensities of samples were calculated by comparing first moments of the derivatives of their resonance with a sample of 0.1-percent pitch in KCl (*10*) (accuracy of about ± 20 percent) when the appropriate filling-factor correction is applied.

Methemoglobin was prepared by oxidizing Hb with excess potassium ferricyanide and removing ionic impurities by dialysis. The MHbNO solution was prepared by adjusting the dialyzed solution with NaOH to *p*H 9 and then treating the solution of MHb with NO as described above for HbNO. Citrated, whole human blood, used without further dilution, also was treated with NO according to the procedure described above.

Ultraviolet absorption spectra were obtained with a Cary spectrophotometer (model 14). The spectrum of the HbNO was in agreement with previously determined values (*11*). The MHb was determined to be at least 95 percent pure by application of Hüfner's ratio (*11*) to the absorption spectrum. The spectrum of MHbNO is quite similar to that of HbNO, except that (i) the extinction for HbNO is generally lower than that for MHbNO in the wavelength range of 2200 to 7800 A, (ii) the farthest red band has a peak at 5710 A for MHbNO and 5730 A for HbNO, and (iii) the extinction coefficients are equal for the bands peaked at 4180 A.

The ESR spectra of solutions of HbNO and MHbNO, and whole human blood treated with NO, exhibit a single resonance with a g-value of 2.03 (about 3300 gauss) and a line width of 83 gauss (between maxima of the differential curve). The spin intensities in these three solutions were equal and corresponded to one unpaired spin per heme. The resonances obtained by prior treatment of the solutions with either N_2 or CO were identical. No resonance was observed, up to fields of about 5000 gauss, for 15-percent solutions of Hb, HbO_2, or MHb; citrated whole blood; or water saturated with NO at 1 atm pressure and room temperature.

The reaction between NO and Hb (or MHb) appears to go to completion within a minute, as judged by the appearance of the bright red color. No reduction in spin intensity was observed during 18 hours for solutions of either HbNO or MHbNO exposed while being shaken in an atmosphere of flowing nitrogen (50 cm^3/min). However, the spin intensity of the resonance slowly decreased when HbNO or MHbNO solutions, flushed extensively with nitrogen to remove dissolved NO, were exposed to air. For example, the spin intensity of such a MHbNO solution remained unchanged for 15 minutes, decreased about 25 percent after 2 hours, and 75 percent after 12 hours. Nitrogen dioxide in small amounts did not affect the spin intensity of buffered HbNO or MHbNO solutions, but when NO_2 made the solution acidic, the intensity decreased and ultimately went to zero; irreversible precipitation and slight release of gas also ensued.

With present ESR equipment, the minimum detectable concentration of NO complex corresponds to about $10^{-5}M$ HbNO or MHbNO, or in whole blood to the detection of 0.1 percent of the Hb as HbNO.

Rats were exposed in a chamber to a dynamic flow of air containing approximately 10 ppm of NO. Blood samples were drawn after 24 hours and after 9 days. Heparinized cardiac blood was introduced immediately into the quartz capillary. Electron spin resonance was not observed either in the blood of these animals or in that of rats unexposed to NO; neither did subsequent exposure to a high concentration of NO that oxidized to a toxic level of NO_2 give detectable ESR in blood.

Results show that ESR's of solutions having equal concentrations of HbNO and MHbNO are indistinguishable, and that the spin intensities correspond to one unpaired electron per heme. A resonance of similar width was reported for crystalline MHb exposed to gaseous NO (*7*). The spin intensity for HbNO in solution is in accordance with the expectation that the resonance is due to the unpaired electron from NO (*6*).

The electron spin of NO in the HbNO and MHbNO complexes in solution must interact strongly with the rest of the molecule. This conclusion is based partly on the essentially isotropic g-value observed for these solutions, compared to the much smaller value for gaseous NO. Also, the short spin-spin and spin-lattice relaxation times of about 10^{-9} second, based on experimental measurements of line width and the radio-frequency power saturation dependence, imply a strong interaction between the electron spin of NO and its environment. The relaxation processes are probably due to spin-orbit coupling of the spin of NO with the hemoglobin molecule and also to thermal motion.

Because the line widths of the resonances of solutions of HbNO and MHbNO are indistinguishable it appears that the dominant relaxation process in each of the two complexes is similar. While it is not possible to distinguish by ESR between solutions of HbNO and MHbNO at room temperature, at low temperatures it may be possible to detect the resonance of Fe^{3+}, which in crystalline MHbNO is reported to be unaffected by NO (*7*).

It is interesting to note that the resonance becomes undetectable for both HbNO and MHbNO solutions when made acidic with NO_2. In these acidic solutions, the globin appears to have been altered, and loss of resonance is probably due to change in the nature of the binding of NO.

In view of the accessibility of Hb in circulating erythrocytes to CO through the respiratory tract (*12*), it would be expected that NO also might gain access and accumulate as HbNO. Assuming complete absorption and transport of inhaled NO, a sufficient volume of air containing 10 ppm of

NO was respired by rats in these preliminary experiments to have achieved detectable levels of HbNO by ESR measurement. On this basis, CO and NO appear not to act similarly in the biological milieu and NO may not achieve contact with blood. On the other hand, the observed decrease in spin intensity of both HbNO and MHbNO solutions in the presence of O_2 suggests slow oxidation of NO or HbNO, which process may also occur in vivo. Animal experiments are being continued along these lines (*13*).

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Sterility in the Mexican Fruit Fly Caused by Chemicals

Abstract. Chlorambucil, 4-{*p*-[bis(2-chloroethyl) amino] phenyl} butyrate, administered in food to the Mexican fruit fly *Anastrepha ludens* (Loew) inhibits the growth of testes and reduces egg hatch to almost nothing. Oviposition is not affected. The compound 4-amino-1H-pyrazolo (3,4-d) pyrimidine sulfate inhibits the growth of ovaries and reduces oviposition and fertility.

Certain chemicals cause sterility in *Drosophila melanogaster* (Meig.) (*1*) and the house fly (*Musca domestica* L.) (*2*). Knipling (*3*) has outlined the concepts of the sterile-male method of insect population control with chemical sterilants.

In Mexico and Hawaii an intensive search is under way to find chemicals capable of causing sterility in or otherwise adversely affecting the reproductive processes of tropical fruit flies. Such chemicals are needed for the laboratory production of sterile flies that can be used in research on the sterilization method of control or eradication.

Candidate chemosterilants (*4*) in acetone mixed with a food (*5*) consisting of granulated sugar and orange crystals, supplemented with protein hydrolyzate, were administered continuously to Mexican fruit flies [*Anastrepha ludens* (Loew)] beginning on the day of emergence. Temperatures ranged from 75° to 80°F and relative humidities from 30 to 60 percent. From 40 to 50 pairs of flies were used in each test.

Eggs were taken (*6*) a few days after sexual maturity and at intervals thereafter during a period of approximately 15 days. The eggs were incubated on pieces of moistened blotting paper. The larvae were reared in a medium consisting of ground carrots and yeast (*7*).

Consumption of Chlorambucil at concentrations of 0.03 and 0.1 percent by parent fruit flies allowed development of some progeny beyond the egg stage and the emergence of a few adults. Consumption at 0.3 percent yielded no adult progeny (Table 1). With 4-amino-1H-pyrazolo [3,4-d] pyrimidine sulfate at 0.3 percent the average oviposition rate was 0.6 egg per day, and some adult development occurred. When these two compounds were combined, each at a 0.15 percent concentration, they had the same effect as 0.3 percent Chlorambucil alone.

Mortalities of adults feeding for 32 days after emergence on the three treated diets approximated the mortality of the controls. Sexual vigor and behavior were normal. At the lowest concentration neither chemical appeared to affect adults of the F_1 generation.

In another test male flies held separately were fed 0.1 percent concentrations of Chlorambucil for the first 14 and 21 days and then mated with females that had eaten only normal foods. Both the fertility of eggs and adult emergence were exceedingly low. A 7-day feeding period by the males prior to mating was not enough to cause appreciable sterility. When females consumed this sterilant for a comparable period before mating with normal males, there was no effect on fertility.

When newly hatched Mexican fruit fly larvae were reared in carrot-yeast media containing 0.0125 to 0.15 percent Chlorambucil, they completed develop-

Table 1. Effect of chemosterilants on reproduction of laboratory populations of the Mexican fruit fly.

Dosage (percent)	Eggs		Percent	
	Total laid	Per fly on days taken	Eggs hatching	Adults developing
Chlorambucil				
0.03	1481	9.1	5.8	1.8
.1	1498	6.2	0.7	0.13
.3	1387	4.8	2.2	0
4-amino-1H-pyrazolo [3,4-d] pyrimidine sulfate				
0.03	1852	7.6	66.1	13.1
.1	1303	5.6	35.7	6.2
.3	172	0.6	8.7	1.7
Mixture equal parts both above materials				
0.03	2236	8.6	56.8	17.0
.1	2230	7.5	6.9	0.4
.3	1430	5.2	1.3	0
Untreated replicates				
1	2003	7.3	83.2	35.0
2	1894	6.8	79.9	34.7
3	1982	7.6	88.1	30.7

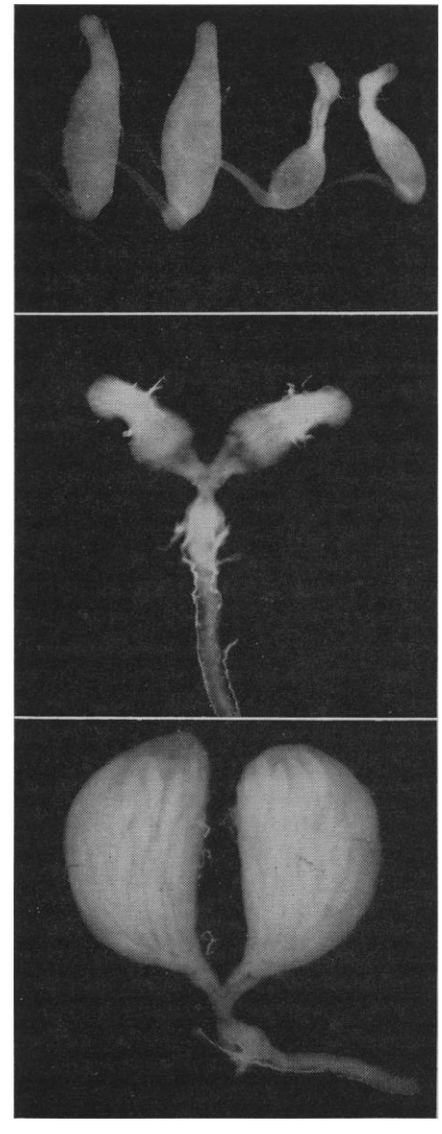

Fig. 1. Testes (top) of Mexican fruit fly, abnormal in treated flies on right and normal on left (× 20). Ovaries (middle) abnormal in treated flies, and (bottom) normal (× 10).