Table 1. Effect on the count rate of an "antistatic" agent applied to the lower surface of a glass planchet containing C^{14}.

Elapsed time (min)	Activity (count/min)	
	Unsprayed	Sprayed
1	59,112	58,774
2	58,346	59,418
3	57,257	59,565
4	55,906	59,389
5	54,864	59,344
6	54,834	59,077
7	53,833	58,963
8	53,812	58,993
9	53,433	59,529
10	53,162	58,914

with an antistatic agent (*7*). Similar results were obtained in the Geiger counter mentioned above.

At lower levels of radioactivity (500 count/min), the statistical fluctuations are too large for successive 1-minute counts to provide meaningful data regarding the possible existence of declining count rates. The average of count rates over 10 minutes, however, should be less for solutions on unsprayed than for sprayed planchets if the effect is common to all samples. To answer this question, the same solution used in the preparation of the planchet of Table 1 was diluted with 80 percent aqueous ethanol, plated on a glass planchet, counted in the proportional counter for 10 minutes, sprayed lightly on the lower surface, recounted for 10 minutes, and, finally, the lower surface was thoroughly wiped with a tissue to remove the spray, grounded to remove any electrostatic charge induced by the wiping, and again counted for 10 minutes. Count rates of 516, 541, and 514 count/min, respectively, were obtained. Since the planchet each time was placed in the same position in the chamber to eliminate geometrical effects, it is clear that the spray did affect the observed count rate at lower, as well as at higher, levels of radioactivity. Further tests in which calibrated $Na_2C^{14}O_3$ solutions (*8*) were used showed that with the sprayed planchets the count rates were consistently higher, and more accurate, than they were with the unsprayed planchets. That the effect of the spray persists for at least 6 months was shown by weekly counting on a sprayed planchet. Spraying the upper surface of the planchet either before or after plating the sample had no effect on the count rate decline.

With Br^{82} and Cs^{137}, nuclides emitting beta particles of considerably higher E_{max} than those of C^{14}, no such counting errors were encountered; indeed, the spray had no effect on the count rates of these nuclides when they were plated on glass, copper, or aluminum planchets. A slight but significant decrease in counting errors was noted when the spray was used for C^{14} plated on copper and aluminum planchets which had not been rigorously cleaned to remove grease prior to plating.

Not all of the phonograph record sprays tried effectively reduced the counting error. Two "antistatic" wiping cloths proved ineffective, and one of these was manufactured by the same company which produced an effective spray. Although there is considerable variation in the nature of the active ingredients (*9*), the composition of only one of the sprays tested was determined (*7*), and the active ingredient was found to be a quaternary ammonium compound.

The mechanism by which this compound decreases these specific counting errors is not clear. Whereas the upper surfaces of the glass planchets used were sand-blasted, the lower surfaces were smooth, and it is possible that the low energy beta particles from C^{14} may interact in some way with the nonmigrating negative ionic charge that was observed by Hubbard and Lucas on the smooth surfaces of silicate glasses (*10*). Such interactions could distort the electric field within the counter. In any event, the use of an "antistatic" agent such as we have described simplifies the counting of C^{14} on glass planchets. The technique may also prove useful for other nuclides, such as H^3, that emit beta particles of low E_{max} and for planchets of different materials.

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Poisoning by Organic Phosphorus Pesticides Potentiated by Phenothiazine Derivatives

Abstract. Repeated administration of the phenothiazine derivatives chlorpromazine and promazine increased the toxicity of a single dose of parathion in male rats. In female rats, a single dose of 5 mg/kg of promazine increased the toxicity of a single dose of parathion, but had no apparent effect on the toxicity of a single dose of Phosdrin. Repeated dosage with chlorpromazine and atropine, or promazine and atropine, after administration of parathion at the same rate, resulted in a slightly higher mortality in female rats than did atropine or no treatment.

In human patients and small animals phenothiazine tranquilizers (*1*) often increase the effectiveness of sedatives, analgesics, and narcotics (*2*). The possibility that promazine may potentiate poisoning in man by organic phosphorus pesticides was suggested by Arterberry *et al.* (*3*). On the other hand, Frada and Gucciardi (*4*) reported that treatment with chlorpromazine prolonged the survival time of guinea pigs poisoned with a mixture of malathion and parathion, but they did not indicate whether any of their treated animals survived. Other investigators have reported that a combination of atropine and chlorpromazine (*5*) or a variety of other phenothiazine derivatives (*5*, *6*) is more effective than atropine alone in preventing poisoning by sarin (*5*) or tabun (*6*).

Adult Sherman rats were used; the males weighed from 266 to 428 g and the females weighed from 175 to 300 g. Technical grade parathion, or 24.3 percent emulsifiable concentrate of Phosdrin, was diluted in peanut oil to the appropriate concentration and administered by stomach tube (5 ml/kg of body weight). Peanut oil alone was given to control rats which did not receive an organic phosphorus compound. Aqueous solutions of promazine and of chlorpromazine, 5 percent and 2.5 percent, respectively, were diluted with 0.9 percent sodium chloride solution to a concentration that permitted administration at a rate of 0.8 ml/kg of body weight. The tranquilizers were given by stomach tube or intraperitoneally. Sodium chloride was administered in like manner to control animals; atropine was given intraperitoneally. LD_{50} values were calculated by the method of Litchfield and Wilcoxon (*7*).

Groups of ten male rats were each given single oral doses of parathion. Each rat was then given 3.0 mg/kg

promazine 30 minutes after parathion, and every 6 hours thereafter until five doses had been given. To a second series of groups of ten rats, chlorpromazine was administered every 3 hours until five doses had been given. The rate of administration of these drugs corresponded to the maximum dosage given to people in cases of extreme anxiety. A third series of groups of ten rats (controls) was given a single dose of parathion followed by 0.9 percent sodium chloride solution given every 6 hours. The results are shown in Table 1.

The LD_{50} values indicated that the treatment with promazine increased the toxicity of parathion by a factor of about two and chlorpromazine increased it by a smaller factor. The LD_{50} value of parathion in the control rats corresponds closely with the value of 13.0 mg/kg for technical parathion which was reported earlier (*8*). Administration of these drugs to parathion-poisoned rats did not alter the time of onset of symptoms or the time of death.

Male rats which were given a single dose of peanut oil alone followed by dosage with promazine or chlorpromazine, as in the first and second series, exhibited marked depression but recovered overnight after treatment was discontinued (Table 1). Single doses of promazine as large as 160 mg/kg produced severe depression, from which all of the rats recovered completely.

A single 5 mg/kg dose of promazine potentiated the toxicity of 2.5 mg/kg of parathion which was given to groups of ten female rats within 30 minutes before to 1 hour after the administration of parathion. There was little effect when the promazine was given 4 hours after parathion.

Repeated dosage with promazine and atropine resulted in slightly higher mortality than atropine alone or no treatment following the same dose of parathion (Table 2). Tests with chlorpromazine gave similar results.

In all these tests, the rats that died exhibited typical symptoms of parathion poisoning.

Groups of ten female rats were each given a single oral dose of 5.0 mg/kg of promazine 30 minutes before, at the same time, and at two intervals after a single 3 mg/kg dose of Phosdrin. This dosage level of Phosdrin is slightly less than the oral LD_{50} value (3.7 mg/kg) for female rats (*8*). Promazine did not appear to have any effect upon the toxicity of Phosdrin. All of these rats exhibited typical symptoms of Phosdrin poisoning; either they died or showed a marked recovery within 2 to 3 hours. After this work was completed, we learned that Mitchell R. Zavon (*9*) had found that chlorpromazine, at dosages higher than those we had used for promazine, increased the mortality of rats previously dosed with Phosdrin (8 mg/kg). He noted the potentiation in each of three series of tests in which the rats were given repeated doses of atropine at the rate of 0, 1, and 2 mg/kg; when results of the atropine tests were combined, the mortality was 1, 7, and 15 out of groups of 15 rats which received repeated doses of chlorpromazine at the rate of 0, 5, and 10 mg/kg.

The results tentatively suggest that, in treatment of parathion or Phosdrin poisoning, tranquilizers derived from phenothiazine should either be avoided or at least used with extreme caution. We have not investigated the possibility that these drugs may increase the toxicity of other pesticides containing organic phosphorus.

Table 1. Effect of promazine (P) and chlorpromazine (CP) in male rats given repeated oral doses (3.0 mg/kg) of these compounds following a single oral dose of parathion. Promazine was given every 6 hours, chlorpromazine every 3 hours; controls were given 0.9 percent NaCl every 6 hours.

Parathion dose (mg/kg)	Mortality (No. dead/No. tested)		
	P	CP	Control
0.0	0/10	0/10	
3.0	0/10		
4.0	1/10		
5.0	7/10		0/10
6.0	5/10	2/10	
7.5		6/10	
8.0	9/10		0/10
9.0		8/10	
10.0	10/10		2/10
12.0		9/10	5/10
15.0			10/10
	LD_{50} (mg/kg)		
	5.4	7.3	12.0
	Confidence limits of LD_{50} (mg/kg)		
	4.7–6.3	5.7–9.4	10.5–13.7
	*Survival time (hr)**		
Range	1–95	2–69	1–69
Mean ± S.E.	15 ± 3.5	13 ± 2.6	14 ± 4.0

* For rats that died.

Table 2. Effect of promazine and atropine treatment in adult female rats given a single oral dose of parathion (3.6 mg/kg). Starting 30 minutes after parathion dosing, the atropine was given intraperitoneally every hour until ten doses were given and the promazine was given intraperitoneally every 3 hours until four doses were given.

Dose (mg/kg)		Onset of symptoms (hr)		Mortality (dead/tested)		Survival time (hr)	
Atropine	Promazine	Range	Mean	Test 1	Test 2	Range	Mean
0	3	1.50–2.25	1.17 ±0.115	4/4	4/4	1–22	11
2	3	1.50–8.25	3.09 ±0.79	2/4	4/4*	12–47	31
2	0	1.50–7.25	4.00 ±0.96	1/4	0/4	93	93
0	0	1.25–3.25	2.03 ±0.24	0/4	2/4	6–70	38
0	3†			0/4			

* Started treatment 30 minutes before parathion. † Peanut oil only in place of parathion.

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Potassium-Argon Dating of Plutonic Bodies in Palmer Peninsula and Southern Chile

Abstract. From the determination of the potassium-argon age of three plutonic bodies late Cretaceous to Cretaceous-Tertiary boundary ages have been calculated.

Potassium-argon determinations for age have been made on unweathered surface samples of three plutonic bodies found in the northern part of Palmer (Antarctic) Peninsula and southern Chile (Table 1).

Argon (Ar) and potassium (K)