Table 1. Comparison of simultaneous filter, gummed-paper, and screen collections.

Date of collection (1955)	Filter collector (disintegration/min)	Gummed paper (disintegration/min)	Stainless steel screen (disintegration/min)	Rainfall (in.)
21–28 Feb.	8,400	340	3,100	1.22 (snow)
28 Feb.–7 Mar.	2,500	200	2,100	1.78
7–14 Mar.	4,900	92	3,400	0.13
14–21 Mar.	5,200	5,100	1,700	1.13
21–28 Mar.	7,500	2,000	11,000	0.43
28 Mar.–4 Apr.	6,700	64	7,700	0
4–11 Apr.	8,000	320	3,800	0.07
11–18 Apr.	14,000	130,000	75,000	1.50
18–25 Apr.	8,900	2,300	1,500	0.47
25 Apr.–2 May	2,000	700	1,900	0.40
2–9 May	9,800	31,000	14,000	0.01
9–16 May	9,300	5,500	2,600	1.62
16–23 May	10,000	9,700	4,700	0.50
23–31 May	110,000	13,000	24,000*	1.11
Total	207,000	200,000	157,000	

* Cloth screen.

During the 2-month period of maximum fallout in the spring of 1955, daily samples of 1-ft² cloth screen gave a total fission-product collection of 2.8×10^5 beta disintegrations per minute as compared with 1.8×10^5 disintegrations per minute using the filter device and 1.9×10^5 disintegrations per minute on standard 1-ft² gummed papers.

In order to get some idea of the efficiency of the screen collector, a composite filter was made up of 7-in. squares of 40-mesh nickel screen on top of 100-mesh copper screen and backed by an efficient filter paper. Air was drawn through this filter at a face velocity of 3000 ft/min by a blower. A rough measure of the efficiencies of the screen filters was obtained from the relative amounts of the cerium-144 (praseodymium-144) isotope deposited on the different filter components. Particle retentions on the screens were compared by assuming that the filter paper was 100-percent efficient. The 40-mesh nickel screen at the top retained 11 percent of the total radioactivity, and the 100-mesh copper screen retained 18 percent, giving a total retention on both screens of 29 percent.

Direct impaction on small fibers is an effective mechanism for deposition of the radioactive particulate matter produced by atomic bombs. While even at highflow rates the collection efficiency of screens is comparatively low, their low airflow resistance and tendency to discriminate against the extremely small particles comprising the natural activities may be advantageous where simple detection of air-borne, fission-product radioactivity is the sole consideration.

Natural filters such as grass or trees may behave like many layers of filter fibers in removing activity carried by surface winds. In this case, the removal of particulates is fairly efficient and may account for a large fraction of the fission-product activity deposited on vegetation, particularly in the absence of precipitation.

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References and Notes

1. W. E. Ranz, *Univ. Illinois Eng. Exptl. Sta. Tech. Rept. No. 3* (1951); J. B. Wong, W. E. Ranz, H. F. Johnstone, *J. Appl. Phys.* 26, 244 (1955).
2. M. Eisenbud and J. H. Harley, *Science* 121, 677 (1955).
3. The cheesecloth used was found to contain a small amount of radioactivity, which could be removed by washing.

19 January 1956

Bonding in the Molecular Addition Complexes of the Alkyl Phosphates and Thiophosphates

In connection with a recent study involving the determination of the solubilities of certain inorganic metal nitrates in the trialkyl phosphates and thiophosphates (*1*), a great difference was observed in the solubilities of the salts in these two solvents. Since the dielectric constants for the two solvents are similar (*2*), the difference in solubility must be due to the difference in bonding between the metal salts and the organic molecules.

To elucidate the nature of this bonding, the solubility of uranium (VI) nitrate hexahydrate, thorium nitrate tetrahydrate, and copper (II) nitrate hexahydrate were determined in tri-*n*-butyl phosphate (TBP) and tri-*n*-butyl thiophosphate (TBPS) (*3*). These two compounds are alike in structure, except that the TBP has a semipolar P→O bond, while the TBPS has a semipolar P→S bond.

The procedure for the solubility determinations consisted of adding about 25 g of the solid salt to 20 ml of the pure solvent contained in a 50-ml bottle, sealing the bottle, and equilibrating the contents on a mechanical shaker for 48 hours at room temperature (25 to 27°C). It was found that this was sufficient time for equilibrium conditions to be established. At the end of this time, three phases were present in the bottle—a solid hydrated-salt phase, an aqueous phase containing a saturated solution of the metal salt, and an organic phase. The organic phase was separated, centrifuged, and analyzed for metal content.

The analysis consisted of weighing out 1- to 4-g duplicate samples of the centrifuged organic phase into separatory funnels containing 25 ml of benzene and 50 ml of water. After equilibration for 2 minutes, the aqueous phase was separated, 50 ml of water was added, and the equilibration was repeated. Two such extractions were sufficient to remove the metal salt from the organic phase. The metal content in the extracted aqueous phases was determined as follows: uranium by the 8-quinolinol method (*4*), thorium by the oxalate method (*5*), and copper by the cupferron method (*6*).

The solubilities of the metal nitrates are given in Table 1. In the TBP, it can be seen that uranium (VI) nitrate is the most soluble. Thorium nitrate is only slightly less soluble than the uranium salt, while copper (II) nitrate is about half as soluble as the other two. The striking observation is that the metal nitrates are only about one-twentieth as soluble in the TBPS as they are in the TBP.

The solubility of uranium (VI) nitrate in TBP has been attributed to the formation of the molecular addition complex, $[UO_2(TBP)_2(NO_3)_2]$ (*7*). The solubilities of the other metal nitrates can also be attributed to this effect but as yet have not been investigated.

The decreased solubilities of the metal

Table 1. Solubilities of the metal nitrates in tributyl phosphate and tributyl thiophosphate at room temperature (25–27°C). The solubilities are expressed in grams of anhydrous metal nitrate per 100 g of solution.

Metal nitrate	TBP	TBPS
$Th(NO_3)_4 \cdot 4H_2O$	42.6 ± 0.2 42.4	2.1 ± 0.2 1.8
$UO_2(NO_3)_2 \cdot 6H_2O$	43.6 ± 0.2 43.4	1.8 ± 0.2 2.3
$Cu(NO_3)_2 \cdot 6H_2O$	21.5 ± 0.1 21.3	0.62 ± 0.05 0.51

nitrate salts in TBPS, as compared with TBP, suggest that the organic molecules are bonded to the metal-nitrate salts in a different manner. Since the only differences in structure in the two organic molecules are the semipolar bonds, P→O and P→S, this would indicate that the interaction involves these atoms with the metal-nitrate salts to form a complex. If the metal-nitrate salt were bonded to the organic molecules through the alkyl oxygen atoms alone, one would not expect any difference in the solubilities of the salts in the two solvents. Exactly how many atoms of the organic molecules are involved in the bonding to the metal-nitrate salts is not known. It is possible that the bonding may involve the semipolar atoms in each of the organic molecules as well as the alkyl oxygen atoms.

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References and Notes

1. W. W. Wendlandt and J. M. Bryant, *J. Phys. Chem.* in press.
2. G. K. Estok and W. W. Wendlandt, *J. Am. Chem. Soc.* 77, 4767 (1955).
3. Kindly furnished by the Virginia-Carolina Chemical Corp., Richmond, Va.
4. A. Classen and J. Visser, *Rec. trav. chim.* 65, 211 (1946).
5. F. Hecht and H. Krafft-Ebing, *Mikrochemie* 15, 43 (1934).
6. I. Belluci and A. Chiucini, *Gazz. chim. ital.* 49, 187 (1919).
7. R. L. Moore, *U.S. Atomic Energy Commission Rept. AECD-3196* (1951).

9 January 1956

Serum Lactic Dehydrogenase Activity in Acute Transmural Myocardial Infarction

During experimental and clinical transmural myocardial infarction, glutamic oxaloacetic transaminase is released from cardiac muscle; this results in increased enzyme activity in the serum (*1*). This fact suggested that other enzymes in cardiac tissue might act similarly during myocardial infarction (*2*). Table 1 lists the activities of lactic dehydrogenase (LD) in dog tissue homogenates. Although the activity in the heart is less than the activity in the kidney, skeletal muscle, and liver, it is reasonable to expect LD to be released into the serum following damage to heart muscle.

The presence of LD in animal and in human-blood serum and whole-blood hemolyzates was previously demonstrated in our laboratory by spectrophotometric assay (*3*). The chemical characteristics of the enzyme in serum were studied and found to be similar to those reported for animal tissues. The normal range of activity in human serum and hemolyzates was established. The level was found to be elevated in certain disease states but notably in patients with acute and chronic leukemia, generalized carcinomatosis, and acute transmural myocardial infarction.

Table 1. Estimated lactic dehydrogenase activity of tissues of the dog.

Tissue	Activity (units/g of wet tissue)
Kidney	640,000
Skeletal muscle	600,000
Liver	390,000
Heart	240,000
Pancreas	150,000
Spleen	140,000
Brain	130,000
Lung	25,000

Serum lactic dehydrogenase was measured by adding serum to a substrate containing pyruvic acid, which oxidizes DPNH to DPN. The resulting change in optical density of the solution was measured in a Beckman DU spectrophotometer (3). The unit of serum LD is defined as the enzyme activity present in 1.0 ml of serum that causes an optical density decrease of 0.001 at a wavelength of 340 mμ in 1 minute under the conditions described. Determinations were made at 23°C. In 243 normal individuals, the range was between 200 and 680 units with a mean of 440 ± 120. The range of serum activity in the normal dog is comparable to the range of activity of human serum.

Serum lactic dehydrogenase was measured in the following: 243 normal individuals, 30 patients with cardiovascular disease that was uncomplicated by acute infarction, 35 with leukemia and malignant lymphoma, 10 with generalized carcinomatosis, 50 with localized carcinoma and other neoplastic disease, 30 with various infectious diseases, and 13 with acute transmural myocardial infarction. Venous blood was obtained for serum-LD determination regardless of the fasting state. The serum was separated from the clotted blood within a period of 2 to 24 hours after collection. It has been found that the activity is essentially unchanged if the separated serum is stored in a refrigerator from 1 to 3 days after collection. When possible, daily bleedings were made during a 5- to 10-day period.

Figure 1 summarizes the serum-LD activity on various days after infarction in 13 patients who had acute transmural infarction. Figure 2 shows the serum-LD activity during a 9-day period in a 58-year-old patient who incurred an acute, transmural, anterior-wall myocardial infarct. The LD activity was 1480 units within 48 hours and gradually fell to normal by the sixth day. The alterations in serum-LD activity in a dog, following closed-chest-wall experimental coronary-artery ligation that resulted in myocardial infarction, are comparable to the alterations seen in human infarction.

In 30 patients with heart disease, including arteriosclerotic heart disease that was associated with coronary insufficiency and/or acute and chronic congestive heart failure but that was not complicated by acute myocardial infarction, the serum-LD activity varied from 300 to 1020 units. In two of these patients, the serum-LD activity was above 680 units/ml. In one of these, chronic heart failure was present in a patient with hypertensive heart disease, auricular fibrillation, and polycystic kidney disease. The second patient had arteriosclerotic heart disease with heart failure.

All patients studied who had acute febrile and chronic infectious diseases had serum-LD activities within the normal range. Normal values were also encountered in anemia, pulmonary infarction, localized neoplastic disease, and chronic disease processes. High levels were encountered in patients with acute and chronic leukemia in relapse, generalized carcinomatosis, and, occasionally, acute hepatitis during its clinical peak, but not in patients with jaundice due to other causes. The serial LD-activity alterations that were noted following myocardial infarction have not been encountered in other clinical settings.

Our observations show that serum-LD activity rises within 24 hours in experimental and human myocardial infarction and returns to the normal range within 48 hours in dogs and within 5 to 6 days in human beings. The mechanism by which the level of enzyme activity is altered is under study but is pre-

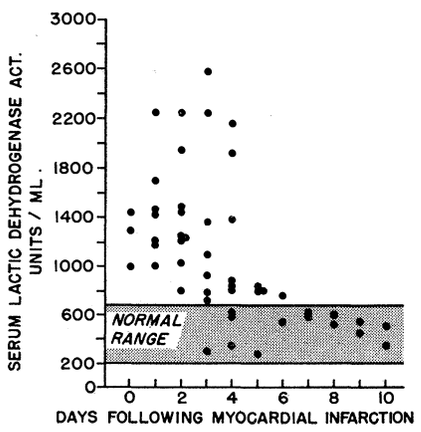

Fig. 1. Serum lactic dehydrogenase activity during the first 10 days following acute transmural myocardial infarction in 13 patients.