acid-precipitable radioactivity was also present only in the polyribosomes and at the top of the gradient, and there was a 20-percent reduction in incorporation of amino acids after a 90-minute exposure to actinomycin (5 μg/ml).

In cells infected with either bacteriophage or vaccinia virus, the formation of infectious virus requires the synthesis of several proteins (*11*). It has been suggested that the sequential synthesis of the different phage proteins may be controlled by the turnover of distinct "messenger RNA's" and the resulting degradation and reassembly of polyribosomes (*12*). A similar type of regulation may operate in vaccinia-infected cells. In these cells, a large fraction of the D-RNA is present in polyribosomes, and more than half of it is degraded to acid-soluble fragments when RNA renewal is blocked by actinomycin. The polyribosomes containing the D-RNA which is degraded in the presence of actinomycin presumably take part in the synthesis of virus structural proteins since the formation of immunologically identifiable viral proteins is interrupted by the presence of actinomycin. Those polyribosomes which remain after exposure to actinomycin are relatively inactive in protein synthesis. In addition to the D-RNA which associates with ribosomes to form polyribosomes, a portion of the D-RNA sediments in the 30 to 74*S* region of a density gradient. Its stability in the presence of actinomycin may be due to association with 45*S* ribosomal subunits or with protein, since its sedimentation coefficient is reduced to 16*S* after deproteinization with phenol or SDS treatment. The D-RNA not in polyribosomes does not direct viral protein synthesis. It may be a precursor of polyribosomes, or involved in the complex sequence of events associated with viral replication.

Table 2. Extent of D-RNA degradation. Infected cells received uridine-2-C^{14} as described (Fig. 1). At the indicated times after addition of 5 μg of actinomycin per milliliter, 5×10^7 cells were fractionated, and SDS (final concentration, 0.5 percent) was added to the cytoplasmic extracts, which were centrifuged (25,000 rev/min, 14½ hours, 15°C) in 5 to 30 percent linear gradients of sucrose dissolved in .01*M* acetate buffer, *p*H 5.1, containing 0.1*M* NaCl and 0.5 percent SDS. The values are the average of two experiments and were obtained by summing the acid-precipitable radioactivity in the 10 to 30*S* region.

Time after adding actinomycin (min)	Acid-precipitable D-RNA remaining	
	(count/min)	(%)
0	14400	100
30	9675	67
60	9625	67
120	8875	62

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11 February 1965

Bird Mortality after Spraying for Dutch Elm Disease with DDT

Abstract. *In Hanover, New Hampshire, where elms were sprayed with DDT, 151 dead birds were found; 10 dead birds were found in Norwich, Vermont, where no DDT was used. Chemical analyses of dead birds, observation of symptoms of DDT poisoning, and a population decline after spraying all indicate severe mortality among certain species in Hanover.*

In an attempt to control Dutch elm disease, the use of DDT (*1*) has become widespread in the United States. While its effectiveness in controlling the disease is uncertain and controversial (*2*), evidence of bird mortality following its use has become extensive (*3–10*). Because no studies have been conducted in the northeastern states (*4*), it has been presumed that abnormal mortality does not occur in this area (*5*); observation of dead birds following annual DDT spraying in Hanover, New Hampshire, however, suggested that this presumption was incorrect. We therefore undertook a study (*11*) to evaluate the effects of DDT on local birds, using the unsprayed town of Norwich, Vermont, 1 mile (1.6 km) west of Hanover, as a reference area.

About 2300 of Hanover's elms, occurring on 670 acres (271 hectares), were sprayed with 1285 lb (583 kg) of DDT by a Rotomist on 15–18 April 1963; dosage averaged 1.9 lb/acre (2.1 kg/hectare), but varied widely because of uneven elm distribution. Such a dormant (prefoliar) spray has been used in Hanover for about 15 years. DDT was replaced by Methoxychlor (*1*) in 1964 and was similarly applied.

From April through July of 1963 and 1964 we counted all birds identified by sight or sound (*12*) in ecologically comparable study areas of Hanover and Norwich. Concurrently, with the aid of residents, dead or dying birds were collected in both towns. Analyses for DDT, DDE, DDD (*1*), fat, and water (*13*) were performed on 106 birds (*14*), including those exhibiting tremors (*15*) prior to death, birds representing a variety of species and feeding habits, some showing injuries, and five reference robins, *Turdus migratorius*, from unsprayed areas. The brain, liver, breast muscle, heart, gonads, and remainder of 48 robins, and whole carcasses of 58 other birds of 25 species, were analyzed.

During 1963, 151 specimens (29 with tremors) of 34 species were found in Hanover; 10 dead birds (none with tremors) were found in Norwich. In 1964, 72 birds (6 with tremors) came from Hanover, while 8 (none with tremors) came from Norwich. Most dead birds, especially small, obscure ones such as sparrows or warblers, will not be found, so these data presumably represent a minor fraction of the total dead birds (*6, 16*).

The robin population in Hanover by 1 June 1963 had fallen 70 percent below the original 1 May population;

this decline was inverse to the rising number of dead robins found. Meanwhile the robin population in Norwich showed no net change. During June 1963, the population in Hanover increased through influx of new robins (*7, 17*), but did not return to the prenesting level. Robin population trends in Hanover and Norwich were similar to each other in 1964.

A comparable population divergence between Hanover and Norwich occurred during 1963 for bark feeders—that is, chickadees, nuthatches, creepers, and woodpeckers. The numbers of bark feeders in Hanover and Norwich study areas were about equal at the time of spraying. Within 3 to 4 weeks, Hanover areas contained one-third as many of these birds as did Norwich areas; dead and tremoring birds of some of these species were found in Hanover. No divergence of bark-feeder populations occurred in 1964.

Evidence indicates that the concentration of DDT residues in the brain is a good criterion for establishing cause of death (*8, 9, 18*). Any dead robin containing more than 50 parts per million in the brain probably died of DDT poisoning (*9*); prior observation of tremors in such a bird would leave little doubt that DDT was the cause of death.

All 16 robins found with tremors (1963), and 20 robins found dead, contained more than 50 ppm of DDT residues (DDT plus DDE plus DDD) in the brain, indicating these birds died of DDT poisoning. Four other robins found dead, and the five reference robins, contained 0 to 5 ppm and died of other causes.

When analyzing whole birds, we suspected DDT poisoning when the residue concentration was 30 ppm or more, since the minimum amount in a tremoring bird was 30.3 ppm (a robin with 70 ppm in the brain); this would assume a similar tolerance among the species represented. Among 99 Hanover birds analyzed, 65 most probably died of DDT poisoning, since all contained more than 30 ppm in the whole bird, and many were seen with tremors or contained more than 50 ppm in the brain. Some of the species represented (for example, myrtle warbler, *Dendroica coronata*, and tree swallow, *Iridoprocne bicolor*) arrived in Hanover several weeks after the time of spraying and feed primarily on living insects in treetops and in the air. We therefore suspect the toxicant was acquired by eating living insects carrying DDT, presenting a paradox of survival of the intended DDT victims and death, instead, of insectivorous birds. Thirty-three other birds, including all those showing injuries, and all Norwich birds, contained 0 to 10 ppm and presumably died of other causes.

Mortality was reduced after the application of Methoxychlor in 1964. Six robins were observed with tremors and others were found dead; these losses are believed caused by residual DDT in soil (*8, 10, 19*).

From the number of dead birds found, the many birds observed with tremors, chemical analyses of these birds, and a population decline among certain species, we conclude that DDT caused severe mortality of resident and migrant birds in Hanover during the spring of 1963.

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References and Notes

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14. Of this total, 103 were found in 1963; three robins were collected in 1964 for reference purposes.
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Lattice Formation in Complement Fixation: Studies with Univalent Rabbit Antibody

Abstract. *Hybrid univalent 6.5S antibody molecules, formed by recombination of half-molecules of rabbit antibody to ovalbumin with those of normal rabbit γ_G-globulin, fail to fix complement in reactions with homologous antigen. Such hybrid molecules, however, block complement fixation by intact antibody to ovalbumin. Molecules of antibody reconstituted in the absence of other protein retain the capacity to fix complement. The data suggest that small complexes containing excess univalent antibody do not fix complement and that lattice formation is required for fixation.*

After mild reduction at *p*H 5 and subsequent acidification to *p*H 2.5, rabbit γ_G-globulin (*1*) dissociates into half-molecules (*2*) consisting of one heavy and one light chain (*3*). On neutralization, such half-molecules recombine in pairs to form a product that has the same sedimentation coefficient, diffusion constant, and molecular weight as untreated γ_G-globulin (*2*,