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of Soviet Literature
on Toxicity and Hazards
of Chemicals

Dimethoate

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Dimethoate

Edited by N. F. Izmerov
Corresponding Member,
USSR Academy of Medical Sciences

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Compiled by professor Y.S. Kagan

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DIMETHOATE

Dimethoate (0,0-dimethyl-S(N-methylcarbomaylmethyl-dithiophosphate) is known in the Soviet Union under the name of "phosphamide". It belongs to the group of organophosphorus pesticides.

Molecular formula: $C_5H_2O_3NS_2P$

Structural formula:

$$(CH_3O)_2\overset{\overset{S}{\|}}{P}SCH_2CONHCH_3$$

Synonyms: rogor, asthoate, BI-58, heterotex, daphene, ditram, EF 590, nalphene, perfekthion, roxion, seniphore, systemine, toxion, phenthione, phosthione, phosphotox, ceryl, cygon

Melting point: 49.9-50.9°C

Density: 1.2770 (at 65°C)

Boiling point: 107°C (at 66.66 Pa) 86°C (at 1.33 Pa)

Water solubility: 39 g/1 [1].

In the USSR the preparation is manufactured as a 40% emulsion concentrate containing, in addition to active substance, an emulsifier (OP-7 or OP-10) and an organic solvent [2].

The commercial product contains not less than 94-96% of the active substance. Methylamine is present there as an impurity whose traces promote the decomposition of dimethoate [3].

PRODUCTION PROCESS (ES)

In the USSR dimethoate is obtained by the following two methods:

(1) by reaction between salts of dimethyl dithiophosphoric acid and N-methylchloracetamide.

$$(CH_3)_2\overset{\overset{S}{\|}}{P}SK + ClCH_2CONHCH_3 \rightarrow (CH_3O)_2\overset{\overset{S}{\|}}{P}SCH_2CONHCH_3 + KCl$$

The reaction proceeds in a medium of water and an organic solvent. The yield of the product is 80-95%.

(2) by reatcion between 0,0-dimethyl-S-(carboxymethyl) or S-(carbalkoxymethyl) dithiophosphate, at low temperature, and aqueous methylamine

$$(CH_3O)_2\overset{\overset{S}{\|}}{P}SCH_2COOC_2H_5 + CH_3NH_2 \longrightarrow (CH_3O)_2\overset{\overset{S}{\|}}{P}SCH_2CONHCH_3 +$$

$$+ C_6H_5OH$$

The yield is more than 90% of theory. In order to stabilize the preparation it should be purified from all impurities and, primarily, from excess methylamine [3].

USE

Dimethoate is a universal insectoacaricide. It is used to fight agricultural pests: European red, brown, fruit, citrus red and hawthorn mites; sugar-beet, apple, green, wooly pear, green peach, black cherry, melon, pea, hop and other aphid; sawfly, leaf roller, codling moth, cherry fruit fly, scale, tortoise scale and thrips. The consumption rate of the preparation ranges from 0.7 to 2 kg/ha. A 40% emulsion concentrate is diluted to 0.1-0.2%. Granulated 1.6% dimethoate is introduced into soil to control pests feeding on sugar beet (100 kg/ha), lucerne (introduced with seeds: 3 parts of the preparation per one part of seeds), and sweed and turnip (20-25 kg/ha) [4].

PATHWAYS INTO THE ENVIRONMENT

When dimethoate was used in gardens it was found in the air of the working zone of tractor drivers in concentrations of the order of tenths of mg/m^3 (average 0.6 mg/m^3); the air in the working zone of fillers contained dimethoate in quantities of 1 to 3 mg/m^3. In an hour, the concentration was 0.1 mg/m^3. After aerial treatment of cotton plantation, dimethoate was not already traced in the air in a few hours.

After a five-fold treatment with the 40% emulsion concentrate (2 kg/ha), the residual dimethoate in apples was 1 mg/kg in 24 hours, 0.6 mg/kg in 10 days, and 0.3 mg/kg in 30 days after the last spraying [5].

Dimethoate concentration of 0.03-0.07 mg/kg was found in apples in the course of 75 days [6], and 0.01 mg/kg, in the period of up to 95 days after treatment. Dimethoate residues only disappeared as late as 120 days after spraying [8].

The preparation was also absent from apples on the 37 day post-treatment. No dimethoate residues were traced in grapes on the 29th day after treatment with a 0,1 and 0,15% solution (700 liters per one ha). Tenth fractions of mg/kg were detected in grape leaves on the 35th day. No dimethoate residues

were observed in citrons fruit on the 24 day following treatment with a 0,2% dimethoate solution (1250 liters per one ha) [7].

In vegetables, (1515 samples), dimethoate was detected in 5.6%, and in fruits (802 samples), in 11.3% of samples. As a rule, dimethoate concentration in fruits and vegetables did not exceed the amounts allowable in the USSR. In some southeast regions of the USSR they reached 1.1 mg/kg and as much as 5 mg/kg in separate samples. Two months after treatment, dimethoate was found also in grapes (up to 1 mg/kg), peaches, apples, cherries, plums and dried appricots (up to 0.5 mg/kg). Dimethoate content of vegetables was higher (up to 1 mg/kg in tomatoes and eggplants) [8].

After intragastric administration of the 40% emulsion concentrate to sheep in doses from 37.5 to 250 mg/kg the dimethoate content of mutton was 0.01 to 0.1 mg/kg, then it dropped to 0.0001 mg/kg and persisted for 25 days. Heat treatment decreased the dimethoate content of mutton but did not eliminate it completely [9].

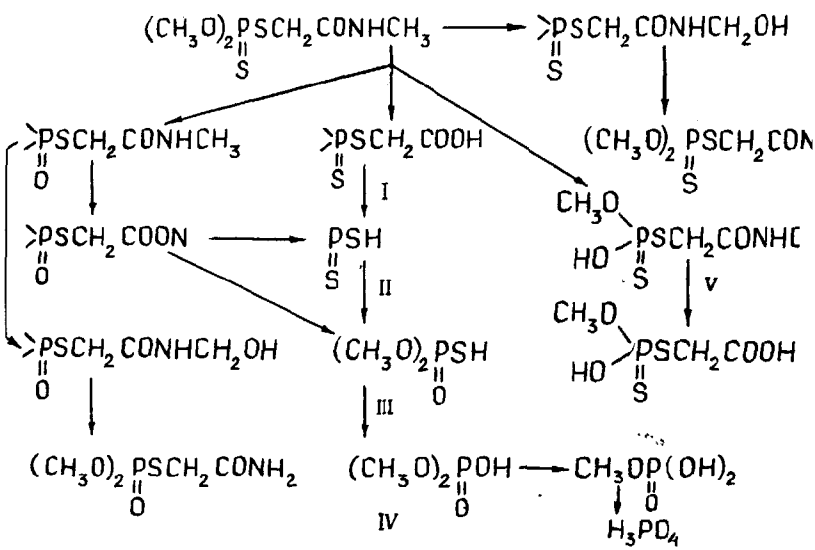
After aerial treatment of cotton plantation, the dimethoate concentration in soil was 0.37 mg/kg; on the 4th day it dropped to 0.1 mg/kg (5). According to the available data [10] with the dimethoate concentration in the soil at a level of 0.15-0.37 mg/kg, its concentration in the cotton ranged from 0 to 0.5 mg/kg.

Dimethoate, as a rule, does not persist in cotton for a long period.

Thus, dimethoate amounted to 0.5 mg/kg in the first two days and it was not traced on the 6th day [5].

ENVIRONMENTAL FATE TEST

Dimethoate transformation in the environment is as follows [12]:



Microorganisms destroy dimethoate in soil. A total of 77% of dimethoate are degraded in 14 days in common soil, 18%, in sterilized soil, and 20%, in gamma-irradiated soil [11].

Photochemical decomposition of dimethoate gives the products of hydrolysis and oxidation [12].

The time of hydrolysis of 50% dimethoate (at 70°C) is 19.9 hours at PH 3, 8.5 hours at pH 7, and 0.8 h at pH 9 [11].

Dimethoate vapour can get into air due to its high volatility [12].

BIOCONCENTRATION/CLEARANCE TIME/MAMMALIAN METABOLITES

Dimethoate is rapidly absorbed from the gastrointestinal tract of warm-blooded animals to inhibit the blood cholinesterase activity during the first 60 min [13]. The preparation is detected in blood 30-40 min after per os administration [14].

Dimethoate is rapidly hydrolized in the animal body to form products that are virtually montoxic. More than 90% of the preparation is excreted in urine during the first 48 h. Animals metabolize dimethoate in essentially the same manner as do plants. Carboxyamidase enzyme is involved in the hydrolysis of dimethoate. The liver is the main organ implicated in the dimethoate metabolism. Oxidative desulfuration of dimethoate occurs in the liver to produce a more toxic P-O analogue. Intensive detoxication of dimethoate takes place in the intestinal wall and blood [14, 15]. Tissues of typhoid fly (Musca domestica) also hydrolize dimethoate [15].

MAMMALIAN TOXICITY ARRAY

LD_{50} of chemically pure dimethoate administered to mice is 135 (152-112) mg/kg, and of commercial dimethoate, 125 (152-98) mg/kg. In albino rat experiments, LD_{50} - 230 (254-206) mg/kg for chemically pure dimethoate, and LD_{50} - 172 (205-130) mg/kg, for commercial dimethoate. In cat experiments, LD_{50} - 150 mg/kg for commercial dimethoate. In skin applications of commercial dimethoate, LD_{50} is 1120 (1460-750) mg/kg for rats, and 1500 mg/kg for rabbits.

Concentrations of dimethoate vapour 20-25 mg/m^3 do not kill rats or cats after a single four-hour exposure. Cats do not die after a single four-hour exposure to dimethoate aerosol in a concentration of 80 mg/m^3 [13].

Intoxication of laboratory animals with dimethoate is esentially the same as that caused by other organophosphorus pesticides and manifested by excitation of central and peripheral muscarine- and nicotine-sensitive choline-reactive systems. Low mobility, supersalivation and lacrimation, nasal discharge, dyspnea, tremor of the head and of the whole body, and fibrillar twitching of muscles of the back and extremities are observed 30-40 min after administration. Animals die from the respiratory standstill.

The cumulative effect of dimethoate in rats is slight. With daily dimethoate doses of 1/10 and 1/15 of LD_{100}, the cumulative coefficient is 9.3 and 6.3, respectivelly. The cumulative properties of dimethoate are more pronounced in cats which die after a cumulative dose of 1 to 6.8 of LD_{100}.

Repeated inhalation of dimethoate vapour in a concentration of 2.3 mg/m^3 elicits a 26% decrease in cholinesterase activity of rat blood serum by the end of the experiment (1.5 months). There was 40-70% decrease in cholinesterase activity of blood serum of cats exposed to dimethoate vapour in a concentration of 5 mg/m^3 and aerosol of 1.5 mg/m^3, as registered by the end of the exposure.

Inhibition of cholinesterase is the earliest sign of dimethoate intoxication. Visible intoxication symptoms develop when cholinesterase activity in blood diminishes by 60-70% [13]. In experiments in vitro, the ability of dimethoate to inhibit the cholinesterase activity in blood is slight. A dimethoate concentration of $1.3 \cdot 10^{-2}$M elicits a 50% inhibition of cholinesterase activity in horse blood serum. Dimethoate is subject to oxidative desulfuration in the animal body to aquire more pronounced cholinesterase properties.

Dimethoate changes the morphological composition of blood, i.e. increases the number of leukocytes and segmented neutrophils and decreases eosinophil and lymphocyte counts. The rise in the hemoglobin content and erythrocyte counts is a compensatory reaction for the developing hypoxia [13]. The protein fractions of blood are affected: the percentage of albumins decreases and that of gamma-globuline increases.

Pathomorphological analysis of internal organs of animals affected by dimethoate revealed stases, plasmorrhage, perivasculites and degenerative changes in the heart, liver, kidneys and the endocrine glands [16].

SPECIAL TOXICITY ARRAY

Carcinogenicity. The carcinogenic effect of dimethoate was shown on mice and Wistar rats. Dimethoate was doubly administered to the stomach (5, 15 and 30 mg/kg) and intravenously (15 mg/kg). The first experimental rat died on the 353d day after a cumulative dimethoate dose of 50 mg/kg. Diagnosis: malignant reticulosis. Four of 19 mice developed leukemia, and one of the animals developed cancer [17].

Mutagenicity. Data are available showing the mutagenic effect of dimethoate on bacteria and insects. A negative result was obtained with laboratory animals [18].

Neurotoxicity/Behaviour. The effect of dimethoate on the animals' nervous system.

Increase in the processes of active inhibition (differentiated and extinctive), lessening of conditioned reflexes and development of protective inhibition were demonstrated by the method of conditioned reflexes. Changes in conditioned reflexes are induced not only by toxic dimethoate doses but also by the doses which do not cause visible signs of intoxication but elicit a 40% (and over) inhibition of cholinesterase activity in erythrocytes and blood

serum [19].

The pathomorphological analysis of the brain of animals which died from dimethoate intoxication, showed plethora of the brain tunic and tissue, differently pronounced degenerative changes, in the cortex ganglionic elements, subcortex nuclei, and to a lesser extent in the cerebellum and medulla oblongata manifested as chromatolysis, caryolysis, caryocytolysis, corrugation and swelling [20].

Potentiation. Cholinolytic preparations (athropine, pentaphene, and amizyl) are the antagonists to dimethoate [21].

Reproduction. Dimethoate is not listed among pesticides having embryotoxic and gonadotoxic properties [22].

Primary irritation. Dimethoate has no irritant effect [13].

EFFECTS ON ORGANISMS IN THE ENVIRONMENT

Lethal concentrations of dimethoate (LC_{50}) are: 20000 mcg/l (24 h exposure) and 8500 mcg/l (96 h exposure) for trout, and 28000 mcg/l (24 h exposure), for carp [1]. Fishes exhibit a drastic depression, slow mobility and flaccid response to external stimuli. Toxic doses for carps in acute experiments with 24-, 48 and 96-hour exposures are 43,40 and 39 mg/l (LC_{50}), respectively. Cumulative properties are slight: dimethoate concentration of 2.1 mg/l (1/20 of LD_{50}), 4.3 mg/l (1/10 of LC_{50}) and 8.6 mg/l (1/5 of LC_{50}) do not kill fishes throughout the experimental period of 30 days [27].

With local affection, LD_{50} of dimethoate for thyphoid flies is 1 mg/kg [15].

SAMPLING/PREPARATION/ANALYSIS

Thin-layer chromatography was used to detect the spots of the preparation in a thin layer of alumina or silica gel treated with a mixture of bromphenol blue and silver nitrate [24]. The determination in air by gas chromatography was made after the absorption the air sample of dimethyl formamide, extraction from the latter by benzene and subsequent determination on a gas chromatograph with the electron capture detector [24]. The colorimetric method of the determination of dimethoate in air and blood is based on the ability of dimethoate to reduce the yellow phosphorus-molybdenum complex to blue phosphorus-molybdenum heteropolycomplex. Sensitivity : 10 mcg of dimethoate in a sample [25]. Nephelometric determination of dimethoate in aie is based on the reaction of dimethoate with Nessler's reagent and the formation of a yellow-brown insoluble compound. Sensitivity : 2.5 mg/kg dimethoate in a sample [26].

TREATMENT OF POISONING

High doses of cholinolytic preparations and cholinesterase reactivators

are used for curing dimethoate intoxication. Therapy of intoxication caused by organophosphorus compounds has been described in detail elsewhere [5, 17, 21, 27]. In experiments on laboratory animals, a benefitial effect was obtained by the use of cholinolytic prepatations, possessing central and peripheral m- and n- cholinolytic effects, atropine sulfate, pentaphene, amizyl and combinations of these preparations with arpenalmethyl sulfomethylate. The most effective are atropine and pentaphene administered in high doses, and also combinations of atropine and amizyl with arpenalmethyl sulfomethylate [27]. Vitamins B and K have a protective effect against dimethoate intoxication [28].

REMOVAL

Dimethoate is detoxicated with alkaline compounds. For this purpose, recommended are the preparation DIAS (a mixture of synthetic surfactants and organic solvents and alkali), 3-5% solutions of potassium hydroxide, soda ash or lime chloride (1 kg in 4 litres of water). The rate of the detoxication increases with heating [29].

The overalls polluted with organophosphorus compounds should be shaken and soaked in soap-and-soda solution for 6-8 hours. Then the overalls should be washed two or three times in a hot soap-and-soda solution and rinsed carefully.

Containers should be decontaminated with a 5% caustic or washing soda (300-500 g per 10 litres of water). The containers should be first filled with this solution, kept for 6-12 hours, then washed with ample water. If soda is not available, wood ash may be used instead [5].

Waste dimethoate and containers should be destroyed in accordance with the sanitary rules [29].

RECOMMENDATIONS/LEGAL MECHANISMS

Adolescents, pregnant and lactating women, men over 55 and women over 50 are not allowed to work with dimethoate. The personnel should undergo annual medical examination before the start of an agricultural season.

Prior to and during the work with dimethoate (once a week) the workers should be examined for cholinesterase activity of blood. Those who exhibit a 25% (and over) decrease in cholinesterase activity must be dismissed from work with dimethoate until the activity of the enzyme is normalized. Also, persons showing the very first signs of illness should be held away from work with dimethoate.

Daily exposure to dimethoate should not exceed 4 hours. During the rest of the workday the personnel should be engaged in some other activities.

The persons handling dimethoate should obligatory be instructed in safety measures. The workers must be provided with individual protective means: RU -60 and RPG -67 respirators with type A cartridges, PO- or PO -3

protective goggles, an overall made of dense or waterproof cloth, rubberized or PCV apron, rubber boots and gloves.

If the above-mentioned respirators are not available gas masks with appropriate cartridges supplemented with filters (white vertical line on the box) may be used.

In addition, signalmen are provided with PCV or oilskin hooded coats.

Dimethoate may be sprayed from airplanes equipped with boxes for insecticides firmly attached to the outer side of the fuselage. The pilot must carefully avoid settlements, water bodies, irrigation ditches and other objects (in accordance with the sanitary rules). For surface spraying, ventilation trailer installations can only be used which are pulled by tractors equipped with closed cabins.

Hand sprayers, knapsack devices, hose sprayers and nonmechanical means are not allowed for use. Particularly dangerous is work with insecticide concentrates which can cause intoxication through contact with the skin or when inhaled or ingested even in small amounts.

Chemical treatment is performed in the morning or at night under the guidance of plant protection experts and general supervision of a physician or an assistant who must pursue the control over the conformity to Sanitary and Safety Instructions [5].

Maximum allowable concentration (MAC) of dimethoate in air of the working zone is 0.5 mg/m^3, (vapour+aerosol) [6]. It belongs to danger class II substances.

MAC in the atmosphere of the populated areas: a maximum single–0.003 mg/m^3, average for 24 hours–0.003 mg/m^3 (danger class II) [5].

MAC in the water in the basins used for drinking and domestic water supply–0.03 mg/l (organoleptic criterion) [5]. Allowable residual concentrations of phosphamide in fruit, citrous plants, potatoes, vegetables and grain is 1 mg/kg (tolerable pesticide residues in food)[5].

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