antidotes. T2 was given in a dose of 0.15 mg/kg *sc* (0.1 LD_{50}), 5 times per week, 4 weeks to adult Wistar rats. Protected animals were given NIM (20 mg/kg *im*) or/and MIN+ (40 mg/kg *po*) each time immediately after T2. Mortality, general condition, body weight gain, food and water consumption and gut alterations of the animals were registered on a daily basis during 4 weeks.

Treatment with NIM or/and MIN+ significantly reduced mortality of the rats treated only with T2. Body weight gain, food and water consumption were significantly decreased in T2-treated animals compared to control ones ($p < 0.001$), what was not the case in the protected rats. In the groups treated with NIM and MIN+ gut alterations were significantly less severe than those observed in animals receiving T2 alone ($p < 0.001$).

These results imply that combinated treatment with nimesulide and zeolite absorbent affords a significant protection against subacute T-2 toxin poisoning in rats.

Key words: T-2 toxin, Nimesulide, Zeolite, Rats

61. ACUTE ORGANOPHOSPHATE POISONINGS: THERAPEUTIC DILEMMAS AND NEW POTENTIAL THERAPEUTIC AGENTS *(13)*

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It has been six decades since synthesis of organophosphates, but this chapter has not yet come to a closure. Toxic effects of organophosphates are well known and the current therapeutic scheme includes supportive therapy and antidotes. There is a dilemma on whether and when to apply gastric lavage and activated charcoal.

According to Position Statement (by EAPCCT) it should be applied only if the patient presents within one hour of ingestion, with potentially lethal ingested dose. Atropine, a competitive antagonist of acetylcholine at m-receptors, which antagonizes bronchosecretion and bronchoconstriction, is the corner stone of acute organophosphate poisoning therapy. There were many attempts to find a more efficient drug, including glycopyrrolate which has been used even in clinical trials, but it still can not replace atropine.

The only dilemma about atropine usage which still exists, concerns usage of high atropine dose and scheme of application. The most efficient atropinization is achieved with bolus doses of 1-2mg of atropine i.v push, with repeating the dose on each 5 minutes until signs of atropinization are registered. Diazepam, with its GABA stabilizing effect, reduces central nervous system damage and central respiratory weakness. Oximes reactivate phosphorilated acetylcholinesterase, which still has not gone ageing, reducing acetylcholine concentration and cholinergic crisis.

These effects are clearly demonstrated in experimental conditions, but the clinical significance of oximes is still unclear and there are still those who question oxime therapy. For those who approve it, oxime dosage, duration of therapy, the choice of oxime for certain OP is still an open issue.

We need new, more efficient antidotes, and those that are in use are only the small part of the therapy which could be used. Experimental studies show favorable therapeutic effect of many agents, but none of them has been introduced in standard treatment of OPI poisoning in the last 30 years.

New potential therapeutic agents for OPI poisoning include: glycopyrrolate as anticholinergic; organophosphorous hydrolases, butyrilcholinesterases and sodium bicarbonate which degrade OPI and accelerate AChE reactivation; reversible anticholinesterases for reduction of AChE reinchibition; NMDA antagonist as neuroprotectors.

Authors from Maryland have proposed the usage of IL-1 Rp antagonists in acute OPI intoxication, a new, original approach to therapy which deserves to be elucidated. For now pharmaceutical industries do not show satisfying initiative in developing new therapeutic agents and antidotes for OPI poisoning. However, randomized, controlled clinical studies, for the beginning with the agents which are in clinical practice, would elucidate their clinical efficacy, reduce the number of lethal pesticide poisonings in developing countries and provide information of special importance for the army and medical service.

Key words: organophosphate poisoning, therapeutic dilemma

62. RADIOLOGICAL DISPERSAL, POLONIUM-210, AND LESSONS FOR PUBLIC HEALTH *(4)*

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On November 23, 2006, Alexander Litvinenko died in London as a result of being poisoned with Polonium-210. Public health authorities in the United Kingdom (UK) subsequently found Polonium-210 contamination at a number of locations in and around London.

UK authorities have determined that citizens of 48 countries other than the UK, including the United States, may have been exposed to this contamination. UK authorities asked the CDC to contact approximately 160 individuals who may have been exposed to Po-210.

These citizens have been advised that their risk of adverse health effects is likely to be low, but, if they are concerned, they should contact their primary health care provider. In turn, physicians are referred to state and local public health departments or CDC for further information on Po-210, including where they can seek testing of 24 hour urine samples for Po-210, if desired.