

# *Current Awareness in Clinical Toxicology*

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## **CURRENT AWARENESS PAPERS OF THE MONTH**

### **Chlorine gas inhalation: human clinical evidence of toxicity and experience in animal models**

**White CW, Martin JG. Proc Am Thorac Soc 2010; 7: 257-63.**

#### ***Abstract***

Humans can come into contact with chlorine gas during short-term, high-level exposures due to traffic or rail accidents, spills, or other disasters. By contrast, workplace and public (swimming pools, etc.) exposures are more frequently long-term, low-level exposures, occasionally punctuated by unintentional transient increases.

Acute exposures can result in symptoms of acute airway obstruction including wheezing, cough, chest tightness, and/or dyspnea. These findings are fairly nonspecific, and might be present after exposures to a number of inhaled chemical irritants. Clinical signs, including hypoxemia, wheezes, rales, and/or abnormal chest radiographs may be present. More severely affected individuals may suffer acute lung injury (ALI) and/or acute respiratory distress syndrome (ARDS). Up to 1% of exposed individuals die.

Humidified oxygen and inhaled beta-adrenergic agents are appropriate therapies for victims with respiratory symptoms while assessments are underway. Inhaled bicarbonate and systemic or inhaled glucocorticoids also have been reported anecdotally to be beneficial. Chronic sequelae may include increased airways reactivity, which tends to diminish over time. Airways hyperreactivity may be more of a problem among those survivors that are older, have smoked, and/or have pre-existing chronic lung disease.

Individuals suffering from irritant-induced asthma (IIA) due to workplace exposures to chlorine also tend to have similar characteristics, such as airways hyperresponsiveness to methacholine, and to be older and to have smoked. Other workplace studies, however, have indicated that workers exposed to chlorine dioxide/sulfur dioxide have tended to have increased risk for chronic bronchitis and/or recurrent wheezing attacks (one or more episodes) but not asthma, while those exposed to ozone have a greater incidence of asthma. Specific biomarkers for acute and chronic exposures to chlorine gas are currently lacking.

Animal models for chlorine gas inhalation have demonstrated evidence of oxidative injury and inflammation. Early epithelial injury, airways hyperresponsiveness, and airway remodeling, likely

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diminishing over time, have been shown. As in humans, ALI/ARDS can occur, becoming more likely when the upper airways are bypassed. Inhalation models of chlorine toxicity provide unique opportunities for testing potential pharmacologic rescue agents.

## **Sensory detection and responses to toxic gases: mechanisms, health effects, and countermeasures**

**Bessac BF, Jordt S-E. Proc Am Thorac Soc 2010; 7: 269-77.**

### ***Abstract***

The inhalation of reactive gases and vapors can lead to severe damage of the airways and lung, compromising the function of the respiratory system. Exposures to oxidizing, electrophilic, acidic, or basic gases frequently occur in occupational and ambient environments. Corrosive gases and vapors such as chlorine, phosgene, and chloropicrin were used as warfare agents and in terrorist acts.

Chemical airway exposures are detected by the olfactory, gustatory, and nociceptive sensory systems that initiate protective physiological and behavioral responses. This review focuses on the role of airway nociceptive sensory neurons in chemical sensing and discusses the recent discovery of neuronal receptors for reactive chemicals.

Using physiological, imaging, and genetic approaches, Transient Receptor Potential (TRP) ion channels in sensory neurons were shown to respond to a wide range of noxious chemical stimuli, initiating pain, respiratory depression, cough, glandular secretions, and other protective responses. TRPA1, a TRP ion channel expressed in chemosensory C-fibers, is activated by almost all oxidizing and electrophilic chemicals, including chlorine, acrolein, tear gas agents, and methyl isocyanate, the highly noxious chemical released in the Bhopal disaster. Chemicals likely activate TRPA1 through covalent protein modification. Animal studies using TRPA1 antagonists or TRPA1-deficient mice confirmed the role of TRPA1 in chemically induced respiratory reflexes, pain, and inflammation in vivo.

New research shows that sensory neurons are not merely passive sensors of chemical exposures. Sensory channels such as TRPA1 are essential for maintenance of airway inflammation in asthma and may contribute to the progression of airway injury following high-level chemical exposures.

## **Amisulpride overdose is frequently associated with QT prolongation and torsades de pointes**

**Isbister GK, Balit CR, MacLeod D, Duffull SB. J Clin Psychopharmacol 2010; 30: 391-5.**

### ***Abstract***

This study aimed to describe the effects of the antipsychotic amisulpride in overdose, including the frequency of QT prolongation and torsades de pointes.

Cases of amisulpride overdose (>1 g) were recruited from 2 state poison centers and a tertiary toxicology unit over 5 years. A 1-page clinical research form was used to collect clinical information. Copies of all electrocardiograms were obtained. Electrocardiogram parameters (QRS and QT intervals) were manually measured as previously described, and plots of QT-heart rate (HR) pairs were compared with the QT nomogram.

There were 83 patients with amisulpride overdoses with a median age of 29 years (interquartile range [IQR], 23-40 years), and 42 (51%) were female. The median dose ingested was 6 g (IQR, 3-13 g, range, 1.2-120 g). The median HR was 66 beats/min (IQR, 60-81 beats/min). Bradycardia occurred in 20 cases (24%), and hypotension in 19 (23%). From 440 electrocardiograms (average of 5 per case; range, 1-15), an abnormal QT-HR pair occurred in 61 cases (73%). Torsades de pointes developed in 6 cases (7%), with doses of 4, 4.6, 18, 24, 32, and 80 g. The patient taking 32 g died after a cardiac arrest. Widened QRS did not occur except transient rate-dependent

bundle-branch block in 3 cases. There were significant associations of bradycardia, hypokalemia, and hypocalcaemia, with QT prolongation and torsades de pointes. Central nervous system effects were uncommon with coma in 7 cases, seizures in 2, and dystonic reactions in 2.

Amisulpride overdose commonly causes QT prolongation, bradycardia, and hypotension. Torsades de pointes occurred commonly enough to suggest that amisulpride is highly cardiotoxic in overdose.

## **L-carnitine for acute valproic acid overdose: a systematic review of published cases**

**Perrott J, Murphy NG, Zed PJ. *Ann Pharmacother* 2010; 44: 1287-93.**

### ***Objective***

To review the evidence supporting the efficacy and safety of L-carnitine in the management of acute valproic acid overdose.

### ***Data sources***

MEDLINE (1950-May 2010), EMBASE (1980-May 2010), and Google Scholar (to May 2010) were searched, using the terms carnitine, valproic acid, and carnitine for valproic acid overdose. Reference citations from identified publications were reviewed.

### ***Study selection and data extraction***

Full-text publications evaluating the use of L-carnitine for management of valproic acid overdose in humans were sought. All studies, regardless of design, case series, and case reports reporting efficacy or safety endpoints were included. All languages were included. Two authors extracted primary data elements including patient demographics, presenting features, clinical management, and outcomes.

### ***Data synthesis***

Seven articles discussing 8 patients and 1 reporting safety data from records of 674 patients were reviewed. Reports covered both pediatric and adult patients with acute exposures to valproic acid mono- and polydrug overdose who were treated with various regimens of L-carnitine. All patients recovered clinically and no adverse effects were noted.

### ***Conclusions***

Published evidence of the efficacy and safety of L-carnitine as an antidote for acute valproic acid overdose is limited. Based on the available evidence, it is reasonable to consider L-carnitine for patients with acute overdose of valproic acid who demonstrate decreased level of consciousness. We recommend intravenous administration of 100 mg/kg once, followed by infusions of 50 mg/kg (to a maximum of 3 g per dose) every 8 hours thereafter, continuing until ammonia levels are decreasing (if they were elevated initially) and the patient demonstrates signs of clinical improvement or until adverse events associated with L-carnitine occur.

## **The value of acute toxicity studies to support the clinical management of overdose and poisoning: a cross-discipline consensus**

**Chapman K, Creton S, Kupferschmidt H, Bond GR, Wilks MF, Robinson S. *Regul Toxicol Pharmacol* 2010; online early: doi: 10.1016/j.yrtph.2010.07.003: 1-26.**

### ***Abstract***

Acute toxicity studies are no longer required to support first clinical trials of pharmaceuticals in man. However, it is unclear in the wording of the revised ICH M3 whether acute toxicity studies are required later in drug development (e.g. phase 3) in order to support the management of overdose.

The NC3Rs held a workshop in January 2010 with representatives from international poison centres, the pharmaceutical and chemical industries, and regulatory and government bodies to explore further whether acute toxicity studies are used to support the clinical management of

overdose of pharmaceuticals and whether this work can be translated to other sectors such as the chemical industry. The consensus formed at the workshop was that acute toxicity studies are not used for managing overdose of pharmaceuticals and are of little value in treating human poisoning from chemicals.

In this paper, the authors describe the key considerations in treating human overdose and poisoning, challenge the value of the classification and labelling process of chemicals for this purpose and describe how acute toxicity studies can be improved to better inform risk assessment

## **Does amyl nitrite have a role in the management of pre-hospital mass casualty cyanide poisoning?**

**Lavon O, Bentur Y. Clin Toxicol 2010; online early: doi: 10.3109/15563650.2010.505573: 1-8.**

### ***Context***

Amyl nitrite has been recommended as a cyanide antidote for several decades. Its antidotal properties were initially attributed to induction of methemoglobin and later to a nitric oxide mediated hemodynamic effect. The ease of administration and alleged rapid clinical effect would recommend its wide use in the pre-hospital management of mass casualty cyanide poisoning; yet there are concerns regarding the use of amyl nitrite for this indication.

### ***Objective***

Review the data on amyl nitrite in cyanide poisoning and evaluate its efficacy and safety in mass casualty cyanide poisoning.

### ***Methods***

A literature search utilizing PubMed, Toxnet, textbooks in toxicology and pharmacology, and the bibliographies of the articles retrieved identified 17 experimental studies and human reports on the use of amyl nitrite in cyanide poisoning, and 40 additional articles on amyl nitrite's properties and adverse effects. One paper was excluded as it was a conference abstract with limited data.

### ***Mechanisms of action***

The antidotal properties of amyl nitrite were attributed initially to induction of methemoglobinemia and later to nitric oxide mediated vasodilation.

### ***Efficacy: experimental studies***

Animal studies on the use of amyl nitrite in cyanide poisoning are limited, and their results are inconsistent, which makes their extrapolation to humans questionable.

### ***Efficacy: human studies***

Clinical reports are limited in number and the part played by amyl nitrite relative to the other treatments administered (e.g. life support, sodium nitrite, and sodium thiosulfate) is unclear.

### ***Adverse effects***

Amyl nitrite can be associated with potentially serious adverse reactions such as hypotension, syncope, excessive methemoglobinemia, and hemolysis in G6PD deficient patients. These effects are more pronounced in young children, in the elderly, and in patients with cardiac and pulmonary disorders.

### ***Dose regimen***

The method of administration of amyl nitrite (breaking pearls into gauze or a handkerchief and applying it intermittently to the victim's nose and mouth for a few minutes) is not easily controlled, might result in under- or over-dosing, can prevent the caregiver from administering life support, and possibly expose him/her to amyl nitrite's adverse effects.

### ***Conclusions***

Administration of amyl nitrite in mass casualty cyanide poisoning can result in unnecessary morbidity and may interfere with the proper management of the incident and the required supportive treatment and rapid evacuation. In the authors' opinion these drawbacks make the use of amyl nitrite in pre-hospital mass casualty cyanide poisoning unwarranted.

## **Bladder cancer risk in painters: a meta-analysis**

**Guha N, Steenland NK, Merletti F, Altieri A, Cogliano V, Straif K. *Occup Environ Med* 2010; 67: 568-73.**

### ***Abstract***

The International Agency for Research on Cancer has classified occupational exposure as a painter as "carcinogenic to humans", largely based on increased risks of bladder and lung cancer.

A meta-analysis, including more than 2900 incident cases or deaths from bladder cancer among painters reported in 41 cohort (n = 2), record linkage (n = 9) and case-control (n = 30) studies, was conducted to quantitatively compare the results of the different study designs and the potential confounding effect of smoking as well as other occupational exposures.

The summary relative risk (meta-RR, random effects) for bladder cancer in painters was 1.25 (95% CI 1.16-1.34; 41 studies) overall and 1.28 (95% CI 1.15-1.43; 27 studies) when including only smoking adjusted risk estimates. The elevated risk persisted when restricted to studies that adjusted for other occupational exposures (meta-RR 1.27; 95% CI 0.99-1.63; 4 studies). The results remained robust when stratified by study design, gender and study location. Furthermore, exposure-response analyses suggested that the risk increased with duration of employment. There was no evidence of publication bias.

Taken together, these results support the conclusion that occupational exposures in painters are causally associated with the risk of bladder cancer.

## **Frequency and predictors of mass psychogenic illness**

**Page LA, Keshishian C, Leonardi G, Murray V, Rubin GJ, Wessely S. *Epidemiology* 2010; online early: PM:20592616:**

### ***Background***

Mass psychogenic illness refers to outbreaks of illness attributed to a toxic agent but for which no plausible organic cause is found. We determined the frequency and predictors of mass psychogenic illness within a sample of chemical incidents.

### ***Methods***

Information was collected on a random sample of 280 chemical incidents. We developed consensus operational criteria for mass psychogenic illness and estimated its frequency. We then assessed environmental, emergency, and health service indicators for their association with mass psychogenic illness.

### ***Results***

Nineteen "chemical incidents" were probable episodes of mass psychogenic illness. This represented 16% of incidents for which people reported symptoms and 7% of all incidents. Odor was a robust predictor of mass psychogenic illness. These illnesses were especially likely to occur in schools or healthcare facilities.

### ***Conclusions***

A substantial minority of chemical incidents may be mass psychogenic illness.

## **Increasing poisoning mortality rates in the United States, 1999-2006**

**Bohnert AS, Fudalej S, Ilgen MA. *Public Health Rep* 2010; 125: 542-7.**

### ***Objectives***

Poisoning mortality rates have increased dramatically in the United States since the 1970s. This trend has been mainly attributed to an increase in accidental medication overdose deaths. The aim of this study was to analyze recent trends in poisoning mortality among U.S. adults using the most recently available data, and to examine gender and age as risk factors.

### **Methods**

Data on injury-based mortality for the entire U.S. were obtained from the Web-based Injury Statistics Query and Reporting System (WISQARS) for 1999-2006. We analyzed poisoning mortality rates by age group, gender, and intent. We modeled time trends in poisoning mortality using Poisson regression.

### **Results**

Although intentional and undetermined poisoning mortality rates remained relatively stable, accidental poisoning mortality rates increased 108.5% between 1999 and 2006, and were significantly higher in each successive year (incidence rate ratio = 1.12 per year increase). Unintentional poisoning mortality rates were higher in men than in women; however, the increase in rate over time was higher in women than in men. The unintentional poisoning mortality rate was highest in individuals aged 40-49 years across all years studied, but we observed large increases in the rate for individuals aged 15-29 and 50-59 years during the study period.

### **Conclusions**

Despite recently raised awareness, rates of unintentional poisoning mortality in the US continued to rise in 2006. Men are at increased risk, but this disparity has decreased over time.

## **The relevance of the individual genetic background for the toxicokinetics of two significant neurodevelopmental toxicants: mercury and lead**

**Gundacker C, Gencik M, Hengstschläger M. *Mutat Res Rev Mutat Res* 2010; online early: doi:10.1016/j.mrrev.2010.06.003: 1-11.**

### **Abstract**

The heavy metals mercury and lead are well-known and significant developmental neurotoxicants. This review summarizes the genetic factors that modify their toxicokinetics.

Understanding toxicokinetics (uptake, biotransformation, distribution, and elimination processes) is a key precondition to understanding the individual health risks associated with exposure. We selected candidate susceptibility genes when evidence was available for (1) genes/proteins playing a significant role in mercury and lead toxicokinetics, (2) gene expression/protein activity being induced by these metals, and (3) mercury and lead toxicokinetics being affected by gene knockout/knockdown or (4) by functional gene polymorphisms.

The genetic background is far better known for mercury than for lead toxicokinetics. Involved are genes encoding L-type amino acid transporters, organic anion transporters, glutathione (GSH)-related enzymes, metallothioneins, and transporters of the ABC family. Certain gene variants can influence mercury toxicokinetics, potentially explaining part of the variable susceptibility to mercury toxicity. Delta-aminolevulinic acid dehydratase (ALAD), vitamin D receptor (VDR) and hemo-chromatosis (HFE) gene variants are the only well-established susceptibility markers of lead toxicity in humans.

Many gaps remain in our knowledge about the functional genomics of this issue. This calls for studies to detect functional gene polymorphisms related to mercury- and lead-associated disease phenotypes, to demonstrate the impact of functional polymorphisms and gene knockout/knockdown in relation to toxicity, to confirm the *in vivo* relevance of genetic variation, and to examine gene-gene interactions on the respective toxicokinetics. Another crucial aspect is knowledge on the maternal-fetal genetic background, which modulates fetal exposure to these neurotoxicants. To completely define the genetically susceptible risk groups, research is also needed on the genes/proteins involved in the toxicodynamics, i.e., in the mechanisms causing adverse effects in the brain. Studies relating the toxicogenetics to neurodevelopmental disorders are lacking (mercury) or very scarce (lead). Thus, the extent of variability in susceptibility to heavy metal-associated neurological outcomes is poorly characterized.

## **Adverse effects of methylmercury: environmental health research implications**

**Grandjean P, Satoh H, Murata K, Eto K. Environ Health Perspect 2010; online early: PM:20529764:**

### ***Objective***

The scientific discoveries of health risks resulting from methylmercury exposure began, in 1865 describing ataxia, dysarthria, constriction of visual fields, impaired hearing, and sensory disturbance as symptoms of fatal methylmercury poisoning. Our aim was to examine how knowledge and consensus on methylmercury toxicity has developed in order to identify problems of wider concern in research.

### ***Data sources and extraction***

We tracked key publications that reflected the new insights into human methylmercury toxicity. From this evidence, we identified possible caveats of potential significance for environmental health research in general.

### ***Synthesis***

At first, methylmercury research was impaired by inappropriate attention to narrow case definitions and uncertain chemical speciation. It also ignored the link from ecotoxicity to human toxicity. As a result, serious delays affected the recognition of methylmercury as a cause of serious human poisonings in Minamata, Japan. Developmental neurotoxicity was first reported in 1952, but despite accumulating evidence, the vulnerability of the developing nervous system was not taken into account in risk assessment internationally until approximately 50 years later. Imprecision in exposure assessment and other forms of uncertainty tended to cause an underestimation of methylmercury toxicity, but repeatedly led to calls for more research rather than prevention.

### ***Conclusions***

Coupled with legal and political rigidity that demanded convincing documentation before considering prevention and compensation, types of uncertainty that are common in environmental research delayed the scientific consensus and were used as an excuse for deferring corrective action. Symptoms of methylmercury toxicity, such as tunnel vision, forgetfulness and lack of coordination, therefore also seemed to affect the environmental health research and its interpretation.

## **Systematic differences between healthcare professionals and poison information staff in the severity scoring of pesticide exposures**

**Adams RD, Gibson AL, Good AM, Bateman DN. Clin Toxicol 2010; online early doi: 10.3109/15563650.2010.491484: 1-9.**

### ***Context***

Severity scores are used in triage and for data comparison in cases of poisoning. Exposure severity scores have not been generally validated and their utilization by healthcare staff other than specialists in poison information (SPIs) is untested.

### ***Objective***

To compare the poisoning severity grading allocated in pesticide exposure cases by healthcare professional enquirers and poison information staff.

### ***Methods***

Pesticide exposures reported to the UK National Poisons Information Service (NPIS) systems in a prospective study were graded for severity by healthcare professional enquirers and NPIS SPIs who used established poisons severity-grading algorithms. The scores were compared in children and adults, for the two professional groupings, both overall and for separate pesticides.

## **Results**

Overall SPIs graded severity resulting from pesticide exposure at a lower level than the enquirer. For children, enquirer mean severity score was 1.62 (95% confidence interval (CI) 1.57-1.66) and SPIs mean severity score was 1.16 (95% CI 1.13-1.19) ( $p < 0.001$ ). For adults, enquirer mean severity score was 1.91 (95% CI 1.84-1.97) and SPIs mean severity score was 1.74 (95% CI 1.69-1.79) ( $p < 0.001$ ). Importantly, the differences in the scores between the two professional groups were greater in children [+0.46 (95% CI 0.41-0.51)] than in adults [+0.17 (95% CI 0.11-0.24)] ( $p < 0.001$ ). Findings for individual pesticides were less consistent but in general showed similar trends. The exception was glyphosate for which severity grading by poison information staff was higher for children [SPIs 1.68 (95% CI 1.38-1.96)] than the enquirers 1.26 (95% CI 1.08-1.44),  $p < 0.02$ ].

## **Conclusions**

Our findings suggest inherent differences in the perception of pesticide toxicity between healthcare professionals and SPIs. There was also a difference in the scoring approach depending on the pesticide involved. Additional investigations are required to define the role and accuracy of severity scoring in different types of poisoning and the applicability to different types of severity assessors.

## **Kinetic analysis of oxime interactions with acetylcholinesterase as a basis for the evaluation of oxime efficacy in organophosphate poisoning**

**Worek F, Eyer P, Aurbek N, Thiermann H. *Curr Bioact Compd* 2010; 6: 16-22.**

### **Abstract**

In the past decades a vast number of oximes have been synthesised in order to identify effective compounds for the reactivation of organophosphorus compound (OP)-inhibited acetylcholinesterase (AChE).

Up to now, oxime efficacy has been tested primarily in animal experiments. However, the accretive evidence of substantial species differences regarding kinetic properties of human and animal AChE led to an increasing number of in vitro kinetic studies quantifying the reactivating potency of oximes. These data were shown to provide a basis for the selection of effective oximes and for defining adequate oxime doses in human OP poisoning.

This review will discuss experimental and theoretical models for the in vitro assessment of oxime efficacy and will give an overview of the present status in the evaluation of oximes as antidotes against OP poisoning.

## **Percutaneous exposure to the nerve agent VX: efficacy of combined atropine, obidoxime and diazepam treatment**

**Joosen MJA, Van der Schans MJ, van Helden HPM. *Chem Biol Interact* 2010; online early: PM:20599844:**

### **Abstract**

The nerve agent VX is most likely to enter the body via liquid contamination of the skin. After percutaneous exposure, the slow uptake into the blood, and its slow elimination result in toxic levels in plasma for a period of several hours. Consequently, this has implications for the development of toxic signs and for treatment onset.

In the present study, clinical signs, toxicokinetics and effects on respiration, electroencephalogram and heart rate were investigated in hairless guinea pigs after percutaneous exposure to 500  $\mu\text{g}/\text{kg}$  VX. We found that full inhibition of AChE and partial inhibition of BuChE in blood were accompanied by the onset of clinical signs, reflected by a decline in respiratory minute volume, bronchoconstriction and a decrease in heart rate. Furthermore, we investigated the therapeutic efficacy of a single dose of atropine, obidoxime and diazepam, administered at appearance of first clinical signs, versus that of repetitive dosing of these drugs on the reappearance of signs.

A single shot treatment extended the period to detrimental physiological decline and death for several hours, whereas repetitive administration remained effective as long as treatment was continued.

In conclusion, percutaneous VX poisoning showed to be effectively treatable when diagnosed on time and when continued over the entire period of time during which VX, in case of ineffective decontamination, penetrates the skin

## **Butyrylcholinesterase as a therapeutic drug for protection against percutaneous VX**

**Lenz DE, Clarkson ED, Schulz SM, Cerasoli DM. Chem Biol Interact 2010; 187: 249-52.**

### ***Abstract***

The administration of purified human plasma-derived butyrylcholinesterase (HuBuChE) as a pretreatment has been demonstrated to enhance survival and protect against decreased cognitive function after exposure to organophosphorus poisons (OPs).

Based on efficacy data obtained with guinea pigs and non-human primates and the lack of behavioral side effects, plasma-derived HuBuChE has been granted investigational new drug status by the US Food and Drug Administration. The recent availability of a recombinant form of HuBuChE (rHuBuChE) from the milk of transgenic goats has now allowed us to determine the pharmacokinetics of that material in guinea pigs and use it as a therapy following exposure to the VX.

The rHuBuChE was expressed as a dimer and following intramuscular (i.m.) administration had more a rapid adsorption and clearance profile in guinea pigs than the plasma-derived material. Based on those data, we administered rHuBuChE i.m. 1 h after a percutaneous exposure of guinea pigs to either 2 x LD<sub>50</sub>) or 5 x LD<sub>50</sub> of VX. Post-exposure therapy with rHuBuChE provided improved survival at both challenge levels, 90% and 33% respectively versus 20% or 0% respectively for animals that did not receive therapy.

These studies showed that BuChE can be efficacious as a therapy against percutaneous exposure to VX.

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### General

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