

Current Awareness in Clinical Toxicology

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CURRENT AWARENESS PAPER OF THE MONTH

Clinical policy: critical issues in the management of patients presenting to the emergency department with acetaminophen overdose

Wolf SJ, Heard K, Sloan EP, Jagoda AS. Ann Emerg Med 2007; 50: 292-313.

Abstract: This clinical policy focuses on critical issues concerning the management of patients presenting to the emergency department (ED) with acetaminophen overdose. The subcommittee reviewed the medical literature relevant to the questions posed. The critical questions are:

1. What are the indications for N-acetylcysteine (NAC) in the acetaminophen overdose patient with a known time of acute ingestion who can be risk stratified by the Rumack-Matthew nomogram?
2. What are the indications for NAC in the acetaminophen overdose patient who cannot be risk stratified by the Rumack-Matthew nomogram?

Recommendations are provided on the basis of the strength of evidence of the literature. Level A recommendations represent patient management principles that reflect a high degree of clinical certainty; Level B recommendations represent patient management principles that reflect moderate clinical certainty; and Level C recommendations represent other patient management strategies that are based on preliminary, inconclusive, or conflicting evidence, or based on committee consensus. This guideline is intended for physicians working in EDs.

A prospective evaluation of shortened course oral N-acetylcysteine for the treatment of acute acetaminophen poisoning

Betten DP, Cantrell FL, Thomas SC, Williams SR, Clark RF. Ann Emerg Med 2007; 50: 272-9.

Study objective: Treatment with a shortened duration of oral N-acetylcysteine (20 to 48 hours) after acute acetaminophen poisoning is effective in the prevention of subsequent hepatic failure and death when administered to individuals meeting appropriate laboratory criteria.

Methods: Individuals with a potentially toxic acetaminophen ingestion according to serum acetaminophen levels were identified prospectively using a large statewide poison control system database throughout a 12-month period. N-acetylcysteine was administered for a minimum of 6 doses (20 hours), after which laboratory studies were obtained. Discontinuation of N-acetylcysteine was recommended by the poison center when 2 criteria were met: serum acetaminophen was undetectable (<10 microg/mL) and liver test results were normal (serum aminotransferase, international normalized ratio). A follow-up questionnaire was administered to

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individuals treated with N-acetylcysteine for 48 hours or less to ascertain the presence of symptoms consistent with progressive hepatotoxicity.

Results: Of 205 acutely poisoned individuals treated with N-acetylcysteine for 48 hours or less, 195 were successfully contacted after discharge, and 187 of 195 (95.9%) reported no symptoms consistent with hepatic failure. Eight individuals (4.1%) reported abdominal pain or vomiting; however, none received further N-acetylcysteine treatment or additional hospitalization.

Conclusion: A shortened duration of treatment with N-acetylcysteine (20 to 48 hours) may be an effective treatment option in individuals considered to be at no further risk of developing liver toxicity according to the fulfillment of appropriate laboratory criteria before N-acetylcysteine discontinuation.

Critical role of c-jun (NH2) terminal kinase in paracetamol-induced acute liver failure

Henderson NC, Pollock KJ, Frew J, Mackinnon AC, Flavell RA, Davis RJ, Sethi T, Simpson KJ. Gut 2007; 56: 982-90.

Background: Acute hepatic failure secondary to paracetamol poisoning is associated with high mortality. C-jun (NH2) terminal kinase (JNK) is a member of the mitogen-activated protein kinase family and is a key intracellular signalling molecule involved in controlling the fate of cells.

Aim: To examine the role of JNK in paracetamol-induced acute liver failure (ALF).

Methods: A previously developed mouse model of: paracetamol poisoning was used to examine the role of JNK in paracetamol-induced ALF.

Results: Paracetamol-induced hepatic JNK activation both in human and murine paracetamol hepatotoxicity and in our murine model preceded the onset of hepatocyte death. JNK inhibition in vivo (using two JNK inhibitors with different mechanisms of action) markedly reduced mortality in murine paracetamol hepatotoxicity, with a significant reduction in hepatic necrosis and apoptosis. In addition, delayed administration of the JNK inhibitor was more effective than N-acetylcysteine after paracetamol poisoning in mice. JNK inhibition was not protective in acute carbon tetrachloride-mediated or anti-Fas antibody-mediated hepatic injury, suggesting specificity for the role of JNK in paracetamol hepatotoxicity. Furthermore, disruption of the JNK1 or JNK2 genes did not protect against paracetamol-induced hepatic damage. Pharmacological JNK inhibition had no effect on paracetamol metabolism, but markedly inhibited hepatic tumour necrosis factor alpha (TNF alpha) production after paracetamol poisoning.

Conclusions: These data demonstrated a central role for JNK in the pathogenesis of paracetamol-induced liver failure, thereby identifying JNK as an important therapeutic target in the treatment of paracetamol hepatotoxicity.

Recovery from a psychotropic drug overdose tends to depend on the time from ingestion to arrival, the Glasgow Coma Scale, and a sign of circulatory insufficiency on arrival

Yanagawa Y, Sakamoto T, Okada Y. Am J Emerg Med 2007; 25: 757-61.

Abstract: The aim of this study was to investigate which factors on arrival correlate with the duration of unconsciousness induced by a psychotropic drug overdose. Patients were 175 consecutive intubated patients unconscious due to psychotropic drug overdose. They were divided into 2 groups, an "early" group in which the patients were extubated within 2 days from hospitalization, and a "delayed" group who were not extubated within 2 days. Glasgow Coma Scale (P = .001) scores in the early group were higher than those in the delayed group. The estimated time from ingestion to admission (P < .0001), creatine kinase level (P < .01), number of cases demonstrating shock (P < .05), shock index (P < .0001), and heart rate (P = .001) in the

early group were smaller than those in the delayed group. Two subjects in the delayed group died of pneumonia and pulmonary embolism. Delayed arrival from ingestion, a low level of unconsciousness, and a sign of circulatory insufficiency in a patient with a psychotropic drug overdose were risk factors of a delayed recovery and death.

Oral antidiabetic agents in pregnancy and lactation: a paradigm shift?

Feig DS, Briggs GG, Koren G. *Ann Pharmacother* 2007; 41: 1174-80.

Objective: To provide information on the use of oral antidiabetic agents in pregnancy and breast-feeding.

Data sources: Primary articles were identified by a MEDLINE search (1966 March 2007) using the MeSH headings: pregnancy in diabetics, pregnancy, polycystic ovary syndrome, hypoglycemic agents, glipizide, glyburide, metformin, rosiglitazone, pioglitazone, clinical trial, controlled clinical trial, multicenter study, randomized controlled trial, case-control studies, and cohort studies.

Study selection and data extraction: All studies using oral antidiabetic agents in pregnancy were evaluated and relevant data were included in the discussion.

Data synthesis: Studies of glyburide and glipizide have found little or no transfer of these drugs across the placenta, whereas metformin and rosiglitazone cross readily. Animal studies have found no evidence to suggest that glyburide, glipizide, metformin, or rosiglitazone are teratogenic. In gestational diabetes, glyburide was safe and efficacious; however, 16-19% of women failed to achieve optimal glucose control. No developmental toxicity in infants was observed when metformin was used before and throughout pregnancy in women with polycystic ovarian syndrome (PCOS). Some of the studies involving patients with type 2 diabetes had methodological problems. A randomized controlled trial using metformin for gestational diabetes in the third trimester is underway. The human information is inadequate to evaluate the risk of glipizide or the thiazolidinediones in pregnancy. In breast milk, 3 studies measured nonsignificant amounts of metformin and one study was unable to detect either glyburide or glipizide.

Conclusions : Neither glyburide nor metformin has caused developmental toxicity in humans. Glyburide has been used for the treatment of gestational diabetes, and metformin has been used in women with PCOS who eventually became pregnant. Additional trials are needed to better define the benefits and risks of oral antidiabetic agents in pregnancy. Metformin, glyburide, and glipizide appear to be compatible with breast-feeding.

Surveillance of prescription drug-related mortality using death certificate data

Wysowski DK. *Drug Saf* 2007; 30: 533-40.

Background: The prescription drugs or drug classes that are most frequently associated with death in the US might be identifiable from death certificate data.

Objective: To identify the drugs/drug classes associated with the greatest numbers of deaths in the US that might be considered as possible targets for prevention.

Study design: US vital statistics data were accessed in order to identify International Classification of Diseases (10th Revision) [ICD-10] codes indicating that prescription drugs had caused or contributed to death and diseases with significant drug-related mortality.

Main outcome measure: ICD-10 codes for primarily prescription drugs that were listed as the underlying cause or as 'total mentions' on death certificates and were implicated in ≥ 1000 deaths in any one year were selected. The annual number of deaths by ICD-10 code was obtained from the Division of Vital Statistics, National Center for Health Statistics. Codes for diseases with significant drug-related aetiologies and involvement in ≥ 1000 deaths in any one year were also identified and analysed separately.

Results: For the selected ICD-10 codes, a total of 25031 deaths were listed as having a

prescription drug as the underlying cause in 2003, compared with 16135 in 1999, a 55% increase. Total mentions of these codes increased from 46523 in 1999 to 72080 in 2003, also a 55% increase. Most codes involved 'poisonings' (overdose or the wrong substance given or taken in error that is accidental, intentional or with undetermined intent). Drugs associated with poisoning deaths had central nervous system effects. Among the codes associated with specified drug classes, poisonings and accidental poisonings involving narcotics, hallucinogens, psychoactive substances and opioids (other than opium and heroin) were associated with the largest numbers of deaths. Drug-related codes associated with the largest percentage increases in deaths between 1999 and 2003 included poisoning due to methadone (275%); poisoning by other and unspecified antidepressants (primarily selective serotonin reuptake inhibitors) [130%]; and poisoning by psychostimulants with potential for abuse (amfetamines and drugs for attention deficit hyperactivity disorder) [117%]. Anticoagulants were associated with the largest numbers of deaths with codes involving "adverse effects in therapeutic use". Among diseases with significant drug-related aetiologies, Clostridium difficile enterocolitis (associated primarily with antibacterials) had the largest percentage increase in total mentions, with a 203% rise between 1999 and 2003.

Conclusions: Deaths due to overdoses are the most prominent cause of drug-related mortality in death certificate data. Certain drugs and drug classes, especially the opioids (e.g. narcotics, methadone), psychoactive drugs (e.g. antidepressants, amfetamines), anticoagulants and antibacterials (which cause or contribute to C. difficile enterocolitis) are associated with large and increasing numbers of deaths and preventive strategies should be considered.

Diffusion-tensor MR imaging for evaluation of the efficacy of hyperbaric oxygen therapy in patients with delayed neuropsychiatric syndrome caused by carbon monoxide inhalation

Lo CP, Chen SY, Chou MC, Wang CY, Lee KW, Hsueh CJ, Chen CY, Huang KL, Huang GS. Eur J Neurol 2007; 14: 777-82.

Abstract: The purpose of this study is to assess the efficacy of hyperbaric oxygen therapy (HBOT) in patients with delayed neuropsychiatric syndrome (DNS) caused by carbon monoxide (CO) inhalation using diffusion tensor magnetic resonance (MR) imaging and neuropsychological test. Conventional and diffusion tensor brain MR imaging exams were performed in six patients with DNS immediately before and 3 months after the HBOT to obtain fractional anisotropy (FA) values. Six age- and sex-matched normal control subjects also received MR exams for comparison. Mini-Mental State Examination (MMSE) was also performed in patients immediately before and 3 months after the HBOT. A significantly higher mean FA value was found in control subjects as compared with the patients both before and 3 months after the HBOT ($P < 0.001$). The mean FA value 3 months after the HBOT was also significantly higher than that before the HBOT in the patient group ($P < 0.001$). All of the patients regained full scores in the MMSE 3 months after the HBOT. Diffusion tensor MR imaging can be a quantitative method for the assessment of the white matter change and monitor the treatment response in patients of CO-induced DNS with a good clinical correlation. HBO may be an effective therapy for DNS.

The management of severe toxic alcohol ingestions at a tertiary care center after the introduction of fomepizole

Green R. Am J Emerg Med 2007; 25: 799-803.

Abstract: Ethylene glycol and methanol ingestions are relatively uncommon but potentially lethal poisonings. Recent trials have demonstrated that fomepizole effectively blocks alcohol dehydrogenase (ADH) in toxic alcohol overdoses, and may eliminate the need for emergent hemodialysis and intensive care unit admission. However, controversy remains in the role of fomepizole in clinical

practice. The purpose of this study was to describe the presentation, management and clinical course of toxic alcohol ingestions at a tertiary care referral center after the introduction of fomepizole to hospital formulary. Data was collected on all patients treated for toxic alcohol ingestions for a 1-year period in a tertiary care referral center. Patients who received fomepizole or ethanol infusions, or who underwent hemodialysis were identified by ED, pharmacy, hemodialysis and ICU databases. The patients' medical records were reviewed, and data was recorded on a predetermined computerized data collection form. Overall, twenty (20) toxic ingestions (14 methanol; 6 ethylene glycol) were identified over the one year period. Fomepizole was used for ADH blockade in 12/20 cases; ETOH infusions in 15/20 cases (combined ETOH and fomepizole use in 7/20). The majority of toxic alcohol exposures were admitted to an intensive care unit (19/20) and received emergent hemodialysis (19/20). All patients were discharged from hospital alive. Patients with methanol and ethylene glycol ingestions who presented to our centers had significant toxicity and received both HD and ICU admission. Further research is required to determine if the method of ADH blockade affects the need for hemodialysis or ICU admission in toxic alcohol ingestions.

State-of-the-science review: does manganese exposure during welding pose a neurological risk?

Santamaria AB, Cushing CA, Antonini JM, Finley BL, Mowat FS. J Toxicol Environ Health B Crit Rev 2007; 10: 417-65.

Abstract: Recent studies report that exposure to manganese (Mn), an essential component of welding electrodes and some steels, results in neurotoxicity and/or Parkinson's disease (PD) in welders. This "state-of-the-science" review presents a critical analysis of the published studies that were conducted on a variety of Mn-exposed occupational cohorts during the last 100 yr, as well as the regulatory history of Mn and welding fumes. Welders often perform a variety of different tasks with varying degrees of duration and ventilation, and hence, to accurately assess Mn exposures that occurred in occupational settings, some specific information on the historical work patterns of welders is desirable. This review includes a discussion of the types of exposures that occur during the welding process – for which limited information relating airborne Mn levels with specific welding activities exists – and the human health studies evaluating neurological effects in welders and other Mn-exposed cohorts, including miners, millers, and battery workers. Findings and implications of studies specifically conducted to evaluate neurobehavioral effects and the prevalence of PD in welders are also discussed. Existing exposure data indicate that, in general, Mn exposures in welders are less than those associated with the reports of clinical neurotoxicity (e.g., "manganism") in miners and smelter workers. It was also found that although manganism was observed in highly exposed workers, the scant exposure-response data available for welders do not support a conclusion that welding is associated with clinical neurotoxicity. The available data might support the development of reasonable "worst-case" exposure estimates for most welding activities, and suggest that exposure simulation studies would significantly refine such estimates. Our review ends with a discussion of the data gaps and areas for future research.

Early thimerosal exposure and neuropsychological outcomes at 7 to 10 years

Thompson WW, Price C, Goodson B, Shay DK, Benson P, Hinrichsen VL, Lewis E, Eriksen E, Ray P, Marcy SM, Dunn J, Jackson LA, Lieu TA, Black S, Stewart G, Weintraub ES, Davis RL, DeStefano F, Vaccine Safety Datalink Team. N Engl J Med 2007; 357: 1281-92.

Background: It has been hypothesized that early exposure to thimerosal, a mercury-containing preservative used in vaccines and immune globulin preparations, is associated with neuropsychological deficits in children.

Methods: We enrolled 1047 children between the ages of 7 and 10 years and administered

standardized tests assessing 42 neuropsychological outcomes. (We did not assess autism-spectrum disorders.) Exposure to mercury from thimerosal was determined from computerized immunization records, medical records, personal immunization records, and parent interviews. Information on potential confounding factors was obtained from the interviews and medical charts. We assessed the association between current neuropsychological performance and exposure to mercury during the prenatal period, the neonatal period (birth to 28 days), and the first 7 months of life.

Results: Among the 42 neuropsychological outcomes, we detected only a few significant associations with exposure to mercury from thimerosal. The detected associations were small and almost equally divided between positive and negative effects. Higher prenatal mercury exposure was associated with better performance on one measure of language and poorer performance on one measure of attention and executive functioning. Increasing levels of mercury exposure from birth to 7 months were associated with better performance on one measure of fine motor coordination and on one measure of attention and executive functioning. Increasing mercury exposure from birth to 28 days was associated with poorer performance on one measure of speech articulation and better performance on one measure of fine motor coordination.

Conclusions: Our study does not support a causal association between early exposure to mercury from thimerosal-containing vaccines and immune globulins and deficits in neuropsychological functioning at the age of 7 to 10 years.

Association of arsenic exposure during pregnancy with fetal loss and infant death: a cohort study in Bangladesh

Rahman A, Vahter M, Ekstrom EC, Rahman M, Golam Mustafa AH, Wahed MA, Yunus M, Persson LA. *Am J Epidemiol* 2007; 165: 1389-96.

Abstract: The authors evaluated the effect of arsenic exposure on fetal and infant survival in a cohort of 29,134 pregnancies identified by the health and demographic surveillance system in Matlab, Bangladesh, in 1991-2000. Arsenic exposure, reflected by drinking water history and analysis of arsenic concentrations in tube-well water used by women during pregnancy, was assessed in a separate survey conducted in 2002-2003. Data on vital events, including pregnancy outcome and infant mortality, were collected by monthly surveillance at the household level. The risk of fetal loss and infant death in relation to arsenic exposure was estimated by a Cox proportional hazards model. Drinking tube-well water with more than 50 microg of arsenic per liter during pregnancy significantly increased the risks of fetal loss (relative risk = 1.14, 95% confidence interval: 1.04, 1.25) and infant death (relative risk = 1.17, 95% confidence interval: 1.03, 1.32). There was a significant dose response of arsenic exposure to risk of infant death ($p = 0.02$). Women of reproductive age should urgently be prioritized for mitigation activities where drinking water is contaminated by arsenic.

A simplified acute physiology score in the prediction of acute organophosphate poisoning outcome in an intensive care unit

Shadnia S, Darabi D, Pajoumand A, Salimi A, Abdollahi M. *Hum Exp Toxicol* 2007; 26: 623-7.

Abstract: Organophosphate poisoning (OPP) occurs frequently and accounts for a large number of intoxication cases treated in intensive care units (ICU). Poisoning by these agents is a serious public health problem. Among pesticides, OPs are the main cause of poisoning and death in Loghman-Hakim Poison Center of Tehran, Iran. The aim of this study was to determine the impact of the Simplified Acute Physiology Score (SAPS II) in the prediction of mortality in patients with acute OPP requiring admission to the ICU of Loghman-Hakim Hospital Poison Center over a period of 12 months. This study was a prospective, case-control of records of patients with acute OPP admitted

to the ICU between January 2006 and December 2006. The Demographic data were collected and SAPS II score was recorded. During the study period, 24 subjects were admitted to the ICU with acute OPP. All 24 patients (15 male) required endotracheal intubation and mechanical ventilation in addition to gastric decontamination and standard therapy with atropine and oximes and adequate hydration. Of these, 24 patients, eight (five male) died. SAPS II score was significantly higher in the non-survival group than the survival group. Mortality following acute OPP remains high despite adequate intensive care and specific therapy with atropine and oximes. One-third of the subjects needing intensive care die within the hospitalization period. SAPS II scores calculated within the first 24 hours were recognized as good prognostic indicator among patients with acute OPP that required ICU admission. It is concluded that SAPS II score above 11 within the first 24 hours is a predictor of poor outcome in patients with acute OPP requiring ICU admission.

Advances in the management of organophosphate poisoning

Peter JV, Moran JL, Graham PL. Expert Opin Pharmacother 2007; 8: 1451-64.

Abstract: Organophosphate (OP) poisoning is commonly encountered in agricultural communities. The mainstay of therapy in OP poisoning is the use of atropine. However, several other therapies have been evaluated. Although oxime has been the most studied antidote, results in humans have been disappointing and limited by the lack of well-designed, prospective, randomised controlled trials. The key factor in determining outcomes in OP poisoning appears to be the timing of antidote administration. Other adjuvants, such as magnesium, fresh frozen plasma and haemoperfusion appear promising, and need to be explored further. A multi-faceted approach may be the answer to improving outcomes in OP poisoning. This review evaluates the advances in OP management over the last 20 years.

Meta-analysis: pesticides and orofacial clefts

Romitti PA, Herring AM, Dennis LK, Wong-Gibbons DL. Cleft Palate Craniofac J 2007; 44: 358-65.

Objective: The risk of orofacial clefts associated with pesticide exposure was examined by conducting a meta-analysis of studies published from 1966 through 2005.

Design: The full text of 230 studies was reviewed in detail, and of these, 19 studies were included in the final analysis. Fixed effects and random effects models were used to calculate pooled odds ratios (ORs) and 95% confidence intervals (CIs), and homogeneity among studies was evaluated.

Main outcome measures: Exposure- and phenotype-specific risks associated with pesticides.

Results: Many of the studies identified as suitable for analysis used a retrospective design with varying sample sizes, levels of exposure assessment, and phenotype evaluation. For all phenotypes combined, maternal occupational exposure was associated with an increased risk of clefting (OR = 1.37; CI = 1.04 to 1.81), whereas the estimates were somewhat weaker for paternal occupational exposures (OR = 1.16; CI = 0.94 to 1.44) or for any residential exposure (OR = 0.77; CI = 0.20 to 2.96). Calculation of pooled estimates for individual cleft phenotypes was mostly limited to studies of paternal occupational exposure; estimates exceeded unity but were not statistically significant.

Conclusions: The results of this meta-analysis suggest that maternal exposure to pesticides is associated with a modest but marginally significant risk of clefting. To better understand the relationship between pesticide exposure and orofacial clefts, future studies should consider evaluation of multiple routes of parental exposure, etiologically homogenous phenotypes, and individual genetic susceptibility.

Acute symptoms following work with pesticides

Solomon C, Poole J, Palmer KT, Peveler R, Coggon D. Occup Med (Oxf) 2007; 57: 505-11.

Background: Serious accidental poisoning by pesticides is rare in the UK, but more minor pesticide-related illness may be under-reported. Anecdotally, use of sheep dip has been linked

with flu-like symptoms.

Aim: To explore the frequency, nature and determinants of acute symptoms following work with pesticides.

Methods: A postal survey of men in three rural areas of England and Wales provided data on occupational use of five categories of pesticide, occurrence of 12 specified symptoms within 48 h of using pesticides and tendency to somatize. Risk factors for pesticide-related symptoms were assessed by modified Cox regression.

Results: Of 10 765 responders (response rate = 31%), 4108 had at some time used pesticides occupationally, including 935 (23%) who reported symptoms following such work on at least one occasion. In two areas, acute symptoms were most frequent following use of sheep dip (29 and 32% of users), but in the third area the rate was significantly lower (13% of users). The relative frequency of symptoms was similar for all five categories of pesticide, and flu-like symptoms did not cluster unusually among users of sheep dip. Risk of pesticide-related symptoms increased with somatizing tendency (prevalence ratio for highest versus lowest category 2.4, 95% confidence interval 2.03.0) and was higher in men who had used pesticides most often or handled concentrate.

Conclusion: Acute symptoms are common following work with pesticides, but in many cases the illness may arise through psychological rather than toxic mechanisms.

Cutaneous and ocular late complications of sulfur mustard in Iranian veterans

Shohrati M, Davoudi M, Ghanei M, Peyman M, Peyman A. *Cutan Ocul Toxicol* 2007; 26: 73-81.

Abstract: Although sulfur mustard (SM) has been used as a chemical warfare agent since the early twentieth century, it has reemerged in the past decade as a major threat around the world. This agent injured over 100,000 Iranians and one-third is suffering from late effects until today. Mustard affects many organs such as the skin, eyes, and lungs, as well as the gastrointestinal, endocrine, and hematopoietic system. In this study we focused on review of the late Cutaneous and ocular complications caused by exposure to SM. All studies regarding long- term ocular and cutaneous effects, which have been done on Iranian population, were collected from domestic and international sources. Pruritus is the most common complain and a malignant change is the most important lesion, which has to be considered. Also this agent is causes of chronic and delayed destructive lesions in the ocular surface and cornea, leading to progressive visual deterioration and ocular irritation.

Our recent experiences with sarin poisoning cases in Japan and pesticide users with references to some selected chemicals

Yokoyama K. *Neurotoxicology* 2007; 28: 364-73.

Abstract: Attention has been paid to neurobehavioral effects of occupational and environmental exposures to chemicals such as pesticides, heavy metals and organic solvents. The area of research that includes neurobehavioral methods and effects in occupational and environmental health has been called "Occupational and Environmental Neurology and Behavioral Medicine." The methods, by which early changes in neurological, cognitive and behavioral function can be assessed, include neurobehavioral test battery, neurophysiological methods, questionnaires and structured interview, biochemical markers and imaging techniques. The author presents his observations of neurobehavioral and neurophysiological effects in Tokyo subway sarin poisoning cases as well as in pesticide users (tobacco farmers) in Malaysia in relation to Green Tobacco Sickness (GTS). In sarin cases, a variety effects were observed 6-8 months after exposure, suggesting delayed neurological effects. Studies on pesticide users revealed that organo-phosphorus and dithiocarbamate affected peripheral nerve conduction and postural balance;

subjective symptoms related to GTS were also observed, indicating the effects of nicotine absorbed from wet tobacco leaves. In addition, non-neurological effects of pesticides and other chemicals are presented, in relation to genetic polymorphism and oxidative stress.

Inhalation toxicology of ricin preparations: animal models, prophylactic and therapeutic approaches to protection

Griffiths GD, Phillips GJ, Holley J. *Inhal Toxicol* 2007; 19: 873-87.

Abstract: Ricin is a toxin and seed protein produced by the castor oil plant, *Ricinus communis*. The toxin is a dimeric protein consisting of an enzymic A chain and a B chain with lectin properties aiding the uptake of the whole molecule into cells. Ricin has been considered a possible military threat for several decades and is now also of some concern as a terrorist agent. The inhalation route is of primary concern in these situations, although previous attacks with ricin have used other approaches. Medical countermeasures against ricin are urgently required and the strategy adopted has been first to understand the nature of the problem, in this case the inhalation toxicology of ricin, followed by the preparation of vaccine antigens. Toxoided ricin and modified recombinant A chain components have been examined in terms of efficacy as potential vaccine candidates in protection of animal models against inhaled ricin, primarily in laboratories both in the United Kingdom and in the United States. One recombinant A chain vaccine has been taken through to clinical trials in the United States and should become commercially available in the next few years. Toxoided ricin has also been used as an antigen to prepare antitoxin antibodies for therapeutic treatment following poisoning. In this review, a synopsis of the inhalation toxicology of ricin and approaches to medical prophylaxis and therapy of poisoning is given, based on work conducted at our laboratory and at other research institutes.

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