



# AMERICAN ACADEMY OF CLINICAL TOXICOLOGY, INC.

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## HERBS & DIETARY SUPPLEMENTS SPECIAL INTEREST GROUP ABSTRACTING SERVICE

February 7, 2010

1. Rudolph T, Knudsen K. A case of fatal caffeine poisoning. *Acta Anaesthesiol Scand.* 2010.

Caffeine is a natural alkaloid methylxanthine that is found in various plants such as coffee or tea. Symptoms of a severe overdose may present with hypokalemia, hyponatremia, ventricular arrhythmias, hypertension followed by hypotension, respiratory failure, seizures, rhabdomyolysis, ventricular fibrillation and finally circulatory collapse. A 21-year-old woman called for the ambulance herself soon after the ingestion of about 10,000 mg of caffeine. At the arrival of the ambulance, the patient went into cardiac arrest almost immediately. After a total resuscitation period of 34 min including seven counter-shocks and 2 mg epinephrine, the patient was stable enough to be transferred to the hospital. The patient soon went into VF again and received two more counter-shocks and 1 mg epinephrine and finally an intravenous bolus dose of 300 mg amiodarone. The initial arterial blood gas showed pH at 6.47, lactate at 33 mmol/l and potassium level at 2.3 mmol/l. Unfortunately, no blood samples for caffeine analysis were taken. Three days after hospital admission, the patient developed myoclonus, which did not respond to medical treatment. Excessive intake of caffeine may produce arrhythmias and pronounced hypokalemia and ensuing ventricular fibrillation. In case of counter-shock-resistant VF, it can be necessary to give an early loading dose of amiodarone. Furthermore, it may be beneficial to replace the potassium as early as possible. Epinephrine and buffer solutions used during resuscitation may further decrease blood potassium levels and should be administered cautiously. Epinephrine can be replaced by other vasopressor drugs, such as vasopressin without effects on beta-receptors.
2. Thomas X, Troncy J. Arsenic: a beneficial therapeutic poison - a historical overview. *Adler Mus Bull.* 2009;35(1):3-13.

Arsenicals have been used since ancient Greek and Roman civilizations and in the Far East as part of traditional Chinese medicine. In Western countries, they became a therapeutic mainstay for various ailments and malignancies in the 19th and early 20th centuries. Fowler's potassium bicarbonate-based solution of arsenic trioxide (As<sub>2</sub>O<sub>3</sub>) solution was the main treatment of chronic myeloid leukaemia until the 1930s. After a decline in the use of arsenic during the mid-20th century, arsenic

trioxide was reintroduced as an anticancer agent after reports emerged from China of the success of an arsenic trioxide-containing herbal mixture for the treatment of acute promyelocytic leukaemia. Arsenic trioxide was first purified and used in controlled studies in China in the 1970s. Subsequently, randomised clinical trials performed in the United States led to FDA approval of arsenic trioxide in the treatment of patients with relapsed or refractory acute promyelocytic leukaemia.

3. Suau GM, Martinez KG. Risk of serotonin syndrome with complementary and alternative medicines: importance to child and adolescent psychiatry. *Bol Asoc Med P R*. 2009;101(1):47-50.

OBJECTIVES: Evaluate how child and adolescent psychiatrists rate themselves regarding their knowledge and clinical skills in assessing interactions between non-prescribed complementary and alternative medicines (CAM) and prescribed medications. METHODOLOGY: A brief questionnaire about the practice of asking patients about CAM use was given to child and adolescent psychiatrists. RESULTS: The questionnaire was completed by 20 child and adolescent psychiatrists. Only 35% of the sample stated that they always asked about CAM use although 55% stated that they aware of the importance of prescribed drug interactions with CAM. Of the sample, 90% stated that they could recognize serotonin syndrome, but only 65% answered correctly to the description of the syndrome. Given a list of possible CAM that could interact with prescribed drugs to produce serotonin syndrome, only 10% identified all the drugs correctly. CONCLUSION: CAM training should be included in training programs and in continued education curriculums for practicing child psychiatrists.

4. Aliyev F, Turkoglu C, Celiker C. Nodal rhythm and ventricular parasystole: an unusual electrocardiographic presentation of mad honey poisoning. *Clin Cardiol*. 2009;32(11):E52-4.

Mad honey poisoning syndrome has been reported in the Eastern Black Sea region and Southeastern regions of Turkey. Herein we report a case of 70-y-old man presented with syncope and severe hemodynamic instability following ingestion of one teaspoon of honey and his unusual electrocardiographic manifestations: nodal rhythm alternating with sinus bradycardia and intermittent ventricular parasystole. In this report, we also tried to explain the possible mechanism responsible for these electrocardiographic findings.

5. Wei X, Chen Z, Yang X, Wu T. Chinese herbal medicines for esophageal cancer. *Cochrane Database Syst Rev*. 2009(4):CD004520.

BACKGROUND: Esophageal cancer is the seventh leading cause of cancer death worldwide. Traditional Chinese herbal medicines are sometimes used as an adjunct to radiotherapy or chemotherapy for this type of cancer. OBJECTIVES: To assess the efficacy and possible adverse effects of the addition of Chinese herbal medicines to treatment with radiotherapy or chemotherapy for esophageal cancer. SEARCH STRATEGY: We searched the Cochrane Upper Gastrointestinal and Pancreatic Diseases Group Trials Register, The Cochrane Library, MEDLINE, EMBASE, AMED (Allied and Complementary Medicine Database), CBM (Chinese Biomedical Database), China National Knowledge Infrastructure, the Chinese Cochrane Centre Controlled Trials Register and CISCOR (The Research Council

for Complementary Medicine) (up to 10 July, 2008). Databases of ongoing trials, the Internet and reference lists were also searched. SELECTION CRITERIA: Randomised controlled trials comparing the use of radiotherapy or chemotherapy with and without the addition of Chinese herbal medicines. DATA COLLECTION AND ANALYSIS: At least two review authors extracted data and assessed trial quality. MAIN RESULTS: We identified 43 trials which claimed to use random allocation. The first authors of all the RCTs we initially identified were contacted by telephone and we discovered that the authors had misunderstood the randomisation procedure. Using this new information, we reassigned all the identified RCTs as non-randomised trials. Because we identified no authentic randomised controlled trials, we were unable to perform a meta-analysis. AUTHORS' CONCLUSIONS: We were unable to find any evidence from RCTs on the effectiveness of TCM in the treatment of esophageal cancer. New trials should be carried out and we recommend that they are large scaled, correctly randomised and that the assessors of the results are blinded to the conditions of allocation.

6. Zheng Y, Gu R, Wu T. Chinese medicinal herbs for measles. *Cochrane Database Syst Rev.* 20094):CD005531.  
BACKGROUND: Measles is an infectious disease caused by the Morbilli virus. Chinese physicians believe that medicinal herbs are effective in alleviating symptoms and preventing complications. Chinese herbal medicines are dispensed according to the particular symptoms. This is an update of a Cochrane review first published in 2006. OBJECTIVES: To assess the effectiveness and possible adverse effects of Chinese medicinal herbs in treating measles. SEARCH STRATEGY: We searched the Cochrane Central Register of Controlled Clinical Trials (CENTRAL) (The Cochrane Library 2009, issue 1) which contains the Cochrane Acute Respiratory Infection Group's Specialised Register; MEDLINE (1966 to March 2009); EMBASE (1980 to March 2009); the Chinese Biomedical Database (1976 to March 2009); VIP Information (1989 to March 2009); and China National Knowledge Infrastructure (CNKI) (1994 to March 2009). We searched the metaRegister of Controlled Trials for ongoing trials. SELECTION CRITERIA: Randomised controlled trials (RCTs) in which patients with measles without complications were treated with Chinese medicinal herbs. DATA COLLECTION AND ANALYSIS: Three review authors (YZ, RG, TW) independently assessed trial quality and extracted data. We telephone interviewed the study authors for missing information regarding participant allocation. Some trials allocated participants according to the sequence they were admitted to the trials, that is to say, by using a pseudo-random allocation method; none of the trials concealed the allocation or blinding method. MAIN RESULTS: We did not identify any suitable trials for inclusion. In this updated review we identified 61 trials which claimed to use random allocation. We contacted 29 trial authors by telephone and learned that the allocation methods used were not randomised. We excluded 34 studies because the patients experienced complications such as pneumonia. Both reasons excluded 10 studies. Another study was excluded because the trial author had not confirmed the diagnosis of measles. We were unable to contact the remaining seven trials' authors, so that they require further assessment and, meanwhile are allocated to the 'Studies awaiting classification' section. AUTHORS' CONCLUSIONS: There is no evidence from RCTs for or against Chinese medicinal herbs as a treatment for

measles. We hope high quality, robust RCTs in this field will be conducted in the future.

7. Teschke R, Wolff A. Kava hepatotoxicity: regulatory data selection and causality assessment. *Dig Liver Dis.* 2009;41(12):891-901.  
BACKGROUND: Kava hepatotoxicity in 20 patients from Germany has been debated worldwide following a regulatory ad hoc causality assessment and ban of kava, an anxiolytic herbal remedy obtained from the rhizome of *Piper methysticum* Forster. AIMS: We assessed causality with a quantitative structured causality analysis in all 20 cases of patients with liver disease, presented by the German regulatory agency that assumed a causal relationship with the use of kava extracts. METHODS: The quantitative scale of CIOMS (Council for International Organizations of Medical Sciences) in its updated form was employed for causality assessment and quality evaluation of the regulatory data presentation. RESULTS: The regulatory information is scattered and selective, and items essential for causality assessment, such as exclusion of kava independent causes, were not, or only marginally, considered by the regulator. Quantitative causality assessment for kava was possible (n=2), unlikely (n=12), or excluded (n=6), showing no concordance with the regulatory ad hoc causality evaluation. CONCLUSION: The regulatory data regarding kava hepatotoxicity is selective and of low quality, not supportive of the regulatory proposed causality; but instead, is an explanation of the overall causality discussions of kava hepatotoxicity. We are proposing that the regulatory agency reports data in full length and reevaluates causality.
8. Smollin CG. Toxicology: pearls and pitfalls in the use of antidotes. *Emerg Med Clin North Am.* 2010;28(1):149-61, ix.  
Although most poisonings require only supportive care, the emergency physician must recognize when the use of an antidote is required, and understand the risks and benefits of the treatment rendered. Although the more commonly instituted specific therapy in acute poisoning is the administration of intravenous fluids followed by the administration of oxygen, in certain circumstances prompt administration of a specific antidote may be required, and failure to identify these circumstances may lead to significant morbidity or mortality. This article describes select antidotes, and discusses their indications and potential pitfalls.
9. Leithead JA, Simpson KJ, MacGilchrist AJ. Fulminant hepatic failure following overdose of the vitamin A metabolite acitretin. *Eur J Gastroenterol Hepatol.* 2009;21(2):230-2.  
Hepatotoxicity associated with the therapeutic ingestion of the vitamin A metabolite acitretin is well recognized. No reported cases of hepatic dysfunction as a consequence of acitretin overdose are, however, present. Here for the first time we report a case of fulminant hepatic failure following an intentional overdose of 600 mg of acitretin. The patient fulfilled the King's College Hospital poor prognostic criteria by 66 h after overdose, but demonstrated a rapid improvement thereafter and did not require liver transplantation. Given the known association between psoriasis and depression, and the possible association of acitretin with psychiatric illness, this is an important potential adverse event.

10. Lam CL, Wong W, Fong DY. Chinese herbal medicine in the treatment of acute upper respiratory tract infection: a randomised, double blind, placebo-controlled clinical trial. *Hong Kong Med J*. 2009;15 Suppl 6(30-4).
11. Hagiya K, Mizutani T, Yasuda S, Kawano S. Nicotine poisoning due to intravenous injection of cigarette soakage. *Hum Exp Toxicol*. 2010.  
A 27-year-old female nurse intravenously injected 5 mL of cigarette soakage solution that contained approximately 5.7 mg nicotine, in a suicidal attempt. Clinical manifestations consisted of nausea, palpitation, abdominal pain, repeated vomiting, and diarrhea. She remained fully conscious during this episode. About 7 hours later, she visited emergency department on foot and received fluid infusion for dehydration. She fully recovered at night of the day. This is the first documented report of acute nicotine poisoning due to intravenous injection of cigarette soakage in humans. Signs and symptoms appeared immediately after the injection, but this case seemed to be relatively mild in terms of clinical manifestation. The elimination half-life of nicotine seems to be short, that is, less than 1 hour. Therefore, if initial treatment is appropriate and the patient can survive acute phase of nicotine poisoning, prognosis is good.
12. Muller D, Desel H. Acute selenium poisoning by paradise nuts (*Lecythis ollaria*). *Hum Exp Toxicol*. 2010.  
Two previously healthy women developed nausea, vomiting, headache and dizziness for several days, a massive hair loss about 2 weeks later and a discoloration of the fingernails. Detailed diagnostic procedures did not reveal any pathological results. Therapeutic measures did not show any effect. Thallium and arsenic were within normal range in plasma. Delayed quantitative determination of selenium in blood, however revealed toxic values (in case I: 479 mug/L of serum, 8 weeks after ingestion, and in case II 300 mug/L of serum, 9 weeks after ingestion). In retrospect, a relation to the ingestion of paradise nuts could be established.
13. Boscolo M, Antonucci S, Volpe AR, Carmignani M, Di Gioacchino M. Acute mercury intoxication and use of chelating agents. *J Biol Regul Homeost Agents*. 2009;23(4):217-23.  
There is a great hazard of mercury intoxication in the third world for artisanal miners using mercury as amalgam for extracting and refining gold. In developing countries, there is the possibility of risk regarding exposure to Hg from amalgam tooth fillings, ethyl-Hg (thimerosal) added as antiseptic to vaccines and methyl-Hg in fish. In one case, a 41-year-old man attempted suicide by ingesting 100 mg of HgCl<sub>2</sub>. After 8 hours, he developed hematemesis and entered the intensive care unit; his urinary Hg was 10.1 mg/l. Treatment with 2,3-dimercaptopropanol (BAL) was started by intramuscular route after 16 hours at the dosage of 5 mg/kg body weight every 4 hours on days 2-3 and 3 mg/kg every 6 hours on days 4-5 and then every 12 hours on days 6-14 without adverse side effects. Acute Hg intoxication can be managed with BAL as first choice chelator, whereas the less toxic 2,3-dimercaptosuccinic acid (DMSA) and 2,3-dimercaptopropane-1-sulfonic acid (DMPS) should be reserved for cases of less severe inorganic Hg or methyl-Hg acute intoxication. Such agents, recommended only for the treatment of acute Hg poisoning, should not be used for patients suffering from neurological diseases in which environmental Hg exposure is

hypothesised.

14. Elikottil J, Gupta P, Gupta K. The analgesic potential of cannabinoids. *J Opioid Manag.* 2009;5(6):341-57.  
Historically and anecdotally cannabinoids have been used as analgesic agents. In recent years, there has been an escalating interest in developing cannabis-derived medications to treat severe pain. This review provides an overview of the history of cannabis use in medicine, cannabinoid signaling pathways, and current data from preclinical as well as clinical studies on using cannabinoids as potential analgesic agents. Clinical and experimental studies show that cannabis-derived compounds act as antiemetic, appetite modulating, and analgesic agents. However, the efficacy of individual products is variable and dependent upon the route of administration. As opioids are the only therapy for severe pain, analgesic ability of cannabinoids may provide a much-needed alternative to opioids. Moreover, cannabinoids act synergistically with opioids and act as opioid sparing agents, allowing lower doses and fewer side effects from chronic opioid therapy. Thus, rational use of cannabis-based medications deserves serious consideration to alleviate the suffering of patients due to severe pain.
15. Fanciullo GJ. Medical cannabis. *J Opioid Manag.* 2009;5(5):245-6.
16. McFee RB, Caraccio TR. Herbal 'Remedy' Poison control deduces true cause of patient's coma. *Jems.* 2010;35(1):34-6.
17. Schubert C. Pandemic blows lid off laws limiting mercury in vaccines. *Nat Med.* 2010;16(1):9.
18. Rahn EJ, Hohmann AG. Cannabinoids as pharmacotherapies for neuropathic pain: from the bench to the bedside. *Neurotherapeutics.* 2009;6(4):713-37.  
Neuropathic pain is a debilitating form of chronic pain resulting from nerve injury, disease states, or toxic insults. Neuropathic pain is often refractory to conventional pharmacotherapies, necessitating validation of novel analgesics. Cannabinoids, drugs that share the same target as Delta(9)-tetrahydrocannabinol (Delta(9)-THC), the psychoactive ingredient in cannabis, have the potential to address this unmet need. Here, we review studies evaluating cannabinoids for neuropathic pain management in the clinical and preclinical literature. Neuropathic pain associated with nerve injury, diabetes, chemotherapeutic treatment, human immunodeficiency virus, multiple sclerosis, and herpes zoster infection is considered. In animals, cannabinoids attenuate neuropathic nociception produced by traumatic nerve injury, disease, and toxic insults. Effects of mixed cannabinoid CB(1)/CB(2) agonists, CB(2) selective agonists, and modulators of the endocannabinoid system (i.e., inhibitors of transport or degradation) are compared. Effects of genetic disruption of cannabinoid receptors or enzymes controlling endocannabinoid degradation on neuropathic nociception are described. Specific forms of allodynia and hyperalgesia modulated by cannabinoids are also considered. In humans, effects of smoked marijuana, synthetic Delta(9)-THC analogs (e.g., Marinol, Cesamet) and medicinal cannabis preparations containing both Delta(9)-THC and cannabidiol (e.g., Sativex, Cannador) in neuropathic pain states are reviewed. Clinical studies largely affirm

that neuropathic pain patients derive benefits from cannabinoid treatment. Subjective (i.e., rating scales) and objective (i.e., stimulus-evoked) measures of pain and quality of life are considered. Finally, limitations of cannabinoid pharmacotherapies are discussed together with directions for future research.

19. Kellerman TS. Poisonous plants. *Onderstepoort J Vet Res.* 2009;76(1):19-23. South Africa is blessed with one of the richest floras in the world, which--not surprisingly--includes many poisonous plants. Theiler in the founding years believed that plants could be involved in the aetiologies of many of the then unexplained conditions of stock, such as gousiekte and geeldikkop. His subsequent investigations of plant poisonings largely laid the foundation for the future Sections of Toxicology at the Institute and the Faculty of Veterinary Science (UP). The history of research into plant poisonings over the last 100 years is briefly outlined. Some examples of sustained research on important plant poisonings, such as cardiac glycoside poisoning and gousiekte, are given to illustrate our approach to the subject and the progress that has been made. The collation and transfer of information and the impact of plant poisonings on the livestock industry is discussed and possible avenues of future research are investigated.
20. Froehlich TE, Lanphear BP, Auinger P, Hornung R, Epstein JN, Braun J, Kahn RS. Association of tobacco and lead exposures with attention-deficit/hyperactivity disorder. *Pediatrics.* 2009;124(6):e1054-63.  
OBJECTIVE: The study objective was to determine the independent and joint associations of prenatal tobacco and childhood lead exposures with attention-deficit/hyperactivity disorder (ADHD), as defined by current diagnostic criteria, in a national sample of US children. METHODS: Data are from the 2001-2004 National Health and Nutrition Examination Survey, a cross-sectional, nationally representative sample of the US population. Participants were 8 to 15 years of age (N = 2588). Prenatal tobacco exposure was measured by report of maternal cigarette use during pregnancy. Lead exposure was assessed by using current blood lead levels. The Diagnostic Interview Schedule for Children was used to ascertain the presence of ADHD in the past year, on the basis of Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, criteria. RESULTS: A total of 8.7% (95% confidence interval [CI]: 7.3%-10.1%) of children met criteria for ADHD. Prenatal tobacco exposure (adjusted odds ratio [aOR]: 2.4 [95% CI: 1.5-3.7]) and higher current blood lead concentrations (aOR for third versus first tertile: 2.3 [95% CI: 1.5-3.8]) were independently associated with ADHD. Compared with children with neither exposure, children with both exposures (prenatal tobacco exposure and third-tertile lead levels) had an even greater risk of ADHD (aOR: 8.1 [95% CI: 3.5-18.7]) than would be expected if the independent risks were multiplied (tobacco-lead exposure interaction term,  $P < .001$ ). CONCLUSIONS: Prenatal tobacco and childhood lead exposures are associated with ADHD in US children, especially among those with both exposures. Reduction of these common toxicant exposures may be an important avenue for ADHD prevention.
21. Holmes P, James KA, Levy LS. Is low-level environmental mercury exposure of concern to human health? *Sci Total Environ.* 2009;408(2):171-82. Mercury has long been recognised as toxic, principally in relation to its effects on

humans following acute or prolonged high-level occupational exposures and, in the latter half of the last century, from a number of environmental incidents. Recognised target organs are the kidneys, central nervous system and thyroid glands. Recently concern has grown about the potential risks to the human population from current background environmental levels, leading bodies such as the World Health Organisation to call for the reduction or, wherever possible, elimination of the use of mercury. This review considers the strength of the epidemiological evidence on the effects of prolonged low-level exposure to the various forms of mercury. The limited research base suggests that several of the potential targets of long-term environmental exposure to mercury are similar to those occurring from occupational exposure including the renal, cardiovascular and immune systems. However, the evidence also suggests that, particularly in the case of organic mercury compounds, the most sensitive endpoint is central nervous system toxicity, especially in relation to exposure during the in utero period and childhood. It also appears that those human populations which have traditionally consumed diets high in seafoods are at greatest risk. While the extent of risk to the general population that may arise from existing environmental exposure levels appears limited, this conclusion is based on an incomplete dataset and therefore the general consensus view that exposure to mercury in its various forms should be minimised where practical, appears to be justified. A number of potential areas of further research are suggested as being pre-requisite to the development of a more rigorous risk assessment.

22. Yorifuji T, Kashima S, Tsuda T, Harada M. What has methylmercury in umbilical cords told us? - Minamata disease. *Sci Total Environ.* 2009;408(2):272-6. Severe methylmercury poisoning occurred in Minamata and neighboring communities in the 1950s and 1960s. The exposed patients manifested neurological signs, and some patients exposed in utero were born with so-called congenital Minamata disease. In a previous report, Nishigaki and Harada evaluated the methylmercury concentrations in the umbilical cords of inhabitants and demonstrated that methylmercury actually passed through the placenta (Nishigaki and Harada, 1975). However, the report involved a limited number of cases (only 35) and did not quantitatively evaluate the regional differences in the transition of methylmercury exposure. Therefore, in the present study, we evaluated the temporal and spatial distributions of methylmercury concentrations in umbilical cords, with an increased number of participants and additional descriptive analyses. Then, we examined whether the methylmercury concentrations corresponded with the history of the Minamata disease incident. A total of 278 umbilical cord specimens collected after birth were obtained from babies born between 1925 and 1980 in four study areas exposed to methylmercury. Then, we conducted descriptive analyses, and drew scatterplots of the methylmercury concentrations of all the participants and separated by the areas. In the Minamata area, where the first patient was identified in 1956, the methylmercury concentration reached a peak around 1955. Subsequently, about 5 years later, the concentrations peaked in other exposed areas with the expected exposure distribution corresponding with acetaldehyde production (the origin of methylmercury). This historical incident several decades ago in Minamata and neighboring communities clearly shows that regional pollution affected the environment in utero. Furthermore, the temporal and spatial distributions of the methylmercury concentrations in the umbilical cords tell

us the history of the Minamata disease incident.

23. Waring WS, Laing WJ, Good AM, Malkowska AM. Acute caffeine ingestion: clinical features in patients attending the emergency department and Scottish poison centre enquiries between 2000 and 2008. *Scott Med J.* 2009;54(4):3-6.  
BACKGROUND AND AIMS: Little information is available regarding the healthcare burden associated with deliberate caffeine ingestion. The present study sought to establish the impact of caffeine ingestion on hospital attendances and Poisons Centre enquiries in Scotland. METHODS: Retrospective analyses of clinical data from patients attending the Royal Infirmary of Edinburgh after acute caffeine ingestion, and TOXBASE enquiries from Scotland regarding caffeine poisoning between 2000-2008 inclusive. Cochran-Armitage trend tests were used to evaluate changes in annual admissions and TOXBASE enquiries. RESULTS: There were 43 hospital attendances due to deliberate caffeine ingestion, representing 0.2% of all poisoning cases. The median (interquartile range) stated dose was 1040 mg (600-1500 mg). Minor gastrointestinal symptoms were common, and no patient developed features of severe toxicity. There were 1418 enquiries to TOXBASE concerning caffeine poisoning, representing 0.2% of all poisoning enquiries from Scotland. The proportions of hospital admissions and TOXBASE enquiries due to caffeine ingestion have remained constant. CONCLUSION: Caffeine ingestion is uncommon, and results in only a small number of hospital attendances and Poisons Centre enquiries. In contrast to patterns reported elsewhere, the prevalence of caffeine abuse has not increased in Scotland over recent years.