Methamphetamine strategy requires evaluation

We read with interest about the strategy recently developed in the York Region of Toronto to curb methamphetamine use, which is based on Vancouver’s 4-pillar drug strategy.1 We recently reported that more than 70% of Vancouver’s street-involved youth have used methamphetamine.2 We have also seen a significant growth in methamphetamine use among Vancouver’s injection drug users, from 2% in 1998 to more than 15% in 2006. These trends have been observed despite Vancouver’s 4-pillar strategy, although we should acknowledge that the enforcement pillar has consumed the overwhelming majority of the local resources devoted to the strategy.

Thus, we wonder if Cronkwright Kirkos and colleagues might be overly optimistic when they state that the supply of methamphetamine can be suppressed “through active and intelligence-led strategic police enforcement.” Unlike heroin and cocaine, which must be farmed illicitly in foreign countries before it is imported, methamphetamine can be inexpensively produced locally from common precursor chemicals. Given the failure to keep heroin and cocaine off North America’s streets,3 the likelihood that law enforcement will curb the growth in the supply of methamphetamine is exceedingly small.4

We also raise caution about untested modes of drug prevention. A study commissioned by the US National Institutes of Health evaluated the United States’ national youth antidrug media campaign and found little evidence of direct favourable effects on youth. Instead, higher exposure to the campaign was associated with a weakening of social norms against illicit drugs.5 Despite ongoing federal funding for such initiatives in Canada, a lack of benefit and evidence of potential harm have also been consistently observed with the drug education tool known as DARE (Drug Abuse Resistance Education).6

Evan Wood MD PhD
Thomas Kerr PhD
British Columbia Centre for Excellence in HIV/AIDS, St. Paul’s Hospital,
Vancouver, BC

Competing interests: None declared.

REFERENCES
1. Cronkwright Kirkos W, Carrique T, Griffen K, et al. The York Region Methamphetamine Strategy. CMAJ 2008;178:1655–6.
2. Wood E, Stoltz JA, Zhang R, et al. Circumstances of first crystal methamphetamine use and initiation of injection drug use among high-risk youth. Drug Alcohol Rev 2008;27:270–6.
3. Wood E, Tyndall MW, Spittal PM, et al. Impact of supply-side policies for control of illicit drugs in the face of the AIDS and overdose epidemics: investigation of a massive heroin seizure. CMAJ 2003;168:165–9.
4. Reuter P, Caulkins JP. Does precursor regulation make a difference? Addiction 2003;98:1177–9.
5. Evaluation of the national youth anti-drug media campaign: 2004 report of findings. Washington: National Institute on Drug Abuse; 2004. Report no. NIDA-8-5063:1–41.
6. Des Jarlais DC, Sloboda Z, Friedman SR, et al. Diffusion of the D.A.R.E and syringe exchange programs. Am J Public Health 2006;96:1354–8.

TASER safety

We are members of the TASER International Scientific and Medical Advisory Board and would like to comment on the review by Nanthakumar and colleagues.1 This review focused on porcine studies; it ignored the 30 papers and abstracts that have provided data on the application of electronic control devices to humans. In addition, there are 4 implications in the review that we believe are erroneous.

The first is that the induction of ventricular fibrillation is relevant to the problem of arrest-related deaths. There are over 700 arrest-related deaths per year in 47 of the 50 United States.2 TASER electronic control devices have been applied to over 1.3 million people. In about 95% of the arrest-related deaths in which an electronic control device was used, the initial rhythm (established by paramedics on the scene) was asystole or pulseless electrical activity.3 These patients typically responded rapidly to atropine and epinephrine, which further differentiates these cases from cases of asystole arising from long-term ventricular fibrillation (lasting about 15 minutes).

The second erroneous implication is that small swine provide a reasonable model with which to measure the risk of electrical induction of ventricular fibrillation in humans. Swine, especially small ones, are extremely sensitive to the electrical induction of ventricular fibrillation.4 In pigs, the Purkinje fibers cross the entire ventricular wall whereas in dogs and humans they are confined to a very thin endocardial layer.5 Activation in swine proceeds from the epicardium to the endocardium, whereas it occurs in the reverse direction in dogs and humans.6 Thus, swine are much more sensitive to external electrical currents. Radiofrequency ablation is routinely done in humans but it will typically produce ventricular fibrillation in swine because they are sensitive to higher frequencies than humans. In addition, the threshold for ventricular fibrillation is directly related to body weight for both utility waveforms and electronic control device waveforms.4,7 In humans, even if the barbs of an electronic control device are placed directly on the cardiac axis, no effect is captured with echocardiographic monitoring.8

Third, it is erroneous to imply that electronic control devices can cause dangerous acidosis. Reports of acidosis induced by the use of an electronic control device come from studies of anesthetized pigs in which the ventilators were turned off.

A final erroneous statement is that the presence of cocaine makes an electronic control device more dangerous.