Heroin overdose

Professor David Clark briefly summarises some of the main research findings focusing on the incidence and possible causes of heroin overdose.

At a recent conference on overdose in Swansea, I was reminded of a number of myths related to heroin overdose that still circulate among users, family members, practitioners and policy makers. These myths hamper the development and implementation of strategies to reduce deaths caused by overdose of heroin and other drugs.

Accidental overdose is the most common cause of death among heroin users. Research studies have revealed that 50-70 per cent of injecting drug users (IDUs) have experienced a non-fatal overdose at some time in their lives, with 20-30 per cent overdosing in the preceding 12 months.

Death from heroin overdose is due to respiratory arrest. Many people who die from heroin overdose do so two to three hours after taking the drug, which means that there is often a long time period during which the person can be helped.

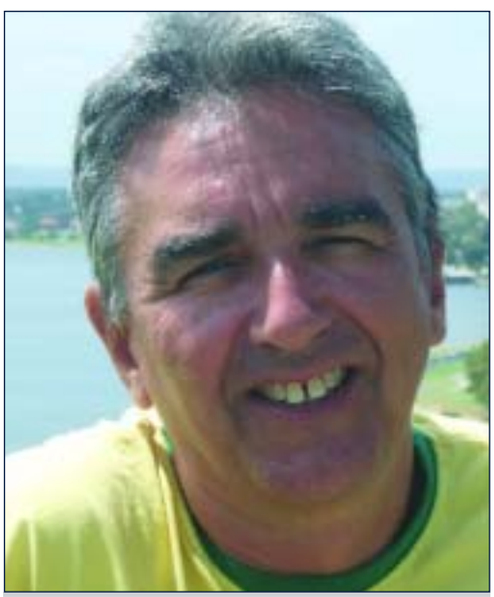
Extensive research has shown the following risk factors for overdose: using the drug intravenously; having a history of heroin dependence; using the drug after a period of non-use or reduced use; not being in treatment for heroin dependence; and concomitant use of depressants, such as alcohol and benzodiazepines, *eg* valium.

It is commonly believed that many overdose deaths occur among young, relatively inexperienced heroin users. However, research has consistently found that most victims of fatal overdose are aged in their late 20s and early 30s. They generally have a long history of heroin dependence. The greater incidence of drug overdose among older users is somewhat counter-intuitive, since one would expect younger users to have less tolerance, and a relatively poor ability to determine the dose of heroin.

Another counter-intuitive finding revealed by research is that at autopsy a large proportion of overdose fatalities have relatively low blood morphine concentrations (heroin is rapidly metabolised to morphine in the body). This finding conflicts with the widely held view that overdose is the result of using a quantity or quality (purity) of the drug in excess of the person's normal tolerance.

Moreover, research has shown only a moderate correlation between the purity of street heroin seizures and the numbers of deaths from overdose. Research has also dispelled the notion that overdose fatalities are related to contaminants in heroin.

What factors underlie the strong age patterns and apparently low blood morphine concentrations found in overdose fatalities?



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A significant number of overdose fatalities occur after periods of reduced use, such as immediately after release from prison. The increased risk for overdose following release is likely to be related to the person taking heroin at a time of low tolerance to the drug, following a period of non-use or reduced use in prison. Hair analysis studies, showing that overdose fatalities were using less heroin than active street users in the months preceding death, confirm this idea.

The loss of tolerance may also be at least partially responsible for the age-related trends in overdose deaths. It has been suggested that, after a decade of use, some users cut down their consumption of heroin as a result of tiring of the rigours of the heroin-using lifestyle.

Entry into abstinence-based treatment also represents a potentially risky period for heroin overdose if the person relapses, since tolerance is lost to the drug or to methadone.

The loss of tolerance that occurs following termination of heroin use may vary for different effects of the drug. Thus, users who reduce their consumption may be at greater risk of overdose as their tolerance to the respiratory depressant effects may have diminished more rapidly than their tolerance to the desired psychological effects of the drug.

Concomitant use of other central nervous system depressant drugs, in particular alcohol and benzodiazepines, is known to increase the risk of heroin overdose, both fatal and non-fatal. Heroin is more likely to cause overdose in people who have been drinking alcohol or taking benzodiazepines because these latter substances can potentiate the respiratory depressant effects of the opiate.

Researchers do not believe that poly-drug use and loss of tolerance can fully explain both the age-dependency of overdose victims and the low morphine concentrations associated with these deaths. They have suggested that mortality from heroin overdose might (also) be associated with systemic disease, some of which may have occurred as a result of previous non-fatal overdose(s). Two examples are provided here.

An involvement of liver disease or dysfunction in heroin overdose is possible, since opiates are metabolised in the liver and drug clearance is reduced in people with liver cirrhosis. IDUs are at significantly increased risk for liver disease, in large part due to their increased likelihood of contracting hepatitis C. Moderate levels of alcohol have been found to exacerbate liver damage arising from hepatitis.

Reduced metabolism of heroin in users with liver damage could prolong the period of heavy intoxication in which they are at risk for overdosing by respiratory depression.

Opiate overdose deaths may also be linked to pulmonary dysfunction, which can result in an increased vulnerability to fatal respiratory depression. Heroin users are likely to exhibit impaired pulmonary dysfunction due to smoking (cigarettes, heroin, crack cocaine), complications of overdose, and their increased susceptibility to infection, the latter arising in part from their poor health and lifestyle.

Recommended reading:
M. Warner-Smith et al (2001), 'Heroin overdose: causes and consequences', Addiction 96: 1113.