indicates that about 2540 men in the sentenced prison population injected drugs in the six months before arrest.[4,5] If we assume from our data that 8% of them were HIV positive that gives an estimated total of 203 HIV positive male drug injectors in the sentenced population. The corresponding figures for women (about 168 recent injectors, and 15% HIV positive) suggest a total of 25 HIV positive women in prison. At any time, therefore, the total number of HIV positive prisoners may be about 228 in a sentenced population of about 36 000.

If drug injecting is found as commonly among remand prisoners as amongst those sentenced the total number of HIV positive drug injectors in the prison system (both sentenced and on remand) is probably about 285 out of a prison population of about 45 000, or 1 in 158. It is more difficult to estimate the prevalence of HIV infection among non-injecting prisoners. We found that 1·7% of the non-injecting subjects were HIV positive but it is difficult to make a projection when prevalence is low.

On the basis of the evidence for drug injectors, we conclude that the prevalence of HIV infection in the prison population is not less than about 1 in 158 (0·6%). This estimate must be compared with the prison department's figure of roughly 50 prisoners known to be positive for HIV antibody per year.

1 Farrell M, Strang J. Drugs, HIV, and prisons. *BMJ* 1991;**302**:1477-8.
2 Johnson AM, Parry JV, Best SJ, Smith AM, DeSilva M, Mortimer P. HIV surveillance by testing saliva. *AIDS* 1988;**2**:369-71.
3 Turnbull PJ, Dolan KA, Stimson GV. *Prisons, HIV and AIDS: risks and experiences in custodial care*. Horsham: AVERT, 1991.
4 Maden A, Swinton M, Gunn J. Drug dependence in prisoners. *BMJ* 1991;**302**:880.
5 Maden A, Swinton M, Gunn J. Women in prison and use of illicit drugs before arrest. *BMJ* 1991;**301**:1133.

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A clinical trial of minocycline in lepromatous leprosy

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Not since 1970, when Rees showed that rifampicin was effective against *Mycobacterium leprae* in infected mice and rapidly bactericidal in patients with lepromatous leprosy,[1] has a new chemotherapeutic agent been introduced to treat leprosy. Drugs for treating leprosy worldwide are limited to dapsone, clofazimine, and rifampicin; this small number is of special concern because of the emergence of drug resistant disease, the general recommendation of multiple drug treatment, and significant side effects and toxicities of each of these drugs, which at times preclude their use. We showed that minocycline at concentrations achievable in humans was active against *M leprae* in mice and consistently bactericidal.[2,3] We therefore initiated a clinical trial of minocycline in patients with lepromatous leprosy.

Patients, methods, and results

Eight consenting adults (seven men and one woman) with lepromatous leprosy (five patients) or borderline lepromatous leprosy (three) who had been previously untreated (six) or whose leprosy had relapsed (two) were treated with 100 mg minocycline alone once daily for three months.

After one week's treatment we observed improvement in either skin erythema or induration in six patients and improvement in both manifestations in two. After three months' treatment skin lesions had noticeably improved in all patients, six patients having complete resolution of all erythema and induration. One patient had mild transient vertigo, which resolved spontaneously without discontinuing treatment. No other adverse reaction was noted. Unexpectedly, none of the patients developed a lepra reaction.

Before treatment and at one week and one, two, and three months after starting treatment skin biopsies were performed, from which 5×10^3 *M leprae* were inoculated into the hind feet of BALB/c mice. If more than 10^5 acid fast bacilli per foot pad were found in pooled foot pads (four) harvested eight and 12 months subsequently or in one foot pad or more of those (generally 10) counted individually 12 months after infection, viable bacilli were considered to have been present in the inoculum.

All of the pretreatment skin biopsy specimens consistently resulted in growth of the bacilli in mice when evaluated by both methods. According to the findings of pooled harvests of foot pads no patient harboured any viable *M leprae* at either two or three months after starting treatment (table). Harvests of individual foot pads were more sensitive in detecting any viable *M leprae*; in six instances these results were positive and those of pooled harvests were negative, and in none was the opposite true (table). Viable bacilli were consistently absent from the skin in only one patient at one week after treatment. At one and two months respectively three and six of the patients had lost detectable viable leprosy bacilli, and by three months none of the eight patients harboured any viable bacilli.

Serum minocycline concentrations determined by an agar disc diffusion method[2] were at the peak (two hours after treatment) 1·84 (SD 0·48) mg/l (range 1·07-2·66 mg/l) and at the trough (24 hours after treatment) 0·43 (0·11) mg/l (range 0·33-0·58 mg/l), well above that (≤0·17 mg/l) previously found to inhibit consistently growth of *M leprae* in mice.[2,3]

Viability of M leprae in skin biopsy specimens from patients with lepromatous or borderline lepromatous leprosy treated with minocycline

Patient No	Pooled harvests* or individual harvest mouse foot pads	Time after starting treatment			
		1 Week	1 Month	2 Months	3 Months
1	Pooled (n=4)	+/+	−/+	−/−	−/−
	Individual (n=10)	5	6	1	0
2	Pooled (n=4)	+/+	−/−	−/−	−/−
	Individual (n=10)	3	4	0	0
3	Pooled (n=4)	−/−	+/−	−/−	−/−
	Individual (n=10)	4	1	0	0
4	Pooled (n=4)	−/−	−/−	−/−	−/−
	Individual (n=10)	1	0	0	0
5	Pooled (n=4)	−/−	−/−	−/−	−/−
	Individual (n=10)	0	0	0	0
6	Pooled (n=4)	−/+	−/−	−/−	−/−
	Individual (n=10)	4	0†	0	0
7	Pooled (n=4)	−/−	+/+	−/−	−/−
	Individual (n=10)	1	5	0	0
8	Pooled (n=4)	+/−	−/+	−/−	−/−
	Individual (n=10)	2	5	5	0

*Eight month harvest/12 month harvest.
†n=8.
+/− Presence/absence of viable *M leprae*.

Comment

The consistent rapidity of the patients' clinical response to minocycline is unique in our experience. The clearance of viable *M leprae* from the skin by minocycline was faster than that reported for dapsone or clofazimine,[4] slower than that for rifampicin,[1,4] and similar to that for pefloxacin and ofloxacin.[5]

Unfortunately, because the safety of prolonged courses of fluoroquinolones has not been established they cannot be recommended for treating leprosy. Minocycline, however, has been used for over two decades, primarily for other bacterial diseases and acne; because of its safety record in long term use our findings strongly support its application to the treatment of leprosy. Minocycline may also be a second bactericidal agent to combine with rifampicin treatment and thereby shorten the duration of treatment required to treat leprosy effectively.

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1 Rees RJW, Pearson JMH, Waters MFR. Experimental and clinical studies on rifampicin in treatment of leprosy. *BMJ* 1970;i:89-92.
2 Gelber RH. Activity of minocycline in Mycobacterium leprae-infected mice. *J Infect Dis* 1987;156:236-9.
3 Gelber RH, Siu P, Tsang M, Alley P, Murray LP. Effect of low-level and intermittent minocycline therapy on growth of M leprae in mice. *Antimicrob Agents Chemother* 1991;35:992-4.
4 Shepard CC. A brief review of experiences with short term clinical trials monitored by mouse foot pad inoculation. *Lepr Rev* 1981;52:299-308.
5 Grosset JH, Ji B, Guelpa-Lauras CC, Perani EG, N'Deli LN. Clinical trial of pefloxacin and ofloxacin in the treatment of lepromatous leprosy. *Int J Leprother Mycobact Dis* 1990;58:281-95.

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Suffocation from misuse of gas masks during the Gulf war

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We report the use of antichemical warfare kits in Israel during the recent Gulf war. The threat of Iraqi chemical missile attacks against the civilian population forced the Israeli civil defence authorities to equip the entire population of about four million with protective devices. People were instructed in advance to "seal" one room in each residence and, on hearing a missile attack alarm, immediately to enter this room and don a gas mask. Subsequent missile alerts and the ensuing confinements in the sealed room ranged from a few minutes to three hours.

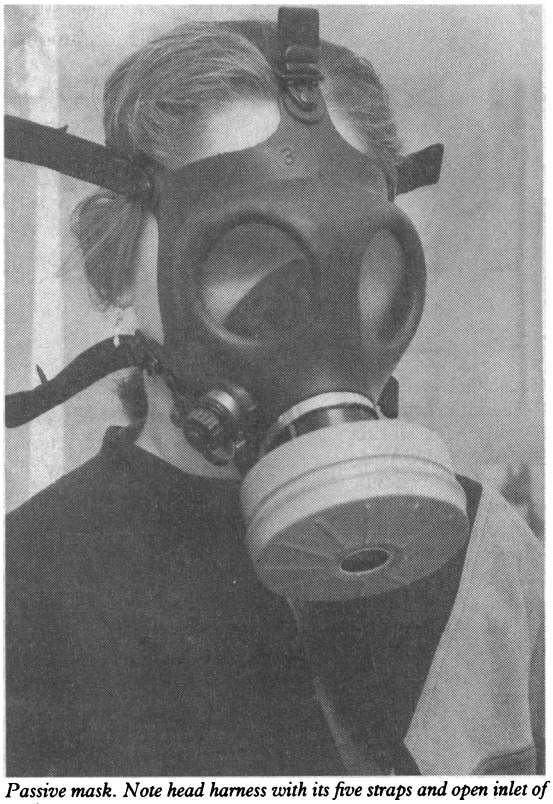
Technical specifications and results

Four types of devices, intended as protection against organophosphorus and dichlorodiethylsulphide compounds were distributed to residents of Israel during the autumn of 1990. These devices were manufactured by Shalom Chemical Industries Ltd. The infant carrier is a large, transparent, plastic box with a passive filter designed to protect the baby's entire body. The active hood is for children 3-4 years of age; it has a battery operated airflow. The passive mask (figure) is for teenagers and adults; it consists of two elements: a rubber face mask with a head harness regulated by five straps, and an attachable filter canister. The canister is tightly screwed into place just before use, after removal of the plastic plug that protects the canister's inlet while it is in its cardboard storage box. The fourth device, the active mask, is for older children and disabled adults. It is essentially the same as the passive mask but with a battery operated airflow.

During the Gulf war and in the wake of 39 missile attacks, 13 people died of suffocation due to mishandling of the protective devices. The bodies of eight of these victims (four males and four females aged between 3 and 78 years), were examined in the Greenberg Institute of Forensic Medicine. In seven, petechiae of the conjunctiva and facial cyanosis were detected, along with visceral petechiae and pulmonary congestion and oedema.[1,2] Pressure marks around the nose and lips caused by the nose cup of the mask were evident in six of the victims.

Comment

Despite detailed instructions and demonstrations by civil defence personnel during the actual distribution of the kits, at least 13 people perished owing to improper handling of the equipment. In all instances where asphyxia was induced by the protective mask the victim was found lifeless with the mask covering the face



Passive mask. Note head harness with its five straps and open inlet of canister

and the plastic plug still intact in the filter canister. A small proportion of the population had reinserted the plastic plug after the first alert, hoping to prolong the filter's durability, and apparently forgot to remove the plug before putting the mask on again. When such a "plugged" mask is attached to the face and the straps of the head harness are tightened the resultant vacuum and negative pressure sucks the mask to the face, thereby impeding its removal at the first signs of dyspnoea and precipitating fatal suffocation. The 3 year old had been fitted by her parents with a plugged mask. Also a 9 month old baby had inadvertently been left for several hours in a passive plastic carrier and had succumbed to hypoxia.

To avoid inadvertent deaths from asphyxia among the users of protective masks, it seems imperative to replace the plastic plug currently supplied with the canister by a readily disposable, non-repluggable cover or lid. The passive baby carriers and even the passive adult masks should be replaced by battery operated, active airflow mechanisms, which will preclude accidental suffocation.

1 Polson CJ, Gee D, Knight B. *The essentials of forensic medicine*. Oxford: Pergamon Press, 1985:449-79.
2 DiMaio DJ, DiMaio VJM. *Forensic pathology*. New York: Elsevier, 1989: 207-50.

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