Agent, Host, and Environment: Hepatitis C Virus in People Who Inject Drugs

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(See the article by Doerrbecker et al, on pages 1830–8, and the brief report by Thibault, on pages 1839–42.)

Hepatitis C virus (HCV) is hyperendemic worldwide in people who inject drugs (PWID) [1]. HCV prevalence rates in PWID typically vary between 40% and 90%, and incidences may fall between 20 and 40 infections per 100 person-years [2–6]. In high-income countries, half of PWID are infected with HCV within 5 years following onset of drug injection, and in low- and middle-income countries, half may be infected within 1 year [1]. A recent publication estimated that, globally, 10 million PWID have been infected with HCV [7].

Infectious disease control strategies typically target features of the agent, host, and environment to interrupt transmission; all such features of HCV favor rapid spread in PWID. The agent is highly efficiently transmitted via parenteral exposure to infectious blood, and it is relatively stable in the environment [8, 9]. Illicit drug injection practices may include the shared use of syringes and drug preparation equipment, such as drug cookers, filtration cotton, and rinse water; each of these host behaviors has been shown to be associated with HCV transmission [5, 6, 10].

Substantial declines in syringe sharing have dramatically reduced human immunodeficiency virus (HIV) incidence in PWID in high-income countries, but the shared use of other drug preparation materials persists [11, 12]; thus, HCV transmission continues at extremely high rates. Owing to HCV’s ability to evade the immune system, 70%–80% of infections become chronic [13, 14]. Thus, the environment—the settings in which PWID inject together—is characterized by a high prevalence of infectious carriers and a wide range of materials that may harbor HCV in the environment—the settings in which HCV may be transmitted among PWID via injection-related materials.

Dörrebecker et al describe their experiments using cell culture–grown HCV (JFH1, derived from a Japanese patient with fulminant hepatitis) to infect hepatoma cells and study infectivity and inactivation of the virus on inanimate surfaces [15]. Using this state-of-the-art method, they revealed that exposure to disinfectants, including those containing alcohol, will effectively inactivate dried HCV on surfaces. Simulating drug injection, containers (spoons or cookers) to prepare drugs for injection were contaminated with HCV in a water solution and heat was applied. Their experiments showed that HCV could survive temperatures up to 65°–70°C, which required between 80 and 95 seconds of heating. In an earlier study in New York and Denver, ethnographers directly observed drug preparation in injection settings and measured heating times and temperatures applied to drug containers [16]. Only 12% of PWID heated drug solutions for >45 seconds, and nearly half heated for <15 seconds; they replicated these conditions in the laboratory and found that HIV was rapidly inactivated when heated, within 7–10 seconds. Thus, this new study by Dörrebecker and colleagues lends additional support to the hypothesis that drug preparation practices that include heating may reduce the risk of HIV transmission via the shared use of containers, but that HCV may still be transmitted. This would also be consistent with substantial epidemiologic evidence that sharing containers is associated with HCV seroconversion [5, 6, 10].

The article by Thibault et al [17] describes laboratory analyses of injection materials (syringes, drug cookers, filtration cotton, used water vials, and alcohol and cotton swabs) collected from PWID in France. Some materials were collected from PWID who reported that they were HIV positive, and others were collected without regard to the HCV status of the individual. Six hundred twenty items were obtained and sorted according to type, and 62 pooled samples acquired from batches of 10 similar items were prepared. HCV RNA could be detected in approximately 30% of pooled samples of syringes and 80% of pooled samples of alcohol and cotton swabs, and the results...
did not differ according to whether or not materials were purposively collected from HCV-positive individuals. None of the pooled samples of water vials or filtration cotton had detectable HCV RNA, and only 1 of 11 pooled samples from drug cooks was positive.

Although detection of HCV RNA is not well correlated with measurement of infectivity, the study raises very interesting questions about the possible role of these swabs in HCV transmission. The first relates to the extent to which reuse of a bloody swab previously used by another injector occurs. Research indicates that few PWID practice or understand proper use of swabs and that unintended use of other injectors’ equipment may occur in the context of an injection setting [18, 19]. The second question is whether alcohol swabs, which contain a 65%-70% solution of isopropyl alcohol (the 2-propyl alcohol shown to be an effective biocide in the article by Dörrbecker and colleagues), will inactivate HCV. This begs a further question about the harms versus the benefits of using an alcohol swab after injection (rather than a cotton swab as currently recommended) and leads to weighing the potential harm in delaying coagulation and risking possible blood exposure to another injector against the potential benefit of using the swab to collect blood from the injection area and inactivate any HCV present. Confirming these results with epidemiologic studies may take time, however. Ideally, one would want to study the association between swab sharing and HCV seroconversion in a cohort of PWID where there is a low prevalence of syringe and container sharing and at least a modest amount of swab sharing. Indeed, the attribution of HCV seroconversion to filter and container sharing in epidemiologic studies was aided by the reduction in syringe sharing that followed the HIV epidemic, which created cohorts that included sufficient numbers of PWID who shared one but not the other. Whether similarly ideal circumstances exist for the study of swabs and HCV is unknown. As was shown in earlier studies of the use of disinfectant bleach to prevent HIV transmission via syringes shared by PWID, a chasm may separate laboratory research findings (effective) from real-life settings (ineffective) [20]. The 2 new studies in this issue of the Journal lean toward bridging this chasm, but confirmatory epidemiologic studies are clearly needed to link virology and ethnography to seroconversion risk.

The Thibault et al study [17] also raises questions about whether the results would differ if the investigators had measured or controlled for the conditions that the equipment had been subjected to during the interval from last use to collection. A recent article by Paintsil et al [21] measured infectivity of laboratory clones of HCV in syringes and reported that “HCV survival was dependent on syringe type, time, and temperature.” Time and temperature may also have affected detection of HCV RNA in different types of equipment to a varying degree. Syringe type can determine the amount of blood remaining in a syringe when the plunger is fully inserted after injection, and this can range between 2 and 84 µL [22]. However, the design of the study did not permit interpretation with respect to these modifying factors.

Let us consider again the agent-host-environment model of HCV transmission as a framework for HCV prevention. Little can be done to alter the infectivity of HCV, which is a function of the relatively high concentration of virus in the blood of chronic carriers. Stability of the virus in the environment can be affected by time, temperature, and the application of biocides; these new studies by Dörrbecker et al and Thibault et al suggest that cleaning cookers or perhaps impregnating injection equipment with safe biocides may help reduce the incidence of new infections. Promoting safe swab use to emphasize avoidance of reuse seems a prudent measure, even in the absence of confirmatory findings. Providing treatment to eradicate HCV infection in large numbers of PWID could conceivably reduce prevalence [23], but modeling suggests that without expansion of programs promoting safe injection, no amount of HCV treatment will reduce the prevalence of infectious carriers to a meaningful degree [24]. Recent studies, including a meta-analysis, showed that multicomponent interventions that support a range of risk reduction strategies (substance use treatment to reduce or eliminate drug injection, adoption of safe injection practices through the provision of sterile syringes and drug-preparation materials, or behavior-change counseling) were highly effective in preventing HCV seroconversion in PWID [25, 26]. Thus, although HCV is a formidable agent seemingly designed to survive in this human niche, new knowledge may reduce the agent’s stability, change host behavior, and lower the prevalence of infectious HCV in people and the environment to ultimately shift the balance in favor of HCV control.

Notes

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References


