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Fire in the vein: Heroin acidity and its proximal effect on users' health

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Abstract

The loss of functioning veins (venous sclerosis) is a root cause of suffering for long-term heroin injectors. In addition to perpetual frustration and loss of pleasure/esteem, venous sclerosis leads to myriad medical consequences including skin infections, for example, abscess, and possibly elevated HIV/HCV risks due to injection into larger jugular and femoral veins. The etiology of venous sclerosis is unknown and users' perceptions of cause/meaning unexplored.

This commentary stems from our hypothesis that venous sclerosis is causally related to heroin acidity, which varies by heroin source-form and preparation. We report pilot study data on first ever *in vivo* measurements of heroin pH and as well as qualitative data on users' concerns and perceptions regarding the caustic nature of heroin and its effects.

Heroin pH testing in natural settings is feasible and a useful tool for further research. Our preliminary findings, for example, that different heroin source-forms and preparations have a two log difference in acidity, have potentially broad, vital and readily implementable harm reduction implications.

Keywords

Heroin; Injection drug use; Risk environment; Skin and soft tissue infection; Venous sclerosis; Vein care; Vein damage; Vein loss

Introduction

The needle and the damage done (Neil Young)

> I've seen the needle and damage done
>
> A little part of it in everyone…
>
> – Neil Young

Access to functioning veins is a primary concern among people who inject drugs (PWID) (Harris & Rhodes, 2012). Loss of peripheral venous access can lead to frustration and pain,

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increased time spent in search of patent veins, multiple injection attempts and the use of smaller veins in the hands, feet and legs in search of a successful injection (Ciccarone, 2013). Ultimately, frustration can lead to desperation and a transition to injection into central veins including the femoral (Coffin, Coffin, Murphy, Jenkins, & Golden, 2012) and neck veins (Bourgois & Schonberg, 2009). PWID may also inject into sub-cutaneous tissue (aka "skin-popping") as well as muscle (aka "muscling") when venous access fails (Ciccarone et al., 2000, 2001).

Venous sclerosis, that is scarring of veins and loss of functionality, can lead to myriad medical consequences for example deep venous thrombosis secondary to repeated femoral injections (Maliphant & Scott, 2005; Mackenzie, Laing, Douglas, Greaves, & Smith, 2000) and particularly the spectrum of skin and soft tissue infections (SSTI) (Ciccarone et al., 2001). In some locations, SSTI, predominantly the clinical entities of abscess and cellulitis, are a common reason for hospitalization of PWID (Ciccarone et al., 2001; Ebright, 2002) and emergency department visits (Ciccarone et al., 2001; Palepu et al., 2001), leading to high societal (Ciccarone et al., 2001; Takahashi, Maciejewski, & Bradley, 2010) and personal costs, such as skin grafting and limb amputation (Bourgois & Schonberg, 2009; Messac, Ciccarone, Draine, & Bourgois, 2013). The prevalence of SSTI varies: in the North American context point prevalence by clinical exam ranges from 10% (Vancouver) (Lloyd-Smith et al., 2008) to 19–32% (San Francisco) (Binswanger, Kral, Bluthenthal, Rybold, & Edlin, 2000; Ciccarone et al., 2000); similarly, self-reported prevalence ranges from 11% (NYC, past 6 months) (Vlahov, Sullivan, Astemborski, & Nelson, 1992) to 27% (San Francisco; past year) (Ciccarone et al., 2000) and self-reported lifetime prevalence approaches 70% (San Francisco) (Ciccarone et al., 2000). In the UK context 29% (Public Health England, 2012) to 36% of surveyed PWID reported an SSTI symptom in the past year (Hope, Kimber, Vickerman, Hickman, & Ncube, 2008). A recent US national study found a doubling of hospitalization rates for heroin-related SSTI and confirmed the geographic disparity noted above: US cities with a dominance of Mexican-sourced "Black Tar" heroin (BTH) had 40% higher rates of SSTI compared with cities supplied by Colombian-sourced powder heroin (Unick, Rosenblum, & Ciccarone, 2015).

Documented infectious agents for SSTI include skin commensal bacteria, for example, *Staphylococcus aureus* (Bassetti et al., 2004; Summanen et al., 1995), including methicillin-resistant *S. aureus* (MRSA) (Fleisch, Zbinden, Vanoli, & Ruef, 2001), as well as oral commensals (Summanen et al., 1995). In the US context BTH, colloquially named by its color and consistency when warm, has been associated in case-series with less common forms of SSTI due to exogenous contamination with hardy spore-forming Clostridium species, including *C. botulinum* (causing wound botulism) (Gollober et al., 1995; Passaro, Werner, McGee, Mac Kenzie, & Vugia, 1998) *C. tetani* (tetanus) (Centers for Disease Control and Prevention, 1998), *C. perfringens* (myonecrosis) (Bangsberg et al., 2002) and. *C. sordellii* (necrotizing fasciitis) (Kimura et al., 2004). In the UK a notable community outbreak of SSTI among PWID had a high mortality rate with evidence of *C. novyi* and *C. perfringens* infections (Centers for Disease Control and Prevention, 2000). Europe has additionally seen a re-emergence of deadly anthrax infections among PWID, with 15 cases reported in the 2012–2013 year; eight in the UK, four in Germany, two in Denmark and one in France (Public Health England, 2012).

BTH may also be linked with common SSTI infections, that is abscess and cellulitis, through injection practices. Ethnographic work among homeless heroin users in San Francisco highlighted practices which likely lead to contaminated injections: reuse of syringes, use of unclean water, cookers or filters, as well as licking needles (Bourgois, 1998). A related, more clinically focused ethnographic study revealed additional injection-related practices which may be involved in abscess formation including: needle contamination by mouth, finger or surface contact; and venous sclerosis leading to multiple injection attempts per session, as well as "missed shots" or perivenous injections (Ciccarone et al., 2000). A large survey of street-recruited PWID in San Francisco found a high frequency of these vein loss-related risk behaviors (past 30 days) among those with clinically diagnosed SSTI: 45% injected subcutaneously/intramuscularly; 35% typically poke their skin >3 times to hit a vein; 25% had >3 missed shots. Interviewed PWID attributed their collapsing veins and abscesses to using BTH (Ciccarone et al., 2000). A systematic review of the published literature (Cohen, Reingold, & Ciccarone, 2012) identified four studies which examined the relationship between subcutaneous/intramuscular injection and SSTI: all were in US locations where BTH is common; all found statistically significant ORs ranging from 2.1 to 15.5 (Binswanger et al., 2000; Murphy et al., 2001; Passaro et al., 1998; Phillips & Stein, 2010). Among UK heroin injectors, injection into the hand, leg or groin (presumably following loss of preferred antecubital (elbow) veins) were independent predictors of self-reported injection site infections (Hope et al., 2008). Other known or hypothesized etiological factors in SSTI include high frequency of injection, lack of skin cleaning prior to injection (Ciccarone et al., 2001; Vlahov et al., 1992) use of unsterile drugs (Ciccarone et al., 2001; Gordon & Lowy, 2005; Murphy et al., 2001), and injection of cocaine (Murphy et al., 2001).

A complete understanding of the geographical disparity and etiology of SSTI eludes us since we cannot completely tie together the distal risks of infectious agent and "risk" behaviors without understanding venous sclerosis and accordingly – a more proximal etiological factor: heroin source, form and chemistry.

Heroin in "Black, Brown and Beige" (Duke Ellington and Mahlia Jackson)

An understanding of the political economy of global heroin is central to elucidating its structural role in the risk environment for PWID (Ciccarone, 2005, 2008; Rhodes, 2004). Heroin is sourced from four geographical regions: Southwest Asia (largely Pakistan and Afghanistan), Southeast Asia (Burma, Laos and Thailand), South America (Colombia) and Central America (Mexico). The expansive forces of globalized commerce combined with contractive and honing interdiction forces have created channels in the distribution of heroin leading to "segmented" markets with regional suppliers intertwined with "exclusive" retail markets (Ciccarone, 2005, 2008; Paoli, Greenfield, & Reuter, 2009). The schematic view of this shows Southwest Asian heroin retailing in Europe and the UK, Southeast Asian heroin going to Australia and Western Canada; and while heroin sourced from Colombia and Mexico all ends up in the US, each goes to its exclusive market: the former to the eastern half and the latter west.

Form follows source: each of these sourced heroins has different physical and chemical properties, including physical state at varying temperatures and chemical character for example HCL salt or base form, which affects water solubility, and purity (Ciccarone, 2008). Function follows form: Southwest Asian heroin from Afghanistan/Pakistan (beige-brown powder with a range of purities), requires moderate heating and the addition of acid to solubilize (that is it has a basic, or high pH, chemistry); Southeast Asian heroin (white fine powder with a range of purity that can go quite high) is cold water soluble in its acidic state that is salt form; heroin from Colombia (tan coarse powder with a wide range of purity) is warm water soluble in its acidic salt form; heroin from Mexico, aka BTH, (brown-black; solid or crumbly, hard at cool temperatures and tar-like when warm/moist; limited in purity to low-moderate) requires high heat to go into solution despite its salt form. Heroin in its base form can be smoked, that is it can handle high heat, vaporizes slowly after melting and yields reasonable heroin smoke concentrations; salt form heroin yields lower smoke concentrations (Strang, Griffiths, & Gossop, 1997). Anecdotally, BTH is reportedly smokable (data unreported) however, there is no published documentation of sustained community practice of heroin smoking in the US.

Ciccarone and Bourgois have developed a structural > chem-chemical > social > behavioral hypothesis to explain the geographic variation in HIV prevalence among PWID in the US (Ciccarone & Bourgois, 2003). In this view, BTH is protective for HIV because its physical/chemical characteristics lead to personal but culturally reproduced behaviors which can reduce HIV transmission. The heating required to put BTH into solution is sufficiently high to kill HIV, its inherent acidity aids in this process; its sticky nature leaves a residue in syringes requiring vigorous rinsing prior to reuse (thus lowering viral load); it also is a structural force in greater syringe turnover. San Francisco PWID' stories and clinical observations support the notion of BTH leading to venous sclerosis, in turn causing dependent users to inject intramuscularly/subcutaneously which is less efficient in viral transmission than intravenous injection. Harm reduction approaches suggest BTH can be smoked, insufflated (nasal administration/snorted) or keistered (anal administration/insertion) although only a small minority of users does so (Ciccarone, 2008). That BTH is associated with bacterial infections due to *Clostridium* (and perhaps common SSTI as well) can also be explained: its tar-like nature picks up all manner of foreign material including soil. Spore-forming bacteria like *Clostridium*, can survive in dry harsh conditions for a while; meanwhile the heating required to put BTH into solution, while insufficient to kill *Clostridium*, may actually awake the spores from their dormant state into a more active pathogenic state. When PWID inject contaminated BTH subcutaneously (into the sub-dermal fat layer) they are placing the infectious agent – an obligate anaerobe – into a well-suited low oxygen state tissue.

The acidity of heroin

How does acidity of heroin and heroin solution play into this? It may be the missing link between different heroin source-forms leading to varying physical damage and the "risk" behaviors that follow the diminution of venous access. In other words the structural risks imposed by heroin type then influence the risk choices of users. Some IDU may transition back to smoking for example, or seek treatment, when their veins become too damaged. In

others, peripheral venous damage can lead to heightened SSTI risk through perivenous, subcutaneous or intramuscular injection, or to potentially greater blood borne virus transmission through greater blood exposure and contamination in the persistent search for patent veins (Harris & Rhodes, 2012).

Venous sclerosis has, in observational reports, been attributed to crack injecting and the overuse of citric acid in heroin and crack, aka “snowball”, preparation in London (Rhodes, Briggs, Kimber, Jones, & Holloway, 2007) and use of BTH (Ciccarone et al., 2000; Ciccarone, 2008), however there has been no systematic study of drug solution acidity. The authors, following independent observations of heroin use in multiple countries and settings, hypothesized a causal link between heroin acidity and venous sclerosis; lead author DC proposed and conceived the following *in vivo* study.

We devised a pilot study to measure street heroin pH (a measure of acidity) and explore users’ perception of the caustic nature of heroin and its effects. We chose Philadelphia and London to start due to access to study populations, different heroin source-forms and the intrigue of the varying acids (by type and amount) London PWID use to solubilize heroin. This study aims to 1, highlight the plausibility of heroin acidity as an explanatory component of the heroin type-to-behavior risk environment equation and 2, document the feasibility of pH testing of street heroin in natural settings.

Methods

This pilot project is an off-shoot of a multidisciplinary exploration of the medical consequences of use of different source-forms of heroin in the US (PI: D. Ciccarone) which combines macro-level epidemiological modeling (see Unick, Rosenblum, Mars, & Ciccarone, 2013; Rosenblum, Unick, & Ciccarone, 2014) with micro-level qualitative investigations in two US cities, Philadelphia and San Francisco, each with an exemplar of heroin type (see Mars, Bourgois, Karandinos, Montero, & Ciccarone, 2014; Rosenblum et al., 2014). The qualitative methods include participant-observation ethnography (Rosenblum et al., 2014), qualitative interviews (Mars et al., 2014) and videography of heroin injection sessions. This pilot study took place in London during July 2012, with comparison data drawn from a field trip to Philadelphia the month prior, and combines qualitative interviews, field notes and *in vivo* pH heroin testing.

Sampling and recruitment

The London participants are a convenience sample of ten individuals recruited by author MH out of her connections with heroin users from her other studies (for example, see Harris & Rhodes, 2012, 2013). Participants were over 18 years of age, spoke English and were predominantly living in council housing in South East and North London; they all injected heroin or heroin/crack mixtures. For comparison, two individuals were recruited who mainly inject prescribed pharmaceutical opiates; one, heroin (UK name: diamorphine), the other methadone. (UK name: dolophine). In Philadelphia, the lead author (DC) with the assistance of local ethnographers recruited a convenience sample of heroin injectors, English speaking and over 18 years of age, from the ongoing US qualitative study to participate in the additional pH pilot.

Data collection

Data collection consisted of in-depth qualitative interviews, videography of injection sessions and micro-sample heroin pH testing. The semi-structured interviews focused on brief life course with heroin including initiation up to current typical use; heroin preparation, for example use of heat and added acid and volume of water; perceptions of heroin quality, source, and changes over time; effects, both desired and undesired on them; injection sites and any scarring/damage; and vein care. Fieldnotes were written after each day of research to ground the participants, researchers and the study in a socio-cultural context and to record recollections, both seen and heard, that may have escaped the audio and video recorders.

pH testing

The demands of *in vivo*, or field-based, illicit drug testing are challenging to meet. One needs a pH meter which is portable and durable yet able to measure minute samples of drug. It also needs to be highly and easily cleanable to avoid cross-contamination of samples and transmission of infectious agents. After exhaustive searching author DC chose the Sentron Hot-line CupFET pH probe: a high temperature rated, non-glass, solid state probe meeting the entire above requirements esp. small sample – it can measure volumes as small as 10 μl (www.Sentron.nl). The sensor was cleaned and calibrated according to the manufacturer's requirements prior to each sample. Participants who consented to the pH study donated 1–2 drops (that is 1–2 "units," ~10–20 μl) from a typical 1 ml (100 units) syringe of their freshly prepared heroin dose, placed into the "cup" of the pH probe. The reading was instantly taken from the pH meter and the sample discarded (rinsed onto absorbent disposable lab towels).

Video recording of injection technique was performed as unobtrusively as possible. Still and video photography focused on preparation of heroin solution, injection sites and procedures; details such as faces, identifying marks and settings were excluded as much as feasible. For the purpose of this pilot study the recordings were used to refresh our memories while writing fieldnotes. The formal comparative analysis of injection techniques is for a future paper.

Analysis

Ten current injectors (street heroin $n = 8$, pharmaceutical heroin $n = 1$, methadone $n = 1$) were interviewed in London and 13 in Philadelphia (all heroin). Audio recordings were transcribed verbatim and NVivo 10 software was used to assist with data organization and retrieval [(2012) QI. *NVivo 10 qualitative data analysis software*. Doncaster, Australia: QSR International Ltd.]. The first author (DC) carried out all of the coding. 'Open coding', described by Strauss and Corbin, was used to identify concepts and categories and systematically apply them throughout the transcripts (Strauss & Corbin, 1990). Fieldnotes, constructed collaboratively by both authors at the time of data collection, were reviewed as part of the analysis. Themes related to heroin's effect on the body, specifically vein health or damage, challenges and responses regarding venous access and care were discussed by the authors and summarized here.

The within-city pH data are presented descriptively. To test the between-city difference in the mean pH of the heroin samples we used both the parametric *T*-test and the Wilcoxon-

Mann–Whitney test. *T*-tests are robust with samples sizes of 15 or more when the data does not contain outliers (Moore & McCabe, 1998). The Wilcoxon-Mann–Whitney test, a non-parametric statistical test, is useful for distinguishing differences in groups with small sample sizes or whose samples are not normally distributed (Conover & Iman, 1981). Both tests were used due to the small sample size and as a robustness check.

Human subjects

Approval for this research was obtained by the institutional review boards of both University of California San Francisco and London School of Hygiene and Tropical Medicine. Given the sensitive nature of the research and vulnerability of the participants, a waiver of written informed consent was obtained. Participants verbally consented to each part of the study separately and could refuse any part. No identifiers were obtained so anonymity could be protected. In the US, a Federal Certificate of Confidentiality was obtained from the National Institutes of Health.

Results

Golden Brown (The Stranglers): Shooting acidic “gear” in London

London street heroin, aka “gear,” reportedly from Afghanistan/Pakistan, required the addition of acid to dissolve into water; most commonly used were pharmacy obtained citric acid powder (common acid typical of citrus fruits) and ascorbic acid (vitamin C) powder. Most participants didn’t choose which acid to use as they simply accepted the one distributed for free, in individual-use sachets, as part of safer injection kits dispensed by pharmacies. The pharmacies in each borough more-or-less only distributed one form of acid and users who wanted a different one paid for it or sought it out from a different borough.

The goal of adding acid was to put the heroin into solution; a magical transformation of chalky brown heroin powder (+ water + acid + heating) into an iced tea-colored transparent liquid. One 44 year-old male respondent using heroin for 20+ years described the color in reference to the Strangler’s 1982 song “Golden Brown” which he states is an allegory about heroin. He tells us that this describes the color of the best heroin: golden or orange-brown.

> Golden Brown finer temptress
>
> Through the ages she’s heading west
>
> From far away, stays for a day
>
> Never a frown with Golden Brown
>
> – Golden Brown – The Stranglers, 1982

Some participants preferred to use ascorbic acid to dissolve heroin and believed that citric acid was responsible for the vein damage they have suffered. For example, this exchange with a 43-year-old female respondent highlights their concern with the effects of “brown” London heroin mixed with citric acid on her veins:

> Q: What substances would you attribute the loss of veins to?

A: The brown heroin. And I knew it was the citric, I was pretty certain it was the combination. Because it just made sense to me, it stung like fuck when you shot it up… And I had no problems with my veins whatsoever when I was in Australia.

Q: What were you using in Australia?

A: White, so you didn't need anything to mix with it, no heat, no nothing just water.

All participants, except one, struggled with venous sclerosis and most injected into their groin, that is femoral, veins. Many, like this 29 y.o. male respondent, complained of vein/subcutaneous tissue burning esp. when injecting with citric-acid-dissolved heroin.

Q: So we were just talking about the use of various acids here to dissolve the heroin. You were going to tell me something about it?

A: I think there's a belief that the Vitamin C is less damaging to the veins than citric acid is because yeah you can physically feel the burn on citric when you're shooting it up whereas with Vit C you don't feel that as much.

Or this male respondent of unknown age:

Q: And what do you think about – you were pointing to the gear on the table – the street heroin?

A: The street gear yeah I don't think that's very clever for your veins because you know of the Vitamin C or citric acid that people are using to dissolve it you know.

Q: If you had a choice which one Vitamin C or citric?

A: I think probably Vitamin C.

Q: Why?

A: I don't know it's just from when I did used to use street smack it just seemed to burn less, you know if you missed.

Alternatively, this 53-year-old woman said this:

Q: Why do you suppose the veins disappear?

A: I don't know. It's a bit of a mystery to me all that stuff. I think citric certainly doesn't do it any favors does it?

A review of the video and still photography clinically confirms apparently sclerosed peripheral veins and swollen hands and legs (due to resultant lymphedema) in most of the London participants.

Strategies to reduce vein damage varied: some preferred ascorbic acid, while others used smaller judicious amounts of either acid. Proponents of using citric acid stated that it always worked to put heroin into solution. Others, like this 44 y.o. male respondent, said they would coach a newer injector to be patient, use incremental amounts of acid and wait for the solution to "clear."

Q: And how did you learn how much citric to use? You sound like you want to use a small amount, you want to use a pinch but you could probably use too little or too much. How do you know how much to use?

A: You've just got to guess really. If you use too little then they'll be a lot of grunge floating on the top of the little cup so you need to use a little bit more. You just put like a little pinch of it in it at a time until you've used the right amount till it's gone clear. But if it stays cloudy I wouldn't touch if it was cloudy, I would just leave it or put more citric in it until it went clear.

The one person, a 57 y.o. male, with little apparent vein damage despite 35 years of heroin injection, was a model of safer injection technique: fastidious hygiene, including robust skin cleaning and use of sterile needles, rotating veins, not using kitchen acids, for example vinegar or lemon juice, to dissolve heroin, high volume (that is dilute) injections, muscular workouts, and cocoa butter moisturizer.

On the "Streets of Philadelphia" (Bruce Springsteen)

At night I could hear the blood in my veins black and whispering as the rain

– Streets of Philadelphia – Bruce Springsteen, 1993

Heroin in Philadelphia is predominately sourced from Colombia. It is already in the hydrochloride (salt) form so additional acid is not required for it to go into solution. The few users who injected heroin exclusively reported few if any vein problems while most of the speedball injectors blamed frequent cocaine use for their vein loss. A 45 y.o. man bluntly declared, "Cocaine is what is killing veins," and while he blamed it on the cut, others claimed a burning quality:

Q: Do you think that heroin affects your veins differently than say cocaine?

A: Differently yeah I think cocaine is worse. I think cocaine just because I think it's more caustic. I think it like it has more of an acid quality where it would eat through your veins more or something.

As veins were lost injection risks intensified. In Philadelphia vein-frustrated injectors resorted predominately to injecting into the external jugular veins in their necks while the preferred larger veins for London injectors were the femoral veins in their groins. Most understood the risks of these central vein injections but were compelled by their dependency and withdrawal symptoms – and lack of patent surface veins – although some preferred neck injections for the more intense rush they provided.

Heroin pH

The pH of street heroin samples in London ($n = 8$) varied from 2.1 (prepared with citric acid) to 3.5 (ascorbic acid). (Table 1) [Note: the pH scale runs from 1 to 14 with 7 being neutral; 1, extremely acidic and 14, extremely basic. It is a log scale so for example a sample with a pH 2 is 10× more acidic than that with a pH of 3.] The mean pH of the citric prepared samples was 2.6 ($n = 5$) – the same acidity level as wine vinegar (Fig. 1). Heroin prepared with ascorbic acid had a mean pH of 3.4 ($n = 3$); almost a log less acidic than those samples prepared with citric acid. Low volume (200–900 μl) heroin solutions appear to be more

acidic than high volume (>1 ml (that is 1000 μl)) solutions; this being more apparent with citric acid prepared solutions. Our single sample of pharmaceutical heroin (diamorphine) in contrast had a pH of 4.8 ($n = 1$) and according to our participant never caused a burning sensation. Even less acidic was the pharmaceutical methadone sample at pH 6.1 ($n = 1$).

In Philadelphia the mean pH of street heroin samples was 4.7 ($n = 13$); similar to that of the UK pharmaceutical heroin sample. The addition of powdered cocaine (hydrochloride salt) increased the acidity of the resultant "speedballs" to a mean pH of 4.2 ($n = 2$). This is in contrast to the London heroin plus crack cocaine (bicarbonate form) "snowball" sample ($n = 1$) which had a pH of 3.2; the addition of crack mellowed out the acidity of that sample of heroin (pH 2.6; $n = 1$). The five cocaine samples in Philadelphia had a mean pH of 3.5; the acidity of which supports users' claims of causticness.

The London heroin samples ($n = 8$) had a lower mean pH of 1.8 units compared with the Philadelphia heroin samples ($n = 13$). The difference between the London sample and the Philadelphia sample is statistically significant using the parametric T-test ($p < 0.001$) and the non-parametric Wilcoxon-Mann–Whitney test ($p = 0.0002$).

Discussion

To our knowledge this paper reports the first ever *in vivo* measurements of street heroin pH. We chose *in vivo* testing, in which we measured the pH of heroin as participants prepared and used it, over *in vitro* testing (where careful measurements are made in a lab controlling for volume of drug, volume of water, temperature, etc.) for veracity sake that is to get an idea of the acidity of heroin as users are actually putting it into their bodies. Our work confirms laboratory, that is *in vitro*, measurements on the varying acidity of brown heroin solutions in the UK (Scott, 2008).

Heroin acidity: A novel concern in users' risk environment

> When I put a spike into my vein and I tell you things aren't quite the same
>
> – Heroin–Lou Reed, 1967

Street heroin in London is highly acidic, with pH close to vinegar when prepped with citric acid. Citric acid heroin solution is almost 10 times more acidic than that with ascorbic acid. Volume of solution matters but perhaps not as much as type of acid used for example small volume (200–900 μl) heroin + citric solutions had a mean pH of 2.3 compared with large volume preparations (1–2 ml) with pH 2.8. Many London participants believe or agree that the citric acid is destroying their veins and they have been advised, or would advise others, to use less. They try to modulate the amount but the anticipation of the shot may lead to excess use in order to speed up dissolving the heroin. No one knows how much acid to add; "less is better," but it is always a guess. Participants have diverse ideas on how to measure out a small dose of acid: a "pinch" (between fingers but what if your fingers are big?) or a "tap" of the sachet (but what if you tap too hard?); both of course are imprecise and likely excessive. Philly heroin users blame vein loss on use of cocaine alone or in speedballs; cocaine is, at the time of this study, very cheap and speedball injecting is very popular. Philadelphia native acidic street heroin is statistically lower in acidity than London user-

prepped street heroin and prescription heroin is descriptively of milder acidity than London heroin dissolved with added acid. Our findings suggest that the “magic” number is a pH of ~4.8 – that of our single pharmaceutical heroin sample. A pharmaceutical textbook states the pH of pharmaceutical heroin is 4 (Florence & Attwood, 1998); our *in vivo* sample may have been buffered with tap water. Solution acidity is not the only consideration in optimum chemical safety for venous infusions; osmolality should also be considered (Kuwahara, Asanami, Tamura, & Kaneda, 1998; Scott, 2008).

Heroin in brown, black and beige (remix)

Reducing the acidity of heroin has salient public health implications. From a harm reduction perspective low-acid heroin can reduce the suffering from vein loss, that is frustration and pain, that PWID experience. In addition if our structural > chem-chemical > social > behavioral hypothesis holds, less vein loss means fewer infections and inflammations for example abscess, cellulitis, phlebitis, etc. Lowering heroin acidity has unknown potential effects on the transmission of HIV. Injection into central for example femoral veins, following peripheral vein loss, was common in our limited London sample. These injections require larger barreled syringes with more “dead space” in the hub connecting syringe and needle. Use of higher dead space syringes has been linked to higher HIV transmission: a two log greater risk compared with low dead space 1 ml TB/insulin syringes, due to holding blood and infectious cells. (Ciccarone, 2013; Bobashev & Zule, 2010; Zule & Bobashev, 2009; Zule, Cross, Stover, & Pretorius, 2013; Zule & Desmond, 1997). In addition, blood borne disease transmission can also be promoted by the messiness of the search for venous access: multiple injection attempts lead to greater blood exposure and risk of cross-contamination of injection paraphernalia (Harris & Rhodes, 2012). On the other hand, *in vitro* experiments show that HIV contaminated syringes are significantly less likely to yield recoverable HIV when rinsed with citric acid solutions of pH at or less than 2.3 (Heimer, Kinzly, He, & Abdala, 2007).

Some source-forms of heroin are smokeable and delaying initiation of injecting or reducing the amount of injecting can reduce the potential of blood borne virus transmission. Heroin can be smoked for many years prior to transitioning to injection (Strang et al., 1997). Heroin in the UK and Europe, originating from Afghanistan/Pakistan, is smokeable in its native basic form (that is prior to the addition of acid). In the UK there is a focus on switching people back from injecting to smoking heroin for example through the supply of high quality foil and teaching people attending syringe exchange how to “chase” that is smoke more effectively (Public Health England, 2014). Colombian-sourced heroin, predominant in the eastern US cannot be smoked as its melting point and vaporization point are too close. And while “Black Tar” heroin in the western US reportedly can be smoked it is undesirable to do so due to irritation and strong smell (data unreported).

Implications

But they forgot to mention the way

That the morphine makes the pain go away

And how I’ll always remember the good times in my spine

And the holes I burned in my brain…

– Acid Song – Johnny Hobo and the Freight Trains, 2005

Basic heroin sourced from Southwest Asia when transformed into injectable heroin solution by typical London injectors is quite acidic. This form of heroin is found throughout Europe so the implications stemming from this study are potentially widespread. Is the acidity of this form of street heroin mutable? Pharmaceutical heroin is less acidic as is Colombian-sourced heroin found in Philadelphia. Indeed one could surmise from our pilot that pharmaceutical heroin's pH of 4.8 should be the goal pH for street heroin. If our pilot findings are found to be reproducible and generalizable then some interventions become apparent. Structurally, one fix is potentially easy: change citric acid distribution to ascorbic acid. Potential concerns of this wholesale change include cost (minimal) and effectiveness (for example stemming from concerns regarding dissolving lower quality heroin), leading some to revert back to using lemon juice or other kitchen acids (and increasing the risk of fungal infections) (Bisbe et al., 1992). Reducing the dose of either acid (not measured in this study) esp. citric, may be helpful. "Do-it-yourself" opioid injectors in New Zealand using very small amounts of citric acid suffer little vein damage and rarely get SSTI (Harris, 2013). This can be accomplished by reducing the size of the packets; supplying instructions that a "pinch" or a "tap" will do; or including a tiny scoop to measure out minute doses. Additional behavioral interventions include: increasing the solution volume to decease acidity; adding acid in small increments; being patient while looking for clearing. Cultural beliefs regarding the effectiveness of ascorbic vs. citric will need to be addressed for example with peer-involved education campaigns utilizing notions of health promotion, injecting ease and pleasure. The pH of heroin using ascorbic acid (3.4) is still too acidic for healthy veins. Finding another mild, yet effective organic acid is desirable. Buffering with sodium bicarbonate (baking soda) is a potential solution but how it affects solubility and users' perception of drug effect are open questions.

Feasibility and future studies

The field measurement of heroin pH is feasible. Users' concerns about loss of drug were minimal and indeed overwhelmed by the positive interest in and curiosity about the study. Infectious safety concerns were theoretical only with the use of strict field-lab protocols. This *in vivo* pH testing has now been demonstrated among housed heroin users in London, as well as open space injectors in Philly.

This study fits into lead author DC's career objective to integrate clinically oriented ethnographic research with public health epidemiology and improve the latter by providing socially plausible notions and measures of risk and thus helping avoid meaningless multivariate associations (Bourgois et al., 2006; Ciccarone & Bourgois, 2003; Ciccarone, 2003). Both authors hope to use insights gained from their social research to aid the provision of meaningful and effective harm-reduction orientated interventions (for example see Messac et al., 2013). A truly integrated social science for harm reduction approach requires cohesive and complex understandings that may involve integration of the basics

sciences (biology, chemistry), clinical sciences (pharmacology, infectious disease), public health (ethnography, social epidemiology) and political economy (politics, economics).

The findings underpinning this Commentary are exploratory and hence limited in their inference and generalizability. The measuring of heroin pH in natural settings entails a loss of control over typical lab conditions including manipulation or measurement of potentially important variables for example solution volume, temperature, osmolality, quantity of added acid, drug diluents and adulterants, etc. This loss should be weighed against the benefit of obtaining more realistic measurements. Naturalistic research of this complexity is challenging on several levels, not the least of which is funding, yet we met one of the initial study objectives that is demonstrating feasibility of *in vivo* heroin solution pH measurements. The fulfillment of the other study aim, that is plausibility of the heroin acidity and venous sclerosis hypothesis, is left up to the judgment of the critical readership. Additional limitations of this preliminary study include its convenience sampling and low sample size. Further evaluation of our hypothesis requires more measurement: sample size needs to be expanded with systematic and rigorous measurements across location. Additional locations and heroin source-forms need investigation; BTH, which has been associated with vein loss and soft tissue infections, is an obvious target. Epidemiological or modeling studies utilizing heroin pH data, or self-reported use of acidifying agents, may begin to establish associations with clinical outcomes of vein loss or SSTI. Each of the suggested structural and behavioral interventions outlined above requires evaluation for effectiveness and acceptability.

Harm reduction has greater potential to affect positive change as its messages are connected to users' concerns (Harris & Rhodes, 2012). Addressing vein health and thus heroin acidity is in line with this philosophy and thus has high potential to affect PWID' lives for the better.

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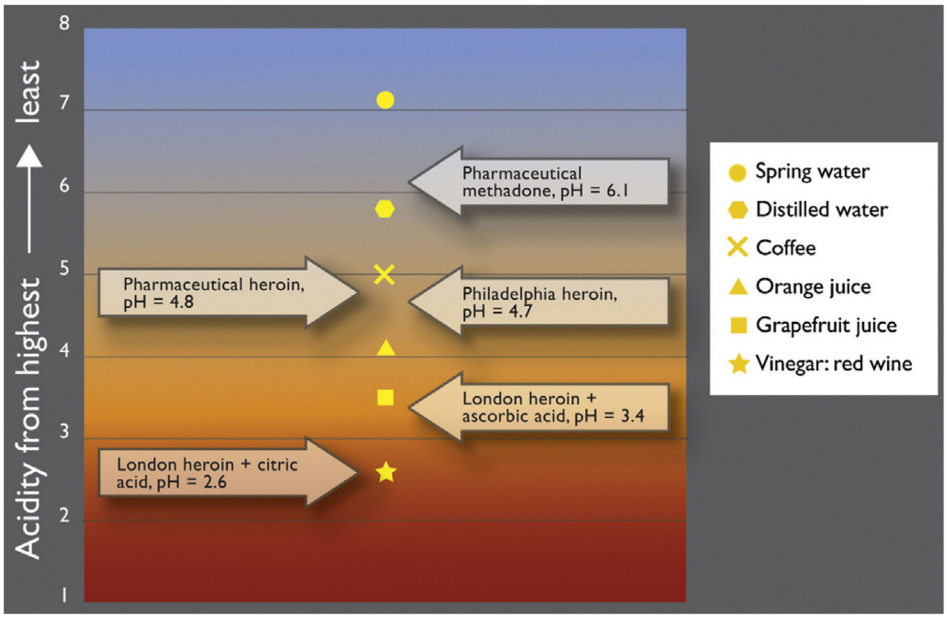

Fig. 1.
pH of heroin solutions vs. common liquids.

Table 1

Heroin pH.

Location	Acid added	Drug	Volume L/H[a]	Sample size	pH
London					
	Ascorbic acid				
		Heroin	Low	1	3.4
		Heroin	High	2	3.5
		Heroin	Combined	3	3.4
	Citric acid				
		Heroin	Low	2	2.3
		Heroin	High	3	2.8
		Heroin	Combined	5	2.6
		Snowball (crack)	High	1	3.2
	None				
		Pharm. heroin	High	1	4.8
		Pharm. methadone	High	1	6.1
Philadelphia					
	None				
		Heroin	Low	13	4.7*
		Speedball (cocaine)	Low	2	4.2
Standards					
		“Spring” water			7.1
		Distilled water			5.8
		Coffee			5.0
		Orange juice			4.1
		Grapefruit juice			3.5
		Vinegar: red wine			2.6
		Coca Cola			2.5

[a]Low volume = 200–900 μl; high volume = 1–2 ml.

*$p \leq 0.001$, compared with mean London heroin pH ($n = 8$).

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