Reply to 'Addiction as a brain disease does not promote injustice'

Hart replies — I appreciate the interest Bedi et al.[1] have taken in my recent publication describing how viewing drug addiction as a brain disease facilitates social injustice[2].

Bedi et al. contend that "neuroimaging studies have shown that neurobiological function in SUDs differs markedly from healthy individuals"[1]. PET studies[3,4] are cited in support of this claim. The first study found no differences in dopamine (D_2) receptor density or affinity when nine alcoholics were compared with eight controls[3]. But, even when differences were observed between the groups on a secondary measure — the ratio between D_2 receptor density and affinity — data for the majority of the alcoholics looked identical to those collected in controls. In the other study, less striatal dopamine was released in cocaine users than controls[4], which could simply reflect adaptation, such as the development of tolerance. It would be a tremendous stretch to argue that these data demonstrate a diseased brain.

It is true that neuroimaging data alone are insufficient to diagnose drug addiction, or Huntington's or Parkinson's diseases — but Bedi et al. argue that drug addiction is analogous to these diseases. Unlike drug addiction, however, where the majority of patients recover without medical treatment, Huntington's and Parkinson's progress inexorably; they are irreversible and fatal. In Huntington's, striatal atrophy progresses steadily through clinical manifestation of the illness[5]. A similar situation occurs with Parkinson's after more than 30% of dopamine neurons in the substantia nigra have been lost[6,7]. There are no comparable neural correlates associated with drug addiction[8].

Bedi et al. also state that I "set up a dichotomy between the social and the biological as mutually exclusive"[1]. This is untrue. I argued that there needs to be greater parity in the way we fund and think about drug addiction, pointing out that the 'diseased brain' perspective has outsized influence. An argument for a more pluralistic view of drug addiction does not exclude a role for neuroscience, as long as there are data justifying that role. Similarly, it is implied that I do not acknowledge the role of biology in mediating drug effects. This implication ignores the first paragraph of my article, in which I state, "cocaine — and other recreational drugs — temporarily alters the functioning of specific neurons in the brains of all who ingest the drug"[2].

Another argument advanced by Bedi et al. is that drug policy and law enforcement are not influenced by over-interpretation of neuroscience data. This point disregards a burgeoning literature showing how politicians enact misguided drug policy based on these data[9]. The recent actions of Philippine President Rodrigo Duterte represents but one example. Less than a year into his presidency, more than 4,000 people accused of using or selling illegal drugs have been killed. Rodrigo Duterte justifies his actions by stating that methamphetamine shrinks the brains of users, and as a result, these individuals are no longer capable of rehabilitation (http://go.nature.com/2uO4xO9).

While I welcome this debate, the critique does not invalidate my original conclusions: (1) there is virtually no evidence indicating that drug addiction is a brain disease; and (2) neuro-exaggerations have had disproportionate influence on drug research funding priorities and drug policies. It's time for a change. ❐

Carl L. Hart

Department of Psychology and Department of Psychiatry, Box 120, Columbia University, 1051 Riverside Drive, New York, NY 10032, USA.
e-mail: c.hart@columbia.edu

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