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Response to 'Fool's gold: diseased marijuana and cannabis hyperemesis syndrome'

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I very much enjoyed reading the 'Letter to the Editor' by Oscar Armando Dorantes, an archaeologist and alumnus of San Diego State University. An individual who has devoted many hours to understanding this 'subculture' of the history and changes in the growing of marijuana. To add further perspective to the history of marijuana I recently found literature reporting that an archeologist in Israel reported that marijuana residue was detected in artifacts from an ancient temple in southern Israel – this was an 8th century BC shrine at Tel Arad, located about 35 miles south of Jerusalem. This provides the first evidence of mind-altering substances as part of the cultic rituals in Judah.

The concept that Dorantes presents from his well-constructed evidence-based and very insightful commentary is right in line with my feelings as the senior author of the article, 'Cannabinoid Hyperemesis Syndrome' published in the December issue of the *Journal of Investigative Medicine* by Mahesh Gajendran et al.[1] The clinical presentation of CHS is a history of smoking marijuana, at least daily and for at least 3 years. There are acute episodes of vomiting accompanied by abdominal pain lasting 1 to 5 days and then periods of weeks with really no symptoms termed as remission. Why are apparently only some people 'chosen' to have this syndrome when many people are smoking? Some begin smoking at high school and some later on, and continue this 'habit' into their daily and professional lives. It is estimated that at least 25%–30% of all people with this typical history of chronic smokers end up in the Emergency Room in this country with the acute symptoms of CHS. We have hypothesized that stress is a key accompanying component and may lower the threshold for vomiting in a genetically predisposed individual. Treatment regimens combine tapering and eventually stopping marijuana while at the same time reducing anxiety with an agent such as Lorazepam and also a centrally acting agent, a tricyclic, (eg, amitriptyline) combining an antiemetic aspect will decrease brain–gut sensitivity and 'block' this brain–gut connection that results in these typical cycles. However, this hypothesis for me was never quite enough to explain the seemingly erratic and random nature of 'who was chosen' to develop CHS.

It is known that marijuana is stored in fat cells in the brain. This led to another theory to be considered – is CHS a dose-response effect-overtime with enough marijuana being smoked that a subset of people reach their threshold for activating the nausea and vomiting center in the central nervous system. This could be a 'cocktail' of the marijuana use with a genetically predisposed subset who do not metabolize marijuana as well and/or are more sensitive and/or in the accompanying setting of severe stressful/anxiety situations the stage is set to activate the vomiting reflex and hence an episode of CHS. This can be somewhat comparable with alcohol where people can consume a lot without having any complications – both acutely and chronically – while others develop the complications of liver disease or pancreatitis, which we see every day in the hospital when alcohol is 'toxic' and/or cumulative in this subset.

Now let's turn to the 'wildcard' theory. People smoking for years have different sources for their marijuana and as well presented by Dorantes we really have no way of monitoring the status of their marijuana source regarding contamination and other toxins. So a theory I have always had is that toxins in the plant from the various soils and preparation and techniques used to grow and fertilize them could be playing a role. Now Dorantes brings to the table a more unifying theory describing the 'deterioration' and real disease that has been permeating the marijuana industry – Hop Latent Viroid which he describes in great detail and depicts pictorially. This Hop Latent Viroid results in 'diseased' plants which, in turn. are being orally ingested and smoked by the public, setting the stage for adverse events.

In response to the question posed by Dorantes on the first page of his Letter to the Editor – were any samples of marijuana analyzed in a patient or from a patient with CHS? The answer is no. Certainly these revelations by Dorantes suggest that we should begin to incorporate in our medical history questions about marijuana quality for each patient, the source of procurement and how the plants might have been grown.

In summary, for a clinician treating this entity for more than 20 years, there is now some real perspective that can be brought to CHS. The nausea and vomiting we see is not always in

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the setting of classic chronic marijuana use as the time of marijuana exposure can vary and actually can sometimes be of short duration. So now we have a potentially unifying hypothesis which does not compete with the ones already espoused but supplements them by providing the important 'MISSING LINK' for this CHS entity –Hop Latent Viroid inducing "diseased" marijuana plants. We thank Dorantes for this very informative and well-crafted information and congratulate him on his pioneering research.

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REFERENCE

1 Gajendran M, Sifuentes J, Bashashati M, *et al*. Cannabinoid hyperemesis syndrome: definition, pathophysiology, clinical spectrum, insights into acute and long-term management. *J Investig Med* 2020;68:1309–16.