Prohibition and its Discourse of Ganja and Madness.

The medical discourse of ganja use, the brain, madness and cannabis use disorder will be deconstructed by focusing on the article titled: "Heavy cannabis use, dependence and the brain: a clinical perspective" by Emese Kroon, Lauren Kuhns, Eva Hoch, Janna Couisjin, first published 13 August 2019. The authors of the article (Kroon et al.) define the aims of the article as follows: "To summarize and evaluate our knowledge of the relationship between heavy cannabis use, cannabis use disorder (CUD) and the brain." The methods utilized by the study is as follows: "Narrative review of relevant literature identified through existing systematic reviews, meta-analysis and a PubMed search." The authors are then reviewing literature on ganja and the brain towards the creation of a discourse of ganja and brain rooted in evidence, this then is the summation of the evidence base of the discourse of ganja, the brain and madness. Kroon et al. presents a review of the evidence base of the discourse of ganja and the brain in all its aspects driven by the hegemonic discourse of prohibition. Kroon et al. never questions the accuracy of the position of prohibition that ganja is a dangerous drug seen in its tenuous clinging to the position of insisting that chronic CUD amounts to a watered down addiction but you don't use the "A" word as they are yet to prove that with CUD the brain is addicted to ganja, but that does not stop you from repeatedly alleging it. What is revealing in Kroon et al. is the research instrument used in the literature reviewed to drive the discourse of ganja use and madness proclaiming it as fact that ganja use sends you mad, with no causal evidence that proves this as fact. The article states in the section Results as follows: "Although causality is unclear, heavy and dependent cannabis use is consistently associated with a high prevalence of comorbid psychiatric disorders and learning and memory impairment that seems to recover after a period of abstinence. Evidence regarding other cognitive domains and neurological consequences, including cerebrovascular events, is limited and inconsistent. Abstinence after treatment is only achieved in a minority of cases; treatment targeted at reduction in use appears to have some success. Potential moderators of the impact of CUD on the brain include age of onset, heaviness of use, CUD severity, the ratio of THCannabinol to cannabidiol and severity of comorbid disorders." From the outset Kroon et al. admits that there is no causal evidence that ganja in fact triggers mental health issues in the human brain. The prohibition discourse of ganja and madness is based on associational evidence of that, which is only circumstantial at best in the absence of studies to prove that ganja does induce comorbid psychiatric disorders in the human brain. Why base a discourse that assaults ganja as a dangerous drug only on circumstantial correlation when we all know correlation is not necessarily causality? This is a war prosecuted by discourse where no evidence is needed, where association is enough to insist ganja is a dangerous drug and in the propagation of the discourse to the masses association becomes causality, fact backed up by the power of the war on drugs. If association points to, hints at causality why then no research to finally prove beyond the shadow of a doubt that ganja sends you mad? Why hasn't the powerful forces of prohibition especially in the North Atlantic

invested the funds to gather this proof once and for all? This is a discourse of power, it doesn't care about truth, what it desires is social control, exerting hegemony, power refuses to be limited by law. There is at this present time no causal proof that ganja induces madness. The lack of causal proof drives an assault on ganja that demands criminalisation and more so of the policing of ganja use by minors and adults in the presence of children and of placing harsher punishment on the use of high potency ganja especially by minors. This medical discourse insists that heavy ganja use triggers cannabis use disorder and one result of heavy use is comorbid psychiatric disorders and learning and memory impairments. In Addictionology comorbidity is a condition where mental illness and addiction simultaneously present in the patient, impact each other and the strategy of the addiction intervention. Without any causal proof that ganja use results in an addicted brain, in psychiatric disorders and learning and memory impairment this discourse of power has now arrived at comorbidity which is rooted in the fact of an addicted brain. Kroon et al. admits that when ganja use is stopped the comorbidity ends which means that it was in the first place not a true comorbidity premised on the interaction of an addicted brain with mental illness. This is why the discourse speaks of disorders and impairments rather than mental illness, mental disease and arrested development for it is all premised on the use of high amounts especially of high potency ganja and when you stop it simply goes away. Just another discourse of reefer madness. The fact that you can just give up on ganja points to the non-addicted brain, which they must counter by insisting that the success rate of giving up ganja is not high, therefore the brain is addicted and this addiction is so potent that the conventional addiction intervention strategy for ganja is not effective. Kroon et al. without a sliver of causal evidence has now torched the edifice of addictions intervention technology by insisting that high potency ganja when used heavily is as addictive as opiates. This they must do as they are yet to present evidence that the mental illness induced by ganja use is organic as the brain becomes diseased, hence it can only be a disorder. More importantly the discourse of ganja and madness is frantically attempting to stem the tide of the acceptance of ganja as a necessary input to human wellness especially in the USA.
The conclusions of the Kroon et al. article reveals the nature of the discourse of the assault on ganja when it states: "Current evidence of long-term effects of daily cannabis use and cannabis use disorder on brain-related outcomes is suggestive rather than conclusive, but use is associated with psychiatric morbidity and with cognitive impairments that recover after a period of abstinence." The evidence that ganja induces mental illness is then suggestive rather than conclusive willingly embraced by prohibition intent on assaulting ganja by any means necessary. An instrument unleashed to justify the failure to legalize marijuana in T&T is predicated on suggestive evidence rather than conclusive evidence but suggestive evidence is the basis of a lynching, of the State executing an innocent man and so it is with ganja and ganja users who are never given the benefit of the doubt. Kroon et al. continues to insist that associational evidence is good enough which demands that this analysis presents what is the research instrument of association and its inherent flaws.

Kroon et al. now presents a summary of the current evidence for the effects of cannabis on the brain. With reference to brain structure: short term effects; there is no evidence to support or refute effects. For long-term effects: With heavy cannabis use there is limited evidence for reduction hippocampal and pre-frontal cortex volume. With CUD there is limited evidence of structural alterations. There is then no causal proof of the ganja addicted brain that has been changed, altered by its addiction to ganja. How ganja impacts the brain, and the mind is still not fully explored by causal science. I suggest Kroon et al. and all the others use the herb and write about their personal experiences as Freud with cocaine. Kroon et al. now deals with cognition/learning and memory: short term effects, where there is sufficient evidence that THC/cannabis impairs (non)-verbal learning and episodic memory, but there is limited evidence of impairment of other types of learning and memory. It impairs non-verbal learning and episodic memory but no other type of learning and memory as verbal learning and long-term memory. Why? Sorry no answer lack of research to find the causal reality which can also debunk, falsify this statement. For heavy cannabis use there is sufficient evidence for impairment but insufficient evidence for lasting impairment after abstinence with evidence for partial recovery. Why does daily ganja use result in impairment but with abstinence the impairment disappears if the brain is addicted to ganja, if ganja has changed the chemistry of the brain and its functions? Sorry no research to uncover the causal dynamic yet you can make these statements of fact which inform legislation to police ganja use. In the case of CUD there is limited evidence of impairment and lasting effects after abstinence which means that the discourse insists that it is persons who consume ganja on a daily basis and especially high potency ganja who are most prone to madness. Kroon et al. now deals with craving which is a key indicator of an addicted brain which indicates the intent to support the designation of ganja as a dangerous drug. The article states that there is sufficient evidence that one of the short term effects is the reduction of craving by ganja. For the heavy ganja user one of the long term effects there is sufficient evidence for is increased craving but there is limited evidence for increased brain activity in reward related areas after ganja use. This is a position entirely at odds with the discourse of addictionology. Craving implies an addicted brain which is the product of the dopamine production mechanism of the brain cells as the two are intractably linked. How then can you crave with ganja use but there is no activation of the dopamine production mechanism of the brain cells? Can you have a craving brain without activation of the dopamine mechanism? No! Then Kroon et al. must present their definition of terms for it is only through unconventional definition of terms can they conjure up a craving brain with a dormant dopamine production mechanism. Another is their definition of evidence. For CUD there is sufficient evidence for increased craving but limited evidence for increased brain activity in reward related areas. The discourse of ganja and madness states with a straight face that a brain exposed to ganja craves ganja but this craving is in no way rooted in the brain utilizing its resources to reward its craving. Heightened craving of an addicted brain arises when it has exhausted its dopamine production resource which triggers the craving for the

drug in an attempt to kick start dopamine production once again. The human is the captive of the addicted brain and does anything to acquire the drug to ease the pain as the addicted brain is literally torturing the human captive for a rush. How then can you have a ganja induced craving and your brain is not demanding its rush? In my life I am yet to observe a heavy ganja user in the same condition as a crack head, a meth head and an opiate shooter.

Kroon et al. now deals with the evidence on cognitive biases but cognitive biases is a discourse of power as it brands human behavior that deviates from what power dictates as the norm, as normal behavior as cognitive biases, deviance and seeks to give a causal explanation for them. This discourse of power wants then to police behavior rooted in a typology of normal and abnormal, it wants then a science of behavior modification to ensure the hegemony of the norm, of normalization. Using ganja is abnormal behavior and it must be normalized and one way to do this is to create the typology of cognitive biases that especially heavy ganja users exercise in their deviation from the norm. With this typology the process of normalization can now assault the ganja users to normalize them. Kroon et al. states that for short term effects there is very limited evidence for cannabis related approach bias and attentional bias. Under long term effects with heavy cannabis use there is sufficient evidence for attentional bias, insufficient evidence for approach bias and no evidence to support or refute lasting effects after abstinence. For CUD there is limited evidence for attentional bias and no evidence to support or refute approach bias. There is no evidence to support or refute lasting effects after abstinence. The cognitive biases of attention and approach have no evidence to prove that they in fact plague ganja users hence ganja users who are all deviants from the norm are doing this for reasons that don't fall under cognitive bias, which points to the power relations of prohibition and the individual and the use of science to assault abnormal behavior.

Kroon et al. now moves to another instrument of power framed to abnormalize the ganja user namely emotional processing which is the theory of the ability of humans to deal with stress, loss and drastic change in life without developing mental health issues. This is the instrument that sets up the play which states that ganja use impacts the brain that results in mental illness through the ganja users inability to process emotion. Kroon et al. states that for short term effects there is consistent but limited evidence that THC impairs emotion recognition particularly negative emotions whilst for long term effects of heavy cannabis use there is limited evidence for impaired emotion identification/recognition and reduced activity in CB1 rich brain areas during emotional processing in current users. No evidence to support or refute lasting effects after abstinence. For CUD there is limited evidence for impaired emotion identification/recognition in CB1 rich brain areas during emotional processing in current CUD. There is then no causal proof, no statement of why the ganja user suffers with an inadequate emotional processing mechanism. What has been shown is that in areas of the brain inundated with THC the brain continues to function normally engaged with the task of emotion processing. Which means that the limited evidence can be the product of a false positive where in your quest to nail ganja as a dangerous drug you blame ganja

when it is not the agent of impaired emotion identification/recognition for trauma is and the person is self-medicating with ganja in response to that trauma. The play to then brands ganja users as being impaired emotion identification/recognition actors with a proclivity to mental illness is then just another three card hustle, snake oil pushed by the snake oil hustler, prohibition.

Kroon et al. now presents the evidence on attentional control, the theory of which focuses on what the individual fixes her/his attention on and what she/he does not, which wraps deviance in the soul of human agency impacted by drug use on the brain. Ganja then impacts the brain to choose what is good for the addicted brain not for the good of the individual and the social order. To justify prohibition of ganja and its policing the studies must show attentional failure under the influence of THC on the brain. Kroon et al. states that with short term effects there is sufficient evidence that THC impairs attentional control and with long term effects with heavy cannabis use there is sufficient evidence for impairments sustained and divided attention in current heavy users. There is insufficient evidence for lasting effects after abstinence with evidence for a partial recovery and for CUD there is no evidence for lasting effects. THC impairs attentional control only when ganja is used and with the cessation of use attentional control changes, recovers which means that ganja does not change the brain and its functioning creating the addicted brain. Just another instrument of power to abnormalize us thereby justifying the policing of the abnormal, or normalization and power is amoral, it lies as its default mode.

Kroon et al. now moves on to working memory another instrument of power vital to insisting that ganja is a dangerous drug as ganja users are incapable of making rational decisions and exhibiting normal behavior. For short term effects Kroon et al. states that there is inconclusive evidence that ganja impairs working memory, whilst for the long term effects on heavy ganja users there is inconsistent evidence for long term working memory deficits and limited evidence for recovery in heavy users. For CUD there is no evidence to support or refute lasting effects. This attempt has failed miserably seen in the inconsistent evidence which speaks to grave methodological issues or a dire lack of research or both.

Kroon et al. next deals with motor inhibition which involves the theory of how a motion in action is aborted by the brain, the spinal column and the nervous system, which is another instrument of power assaulting ganja and ganja users where ganja use impairs the ability of the body to inhibit its motion in action. Kroon et al. reports that for short term effects there is sufficient evidence that THC/cannabis impairs inhibition ongoing responses (stop signal tasks) but there are inconsistent results with other inhibition tasks. But with long term effects with heavy cannabis users there is limited and inconsistent evidence for impairment as for CUD. How can you have sufficient evidence for motor impairment in the short term but not so for long term effects on heavy users or with CUD? There is then something fundamentally wrong with the research instrument used in these studies which is magnified by the insignificant evidence for impairment of other

inhibition tasks. Another instrument of power assaulting ganja that has failed in its intent buttressed by lies.

Kroon et al. now deals with decision making which reveals the intent to insist that ganja users have impaired decision making processes which results in reckless choices that place the user, their dependents, their property and the social order at risk which demands State intervention. Ganja users are then in a condition of dependency worse than minors which demands State tutelage based on the abrogation of their rights. Kroon et al. reports that for short term effects there was insufficient evidence that THC/cannabis impairs decision making. For long term use by heavy cannabis users there is insufficient and inconsistent evidence for impairment and for CUD limited and inconsistent evidence for impairment. The attempt to validate the assault on ganja through decision making impairment has then failed as the research instruments used have produced insufficient and inconsistent evidence as the studies have failed to prove their position and there is no homogeneous discourse produced by the evidence, but this does not defeat the assault of prohibition.

The final cognitive function of the research reviewed by Kroon et al. is intelligence and its impairment by ganja use, again another assault on ganja and the user as impaired intelligence renders the ganja user a less than competent individual endowed with rights demanding State tutelage. Kroon et al. reports that for short term effects there was no evidence to support or refute effects, whilst for long term effects for heavy cannabis users there is insufficient and limited evidence for reduced intelligence and for CUD there is limited and insufficient evidence for reduced intelligence. The research into ganja use and cognition is then eminently political seeking to produce evidence to enhance the power of the State over the ganja using individual, by reducing them to an abnormal human in need of State tutelage which ultimately involves the usurpation of their human rights, their self-determination. The research activity has failed but that does not stop the assault of prohibition and the encroach of the State on the human rights of ganja users in its ever present quest for normalization.

Kroon et al. now presents the most crucial aspect of their article: the evidence of the link between ganja use and the occurrence of specific types of mental illness, this then is the evidence that proves or refutes the discourse of ganja use and madness. Kroon et al. names this section as psychiatric comorbidity which is simply an addicted brain presenting with mental illness after having failed to prove that the brain of the ganja user is in fact an addicted brain. Kroon et al. reports for depression and the short term effects of ganja use that there is no evidence to support or refute effects, whilst for the long term effects of heavy cannabis use there is sufficient statistical association but causality is unclear and for CUD there is sufficient statistical association but causality is unclear. Statistical association is not causality and the nature of the research instrument that conjures up statistical association will be critiqued in this study. Kroon et al. now reports for bipolar disorder and short term effects of ganja use that there is no evidence to support or refute effects, whilst for long term effects of heavy cannabis users there is sufficient evidence of statistical association but causality is unclear and for CUD there is

sufficient evidence of statistical association with causality unclear. Again, for bipolar disorder there is no causal link proven only statistical association and this is accepted as causality because it is ganja use under assault.

Kroon et al. now deals with ganja users and anxiety disorders and panic attacks by reporting that with short term effects of ganja use there is sufficient evidence that ganja increases risk anxiety and panic attacks, whilst for long term effects of heavy cannabis use there is sufficient evidence of statistical association but causality is unclear and for CUD the same applies. The evidence for ganja use and anxiety disorders and panic attacks is again statistical association not hard core causal proof which shows the paucity of the research undertaken. The research is then dominated by the utilization of the research instrument of statistical association as a final end point research method when this should inform causal research not replace it. But with the assault on ganja anything is permitted to constitute accepted, normal truth.

Kroon et al. now deals with ganja and post traumatic stress disorder (PTSD) insisting that ganja induces PTSD which openly contradicts the word of veterans of war who use ganja to relieve their symptoms of PTSD. Kroon et al. reports on short term effects as having no evidence to support or refute effects, whilst the long term effects of heavy cannabis use has sufficient evidence of statistical association but causality is unclear and for CUD the same applies.

Kroon et al. now reports on Psychosis and Schizophrenia and ganja use which is the primary weapon used in the assault on ganja use in the discourse of ganja and madness. This is prohibition's Holy Grail and when it is falsified the assault is unmasked revealing raw, naked, amoral power with impunity, no limits at work. Kroon et al. reports that for short term effects there is sufficient evidence that ganja increases the risk of transient positive symptoms, whilst there is limited evidence for an increased risk for negative symptoms. The long term effects of heavy cannabis use has sufficient evidence for association psychosis but causality is unclear and for CUD there is sufficient evidence of statistical association but causality is unclear. The link between ganja use and psychosis is then premised on statistical association not causal evidence which is acceptable evidence for the position that ganja use generates the mental illness of psychosis/schizophrenia in users. The manner in which this evidence based on statistical association was generated is very instructive.

The final area in this section is "other substance abuse disorders" where there is no report under short term effects, whilst under long term effects with heavy use there is sufficient evidence of statistical association but causality is unclear which also applies to CUD. With reference to long term effects of heavy ganja use Kroon et al. reports that there is limited and inconsistent evidence that ganja is a gateway drug. The studies were then unable to prove even with statistical association that ganja use led to the use of cocaine, heroin meth, opiates etc, alcohol and tobacco. Those who still use the gateway drug discourse are then stuck in a time warp a la reefer madness.

Kroon et al. ends their summary of current evidence of ganja and the brain by reporting on neurological disorders as cerebrovascular accidents and brain tumors. On cerebrovascular accidents Kroon et al. reports that for short term effects there is limited evidence that ganja use increases the risk of this accident, whilst for long term effects of heavy ganja use there is no evidence to support or to refute effects and the same applies to CUD. Again, another area that demands causal research sacrificed on prohibition's altar of expediency. For brain tumors there is no evidence for short term effects, whilst for long term effects of heavy ganja use and for CUD there is no evidence to support or refute effects.

The abiding lesson of Kroon et al. is the paucity of the evidence presented in support of ganja as being dangerous to the mental health of the user. This assault on ganja is rooted on the insistence on the link between ganja use and psychosis as fact, as truth when the only evidence offered in support is derived from statistical association not causal research and experimentation. The final nail in the coffin of the discourse of ganja and madness is an analysis of how statistical association is manufactured and passed off as evidence.

Prohibition and its discourse of ganja and madness is not interested in settling the issue of ganja and madness, once and for all, with the funding of research into the existence of a causal link between ganja use and madness. The discourse of the scientific validity of the statistical association of ganja use and madness serves the strategic aim of prohibition to assault ganja users and there is no systematic rebuttal of this false scientific discourse by those who are defenders of ganja use. In addition the new ganja oligarchs created by prohibition are not willing to invest in the research needed to settle this issue once and for all, for they must also protect their market and the windfall profits afforded them by prohibition, especially with medical marijuana. What is apparent today is that statistical association is the hegemonic norm driven by an unproven truism that ganja use results in mental illness. Any research that questions this discourse will receive no funding from prohibition and the academic careers of the researchers will be mortally wounded. The scientific and medical community is then policed by prohibition to ensure its hegemony which results in so called medical experts peddling fantasy for facts and snake oil for science, science is then a discourse of power.

The article titled "The contribution of cannabis use to variation in the incidence of psychotic disorder across Europe (EU-GEI): a multicentre case-control study" by Marta Di Forti et al. March 19, 2019 accepts from the outset as scientifically verified fact that ganja use does result in psychosis, this is a certainty, never questioned, an article of faith, a linchpin of dogma. Di Forti et al. states: "Cannabis use is associated with increased risk of later psychotic disorder but whether it affects incidence of the disorder remains unclear." For the authors an association between ganja use and psychotic disorder is adequate and effective for further research into an association. Research founded on a causal link in the case of ganja use is not necessary as one can continue to use a statistical association as causal fact to drive further research towards confirming the association as truth. This is self into self, utter and total delusion. Di Forti et al. states

“We aimed to identify patterns of cannabis use with the strongest effect on odds of psychotic disorder across Europe and explore whether differences in such patterns contribute to variations in the incidence rates of psychotic disorder.” Statistical association is then a self-fulfilling prophecy which drives an edifice of research to confirm the accuracy of the prophecy whilst expanding its ambit. This is indicated potently by the methodology of the research which sampled patients of psychiatric services who presented with first episode psychosis who self-reported ganja use and non-users. This study accepts as truth ganja use and psychosis hence it is not seeking causality as association is enough. What it is looking at is the pattern of use and the type of ganja used and available and its impact on psychotic disorder in the sample rooted in the fact of ganja use and psychosis, this is dogma. But the methodology is the same where you sample those with psychotic disorder ask them if they are ganja users then do the statistical analysis of the sample and come up with the correlation between ganja use and psychosis. If coffee consumption was prohibited you can do the same with cancer victims, ask them if they consume coffee then come up with the statistical association between coffee consumption and cancer. This is not causal evidence just inference which has to be put under the gaze of research, experimentation. Di Forti et al. now goes one step further by presenting further statistical association proving the association between high potency ganja use and psychotic disorder whilst the causal link between ganja and madness remains unproven, which is accepted as evidence by prohibition as it must for it serves power. This is the second dangerous discourse of prohibition that drives an instrument of power in the war against ganja that is rooted in bogus, snake oil science. What is noteworthy is the failure of the counter discourse for ganja use to engage with these discourses of prohibition. There can be no liberation when you are dominated at the level of the idea.