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**From:** Peter Thiel [REDACTED]  
**Sent:** Sunday, September 14, 2014 4:15 PM  
**To:** Jeffrey Epstein (jeevacation@gmail.com)  
**Subject:** FW: Reply to Benzo study

Quick feedback from my on-staff health expert...

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Last night was a lot of fun, as always!

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<= class=MsoNormal>From: Jason Camm  
Sent: Sunday, September 14, 2014 8:44 AM  
To: Peter Thiel  
Subject: Reply to Benzo study

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Yes, I saw this a few days ago.

Initial thoughts, Benzodiazepines act on similar receptors to GHB. Whilst they are both CNS depressants, Benzo's actually reduce the amount of stage 3 and 4 sleep (deep sleep associated with growth hormone release and memory consolidation) to almost negligible levels. GHB, as we have discussed before, is actually a potent initiator of stage 3 and 4 sleep.

Whilst I think the association does deserve further study, there are a few explanations that can be considered from this paper:

1. A true association that reflects Benzo use in the early stages of AD
2. Benzo's may cause minor brain damage additive to AD
3. A direct relationship between Benzo and the AD disease process
4. This could all be a false positive/ statistical fluke

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A few points of note:

the study only finds an association with AD (to medications you are not prescribed (Ambien/ Zolpidem/ Lunesta are NOT considered Benzo's in this study)).

- Non-specific symptoms have been shown to arise over a decade before AD diagnosis, a 5 year study is not long enough to exclude reverse causality.

Difficulty initiating sleep is also an early sign of AD - it is likely more people with AD are being prescribed the drug in the first place.

Benzo's have been shown to repeatedly impair memory through their effect on the brain. I have a number of hypothesis on this.

After the Metamed report, I did some further research on alternatives to GHB via sleep specialists. Non-benzo's (a newer class of drugs) such as Ambien, Zolpidem or Lunesta allow some stage 3 and 4 sleep to occur and are considered to be more mimetic of a natural sleep cycle. These are currently less commonly prescribed in the UK (where the study was performed) than the US.

In addition to your sleep cycle there are a number of other risk factors for AD relating to sleep. These include:

- Jet lag

- Sleep deprivation

Excessive daytime sleepiness

- Sleep fragmentation

We have seen on a number of occasions that your recovery time from long haul flights is generally better than most people can expect. You generally try to aim for 6-8 hrs of sleep a night. You do not report excessive sleepiness throughout the day.

It is unclear if sleep fragmentation is actually an issue for you because of the way GHB use affects sleep architecture (the proposed mechanism is that memory consolidation and formation is primarily achieved at night in stage 3 and 4 of sleep – in fragmented sleep these stages do not occur. The use of GHB changes sleep architecture such that you have a great deal of stage 3 and 4 sleep but also fragment your sleep – there are currently no studies assessing this, nor does anyone seem to have a good opinion).

Considerations specific to you:

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\*=nbsp; We already have you using a new class of drugs not associated with the risks measured in this study (Ambien/ zolpidem on days where you do not use GHB).

· =/span>We should consider a sleep study which should measure:

1) sleep architecture across 3 nights. (night one take Xyrem, night two do not)

2)=span style='font:7.0pt "Times New Roman"'> =If your sleep architecture is in fact altered resulting in greater amounts of REM sleep, does taking Zolpidem on night 3 change this?

3) &n=sp; any apnea events

Having this info would allow us to customize any changes that may in fact reduce risk of disease as well as potentially improving quality of life.

Let me know your thoughts,

Jason

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Jason Camm

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