Dear Colleagues,

Canadian researchers have now found in the brain of mouses a molecule FXR1P that appears to switch on total recall in the brain, and it could be used to help treat people with neurodegenerative diseases.
More about this you can see at:
http://www.cell.com/cell-reports/fulltext/S2211-1247%2814%2900882-1
Suppose now needed to work out whether FXR1P molecule plays the same role in humans.
I would like quantum mechanically investigate how this FXR1P molecule works in synapses and in neuronal networks of humans prior the medical experiments will be done. I will check also the similar molecules which possesses the similar electronic structure, frontier molecular orbitals and quantum entangled electron as well as spin density transfer in excited states, as possesses FXR1P molecule for the reason that maybe these new designed molecules are more effective in human synapses and in neuronal networks of humans in comparison with that FXR1P molecule is doing in mouses brain.

With best regards,

Arvydas Tamulis

In reply:

Dear Arvydas Tamulis and friends:

Thank you for this fascinating information! Clearly there are many potential therapeutic benefits to the study of this mechanism. A demonstrable increase in mnemic functioning! From a quick read it appears that the purpose of the mnemic limiting function of FXR1P may be to aid in the proper prioritizing of information. I infer this from the following: "…loss of FXR1P specifically enhances hippocampal protein synthesis-dependent late-phase long-term potentiation (L-LTP), spatial memory, and expression of the AMPA receptor subunit GluA2. …Thus, FXR1P has a critical role in limiting synaptic plasticity and memory storage in the brain…However, unlike WT mice, cKO mice failed to shift their preference to the new platform location during the reversal probe test. Measuring the average latency to the first platform crossing further revealed that cKO mice showed an equal latency to first crossing of the original and reversal platform locations, unlike WT mice that immediately targeted the location of the reversal platform (Figure 2G). Together, these results indicate that FXR1P cKO mice have enhanced spatial memory without perseverative behaviors."

I note: ***the last sentence implies a possible treatment for OCD.***

I have recently read a paper, on the persistence of long term memory in Aplysia in the condition that mnemic neural connectivity is disrupted. Reinstatement of long-term memory following erasure of its behavioral and synaptic expression in Aplysia Shanping

Chen, Diancai Cai, Kaycey Pearce, Philip Y W Sun, Adam C Roberts, David L Glanzman DOI: http://dx.doi.org/10.7554/eLife.03896 It appears the the epigenetic portion of DNA itself holds the information apart from the neural connections. Only a small bit of stimulus is needed to create the extinguished behavior. This looks to me like a dead ringer for the mechanism of instantiation of instinct and or phylogenetic memory itself. Perhaps the author has already found the mechanism?

"these results imply that the persistence of memory does not require the stability of particular synaptic connections.....suggest that the persistence of sensitization related LTM in Aplysia does not require the persistence of the synaptic connections generated during learning. Rather, LTM appears to be regulated by a homeostatic mechanism that specifies the net synaptic strength according to experience. …According to this scheme, synapses serve merely to express LTM, they are not sites of LTM storage." I say: Line 379 to 386 indicate that genetic storage of information…specifically the epigenetic portion is the store!! And this: "Although early evidence indicated that learning -induced DNA methylation in the hippocampus was transient and readily reversible (Miller & Sweatt 2007), a more recent study has reported that contextual fear conditioning in rats induces DNA methylation of the gene for calcineurin in cortical neurons that persists for at least a month (Miller et al 2010). …Thus, DNA methylation may constitute an epigenetic mechanism for the lifelong storage of memory (Day & Sweatt 2010)."

It seems like the sensitivity to the stimulus which can be elicited, as a waiting memory, even after the neural connectivity is disrupted, is a good bet for a system with two layers of information protection. That which is epigenetically encoded can then be retrieved to provide a systemic informational back up if systemic disruption is encountered. Only a bit of stimulus…and presto…a patterned behavior emerges from encoded epigenetic mnemic DNA storage in response to the familiar condition. Now, along with this, it is possible, and not entirely unlikely after understanding something of the intra-connectivity of the quantum coherent bio-system, that another layer of functionality is possibly implied by the very familiar pattern of "ripe" response, just waiting for conditions to cause its pre-patterned emergence: instinct and the phylogenetic. A behavior just waiting to be brought to fruition. So, one group of Aplysia are given stimulus over and over spanning many generations. The young then, should display instinct once exposed to same stimulus…the response waiting in the genes as sensitivity and predisposition toward behavior…ripe fruit. The next step is to repeat the experiment with an agent which to some extent blocks the DNA methylation process. No instinct should be present. As to the phylogenetic, let us imagine that half the information is transferred by each parent as one would expect. If the RACE had encountered the SAME stimulus…the trait would indeed be passed on as it would be independently encoded in full in each parent…the phylogenetic.

I would be quite curious as to if you imagined there is any possible truth to my speculations. If we know how it LTM encoded into ontology and phylogeny, limited by factors such as FXR1P and strengthened by its removal: Perhaps we could determine the way to alter traumatic long term memory, not only for victims of the usual sort, but for

the race as well.

Thank you for indulging these ideas!

Rich