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GUT DYSBIOSIS IN DOGS WITH SPINAL CORD INJURY: IMPACT OF POLENOPLASMIN

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Intervertebral disc disease is a common cause of spinal cord injury in dogs and significantly impacts quality of life.

Canine studies have demonstrated that inflammatory mechanisms may play a critical role in canine with spinal cord injury associated with intervertebral disc disease.

Studies in dogs have demonstrated the presence of gut dysbiosis (alterations in gut bacterial homeostasis) secondary to spinal cord injury.

Therefore, targeting gut dysbiosis could have significant therapeutic value in the management of spinal cord injury.



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Several methods for treating the contusive injury have been investigated in dogs but an optional treatment has yet to be determined.

The dysbiosis is thought to impair recovery by decreasing the production of short-chain fatty acids which play a role in suppressing inflammation within the central nervous system.

Thus, the dysbiosis contributes to the onset and progression of intraspinal pathology after spinal cord injury.



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There is increasing interest in our microbiomes and the connection between gut and brain health.

The purpose of this study is to determine if gut dysbiosis occurs in dogs with spinal cord injury secondary to intervertebral disc disease compared to healthy dogs.

The objective of this study was to characterize the gut microbiota in dogs with spinal cord injury.



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The results of this study would be of interest since to our knowledge, microbiome-associated studies targeting spinal cord injury dogs are non-existent and the results might help explain possible implications of gut microbiome in spinal cord injury.



We found that gut microbes that metabolize tryptophan - an essential amino acid - secrete small molecules called indoles, which stimulate the development of new brain cells in adults.

We demonstrated that the indole-mediated signals elicit key regulatory factors known to be important for the formation of new adult neurons in the hippocampus, an area of the brain also associated with memory and learning.



This finding is exciting because it provides a mechanistic explanation of how gut-brain communication is translated into brain cell renewal, through gut microbe produced molecules stimulating the formation of new nerve cells in the adult brain.



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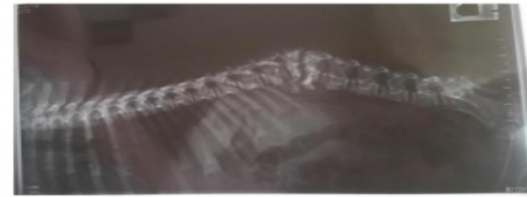


These findings bring us closer to the possibility of novel treatment options to slow down memory loss, which is a common problem with aging and neurodegenerative diseases including but not limited to Alzheimer's disease.

These include drugs to mimic the action of indoles to stimulate the production of new neurons in the hippocampus or to replace neurons damaged by stroke and spinal cord injury, as well as designing dietary intervention using food products enriched with indoles as a preventive measure to slow down aging.

An interesting case of a puppy from Cyprus, who was hit by a car was broken in his spine and was paralyzed with his back legs.

https://www.deniplant.ro/polenoplasmin_catel.htm



www.deniplant.ro/catelusa.mp4

California, 2019



For 4 months the puppy was treated with Polenoplasmin, in addition to the physical recovery treatments and the dog was able to walk again. The puppy lives and walks alone and today as can be seen in the following video:

<https://youtu.be/OcQ2NXgZnXs>

after 6 years the puppy is healthy and can run freely.

<https://youtu.be/lwzywDfKsnI>

<https://youtu.be/Z7fcuVWesMc>



Conclusion

The work reported in this paper addresses the formation of neurons in the dog brain.

We are currently assessing whether indoles can also stimulate early formation of neurons during brain development.

Another area of potential intervention interest is in situations of spinal cord injury where there is an urgent need to generate new neurons.

This study with Polenoplasmin is another intriguing piece of the puzzle highlighting the importance of lifestyle factors and diet.

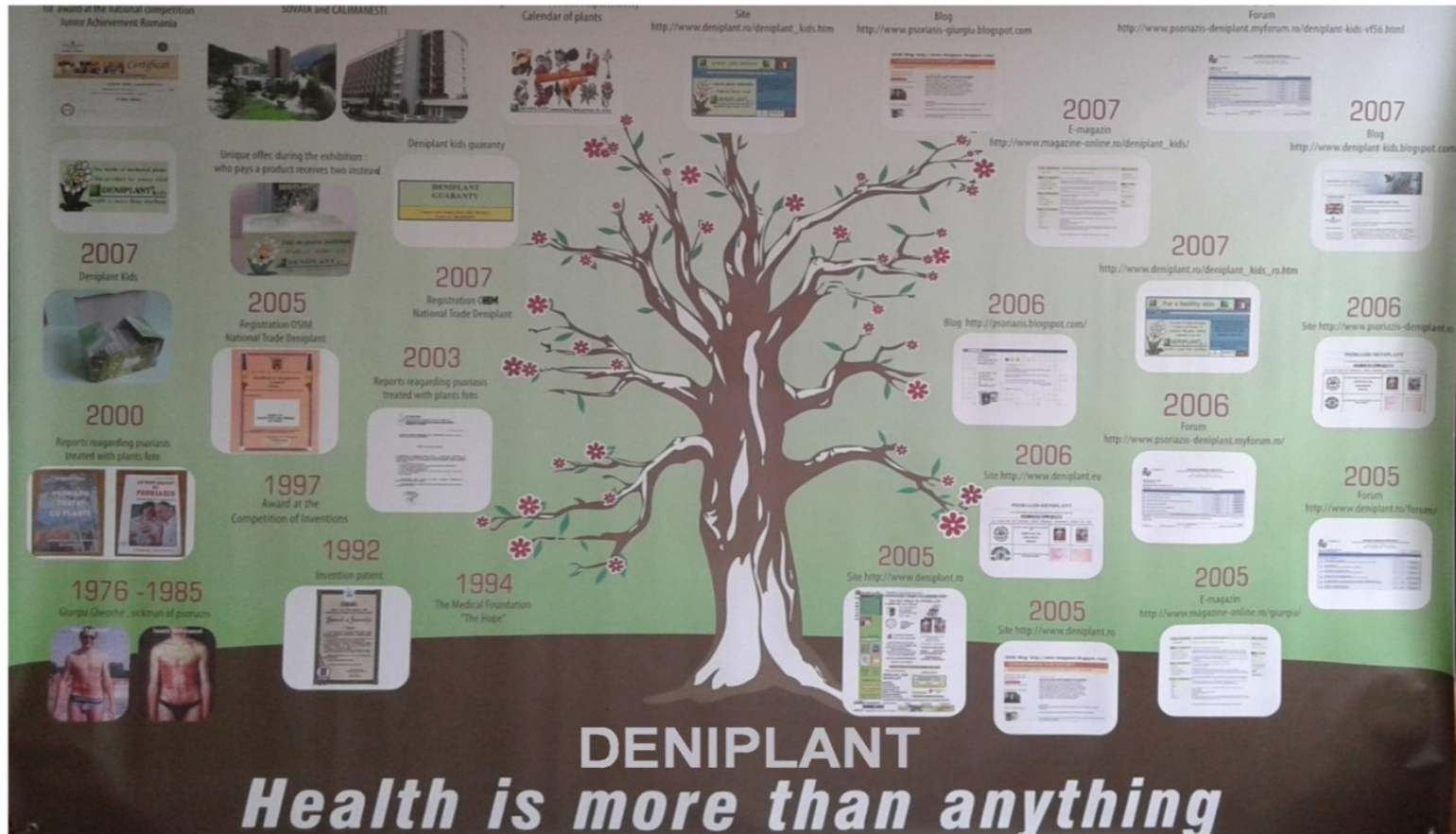


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