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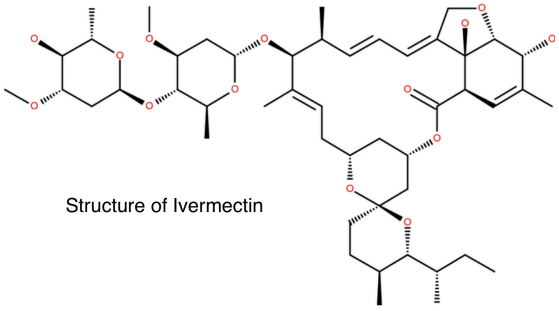
BC368 – Biochemistry of the Cell II
Case Study #2: Scabies to Coma

Case Background

A 13-year-old boy is admitted to your hospital with chief complaint scabies. To treat the scabies you order an oral dose of ivermectin given at 0.23mg per kilogram of bodyweight (the standard dose). Two and a half hours after administration of medication the boy displays signs of impaired consciousness. Three and a half hours after that, his condition continued to deteriorate with persistent signs of encephalopathy (impaired function of the brain), specifically coma, slurred speech, ataxia (lack of voluntary muscle coordination), and binocular diplopia (double vision) as well as abdominal pain and vomiting.

Lab Findings:

All vitals (blood pressure, heart rate, body temperature) recorded during the time of stay were within normal limits, toxicology screenings came out all clear. Genetic sequencing identified the child as a compound heterozygote (two different mutated alleles at a specific locus) of *ABCB1*.

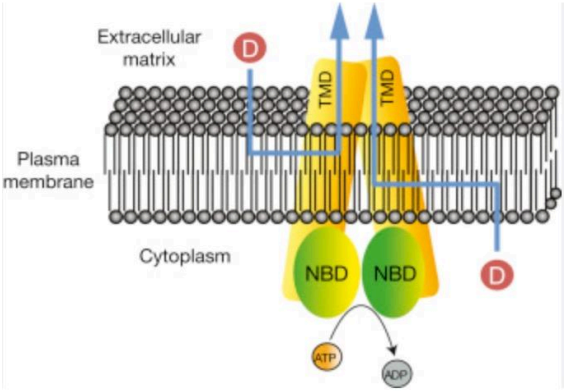

Structure of Ivermectin

History Findings:

At 4 years old, the boy was admitted to an emergency unit for progressive consciousness disorders similar to those seen in the ER today. The only medication recorded at that time was local topical applications of fusidic acid ointment for ten days for an eyelid wound. He spontaneously recovered at the disruption of treatment.

Biochemistry:

The gene, *ABCB1,* encodes for the ABC transporter protein, ABCB1, also known as MDR1 (Multi-Drug Resistance 1). MDR1 effluxes toxins in the BBB and is also responsible for resistance to chemotherapeutic drugs in the brain. In some breeds of dogs such as collies, which are homozygotes for a nonsense mutation in *ABCB1*, and in *Abcb1*-knockout mice, ivermectin induces neurologic disorders that can be fatal.



Vocabulary:

Encephalopathy, ataxia, binocular diplopia, compound heterozygote, agonist

Analysis:

Q1: Based on the fact that mutation of *ABCB1* causes a truncated transport protein lacking the C-terminal nucleotide-binding domain, what can you conclude about the function of the patient's MDR1?

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Q2: What does your conclusion from Q1 reveal about how ivermectin is being processed in the patient's body?

Q3: (Challenge!!) The neurotransmitter GABA (gamma-aminobutyric acid) causes hyperpolarization of the postsynaptic membrane of neurons when bound to the GABA-receptor. Ivermectin is a known GABA agonist, meaning it increases the activity of the GABA Receptor. What could be a possible mechanism for this hyperpolarization of the membrane and how would it affect neural signaling? How can this explain some of the symptoms the patient is experiencing?

References:

- Baudou, E., Lespine, A., Durrieu, G., André, F., Gandia, P., Durand, C., & Cunat, S. (2020). Serious Ivermectin Toxicity and Human *ABCB1* Nonsense Mutations. *The New England journal of medicine*, *383*(8), 787–789. https://doi.org/10.1056/NEJMc1917344
- Krůsek, J., & Zemková, H. (1994). Effect of ivermectin on gamma-aminobutyric acid-induced chloride currents in mouse hippocampal embryonic neurones. *European journal of pharmacology*, *259*(2), 121–128. https://doi.org/10.1016/0014-2999(94)90500-2