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Pharmacoresistance is not associated with changes in P-gp expression at the level of the blood-brain barrier

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Increased expression of P-glycoprotein (P-gp) at the blood-brain barrier (BBB) in pharmacoresistant epilepsy has been reported. It is thought to cause insufficient antiepileptic drug brain concentrations, and thereby decreased seizure control. However, the question remains how BBB P-gp expression changes with time, and if it is a reliable indicator for changes in BBB P-gp function.

In rats, the relationship between P-gp expression (*ex-vivo* by immunohistochemistry) and function (BBB transport of quinidine, with or without P-gp inhibition by tariquidar) was determined up to 3-5 weeks following kainate induced Status Epilepticus (SE). SE was induced by intraperitoneal injections (IP, group A) or unilateral intrahippocampal injection (IH, group B). In addition, IH injection was followed by *in vivo* bilateral hippocampal microdialysis experiments (IH, group C).

Following SE, for all groups, an initial increase in P-gp expression was followed by a subsequent decline. Group C showed a lack of relation between BBB P-gp expression and BBB P-gp function in individual rats.

In conclusion, there is no relationship between BBB specific P-gp expression and function, and pharmacoresistant epilepsy is related to temporal changes in BBB P-gp expression, but is not related to changes in BBB p-gp function, but merely at the brain parenchymal cell level.

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