Akademie für Integrative Medizin,
Zahnmedizin und Bewusstseinstechniken

<u>Beispiel:</u> „Aktivierte T-Zellen vermitteln Zelltod und Dysfunktion an den Endothelzellen der Blut-Hirn-Schranke“

Activated T cells mediate direct blood-brain barrier endothelial cell death and dysfunction. Neuroreport 2002 Dec 20;13(18):2587-91 (ISSN: 0959-4965) Tan KH; Purcell WM; Heales SJ; McLeod JD; Hurst RD
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Neuro-inflammation is characterized by immune cell infiltration across the **blood-brain barrier**, a process instrumental in neuronal cell death. In neuro-inflammation the **blood-brain barrier** is also damaged and the consequences of activated lymphocytes on the integrity of the **blood-brain barrier** is not well characterized. Utilizing an **blood-brain barrier** model we demonstrate that endothelial cell viability and **barrier** integrity are directly altered following lymphocyte exposure. The effect of **activated lymphocytes** is cell number dependent, mostly mediated by direct contact, and is not associated with the pro-inflammatory cytokine TNF-alpha. For the successful treatment of neuro-inflammatory disease, intervention of this direct effect at the **blood-brain barrier** is warranted.

<u>Kommentar:</u> Die zitiere Veröffentlichung beweist die mögliche Vernetzung von

- Hypersensibilisierung (=T-Zellen Aktivierung) und
- Erkrankungen der Blut-Hirn-Schranke verbunden mit
- Entzündungsprozessen im Bereich des Gehirns.

<u>Beispiel:</u> „Quecksilberverbindungen indizieren Metallothioneine im Gehirn von Ratten“

Induction by mercury compounds of brain metallothionein in rats: Hg0 exposure induces long-lived brain metallothionein. Arch Toxicol 1998 Mar;72(4):187-91 (ISSN: 0340-5761) Yasutake A; Nakano A; Hirayama K Biochemistry Section, National Institute for Minamata Disease, Kumamoto, Japan.

Metallothionein (MT) is one of the stress proteins which can easily be induced by various kind of **heavy metals**. However, MT in the **brain** is difficult to induce because of **blood-brain barrier** impermeability to most **heavy metals**. In this paper, we have attempted to induce **brain** MT in rats by exposure to methylmercury (MeHg) or metallic **mercury** vapor, both of which are known to penetrate the **blood-brain barrier** and cause neurological damage. Rats treated with MeHg (40 micromol/kg per day x 5 days, p.o.) showed **brain** Hg levels as high as 18 microg/g with slight neurological signs 10 days after final administration, but **brain** MT levels remained unchanged. However, rats exposed to Hg vapor for 7 days showed 7-8 microg Hg/g **brain** tissue 24 h after cessation of exposure. At that time **brain** MT levels were about twice the control levels. Although **brain** Hg levels fell gradually with a half-life of 26 days, MT levels induced by Hg exposure remained unchanged for > 2 weeks. Gel fractionation revealed that most Hg was in the **brain** cytosol fraction and thus bound to MT. Hybridization analysis showed that, despite a significant increase in MT-I and -II mRNA in **brain**, MT-III mRNA was less affected. Although significant Hg accumulation and MT induction were observed also in kidney and liver of Hg vapor-exposed rats, these decreased more quickly than in **brain**. The long-lived MT in **brain** might at least partly be accounted for by longer half-life of Hg accumulated there. The present results showed that exposure to Hg vapor might be a suitable procedure to provide an in vivo model with enhanced **brain** MT.

Kommentar: Die zitiere Veröffentlichung beweist die

- Zerstörung der Blut-Hirn-Schranke durch Quecksilberdampf
- im Gegensatz zu organischem Quecksilber und zeigt
- die Anregung von thiolgebundenen Entgiftungssystemen im Gehirn nach Quecksilberdampf.

Gibt es noch andere Literatur zur Schädigung der Blut-Hirn-Schranke durch Zahnmetalle?

Zheng W et al.: Choroid plexus protects cerebrospinal fluid against toxic metals. FASEB J 5 (1991) 2188-2193
124. Aschner M: Methylmercury in astrocytes -what possible significance? Neurotoxicology 17 (1996) 93-106
125. Walum E et al.: Use of primary cultures and continuous celllines to study on astrocytic regulatory functions. Clin Exp Pharmacol Physiol 22 (1995) 284-287