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Poster presentation

Cerebral net exchange of Brain-Derived Neurotrophic Factor (BDNF) during experimental systemic inflammation and hypoxaemia in humans

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Sepsis is notably associated with neuronal damage, an effect that may be exacerbated by hypoxaemia. The present study was designed to investigate if this interrelationship involves an effect on brain-derived neurotrophic factor (BDNF), an intrinsic neuroprotective agent which is released from the brain under normophysiological conditions [1]. We hypothesised that experimental systemic inflammation and hypoxaemia would abolish this release.

36 healthy male volunteers aged 25 (SD, 4) years were randomised to:

1. normoxia for 12 hours and a four-hour intravenous infusion of lipopolysaccharide (LPS) from 4–8 h (total dose 0.3 ng/kg), N = 12

2. hypoxia for 12 hours (12.9% O2) and a four-hour intravenous infusion of saline from 4–8 h, N = 11

3. hypoxia for 12 hours (12.9% O2) and a four-hour intravenous infusion of LPS from 4–8 h (total dose of 0.3 ng/kg), N = 13

Cerebral blood flow (CBF) was measured by the Kety-Schmidt technique, and arterio-jugular venous concentrations of BDNF were determined at baseline and 9 h. The cerebral net exchange was calculated by multiplying CBF with the arterio-jugular concentration differences of BDNF.

A cerebral release of BDNF was present at baseline ($P < 0.005$). This was attenuated in all three groups, but with no difference in the cerebral net exchange values from baseline or between interventions (NS, MANOVA). There was no effect of any of the interventions on the arterial levels of BDNF (NS, MANOVA).

In conclusion, systemic inflammation and hypoxaemia may abolish the net cerebral release of BDNF in healthy humans.

References

1. Krabbe KS, *et al.*: *Diabetologia* 2007, **50(2):**431-8.