

Investigation of Caffeine's Impact on Cerebral Physiology Using SWI (Susceptibility-Weighted-Imaging)

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Introduction: Caffeine decreases the BOLD (blood oxygenation level dependent) signal of cerebral venous vessels by acting as an adenosine antagonist and thus decreasing the CBF (cerebral blood flow) [1]. Sedlacik *et al.* [2] have shown that caffeine has a contrast enhancing effect on cerebral veins using SWI. Aim of this study was to investigate the time course of signal change comparing caffeine-tolerant volunteers who have been consuming caffeine for more than 8 weeks and abstinent volunteers.

Methods/Materials: High resolution T2*-weighted images were acquired in two groups consisting of 12 caffeine-consuming and 15 caffeine-abstinent volunteers on a 1.5T system using a velocity compensated 3D gradient echo imaging sequence. Sequence parameters were: TE/TR/FA=40ms/57ms/20°, FoV= 256x192x76, matrix=512x256x38, acquisition time TA=9min:15sec. After a native scan was performed, the subject was given 200mg caffeine orally directly in the scanner. Four subjects of each group were re-measured with 100mg. The scan was then repeated for about an hour and the acquired 3D datasets were realigned. BOLD-signal changes were analysed and statistically evaluated by using ROIs in venous vessels, ventricles and segmented grey and white matter.

Results: Maps of signal difference clearly visualized the caffeine-induced response of venous vessels (Fig.1). No significant signal change was detected in ventricles, grey and white matter. Maximum signal decrease occurred after approximately 40 min (Fig.2). This reflects the physiological effect of caffeine, which reaches the maximum plasma level after about 40-45 min [3]. No difference in time was observed between the two groups. The decrease reached -17% with the inured and -23% with the abstinent subjects in the vessels (Fig.2). This difference was statistically significant ($p=0.021$, Student's t-test, one-tailed, unpaired samples, unequal variances). It was further possible to fit a mono-exponential function to the signal decrease (Fig.2, $r^2>0.99$) which confirms a linear pharmacokinetic model of the oral absorption of caffeine [4]. Reducing the caffeine dose to 100mg did not show significant differences of the signal change in veins.

Discussion/Conclusion: SWI contrast enhancement with caffeine was displayed and quantified. The less distinct signal change due to receptor up-regulation after frequent caffeine ingestion of about 6-8 weeks [5] was shown. As expected, there was no significant signal change in ventricles.

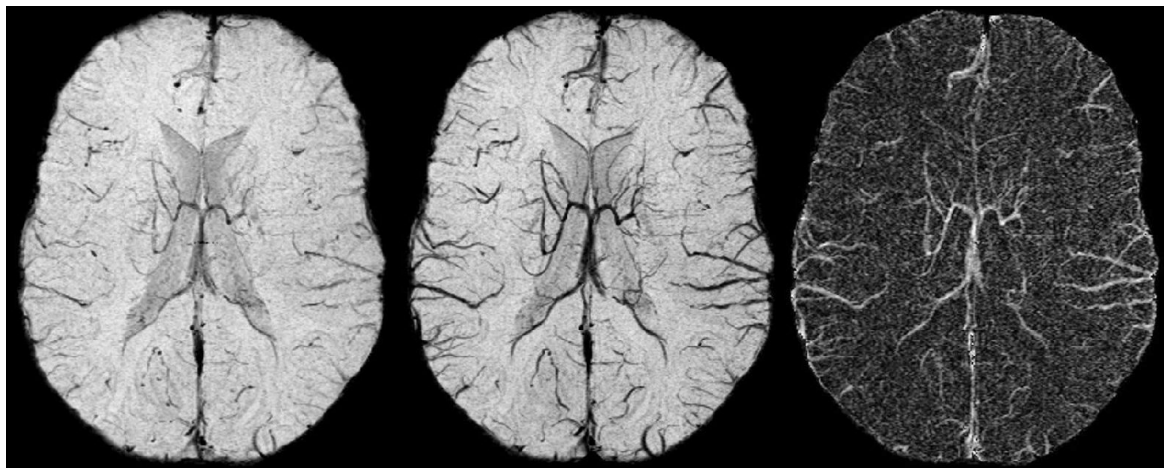


Fig.1: SWI scan native (left) and 45 min post caffeine (middle) both identically processed. Right: subtraction between both SW-images.

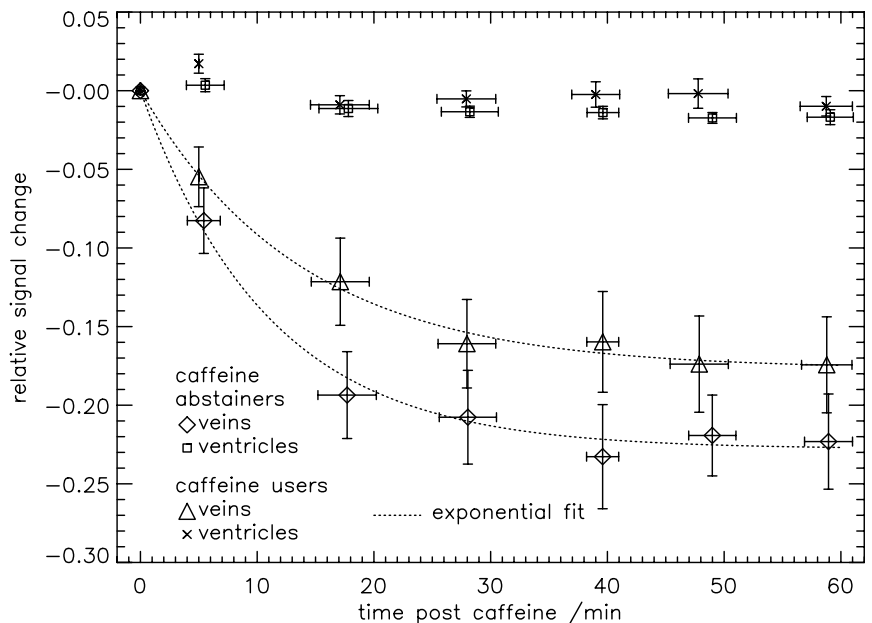


Fig.2: Signal change for caffeine-tolerant and abstinent volunteers in veins and in ventricles.

Literature: [1] B Johansson, et al., Brain Res, 762:156-164,(1997); [2] J Sedlacik, et al., Proc. of 8th annual meeting of the German chapter of the ISMRM (2005). [3] BB Fredholm, et al., Pharmacol Rev, 51:83-133, (1999). [4] R Urso et al., Eur Rev Med Pharmacol Sci, 6, 33-44 (2002). [5] SR Dager, et al., Am J Psychiatry, 156: 229-237, (1999).