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Abstract Title:	Properties of cortical spreading depression across visual cortex in mice with spontaneous mutations in P/Q-type Ca ²⁺ channels.
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Migraine, the most common neurological disorder, afflicts 10-15% of the general population. Cortical spreading depression (CSD), a wave of depolarization spreading slowly across the cortex, is thought to mediate aura that sometimes precedes migraine. However, whether CSD actually triggers migraine remains controversial. Multiple mutations in α_{1A} , the pore-forming subunit of P/Q-type Ca²⁺ channels, are found in familial hemiplegic migraine (FHM), a rare hereditary migraine with aura. Elsewhere, we report that T666M and certain other FHM mutations lead to decreased Ca²⁺ influx, qualitatively similar to α_{1A} subunit mutations in tottering (*tg*) and leaner (*tg^{la}*) mice. Thus, a first approach to relating α_{1A} mutations and spreading depression is to compare CSD in wild type, *tg* and *tg^{la}* mice. We studied visual cortex since auras appear primarily as visual disturbances. Increasing concentrations of KCl (100 mM – 2M) were applied to a 0.5 mm² region of V1 cortex, while voltage changes were recorded in V2, 2.3 mm away. The lowest KCl concentration that first caused CSD was defined as the CSD threshold. We found that CSD in visual cortex was not significantly easier or harder to trigger in *tg* than in wild type mice. Likewise, there was no significant difference of CSD threshold between *tg^{la}* and wild type mice. Interestingly, repetitive applications of KCl failed to elicit more than one CSD in *tg^{la}* mice, whereas wt and *tg* visual cortex responded repeatedly and reliably; preliminary data suggested that CSD amplitude may be smaller in *tg^{la}* mice. The lack of change in CSD threshold in *tg* and *tg^{la}* leaves room for the possible involvement of an additional predisposing condition, promoted by α_{1A} mutations. Alternatively, their contribution to migraine may lie downstream or be independent of the CSD event itself.

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