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Reply to Letter to the Editor

We appreciate the pertinent comments by Tringali and colleagues agree that the contribution of rTMS-related noise to tinnitus exacerbation should not be ignored. Indeed there is evidence in animal models supporting the notion of acoustic trauma by TMS [1–3]. However, some caution should be taken in interpreting these studies as the TMS coil was discharged right over the ear and the experiments were done in Albino animals that are particularly sensitive to acoustic trauma [4–6]. Therefore, the translation to normal humans is unclear. In any case, it does appear that the use of hearing protection prevented hair cell loss and neuroanatomical damage in the same animal model [7], and studies in humans support the same claim [8,9]. It thus seems that the potential impact of acoustic trauma can be minimized, but for example in pediatric populations, the situation may be different, given higher sensitivity to injury by acoustic trauma and lack of normal hearing protection [8,10].

The pathophysiologic mechanisms for tinnitus induced by TMS in humans remain unclear. The aim of our review was to highlight the safety of TMS in disease states where site-specific neuronal activity is altered significantly. Along these lines we sought to underscore adverse events from the perspective of brain mediated mechanisms. However, we did not intend to suggest that acoustic trauma does not play a role, and appreciate Tringali's letter which enables us to clarify this point. In addition to acoustic trauma, TMS-induced alterations in auditory cortical excitability and plasticity, direct stimulation of inner ear structures or acoustic nerve, sensory input via the trigeminal nerve, or activation of other cranial nerve systems, may all be relevant contributors [11–13]. Further work is certainly needed to determine the true cause of tinnitus induction or exacerbation by TMS.

We agree that hearing safety remains a concern in TMS, even when the proper hearing protection is provided [14]. The need for precautionary hearing protection is of the utmost importance in patients with tinnitus or other auditory dysfunction. This issue also reinforces our claim that adverse events are too often underreported or inadequately documented. Without attention to such detail, progress in the field cannot be achieved.

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Available online 23 March 2012

http://dx.doi.org/10.1016/j.brs.2012.01.003

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Predict the outcome of depression after rTMS using neuroimaging: Issue of response or non-response?

We read with great interest the recent article of Kito et al. [1] suggesting that SPECT cerebral blood flow ratio of the dorsolateral prefrontal cortex to the ventromedial prefrontal cortex predicts response to repetitive transcranial magnetic stimulation (rTMS) in depressed patients. This elegant study highlights the growing interest in clinical use of functional neuroimaging, and in particular of brain SPECT, to predict or evaluate the treatment response [2–6].