**Supplementary Introduction**

It remains unclear how tinnitus is generated by the “tinnitus network” and modulated by rTMS. Several studies have demonstrated a relationship between brain resting oscillation and tinnitus, and reported that tinnitus patients have an increased oscillation power in the delta-band (<4 Hz) and a decreased oscillation power in the alpha-band (8–12 Hz).[Lorenz et al., 2009; Schlee et al., 2009; Vanneste et al., 2010] Since alpha oscillation inhibits areas of the cortex that are not in use and plays an active role in network coordination and communication,[Palva and Palva, 2007] decreased alpha oscillation may reflect imperfect inhibition or coordination of phantom sound perception (tinnitus). A magnetoencephalography (MEG) study also suggested an increase in gamma-band (40–90 Hz) oscillation power activity in chronic tinnitus patients.[Lorenz et al., 2009; Weisz et al., 2007b] Importantly, decreased alpha-band oscillation power and increased gamma-band oscillation power are reproducible findings in tinnitus patients.[Schlee et al., 2009] The second aim of this study was to elucidate the changes in cortical oscillation power before and after rTMS in chronic tinnitus patients. We aimed to elucidate the changes in the inter-areal functional connection of brain oscillations before and after active dual-site rTMS in the same subject.

In this study, we focused particularly on the dorsolateral prefrontal cortex (DLPFC) as several cross-sectional studies suggested that modulating the DLPFC has a significant effect on chronic tinnitus.[De Ridder et al., 2013; Vanneste and De Ridder, 2012] The DLPFC is related to auditory memory, inhibiting the primary AC, and the top-down modulation of auditory attention.[Alain et al., 1998; Knight et al., 1989; Mitchell et al., 2005] The application of rTMS to the DLPFC may either directly modulate the tinnitus network or indirectly influence the tinnitus network.[Noh et al., 2017a]

**Supplementary Methods**

**Study 1**

**Inclusion and exclusion criteria**

All patients had tried some of several standard treatment modalities such as vasodilators, antidepressants, hearing aids, noise generators, and tinnitus retraining therapy, but were unsatisfied. Patients with a history of seizures, suspected diagnosis of organic brain damage, cardiac pacemakers and other electronic implants including cochlear implants, and intraocular ferromagnetic materials and particles were excluded. Patients who were taking concomitant medications such as antidepressants and antipsychotics or who had serious heart disease or other unstable major medical conditions were excluded. A normal middle ear status was demonstrated by audiogram and otoscopy. Abnormal psychologic conditions such as depression, anxiety, and insomnia were screened using the validated Beck’s Depression Inventory (BDI) for depression, State-Trait anxiety inventory (STAI) for anxiety, and Pittsburgh sleep quality Index (PSQI) for sleep quality.

**Randomization**

The patients were randomly assigned to receive either dual-site rTMS (Group 1, n = 17) or sham rTMS (Group 2, n = 13) using an unrestricted randomization technique. Sealed opaque envelopes containing information regarding which group each patient belonged to were used for the randomization; except for the individual (T.S.N.) who performed the rTMS, all other researchers and patients were blinded to the treatment method. The treatment outcomes were evaluated by a physician (M.W.S.) who was also blinded to the treatment group.`

**rTMS treatment**

Magnetic stimulation was administered at a frequency of 1 Hz using a Medtronic system (MagPro, Medtronic, Minneapolis, MN, USA) with a figure-of-eight coil (MCF-B65, 90-mmouter diameter, Medtronic). The stimulation intensity was set at 110% of the resting motor threshold (RMT). The RMT was determined in the right abductor pollicis brevis and was defined as the lowest intensity at which at least four of eight consecutive motor evoked potentials (MEP) were ≥50 μV in amplitude when the investigated muscle was at rest.[Rossini et al., 1994]

**Pure Tone Audiometry**

Pure tone audiometry was conducted according to the ANSI S3.6-2004 using an AD229b audiomer (Interacoustics, Denmark) in a double walled soundproof booth (fulfilling ANSI S3.1-2013). The pure tone audiometry for each of the 6 frequencies (range: 0.25, 0.5, 1, 2, 4, and 8 kHz) was measured for all subjects. The participants started the test by listening to a 1 kHz tone; the test tone was reduced by 10 dB or increased by 5 dB according to the presence or absence of a response. The hearing threshold was defined as the lowest intensity level (in dB HL) at which the participant responded to 50% of the stimuli.

**Study 2**

**Inclusion and exclusion criteria**

The number of subjects was small because we used strict selection criteria. The exclusion criteria were the same as in study 1, but additional inclusion criteria were applied. The subjects were all right-handed and had no previous history of neurologic or psychiatric disorders. The tinnitus frequency was also required to match a certain type of pure tone. Subjects who could not precisely match or describe their tinnitus and who complained of a tinnitus that was similar to narrow band noise or broad band white noise were excluded from study 2. The selection criteria for this study was strict compared to the former studies. Accordingly, the outcome of this study may not reflect the whole tinnitus patient population. But we believe the strict inclusion criteria will increase the reproducibility of this study.

**Subjects**

The demographics in terms of gender (M:F; 3:2), age (49.4 ± 16.1), duration of tinnitus (43.8 ± 47.7), threshold of pure tone audiometry (mean of 500/1000/2000, 4000, and 8000 Hz was 29.7 ± 16.6, 46.0 ± 26.8, and 57.5 ± 26.5 respectively), BDI (12.2 ± 9.8), STAI-X1 (42.0 ± 14.8), STAI-X2 (39.0 ± 13.4), PSQI (9.2 ± 2.6), and pre-treatment THI scores (52.6 ± 21.9) were not statistically different from those of study 1.

**rTMS treatment**

Structural T1-weighted high-resolution MRI (3 Tesla Magnetom Tim Trio Scanner, Siemens Medical Solutions, Erlangen, Germany) was performed in all subjects. The rTMS coil was navigated to the primary AC [Saenz and Langers, 2014] by a neuronavigation system (Cybermed, In2vision, Seoul, Korea). The primary AC was defined as the first traverse gyrus of Heschl in the MRI.

In study 2, the subjects received dual site (AC and FC) rTMS stimulation: same as the subjects in study 1. The only difference was that the neuronavigation system was used as a supplementary tool to check if an imaginary line perpendicular to the figure-of-eight coil passed through the Heschl's gyrus. The neuronavigation system was used to double check that the AC rTMS was not given to a totally nonsense location. The neuronavigation system was not used for the FC localization.

**MEG recoding and data acquisition**

The MEG had 102 identical triple sensor elements (one magnetometer and two gradiometers that were oriented perpendicular to each other) in a helmet-shaped array. MEG signals were recorded using a 0.1–200-Hz band pass ﬁlter at a sampling rate of 600 Hz. Four head position indicator coils were attached to the subjects’ scalps to align the head position in the sensory array. The coil location was identified with respect to three anatomical landmarks (the nasion and the two preauricular points) using a 3D digitizer (Fastrak, Polhemus, Burlington, VT, USA). This information was used to co-register the MRI, MEG and head coordinates to enable the MEG signal sources to be superimposed on individual T1 MRI images.

**MEG data analysis**

First, the power spectral density in each node was calculated using fast Fourier transform and Welch’s approach with a window of 1 s and an overlap of 50%. Five frequency ranges of brain oscillation were selected based on previous tinnitus power spectra analysis reports[Schlee et al., 2009; Vanneste et al., 2011] and our prior study as follows: alpha band, 8–12 Hz; beta 1 band, 13–18 Hz; beta 2 band, 18.5–21 Hz; beta 3 band, 21.5–30 Hz; and gamma band, 38–72 Hz. Second, functional connectivity analysis of the individual edges was performed. We calculated an indicator for the centrality of each node. The 'centrality' of a node is high when a node connects to many other nodes and is low if the nodes connect to only few other nodes.[Schlee et al., 2009] Degree centrality for each node is calculated by summing the number of linked edges from the node.[Schlee et al., 2009]
1) The right primary AC (Montreal Neurological Institute [MNI]; coordinates 48, –25, 13). 2) The right DLPFC (43, 21, 38). 3) The right inferior parietal cortex (40, –67, 32). 4) The dorsal anterior cingulate cortex (40, –67, 32). 5) The left primary AC (–49, –26, 12). 6) The left DLPFC (–48, 21, 38). 7). The left inferior parietal cortex (–31, –68, 32). Then, the source activity was extracted using a multi-dipole method with BESA (Brain Electrical Source Analysis, MEGIS Software, Munich, Germany)

**MEG analysis Methods**

The power spectral density in each node was calculated using fast Fourier transform and Welch’s approach with a window of 1 s and an overlap of 50%. A Hanning window was then applied and the grand average of the power spectra was produced over all seven nodes. Five frequency ranges of brain oscillation were selected based on previous tinnitus power spectra analysis reports[Schlee et al., 2009; Vanneste et al., 2011] and our prior study as follows: alpha band, 8–12 Hz; beta 1 band, 13–18 Hz; beta 2 band, 18.5–21 Hz; beta 3 band, 21.5–30 Hz; and gamma band, 38–72 Hz (Supplementary Fig. 1). Correlation coefficients between nodes were estimated using mean band powers of every segment in the power spectral density analysis. Changes in connectivity strength (Δconnectivity = post-connectivity – pre-connectivity) were set as the independent variable. To correlate the functional connectivity with the clinical outcome, the ΔTHI (=post THI – pre THI) was set as the clinically relevant primary outcome measure (dependent variable). The edges that had a significant correlation between the Δconnectivity and ΔTHI were identified in each frequency range (alpha, beta 1, 2, 3, and gamma band). The correlation between the connectivity change and THI was assessed using Spearman’s analysis. The significant correlation between the connectivity change and THI was demonstrated (Supplementary Fig 2 and 3).

**Supplementary Results**

VAS outcome

**Table 2. Visual analog scales (VAS) score for tinnitus symptoms before and after rTMS**.

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| **VAS** | Pre-treatment | 1 wk. | 2 wk. | 4 wk. | 8 wk. |
| **Group 1** |  |  |  |  |  |
| Awareness | 7.5±2.5 | 5.8±3.0**\*** | 5.2±2.9**\*** | 6.2±3.1**\*** | 5.8±3.2**\*** |
| Annoyance | 6.1±2.3 | 4.8±2.4**\*** | 4.3±2.4**\*** | 4.3±2.0**\*** | 4.1±2.0**\*** |
| Loudness | 6.3±1.6 | 5.5±2.2 | 4.4±1.6**\*** | 4.7±2.0**\*** | 4.4±1.6**\*** |
| Effect on daily life | 4.8±1.9 | 3.9±2.1**\*** | 3.4±1.5**\*** | 3.2±2.2**\*** | 3.5±2.1 |
| **Group 2** |  |  |  |  |  |
| Awareness | 7.8±2.7 | 7.3±2.8 | 7.1±3.1 | 6.7±2.7**\*** | 6.8±2.2 |
| Annoyance | 6.5±2.6 | 6.7±1.9 | 5.8±2.0 | 5.6±2.1 | 5.8±1.9 |
| Loudness | 6.9±2.0 | 6.5±2.0 | 6.2±1.7 | 6.5±1.8 | 5.8±2.1 |
| Effect on daily life  | 5.3±2.4 | 6.2±1.9 | 5.8±2.1 | 5.1±2.0 | 4.7±1.7 |

\*Statistically significant improvement vs. pre-treatment VAS score (*p*<0.05); wk., weeks after treatment. Data are presented as means ± standard deviations.

When the VAS score was compared between pre- and post-treatment, a significant improvement in VAS was observed in group 1 at post-treatment weeks 1–8 (Table 2). In contrast, there was no improvement in group 2, except for the VAS on awareness of tinnitus at post-treatment 4 week. When the changes in the VAS scores (ΔVAS) were compared between the two groups, the treatment outcome was significantly better in group 1 compared with group 2 in the loudness scores at 2 (*p* = 0.003) and 4 (*p* = 0.032) weeks after rTMS treatment. When the proportion of patients with a clinically meaningful improvement (ΔVAS for loudness ≤–3)[Ahnblad and Nordkvist, 2017; Dewyer et al., 2015] was evaluated at 1-4 weeks post-treatment, the response rate was 52.9% in group 1 and 15.4% in group 2.

**Supplementary Figure Result:** When each node was analyzed individually, the increased alpha band power was more pronounced in the left cortices (5, left primary AC; 6, left DLPFC; and 7, left inferior parietal cortex) compared with the right cortices (1, right primary AC; 2, right DLPFC; and 3, right inferior parietal cortex, respectively). When the clinically relevant functional architecture of the networks (clinically relevant alpha, beta, and gamma network) was plotted on a 3D rendered brain (Fig. 3), all the significant edges were connected to the (5) left primary AC or (6) left DLPFC. That is, the degree of node was highest in the (5) left primary AC (degree of 5) and (5) left DLPFC (degree of 4). The location of these two nodes, which had the highest centrality, matched the sites to which rTMS stimulation was applied. The degree of the node was between 1 and 3 in the (7) left inferior parietal cortex, (4) dorsal anterior cingulate cortex, and (3) right inferior parietal cortex. It was 0 in the rest of the nodes.

**Supplementary discussion:**

The DLPFC has been studied as the most probable non-auditory target for tinnitus modulation. The DLPFC also inhibits input to the primary auditory cortex[Knight et al., 1989] and is associated with auditory attention,[Voisin et al., 2006] resulting in the top-down modulation of auditory processing.[Mitchell et al., 2005; Vanneste et al., 2011] In 2008, the Regensburg group reported the results of the first pilot study using dual-site rTMS. The rTMS treatment strategy consisted of a combination of high-frequency prefrontal and low-frequency temporal rTMS.[Kleinjung et al., 2008] The combined TMS to the temporal cortex and DLPFC had better long-lasting results for tinnitus-related distress compared with TMS on the temporal cortex alone.[Kleinjung et al., 2008; Vanneste et al., 2011] In this study, unlike the Regensburg study, tinnitus seemed to improve progressively over 8 weeks. The reason why tinnitus improved progressively (getting better over time) over 8 weeks in this study, is probably because the true treatment outcome was underestimate by THI at week 2. That is, the true treatment effect at week 2 might actually be better than what has been acquired in this study, due to inter-study variability or slight difference in the base line THI score. In our former studies with the same rTMS protocol, the improvement in THI score at week 2 was always 15 points.[Noh et al., 2017a; Noh et al., 2017b; Park et al., 2015] But in this study, the improvement in THI score at week 2 was only 10 points. Due to this limited improvement at week 2, the treatment outcome might seem as if it gets better over time at week 4 and week 8. If we presume that the true improvement in THI score was 15 points at week 2 (reaching THI score of 30), the same pattern of improvement over time would have been replicated: best treatment outcome at week 2 and worse outcome with time after 4-8 weeks.

According to the current double-blind randomized controlled trial, active rTMS was superior to sham treatment. The post-treatment THI score improved after rTMS only in group 1 (Fig. 1). An improvement in tinnitus-related VAS (awareness, loudness and annoyance) score was also found only in group 1 (Table 2). The proportion of responders was also higher in group 1 compared with group 2. These results suggest that dual-site rTMS has the ability to suppress tinnitus. We then asked how rTMS worked on the brain to suppress tinnitus. Although this question cannot be fully answered by this study alone, it seems that rTMS potentiates alpha activity (inhibitory oscillations) in the tinnitus network. The grand averaged power spectra analysis revealed that an alpha band oscillation was related to tinnitus (Fig. 2). It was also noticeable that the functional architecture of the connectivity network was concentrated in the left primary AC and left DLPFC, which are the two sites to which rTMS stimulation was applied in the current study.[De Ridder et al., 2011; Kleinjung et al., 2008; Langguth et al., 2014; Park et al., 2013]

Study 2 was an open label pilot study of 5 treatment responders from study 1 without a control; performed to gain insight into how tinnitus might have changed in response to treatment. When the individual functional connection that had a significant correlation with THI was analyzed, the alpha band Δconnectivity was negatively correlated with ΔTHI (alpha). That is, a stronger interareal neuronal connection in the alpha band (positive Δconnectivity) was correlated with a better treatment outcome (negative ΔTHI). This result was consistent with the grand averaged power spectra analysis, since the post-rTMS beneficial effects were closely related to the enhanced alpha band oscillation power. When the location of the clinically relevant interareal neuronal connection was explored, the clinically relevant alpha network was found in the edge between the (6) left DLPFC and (3) right inferior parietal cortex (alpha).

While only the alpha band showed a pronounced change in the grand averaged power spectra analysis, additional clinically relevant beta and gamma connections were found when the individual connectivity was analyzed between each node. For the beta band, Δconnectivity was positively correlated with ΔTHI (beta). That is, an increased beta 1, 2, and 3 band connectivity (positive Δconnectivity) was correlated with a worse treatment outcome (positive ΔTHI). This clinically relevant beta network was located in the edges that connected the (5) left primary AC to the (6) left DLPFC and (3) right inferior parietal cortex (beta).

For the gamma band, Δconnectivity was positively correlated with ΔTHI (gamma). That is, increased gamma band connectivity (positive Δconnectivity) was correlated with a worse treatment outcome (positive ΔTHI). This clinically relevant gamma network was located in the edges that connect the (5) left primary AC to the (7) left inferior parietal cortex and (3) right inferior parietal cortex (gamma). The edge between the (6) left DLPFC and the (4) dorsal anterior cingulate cortex was also positively correlated in the gamma band.

Gamma activity, which can be considered a sign of the enhanced synchronized firing of neurons, appears to be involved in the formation of a conscious perception of tinnitus.[Weisz et al., 2007a] The “oscillatory model of tinnitus” suggests that hearing loss reduces the spontaneous firing of inhibitory neurons (marked by decreased alpha activity). This condition leads to the “release of inhibition”, increased neuronal activity with synchronization in the gamma band,[Weisz et al., 2007a] and the generation of conscious sound perception (tinnitus). When the current results are applied to the model, it seems that active rTMS can potentiate (normalize) the decreased alpha activity in the tinnitus network. This leads to inhibition (normalization) of the abnormally increased gamma activity and, consequently, the suppression of tinnitus. Since MEG recordings were made both before and after rTMS in the same subjects, the current study may provide stronger evidence to support the “oscillatory model of tinnitus”. Previous studies compared the difference in functional connectivity between different subjects (tinnitus patients vs. controls; responders vs. non-responders). In the current study, we were able to elucidate the changes in cortical oscillation activity and functional connectivity before and after rTMS in the same subject.

 There are several limitations to the present study. First, the sample size was small, particularly in study 2, due to limited funding. A study with a larger number of subjects should be performed in the future to draw firm conclusions. However, the use of a within subject design where we compared the pre- and post-treatment MEG recordings in the same subject increased statistical power and reduced the probability of a type-2 error. Second, the source montage that we used in the current study covered only seven nodes of interest. Due to the small number of nodes and the technical constraints that are inherent to the inverse modeling used in MEG,[Schlee et al., 2009] the interpretation of the location of the connections may not be precise. Nevertheless, we believe this shortcoming does not undermine the main finding of the study since we focused on the oscillations and connectivity rather than the precise location of the activity (supplementary material Fig.2). Third, the fact that there was no control group in study 2 is a great shortcoming of this study. It would be highly desirable to have a control group without tinnitus to identify which alterations in the MEG data are related to tinnitus and to analyze whether rTMS treatment contributes to a normalization of the tinnitus related alterations in the future study. Fourth, the sham condition may have been detectable: the contact sensation of the rTMS coil to the scalp can be similar, but the sensation of magnetic pulse cannot be the same. Fortunately, no subject had prior knowledge on the feeling of rTMS. So it would have been difficult for the patients to figure out which group they were allocated to. We have checked this point by unrevealing the group at the end of the trial (8-12 weeks later). And all the subjects in the sham group did not notice that they participated as a sham treatment group subject. But it is true that this factor may have influenced the outcome of our study. Fifth, group 1 had many more men than women than did Group 2. Also, more subjects dropped out of Group 2 than Group 1 (4 versus 0). We are not aware of any literature that proved a difference in rTMS effect for tinnitus depending on the gender. As for the drop-outs, all four drop-out subjects refused to participate in the study for reasons unrelated to the rTMS treatment effect (2 for personal reasons, 2 for hearing discomfort). But it may be true that the difference in gender and drop-out rate can make the comparison difficult. Sixth, there was a large variability in the THI score for both groups as the standard deviation were almost half the size of the mean values when measured at each time point. The large variability is a shortcoming and was not a pliable factor in this clinical trial.

**Conclusion**

We performed a prospective double-blind randomized controlled trial to elucidate the effects of dual-site (AC+FC) rTMS on tinnitus control. A beneficial effect of rTMS on tinnitus suppression was found in the dual-site active rTMS group, but not in the sham rTMS group. We next asked how rTMS works on the brain to modulate tinnitus. When the mean band power changes were compared between pre- and post-treatment, an increased oscillation power was observed in the alpha band after rTMS. However, our study has various limitations that may make it difficult to reach a firm conclusion. For instance, number of subjects was small, there was no control group in study 2, the sham condition may have been detectable in some subjects, and there was a large variability in the THI score. Additional studies that overcome the stated limits are needed to further confirm our outcome.

**Figure.1 Legend**

**Changes in the oscillation power at each node.** When the oscillatory power was analyzed at each node individually, the increased alpha band power was more pronounced in the left cortices (5, left primary AC; 6, left DLPFC; and 7, left inferior parietal cortex) compared with the right cortices (1, right primary AC; 2, right DLPFC; and 3, right inferior parietal cortex, respectively). It is noticeable that rTMS was only applied to the left hemisphere and that the pronounced enhancement in alpha band oscillation was also observed in the left cortices.

**Figure.2 Legend**

**Clinically relevant functional connectivity.** The alpha band Δconnectivity (= post-connectivity – pre-connectivity) was negatively correlated with ΔTHI (= pre-THI – post-THI). That is, a stronger interareal neuronal connection in the alpha band (positive Δconnectivity) was correlated with a better treatment outcome (negative ΔTHI). When the location of the clinically relevant interareal neuronal connection was explored, the clinically relevant alpha network was found in the edge between the 6) left DLPFC and 3) right inferior parietal cortex. Meanwhile, the beta 1, 2, 3, and gamma band Δconnectivity was positively correlated with ΔTHI. That is, an increased beta and gamma band connectivity (positive Δconnectivity) was correlated with a worse treatment outcome (positive ΔTHI). This clinically relevant beta network was located in the edges that connected the 5) left primary AC to the 6) left DLPFC, 3) right inferior parietal cortex, and 7) left inferior parietal cortex. The edge between the 6) left DLPFC and the 4) dorsal anterior cingulate cortex was also positively correlated in the gamma band.

**Figure 3 Legend**

**Clinically relevant functional architecture of the tinnitus networks.** The clinically relevant alpha, beta, and gamma networks were plotted on a 3D rendered brain. All the meaningful edges were connected to the 5) left primary AC or 6) left DLPFC. When “degree of node” was used as a measure of centrality, it was highest in the 5) left primary AC (degree of 5) and 6) left DLPFC (degree of 4). The location of these two nodes, which had the highest centrality, matched the sites of rTMS stimulation. RA, 1) right primary auditory cortex (AC); RF, 2) right dorsolateral prefrontal cortex (DLPFC); RP, 3) right inferior parietal cortex; ACC, 4) dorsal anterior cingulate cortex; LA, 5) left primary AC; LF, 6) left DLPFC; LP, 7) left inferior parietal cortex; ant, anterior; post, posterior.

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