Brain mapping after prolonged cycling and during recovery in the heat

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De Pauw K, Roelands B, Marušič U, Tellez HF, Knaepen K, Meuesen R. Brain mapping after prolonged cycling and during recovery in the heat. J Appl Physiol 115: 1324–1331, 2013. First published August 29, 2013; doi:10.1152/japplphysiol.00633.2013.—The aim of this study was to determine the effect of prolonged intensive cycling and postexercise recovery in the heat on brain sources of altered brain oscillations. After a max test and familiarization trial, nine trained male subjects (23 ± 3 yr; maximal oxygen uptake = 62.1 ± 5.3 ml·min⁻¹·kg⁻¹) performed three experimental trials in the heat (30°C; relative humidity 43.7 ± 5.6%). Each trial consisted of two exercise tasks separated by 1 h. The first was a 60-min constant-load trial, followed by a 30-min simulated time trial (TT1). The second comprised a 12-min simulated time trial (TT2). After TT1, active recovery (AR), passive rest (PR), or cold water immersion (CWI) was applied for 15 min. Electroencephalography was measured at baseline and during postexercise recovery. Standardized low-resolution brain electromagnetic tomography was applied to accurately pinpoint and localize altered electrical neuronal activity. After CWI, PR and AR subjects completed TT2 in 761 ± 42, 791 ± 76, and 794 ± 62 s, respectively. A prolonged intensive cycling performance in the heat decreased β activity across the whole brain. Postexercise AR and PR elicited no significant electrocortical differences, whereas CWI induced significantly increased β3 activity in Brodmann areas (BA) 13 (posterior margin of insular cortex) and BA 40 (supramarginal gyrus). Self-paced prolonged exercise in the heat seems to decrease β activity, hence representing decreased arousal. Postexercise CWI increased β3 activity at BA 13 and 40, brain areas involved in somatosensory information processing. A training schedule comprises exercises at different intensities to enhance exercise performance. This obviously affects the postexercise recovery process and subsequent exercise performance. The rationale for the use of different recovery interventions to enhance exercise performance is based on the assumption that they will confer an ergogenic effect beyond that afforded by passive rest (PR) alone. Two frequently applied recovery interventions in the field are active recovery (AR) and cold water immersion (CWI). The underlying idea is to fasten blood lactate removal, although lactate removal may not be a valid criterion for assessing recovery (2, 11). Immersion in cold water rapidly decreases the postexercise thermal stress (TS), which is especially beneficial in hot environments, but studies also acknowledge its ergogenic effect in a thermoneutral environment (47, 48). Regarding the postexercise recovery period, little or no data are available concerning central aspects. However, revealing underlying central mechanisms involved in recovery might be relevant to the field, because recovery does not solely imply the physiological return to homeostasis, but also psychological factors (mental preparedness, motivation, and mood), before a subsequent exercise performance. Since CWI activates several distinct somatosensory modalities, including tactile, pressure, and thermal sensations, it is suggested that cerebrocortical processing is altered in the parietal somatosensory cortex (brain areas 1 and 3b) (42).
Although evidence is available that exercise-induced fatigue in the heat decreases the relative β-power at several electrode sites (F3, F4, Cz, and Oz) (28, 30), no study included whole brain EEG analysis for the identification of the source of the altered brain activity. Additionally, there is a research-knowledge gap regarding the impact of different postexercise recovery interventions on brain functioning. Therefore, the aim of this experiment is to determine the effect of intensive prolonged cycling and postexercise recovery in the heat on sources of altered brain activity. Despite the introduction of improved imaging techniques, it remains challenging to clearly display neural changes within the human cortex during whole body exercise (45). Therefore, EEG was applied before and after an intensive prolonged cycling performance, as well as during the postexercise recovery period in the heat. sLORETA was used to accurately pinpoint and localize altered neuronal activity. From the available literature, it is expected that prolonged self-paced endurance performance in the heat decreases β activity in specific brain areas, mainly the frontal brain area. It is also expected that postexercise CWI would increase cortical activation and, therefore, β activity in somatosensory brain areas.

MATERIALS AND METHODS

Subjects. Nine male subjects [age = 23 ± 3 yr; weight = 72.6 ± 5.2 kg; relative maximal oxygen uptake = 62.1 ± 5.3 ml·min⁻¹·kg⁻¹; absolute maximal power output = 333 ± 37 W] classified as “performance level 3” (12) performed a max test, familiarization trial, and three experimental trials. Subjects were fully informed of the risks and discomforts associated with the experiments before giving their informed consent to participate. Subjects were required to abstain from caffeine and other psychoactive substances and also from intensive training the day before the experiment. The experiment was approved by the institutional medical ethics commission.

Experimental design. Preceding the experiments, subjects underwent a medical screening. If no contraindication was found, a maximal cycle test was performed. Subjects had no previous experience with a time-trial protocol and EEG setup; therefore, a familiarization trial was included. Subjects completed all trials on the same electromagnetically braked lower extremity cycle ergometer (Lode, Excalibur Sport, Groningen, the Netherlands) in a temperature-controlled chamber (temperature: 30°C, and relative humidity: 43.7 ± 5.6%). Subjects reported to the laboratory at the same time of the day and the same day of the week for 5 consecutive weeks. During the maximal cycle test, the resistance was set at 80 W, and every 3 min the resistance was increased by 40 W until volitional exhaustion. Maximal oxygen uptake was analyzed by a breath-by-breath ergospirometric device (MetaMax 3B, Cortex Biophysik). The individual maximal external power (Wmax) obtained from the maximal test was used to calculate 55 and 75% of the Wmax (W), and the target amount of work (kJ) for the simulated time trial (18). Wmax was defined as the highest power output that could be reached during the maximal test and was determined by the formula: 

\[ W_{\text{max}} = \frac{W_{\text{out}}}{t/180} \times 40 \] 

[where Wout workload of the last completed stage, and t is time (seconds) in the final stage].

Each experimental trial (Fig. 1) consisted of a constant-load trial (55% Wmax) lasting 60 min, immediately followed by a 30-min simulated time trial (TT1). Subjects were instructed to cover a fixed amount of work as fast as possible during the TT1, that began at a workload corresponding to 75% of Wmax, but were free to alter their power output as desired from the outset, meaning that, if the power output was decreased, TT1 duration increases. A computer program displayed a bar indicating the percentage of total work completed to give the subject an indication of his progress. Throughout the protocol, no feedback was provided regarding time lapsed, power output, pedal cadence, or heart rate (HR). During exercise, subjects had ad libitum access to plain water.

After the TT1 completion, a recovery intervention was applied for 15 min in the same environmental conditions. The control intervention was PR, i.e., sitting on a comfortable chair. During CWI, subjects were immersed until the sternum in an inflatable bath (iCool Sport) and the water temperature was set at 15°C. During AR, subjects cycled at a constant resistance of 80 W. For each experimental trial, subjects were assigned to one of the three recovery interventions in a randomized, crossover manner. After the recovery intervention, subjects had to lie down and rest for 45 min in the climate chamber. The second exercise bout consisted of a 12-min simulated time trial (TT2) that was started at 85% of the Wmax. Again subjects had to cover a certain amount of work as fast as possible and were free to increase or decrease their power output as desired from the outset. This protocol was validated (18) and used in several experiments of our laboratory (39, 40). TT2 performance (in seconds and average power output) was an important outcome measure. At regular intervals, the blood lactate concentration ([BLa]), HR, rating of perceived exertion (RPE), TS, and rectal temperature (Trect) were registered. [BLa] was taken with a capillary earlobe sample and analyzed by a Biosen 5030 (EKF, Magdeburg, Germany). For HR, a Geonaute CW T500 HR monitor (Decathlon, Villeneuve d’Ascq, France) was used. The RPE was rated on a scale of 6 (“no exertion”) to 20 (“maximal exertion”) (5), and perceived TS on a 21-point scale of +10 (very, very hot) to −10 (very, very cold) (35). The Trect was monitored via a rectal probe (Gram LT-8A, Saitama, Japan) inserted 10 cm beyond the anal sphincter before testing. During the constant-load trial and the rest period, HR, [BLa], RPE, TS, and Trect were recorded every 10 min. During TT1 and the recovery period, the same parameters were obtained every 5 min, and during TT2 every 3 min.

EEG recordings and electrophysiological analysis. Continuous EEG data were derived from 32 active Ag/AgCl electrodes attached on the subjects’ head (Acticap, Brain Products, Munich, Germany), according to the “10–20 International System” (17). The sampling rate was set at 500 Hz (Brain Vision Recorder, Brain Products, Munich, Germany). Electrode impedance was kept <5 kΩ throughout the recording. During EEG recordings, subjects had been instructed to relax, close their eyes, and maintain the same posture. EEG datasets were obtained at baseline when subjects sat on the bicycle. EEG was also applied during the recovery interventions (Fig. 1). During AR, subjects were instructed to maintain the same body and head posture and pedal cadence to minimize movement and muscle artifacts.

EEGlab was used to preprocess the datasets. Data were re-referenced to an average reference of all electrodes. Bandwidth was defined between 1 and 45 Hz, and notch filter was applied at 45–55 Hz. The filtering method (matlab function fir: EEGfilt) involves the weighted least squares linear-phase finite impulse response filter design. Artifacts were visually removed using Independent Component Analysis (infromanica). Independent components were cate-

![Fig. 1. Design of one experimental trial. *1 represents the used EEG datasets of the passive rest (PR) group to determine the effect of prolonged cycling in the heat on brain functioning. *2 represents the EEG datasets used to compute comparisons within each recovery intervention. Recov, recovery; R, rest; TT2, second time-trial performance.](http://jap.physiology.org/ by 10.220.33.6 on November 4, 2016)
gorized as brain or nonbrain activity, according to the component properties (scalp topographies and activity spectra) and visual inspection of the time course.

Brain imaging technique sLORETA. The source localization method sLORETA attempts to solve the inverse problem by assuming related orientations and strengths of neighboring neuronal sources (34, 36) and proceeds at the voxel (volume element) level. Thus sLORETA analysis was performed to answer the questions whether brain frequency bands differ between different time points and where the differences take place (brain localization). Time points during the experiment were baseline and the first part of the postexercise PR intervention. The source localization

time interval set at 0.017.

Statistical analysis. Data (TT2 performance, \([BLa]\), HR, RPE, TS, Trect, and skin temperature) were not normally distributed, and therefore nonparametric Friedman tests for each dependent variable were performed to observe differences between interventions for each time point. Post hoc analysis with Wilcoxon signed-rank tests was conducted with a Bonferroni correction applied, resulting in a significance level set at \(P < 0.017\).

sLORETA statistical analyses are performed at voxel level, involving the formation and assessment of a statistic image or statistical nonparametric map, which showed the highest possible statistical power (27). As shown in Fig. 1, sLORETA files were used at baseline and the first part of the PR group to outline the exercise-induced alterations of the frequency bands and responsible brain areas. Paired comparisons (with one single test: log of ratio of averages) were performed for all time frames and selected frequency bands. To localize recovery-induced altered frequency bands, the datasets of the first and third part of each recovery period (within-group comparisons) were used. No a priori anatomical hypotheses existed, and, therefore, multiple tests needed to be performed at all voxels simultaneously (27). To correct for multiple comparisons, the statistic program of sLORETA is based on Fisher’s permutation test (15) and relies on a bootstrap method with 5,000 randomizations.

An important outcome of sLORETA statistics was the critical threshold \((t_{\text{critical}})\). Voxels with statistic values exceeding the \(t_{\text{critical}}\) have their null hypotheses rejected. The omnibus null hypothesis (combined voxel hypotheses) states that there was no activation anywhere in the brain, and, if rejected (at \(P < 0.05\), a significant difference in a specific frequency band existed at these voxels between two conditions. The statistical nonparametric map method provided voxel information i.e., Montreal Neurological Institute/Talairach coordinates, Brodmann area (BA), lobe, and structure.

RESULTS

**TT2 performance and physiological responses to recovery.** Friedman tests showed no significant TT2 performances between the recovery interventions. After CWI, PR and AR subjects completed TT2 in 761 ± 42 s (average power output: 269 ± 37 W), 791 ± 76 s (average power output: 259 ± 39 W), and 794 ± 62 s (average power output: 256 ± 33 W), respectively. The physiological responses to recovery are presented in Table 1.

**Prolonged cycling in the heat and brain mapping.** Statistical overall analysis by means of the omnibus significance test revealed decreased \(\beta1, \beta2, \text{ and } \beta3\) activity in the whole brain area after a prolonged intensive cycling performance in the heat (two-tailed \(t_{\text{critical}}\); \(P < 0.05 = 2.332\)). As shown in Table 2, the highest number of significant voxels for \(\beta2\) and \(\beta3\) activity (Fig. 2) were observed in the temporal lobe at BA 20 (number of significant voxels for \(\beta2\): 175, highest statistical voxel value: 2.807; \(\beta3\): 221, highest statistical voxel value: 2.828), BA 21 (number of significant voxels for \(\beta2\): 199, highest statistical voxel value: 2.897; \(\beta3\): 219, highest statistical voxel value: 2.833), BA 22 (number of significant voxels for \(\beta2\): 84, highest statistical voxel value: 2.907; \(\beta3\): 112, highest statistical voxel value: 2.770), and BA 38 (number of significant voxels for \(\beta2\): 116, highest statistical voxel value: 2.667; \(\beta3\): 158, highest statistical voxel value: 2.694). High statistical voxel values were also found in BA 40 (parietal lobe), BA 41, BA 42 (temporal lobe), and BA 43 (highest statistical voxel value range: 2.648–2.894). The \(\beta1\) band
Table 2. Brodmann areas (plus number of voxels) that showed significantly decreased β2 and β3 activity post- compared with preexercise conditions

<table>
<thead>
<tr>
<th>BA</th>
<th>Frequency Band</th>
<th>Highest Statistical Voxel Value</th>
<th>MNI Coordinates (X, Y, Z) mm</th>
<th>No. of Voxels</th>
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<td>4</td>
<td>β2</td>
<td>2.66083</td>
<td>60, -5, 15</td>
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</tr>
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<td>40, -20, -10</td>
<td>12</td>
</tr>
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<td>40, -20, -10</td>
<td>33</td>
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<td>65, -25, -20</td>
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<td>45, 5, -45</td>
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<td>β3</td>
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<td>β3</td>
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<td>55, -20, 5</td>
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<tr>
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<td>20</td>
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<td>β2</td>
<td>2.89446†</td>
<td>65, -15, 10</td>
<td>28</td>
</tr>
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<td>65, -15, 10</td>
<td>31</td>
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<td>65, -15, 15</td>
<td>13</td>
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<tr>
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<td>β3</td>
<td>2.56041</td>
<td>-40, 20, -20</td>
<td>70</td>
</tr>
</tbody>
</table>

BA, Brodmann areas; MNI, Montreal Neurological Institute. β2 and β3 activity was significantly decreased (P < 0.05; tP < 0.01) for post- compared with preexercise conditions [two-tailed critical threshold (t_critical) for P < 0.1 = 2.078; P < 0.05 = 2.332, and P < 0.01 = 2.854]. Voxels values above 2.5 are presented. MNI coordinates of the voxel with the highest statistical value are shown. Number of voxels with a statistical value higher than t_critical are shown.

Also showed decreased activity in the temporal lobe (BA 20, 21, 22, 38, 41, 42, and 43; highest statistical voxel value range: 2.34380–2.49272).

Postexercise recovery and brain mapping. The recovery period was divided into three equal parts to outline recovery-induced changes of the frequency components and responsible brain areas. Therefore, comparisons between the first and third (last) part of each recovery intervention were computed within each postexercise recovery intervention. Interestingly, during AR and PR, no significant differences in electrical neuronal activity were observed, whereas CWI induced significantly increased β3 activity (two-tailed t_critical, P < 0.05: 0.945). The most active neuronal generators are distributed in BA 13 (insular cortex, posterior part, highest statistical voxel value: 0.949) and BA 40 (polar lobe, supramarginal gyrus, highest statistical voxel value: 0.950) (Table 3, Fig. 3).

DISCUSSION

The present study examined and located altered brain oscillations due to prolonged intensive cycling performance and postexercise recovery in the heat using sLORETA. Although no significant subsequent time-trial performance differences were observed among the intervention groups, CWI, subjects were able to maintain a high-power output from the outset of exercise compared with an immediate decrease of the power output after AR and PR. Thus an altered pacing strategy during subsequent time-trial performance emerged. We redirect the reader to De Pauw et al. (13) for more detailed information regarding the physiological parameters and pacing strategy during TT1. To our knowledge, the present study is the first to apply a brain imaging technique to artifact-free EEG datasets after prolonged intensive cycling performance and during the postexercise recovery period in the heat. Most studies regarding exercise and brain functioning examined the α- and β-frequency band (4, 23, 28), but failed to provide more detailed information within the frequency bands. One of the main findings of our study was that prolonged cycling in the heat decreased β activity, especially in the higher frequency ranges (β2 and β3), across the whole brain area. Another finding was that postexercise AR and PR in the heat did not affect the electrical neuronal activity, whereas CWI increased β3 activity at BA 13 (posterior insular cortex) and BA 40 (supramarginal gyrus), brain areas involved in the integration of somatosensory information.

Exercise stresses the homeostatic status of an athlete and increases the arousal level, defined as the current energetic level of the organism (3). Bonnet et al. (4) observed that increased physiological arousal predominantly results in low-amplitude, high-frequency waves, and Moraes et al. (26) suggested that the augmented β-frequency waves are also related to greater cortical activation, increased attentional demand, metabolism, and body temperature. In contrast to the aforementioned experiments, the present study was performed in the heat, and observations show that self-paced high-intensity cycling in the heat decrease β activity over the whole brain area. Two possible underlying mechanisms might explain this observation.

First, it is widely acknowledged that performance in the heat is attenuated compared with exercise performance in cold or temperate conditions. In the experiment of Nielsen et al. (28), subjects performed a prolonged cycling exercise in the heat and showed a progressive decrease of the relative β activity. Higher activity in the β-band is believed to reflect corticocortical and thalamocortical transactions related to specific information processing and vice versa (16). Since a combination of body and brain temperature, and an indirect effect of different information trafficking from nociceptors and thermo- and mechanoreceptors to the brain presumably lead to increased central fatigue, observed performance decrements in the heat are most likely due to inhibitory signals from the thalamus-hypothalamus to different brain areas involved in sensory and motor processing. Possibly these inhibitory signals lead to the observed decreased β activity (28). Caution is needed regarding the results of Nielsen et al. (28), because no more than two electrodes (F3 and F4) were used to compute the relative power spectrum (using fast Fourier transform).

Second, the intensity of the cycling bout might explain the decreased β activity along the whole brain area. Interesting experiments of Schneider et al. (44) and Brümmer et al. (6) showed similar findings after self-selected high-intensity exercise in a thermoneutral environment. They subjected regular
runners to (self-selected) high- and low- or moderate-intensity running and observed decreased activity after the high-intensity running at 5 of 19 electrode sites (F7, C3, C4, P8, O2) (44) and brain areas involved in emotional processing (BA 11, 25, 47) (6). They postulated that higher intensity exercise in the preferred sport leads to a higher feeling of calmness, a sense of well being, and/or positive emotions. In the present study, regular cyclists were included and subjected to a self-paced, high-intensity cycling. Thus a relaxational effect after high-intensity exercise may have influenced activity, perceived psychological strain, and motivational state (44). Although Brümmer et al. (6) and Schneider et al. (44) solely observed alterations in brain regions linked with emotional processing during experiments in thermoneutral temperature, findings of the current experiment in the heat showed the involvement of the whole brain, hence representing a more general decrease in arousal.

When focusing on the postexercise recovery period, AR and PR did not elicit electrocortical alterations. CWI induces tactile (hydrostatic pressure of water) and thermal sensations (cold water) and affects cerebrocortical processing in the somatosensory cortex (14) and cardiovascular parameters (decreased HR, increased stroke volume) (13, 47, 48). sLORETA revealed that CWI increased β3 activity in the posterior margin of the insular cortex (BA 13) and the supramarginal gyrus (BA 40), regions associated with processing of somatosensory activity (24). In this case, the term activation is more appropriate, reflecting

Table 3. CWI-induced alterations in the β–frequency range between the last and first part of the recovery period

<table>
<thead>
<tr>
<th>Frequency Range</th>
<th>BA</th>
<th>Statistical Voxel Value</th>
<th>MNI Coordinates (X, Y, Z), mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>β3 60-40</td>
<td>40</td>
<td>0.950062</td>
<td>−60, −45, 20</td>
</tr>
<tr>
<td>β3 60-30</td>
<td>13</td>
<td>0.949223</td>
<td>−55, −45, 20</td>
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</table>

Two-tailed t adjusted for \( P < 0.10 = 0.097; P < 0.05 = 0.945, \) and \( P < 0.01 = 0.982. \) MNI coordinates of the voxel with the statistical value are shown.
regional brain processing, because the stimulus altered brain processing in specific brain regions (3). In the past, Craig (8), Egan et al. (14), Menhert et al. (22), and Minoshima and Casey (25) applied functional imaging techniques during cold stimuli and revealed high activation of the posterior insular cortex. Thus evidence suggests the involvement of the posterior insular cortex rather than parietal somatosensory cortices in temperature sensation.

The posterior insular cortex, a cortical “island” buried within the lateral sulcus, has been linked to cardiorespiratory activity (29), emotion (38), temperature sensation (9, 32), and the modulation of the body’s homeostasis (10, 33) and has close connections with the hypothalamus (8). Since CWI rapidly declines $T_{rect}$ and TS, it is worth mentioning that the hypothalamus houses temperature centers that integrate efferent and afferent signals for the control of the autonomic and behavioral thermoregulatory responses (28). Another interesting observation from the study of Johannsen et al. (19) is the activation of BA 40 in tasks involved with sustained and vibrotactile attention. The present study focused on brain functioning, although it might be assumed that recovery also influences psychological factors, such as behavior, motivation, attention, and mood (49). Future research should, therefore, aim at identifying links between recovery, psychological status, and altered exercise performance.

As mentioned before, it remains challenging to clearly display neural changes within the human cortex during whole body exercise (45). Under specific circumstances, such as exercising in the heat, a high perspiration rate will increase the risk of electrode bridging, which results in a distortion of the signal. Exercise might also induce muscle and electrode movement artifacts. Therefore, research involving EEG and exercise is mainly limited to differences between pre- and postexercise conditions (23, 44). A limitation of pre- and postexercise measurements is that a loss of information might occur due to a time delay between the postmeasurement and the end of the first cycling bout. Combining EEG with near-infrared spectros-
copy method (37, 42) might be appropriate for studying corti-
cal activation during exercise in future studies.

In conclusion, prolonged, intensive, self-paced cycling per-
formance in the heat decreased β activity along the whole
brain, hence representing decreased arousal. Postexercise AR
and PR did not elicit altered brain oscillations, whereas CWI
increased β3 activity at the posterior part of the insular cortex
(BA 13) and the supramarginal gyrus (BA 40), brain areas
linked to somatosensory information processing.

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DISCLOSURES

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AUTHOR CONTRIBUTIONS

Author contributions: K.D.P., B.R., and K.K. interpreted results of experiments; K.D.P. performed experiments; K.D.P., U.M., and H.F.T. analyzed

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