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Acute exercise modulates feature-selective responses in human cortex

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An organism’s current behavioral state influences ongoing brain activity. Nonhuman mammalian and invertebrate brains exhibit large increases in the gain of feature-selective neural responses in sensory cortex during locomotion, suggesting that the visual system becomes more sensitive when actively exploring the environment. This raises the possibility that human vision is also more sensitive during active movement. To investigate this possibility, we used an inverted encoding model (IEM) technique to estimate feature-selective neural response profiles from electroencephalography (EEG) data acquired from participants performing an orientation discrimination task. Participants \((n = 18)\) fixated at the center of a flickering \((15\) Hz) circular grating presented at one of nine different orientations and monitored for a brief shift in orientation that occurred on every trial. Participants completed the task while seated on a stationary exercise bike at rest and during low- and high- intensity cycling. We found evidence for inverted-U effects; such that the peak of the reconstructed feature-selective tuning profiles was highest during low-intensity exercise compared to those estimated during rest and high-intensity exercise. When modeled, these effects were driven by changes in the gain of the tuning curve and in the profile bandwidth during low-intensity exercise relative to rest. Thus despite profound differences in visual pathways across species, these data show that sensitivity in human visual cortex is also enhanced during locomotive behavior. Our results reveal the nature of exercise-induced gain on feature-selective coding in human sensory cortex and provide valuable evidence linking the neural mechanisms of behavior state across species.
INTRODUCTION

The behavioral state of an organism has dramatic effects on sensory evoked brain responses. Clear demonstrations of these effects come from studies that compare visual cortical activity in awake and anesthetized animals (Greenberg, Houweling, & Kerr, 2008; Sellers, Bennett, Hutt, Williams, & Fröhlich, 2015). Recent neural recordings in awake and behaving animals and invertebrates also reveal robust modulation of neural activity as a function of behavioral state, with evidence that locomotion can influence response gain in visual cortex and subcortical structures (Ayaz, Saleem, Schölvinck, & Carandini, 2013; Chiappe, Seelig, Reiser, & Jayaraman, 2010; Fu et al., 2014; Keller, Bonhoeffer, & Hübener, 2012; Maimon, Straw, & Dickinson, 2010; Niell & Stryker, 2010; Polack, Friedman, & Golshani, 2013; Saleem, Ayaz, Jeffery, Harris, & Carandini, 2013). In the human, not only is cognitive performance influenced by changes in behavioral state that occur with physical activity (Chang, Labban, Gapin, & Etnier, 2012; Lambourne, Audiffren, & Tomporowski, 2010), but a number of studies have used electroencephalography (EEG) to reveal modulation of brain activity during exercise (Cheron et al., 2016). These investigations indicate that physical activity impacts upon global oscillatory brain activity (Bailey, Hall, Folger, & Miller, 2008; Fumoto et al., 2010; Hottenrott, Taubert, & Gronwald, 2013; Ludyga, Hottenrott, & Gronwald, 2016) as well as patterns of activation that relate to specific stages of cognitive function (Bullock, Cecotti, & Giesbrecht, 2015; De Sanctis, Butler, Malcolm, & Foxe, 2014; Grego et al., 2004; Pontifex & Hillman, 2007).

While there is evidence that locomotion may act as a gain control mechanism in mouse visual cortex (Ayaz et al., 2013; Keller et al., 2012; Niell & Stryker, 2010; Polack et al., 2013; Wilson & Glickfeld, 2014) other evidence suggests that the mechanism is not consistent across all cell types or at all stages of the visual pathway (Erisken et al., 2014; Saleem et al., 2013).
Recent data from mouse auditory cortex also suggest a non-linear “inverted-U” relationship (Yerkes & Dodson, 1908) between the intensity of physiological arousal and the neuronal response, such that sensory responses were largest and most reliable during moderate arousal when compared to at rest and high arousal (McGinley, David, & McCormick, 2015). Given the vast differences between rodent and primate visual pathways, there is no a priori reason to expect that the human visual system should exhibit the same response to physical activity that has been observed in other species (Laramée & Boire, 2015). However, recent data collected from human participants revealed exercise-induced increases in the scalp-recorded visual evoked P1 event-related potential (ERP) during moderate-intensity exercise when compared to rest and intense exercise (Bullock et al., 2015). These data suggest that physical activity may induce sensory gain in a manner consistent with the inverted-U model, but they do not inform us of the specific nature of the gain and how this impacts upon feature-selective coding in sensory cortex.

Thus, the mechanisms that mediate changes in visual processing that occur with physical activity in human visual cortex are unclear.

Here we tested the effects of physical-activity induced changes in behavioral state on feature-selective, population level neural encoding of visual information in the human brain. To estimate feature-selective response profiles, we applied a computational technique that uses spatially specific patterns of neural activity recorded via non-invasive human neuroimaging techniques. This computational technique, referred to as an inverted encoding model (IEM) has been applied to the BOLD signal measured with fMRI to estimate feature and spatially selective neural response profiles in retinotopically organized visual cortex (Brouwer & Heeger, 2009, 2011, 2013; John T. Serences & Saproo, 2012; Sprague, Saproo, & Serences, 2015). More recently, this method has been used with scalp-recorded EEG to uncover the temporal dynamics
of feature-selective processing in the human brain (Garcia, Srinivasan, & Serences, 2013) and
the contents of spatial working memory (Foster, Sutterer, Serences, Vogel, & Awh, 2015). We
recorded EEG while participants performed an orientation discrimination task during three
different behavioral states: at rest and during low- and high-intensity bouts of cycling exercise.
We then employed an IEM approach to reconstruct orientation-selective population level
response profiles for each condition. Given that previous work with both humans and mice
demonstrates inverted-U shaped gain in cortex as a function of exercise intensity (Bullock et al.,
2015; McGinley et al., 2015) we predicted similar U-shaped effects may also occur in the present
data. Consistent with our predictions, we found evidence for multiplicative gain during low-
intensity exercise when compared to rest and high-intensity exercise. Furthermore, we observed
reduced tuning profile bandwidth during low-intensity exercise relative to rest. These data reveal
the effects of physical activity on feature-selective coding in human sensory cortex and provide
valuable evidence linking the neural mechanisms of behavior state across species.

METHODS

Participants. 18 adult volunteers from the University of California, Santa Barbara
community took part in the study, either in exchange for course credit or for financial
compensation of $20 per hour. The sample size was determined based on previous studies that
have applied an IEM approach to EEG data (Foster et al., 2015; Garcia et al., 2013) as well as
investigations of exercise effects on cortical activity (e.g. Bailey et al., 2008; Hottenrott et al.,
2013; Ludyga, Hottenrott, et al., 2016). Demographic and physiological data are reported in
Table 1. All participants reported having normal vision. All participants completed the Physical
Activity Readiness Questionnaire (PAR-Q; National Academy of Sports Medicine) in order to
determine their eligibility to participate in aerobic activity. Informed consent was obtained
before the start of the experiment. All procedures were approved by the UCSB Human Subjects Committee and the US Army Human Research Protection Office.

**Visual stimuli.** Visual stimuli consisted of a circular, square wave grating (spatial frequency of 2 cycles per degree, subtending 7° of visual angle) superimposed with a central fixation point (subtending 0.5° of visual angle). Stimuli were presented on an 19 inch ViewSonic E90f CRT monitor with custom scripts that utilized the Psychophysics Toolbox for MATLAB (Brainard, 1997). Participants viewed the screen at a distance of 110 cm. The eye-tracker was positioned 60 cm from the eye.

**Stationary bike set-up.** The stationary bike was a CycleOps 400 Pro Indoor Cycle (Saris Cycling Group, Madison, WI, USA). T2+ Profile Design Aero Bars (Profile Design, Long Beach, CA, USA) were attached to the handlebars and a Logitech Trackball Mouse (Logitech, Newark, CA, USA) was fixed to the end of the bars. The equipment setup is shown in Fig. 1a. The addition of the aero bars allowed participants to lean their body weight onto the elbow pads, leaving their hands free to press mouse buttons during the experiment. The bars also helped stabilize the participant and reduce movement of the head and upper body, which is important for reducing the motion artifact during EEG recording. Trainer Road software (Trainer Road, Reno, NV, USA) was used to control the stationary bike, and a CycleOps wireless heart rate monitor was used to monitor heart rate. Our equipment setup is based on the setup described in (Pontifex & Hillman, 2007) and we have used a similar setup in two previous studies (Bullock et al., 2015; Bullock & Giesbrecht, 2014).

**Procedure.** Each participant volunteered for two sessions, a pre-testing session and the main testing session. The pre-testing session was conducted ~1 week prior to the main session. The pre-testing served four key purposes. First, the eye-tracker (Eyelink 1000 Plus, SR
Research, Ltd., Mississauga, Ontario, Canada) was tested to ensure that each participant’s eye position could be tracked successfully in remote mode. Second, the level of task difficulty for the orientation task was determined for each individual using the method of constant stimuli. Third, each participant’s VO2max was estimated using the Astrand-Rhyming submaximal bike test (Åstrand & Ryhming, 1954). Fourth, seat height and bar position was set to ensure that each participant was comfortable on the stationary bike and able to maintain a steady pedaling cadence at ~50 RPM.

At the start of the pre-testing session participants mounted the bike and were familiarized with the task and given blocks of practice trials. They then completed a method of constant stimuli procedure consisting of 54 trials of the rotation offset task (see main-testing session procedures) presented at six different difficulty levels (ranging between 1° to 8° of rotation offset) in a fully randomized order. This procedure lasted ~40 minutes and was completed at rest (not pedaling). A Weibull function was then fit to the data in order to estimate an orientation deviation that results in 80% accuracy. Participants then completed the Astrand-Rhyning submaximal bike test (Åstrand & Ryhming, 1954; Bullock & Giesbrecht, 2014). During the test participants were coached to minimize upper body and head movement, and they were instructed to maintain a smooth pedaling cadence of 50 revolutions per minute (RPM) in time to a metronome that sounded at 100 beats per minute (BPM). The values obtained from this procedure provided an estimate of maximal oxygen consumption (VO2max).

In the main testing session, EEG data were recorded for each participant using a Brain Products ActiCHamp system (Brain Vision LLC, Morrisville, NC) consisting of 64 active electrodes arranged in an actiCAP elastic cap and placed in accordance to the 10-20 System. The TP9 and TP10 electrodes were adhered directly to the right and left mastoids. Data were
sampled at 500 Hz and referenced to the average mastoid signal. At the beginning of each investigation all impedances were < 15 kΩ. Participants were then familiarized with the Ratings of Perceived Exertion (RPE) scale (Borg, 1970). RPE is a subjective rating of the intensity of physical sensations a person experiences during physical activity, including increased heart rate, respiration rate, muscle fatigue and physical discomfort. Participants reported their RPE throughout the experiment by viewing the Borg scale and reporting a number between 6 (no exertion) and 20 (maximum exertion).

After the EEG electrodes and wireless heart rate monitor were mounted, participants then mounted the stationary bike and completed trials of the rotation offset task (Fig. 1b). Each trial began with the participant fixating on a centrally presented fixation cross. The trial was initiated by pressing the right mouse button, and after a brief pause (500 ms) a circular grating stimulus appeared and cycled on/off with a blank grey screen at 15 Hz for 3 seconds. The 15 Hz flicker was intended to induce a steady state response that served as the basis for our analysis. The grating was comprised of alternating black and white bars presented at one of nine different orientations, ranging from 0° - 160°. During the trial the grating rotated either counterclockwise (CCW) or clockwise (CW) for three stimulus cycles, and at the termination of the trial the participant pressed a button to indicate the direction of the rotation offset. The size of the target rotation offset was determined on an individual basis according to each participant’s 80% performance threshold calculated using the method of constant stimuli during the pre-testing session. The target occurred towards the end of the trial (randomized between 2000 ms and 3000 ms) on 80% of trials so as to minimize contamination of the orientation selective response profile. On 20% of trials the target occurred earlier in the trial (between 133 ms and 2000 ms) to ensure that participants did not develop a strategy of only attending to the final 1000 ms of the
Eye position was sampled at 500 Hz throughout each trial using an Eyelink 1000 plus eye-tracker (SR Research, Ltd., Mississauga, Ontario, Canada) in “remote” mode. If the participant blinked or moved their eyes more than 1.75° away from the central fixation cross the trial was immediately terminated and the message “Broken Fixation!” appeared on the screen. Aborted trials were appended to the end of each block, thus ensuring that each participant completed exactly the same number of trials in each condition. Head position was also sampled at 500 Hz throughout each trial by logging the position of a small target sticker placed on the participant’s forehead, relative to the position of the eye-tracker.

Each participant performed the orientation task at rest and during low- and high- intensity bouts of cycling exercise. At rest the participant did not pedal; during low-intensity exercise the resistance on the bike was fixed at a minimal level (50W); during high-intensity exercise the resistance was set at a level that participants self-reported to be “somewhat hard” (12-14 on the rating of perceived exertion (RPE) scale; Borg, 1970). Participants were instructed to pedal at 50 RPM to the beat of a metronome in order to keep cadence consistent across participants and conditions. Exercise condition order was fully counterbalanced across the sample. Participants performed the task until they had completed 36 unbroken trials per block and 10 blocks per condition (broken trials per block (Mean ± SEM): Rest: 12.9% ± 1.48, Low: 18.47% ± 3.50, High: 19.12% ± 3.84).

Each condition took ~40 minutes to complete, excluding warm up and cool-down time in the low- and high- intensity exercise conditions. Prior to the first active condition (either low- or high- intensity exercise, depending on counterbalancing order) participants warmed up for ~5 minutes while being given further coaching to ensure they maintained a smooth pedaling cadence to the beat of the metronome and minimized head and body movement. Care was taken
to ensure that heart rate returned to within 15 BPM of resting heart rate after the completion of
one active condition before starting the next condition. The entire EEG session took ~5 hours.

**EEG data preprocessing.** MATLAB (version 2013b, Massachusetts, The MathWorks Inc.) was used for offline processing of the EEG data, along with the EEGLAB toolbox (Delorme & Makeig, 2004). The continuous data were low-pass filtered at 30 Hz to remove high-frequency muscle movement artifacts (Bullock et al., 2015; De Sanctis et al., 2014; Pontifex & Hillman, 2007) and high pass filtered at 4 Hz to remove low-frequency activity caused by sweating. The data were epoched between -.5 and 2.5 seconds, trials with blinks/broken fixations were removed and the data were then submitted to a threshold rejection routine, whereby any electrode with a kurtosis distribution exceeding 5 standard deviations from the mean was excluded (mean number of electrodes excluded: rest 3.9 ± .4 electrodes; low 3.5 ± .5 electrodes; high 3.4 ± .4 electrodes) and trials exceeding ±150 µV in remaining channels were excluded (mean number of trials excluded: rest 4.4 ± 2.2; low 10.9 ± 5.3; high 4.3 ± 2.3). Trials with early target onsets (<2000 ms) were excluded and the remaining trials were cropped to precisely 2 seconds (30 complete stimulus presentation cycles at 15 Hz), thus removing any contamination of the neural response by the target rotation onset. The final step of preprocessing involved converting the 2 s of pre-target data from the included electrodes and each included trial into the single-sided Fourier spectrum using the standard FFT function in MATLAB (fft.m).

**Pattern classification analysis.** To determine the extent to which the stimulus evoked responses carried information about orientation, a linear discriminant classifier was trained on the estimates of power and phase angle based on the real and imaginary components of the Fourier coefficients at the stimulation frequency (i.e., 15 Hz). Leave-one-out cross validation was used to train and test the classifier. Classifier performance was measured by converting
correct classifications to proportion correct (n correct classifications/total classifications) and comparing to chance (1/9=0.111). Hypothesis tests were evaluated against an empirical null distribution estimated using resampling (see **Hypothesis testing**).

**Inverted encoding model.** We used an IEM to reconstruct orientation selective tuning profiles based on the spatial distribution of stimulus-evoked activity across the scalp. The goal of the first part of the model is to estimate the extent to which the linear combination of *a priori* canonical responses (i.e., a basis set) captures the underlying structure in the observed data. The goal of the second part is to determine how much information the response pattern contains about the stimulus features, to the extent that it supports an accurate stimulus reconstruction. This essentially allows the overall shape of the reconstruction to be quantified. The method adopted here was initially used in fMRI studies (Brouwer & Heeger, 2009, 2011; Ester, Sprague, & Serences, 2015; Naselaris, Kay, Nishimoto, & Gallant, 2011; John T. Serences & Saproo, 2012) and recently applied to scalp-recorded EEG (Foster et al., 2015; Garcia et al., 2013). Like decoding (i.e., pattern classification), inverted encoding models involve both training and testing. Here, training was performed using all trials but one and testing was performed on the single trial left out. More specifically, for a given individual and condition, \( m \) represents the number of EEG electrodes in each dataset, \( n_1 \) the number of trials in the training set (~281 trials,) and \( n_2 \) be the number of trials in the testing set (1 trial). Let \( j \) be the number of hypothetical orientation channels (\( C_j \times n_1 \)), composed of half-sinusoidal functions raised to the seventh power as the basis set. In the current study 9 equally spaced orientations were used (i.e., \( j=9 \)). Raising the functions to the seventh power was intended to approximate the orientation bandwidth of orientation selective cells in primate visual cortex (Gur & Snodderly, 2007; Ringach, Bredfeldt, Shapley, & Hawken, 2002; Ringach, Shapley, & Hawken, 2002). For each cross-validation step,
the data were separated into independent training and testing sets. For each train-test iteration, $B_1 (m \times n_1)$ represents the training set and $B_2 (m \times n_2)$ the test set. A standard implementation of the general linear model (GLM) was then used to estimate the weight matrix $(W, m \times j)$ using the basis set $(C_1)$. More specifically, using the GLM:

$$B_1 = WC_1 \quad \text{(Equation 1)}$$

Then, the ordinary least-squares estimate of $W$ can be computed as:

$$\hat{W} = B_1 C_1^T (C_1 C_1^T)^{-1} \quad \text{(Equation 2)}$$

Using the estimated weight matrix $(\hat{W}, \text{equation 2})$ and the test data $(B_2)$, the channel responses $C_2 (j \times n_2)$ can be estimated by:

$$\hat{C}_2 = (\hat{W}^T \hat{W})^{-1} \hat{W}^T B_2 \quad \text{(Equation 3)}$$

After the $\hat{C}_2$ was solved for each orientation, the channel response function on each trial was then circularly shifted to a common stimulus-centered reference frame, and the centered response functions were averaged. Thus, by convention, the $0^\circ$ point on the x-axis refers to the orientation of the stimulus that evoked the response profile. The final step was then to square the absolute value of the stimulus centered response function to obtain a measure of power ($\mu V^2$).

To investigate the source of potential feature-selective modulations, we fit a von Mises distribution to the observed data. Doing so allowed us to estimate tuning profile response bandwidth; an approach that is consistent with measures of orientation-selectivity used in both population level human fMRI and single-unit animal studies (e.g. Niell and Stryker, 2008, 2010; Serences et al., 2009; Byers and Serences, 2014), as well as gain factor and baseline. The CTFs for each participant and exercise condition were independently fit with a von Mises function (Equation 4) with mean ($\mu$), concentration ($k$), gain ($g$) and baseline ($b$) as independent free parameters that reflect distinct attributes of the function. The parameter $\mu$ is analogous to the
mean in the normal distribution and $k$ is analogous to the inverse of the variance. Thus, $k$
represents tuning bandwidth (a larger $k$ value reflects increased concentration around the mean, hence reduced bandwidth).

$$f(\theta) = g \cdot e^{k[\cos(\mu - x) - 1]} + b \quad \text{ (Equation 4)}$$

The von Mises function was fit to the data for each participant/condition 150 times using initial seed values for $g$ (0 to 2), $k$ (0 to 8) and $b$ (-3 to 3). The $\mu$ seed value was fixed at $\pi/2$. Ranges of initial seed values were used to help ensure that the fitting algorithm did not get consistently stuck in a local minimum. The set of parameters for each participant and condition that yielded the lowest root mean squared error across the 150 iterations were then used for subsequent analyses.

**Pupil area, gaze position, and head motion.** Pupil area, gaze position, and head-motion data were extracted from the eye-tracking log file. Pupil area was recorded in arbitrary units; eye gaze position in X and Y screen coordinates which were converted to degrees of visual angle; and head position in arbitrary X (horizontal travel) and Y (vertical travel) units. Pupil area and head motion data were normalized between 0 and 1 using the equation

$$x_{new} = \left( x - x_{\min} \right) / \left( x_{\max} - x_{\min} \right).$$

For consistency with the EEG data, trials with early target onsets (<2000 ms) were excluded and remaining trials were cropped to precisely 2 seconds. Head-motion data from 5 of the 18 participants were not logged, so the remaining 13 participants data were analyzed. To quantify changes in gaze and head motion, a measure of **sample dispersion** (the distribution of sample cluster around its mean) was calculated for each trial.

**Sample dispersion** $S_d$ is defined according to the equation $S_d = \sqrt{S_x^2 + S_y^2} / 2$ where $S_x^2$ and $S_y^2$ represent the horizontal and vertical variances of the sampled cluster (Juni, Gureckis, &
\(S_d\) scores were then averaged across trials to obtain mean gaze dispersion and head motion dispersion scores for each participant and condition.

**Hypothesis testing.** Statistical significance of the hypothesis tests was assessed using a non-parametric permutation based resampling technique to empirically approximate null distributions for the \(F\) and \(t\) statistics (Foster et al., 2015). This approach has the advantage of being robust to violations of normality. The null distributions were generated according to the type of data being analyzed. Specifically, for the univariate repeated-measures analyses we shuffled the condition labels within subjects and ran 1000 iterations of the appropriate repeated measures ANOVA and post-hoc \(t\)-tests, which we then used to generate null distributions of \(F\) values and \(t\) statistics. For the multivariate decoding and IEM analyses we shuffled the orientation labels and ran 1000 iterations of the model, reshuffling the labels with every new iteration to create a matrix of 1000 null CTFs for each subject and condition. Where appropriate we then ran repeated-measures ANOVAs, one-sample \(t\)-tests, or paired sample \(t\)-tests on each of the 1000 iterations to generate null distributions of \(F\) values and \(t\) statistics. Once we obtained null distributions for each of our data sets, we then tested for reliable difference by calculating the probability of obtaining \(F\) and \(t\) statistics from each of the null distributions that were greater than the observed \(F\) and \(t\) statistics. The standard observed \(F\) and \(t\) statistics for each test are reported in the text, along with the critical \(p\)-value (labeled \(p_{null}\)), which represents the probability of observing a value greater than this in the null distribution. To give a more precise sense of the position of the observed statistic in the null distribution we report the tests as \(p_{null}<.05, p_{null}<.01, \) or \(p_{null}<.001\). If a \(p_{null}\) value of \(>.05\) is reported, then the effect was not considered to be statistically reliable. To provide an indication of effect size, partial eta squared \((\eta^2)\) is reported for ANOVA results and Cohen’s \(d\) for all \(t\)-tests. To test for relationships
between variables we used a bootstrap resampling procedure with 1000 iterations to compute
mean correlation coefficients and 95% confidence intervals, whereby confidence intervals
overlapping zero indicates a non-significant result.

RESULTS

Exercise physiology and task performance. We used several measures to confirm the
efficacy of our exercise intensity manipulation. First, we compared the exercise intensity
conditions [rest, low, high] in terms of heart rate and pupil area. Heart rate (Fig. 2a)
significantly increased as a function of exercise, \[ F(2,34) = 187.31, p_{\text{null}} < .001, \eta^2 = .92 \], such that
each step-wise increase in intensity caused a significant increase in heart rate (rest vs. low-intensity \[ t(17) = 9.85, p_{\text{null}} < .001, d = 1.97 \]; low vs. high intensity \[ t(17) = 9.59, p_{\text{null}} < .001, d = 1.04 \]). Second, we compared exercise-induced changes in pupil area (Fig. 2b). The goal behind
analyzing the pupil data was to determine the effects of exercise-induced arousal on raw pupil
area, so the data were not baseline corrected. The data for each subject and condition were
collapsed across the 2 s trial epoch for analysis purposes. Pupil area increased as function of
exercise \[ F(1,17) = 41.09, p_{\text{null}} < .001, \eta^2 = .71 \], again with each increase in exercise intensity
resulting in a significant increase in pupil area (rest vs. low-intensity \[ t(17) = 7.84, p_{\text{null}} < .001, d = .53 \]; low- vs. high- intensity \[ t(17) = 2.26, p_{\text{null}} < .05, d = .15 \]). These results are consistent with
converging evidence from human and animal studies showing that in addition to variation with
ambient light, pupil dilation can also be used to index arousal (Bradley, Miccoli, Escrig, & Lang,
2008; Erisken et al., 2014; Gilzenrat, Nieuwenhuis, Jepma, & Cohen, 2010; McGinley et al.,
2015). However, an important consideration is that due to the dual-task nature of our study, the
step-wise increase in physical effort is likely accompanied by an increase in mental effort, and
this may also contribute to the increased pupil size (Laeng, Sirois, & Gredeback, 2012).
Third, we compared pedaling power output between the two conditions in which the
participants were pedaling and observed that power output was greater during high-intensity
(88.21 ± 21.5 W) [mean ± SEM] compared to low-intensity exercise (50.23 ± 2.44 W), \[t(17) =
7.56, p_{null} < .001, d = 2.48\]. In addition, participants’ self-reported exertion was greater during
high-intensity (RPE 13.33 ± 0.12) compared to low-intensity (RPE 7.63 ± 0.17) exercise \[t(17) =
27.37, p_{null} < .001, d = 9.28\]. There was a small but significant increase in cadence during low-
(53.47 ± 2.66 RPM) compared to high- (51.76 ± 2.21 RPM) intensity exercise \[t(17) = 4.29, p_{null}
< .01, d = .69\], likely due to participant's tendency to increase cadence when pedaling resistance
was minimal.

Analysis of the task performance data (Fig. 2c) revealed a general impairment of
orientation discrimination accuracy during exercise compared to rest \([F(2, 34) = 6.71, p_{null} < .01,
\eta^2 = .28]\), such that accuracy was lower during both exercise conditions (low- and high- intensity)
compared to rest \([t(17) = 3.36, p_{null} < .001, d = .72; t(17) = 2.53, p_{null} < .05, d = .62, \text{ respectively}]\),
but accuracy in the exercise conditions did not differ \([t(17) = -.78, p_{null} > .05, d = -.14]\).

**EEG power and pattern classification.** After preprocessing the EEG data, single-trial
power between 4 and 30 Hz was estimated using a Fast Fourier Transform (FFT) (see Methods).

Our stimulus stream cycled on/off with a blank grey screen at 15Hz and thus evoked a robust
spike in power at the 15 Hz stimulation frequency that was focally distributed at the occipital and
parieto-occipital electrodes in each of the three exercise conditions (Fig. 3). Mean power at 15
Hz was modulated by exercise intensity \([F(2,34) = 6.48, p_{null} < .01, \eta^2 = .28]\), such that power was
significantly higher during both exercise conditions (low- and high- intensity) compared to rest
\([t(17) = 2.52, p_{null} < .05, d = .19; t(17) = 2.93, p_{null} < .05, d = .22, \text{ respectively}]\). Power did not
differ between exercise conditions \([t(17) = .58, p_{null} > .05, d = .02]\). Mouse studies that have
manipulated behavioral state using locomotion have reported reductions in low frequency local field potential power (<30 Hz) during locomotion when compared to stationary periods (Niell & Stryker, 2010; Polack et al., 2013). We conducted this analysis on our Fourier coefficients measured at occipital and parieto-occipital channels with a frequency resolution of .17 Hz, and revealed that estimates of power across the lower frequency bands [theta: 4-8 Hz, alpha: 8-13 Hz and beta: 16-30 Hz] were modulated by exercise \[F(2,34) = 30.9, \ p_{null} < .001, \ \eta^2 = .65\], with increased power during low- and high- intensity exercise when compared to rest \[t(17) = 6.16, \ p_{null} < .001, \ d = .55; \ t(17) = 6.70, \ p_{null} < .001, \ d = .67\] but no difference in power between exercise conditions \[t(17) = 1.46, \ p_{null} > .05, \ d = .11\]. This result contradicts the mouse studies, but is consistent with previous recordings of EEG activity in humans that show increases in alpha, beta and/or theta frequency bands as a function of exercise (Bailey et al., 2008; Hottenrott et al., 2013).

To determine the extent to which the single trial EEG data carried information about stimulus orientation, estimates of power and phase angle were entered into a linear discriminant classifier using a leave one trial out cross-validation procedure (see Methods). Overall classifier accuracy was above chance (chance=1/9) in all conditions \[rest: \ t(17) = 6.43, \ p_{null} < .001, \ d = 3.03; \ low: \ t(17) = 6.89, \ p_{null} < .001, \ d = 3.25; \ high: \ t(17) = 5.57, \ p_{null} < .001, \ d = 2.63\] (Fig. 4).

Pair-wise comparisons of classifier performance indicated that decoding accuracy was higher during rest compared to high intensity exercise \([t(17) = 2.36, \ p_{null} < .05, \ d = .54\], but neither rest nor high intensity exercise were reliably different from low-intensity exercise \([t(17) = -.04, \ p_{null} > .05, \ d = -.01; \ t(17) = 2.01, \ p_{null} > .05, \ d = -.57, \text{respectively}\].

**Inverted encoding model.** To estimate orientation selective tuning profiles at the population level we used an IEM modeling technique (e.g Brouwer and Heeger, 2009, 2011,
Here we used complex Fourier coefficients estimated at the artifact-free electrodes from the first 2000 ms of trials on which the target occurred over 2000 ms after trial onset (80% of trials). Doing so ensured that the neural response was not contaminated by target rotation activity. A set of training trials (all trials except for 1) was used to estimate the magnitude of the response at each electrode as a linearly weighted sum of the basis set (an idealized set of orientation tuning functions represented in Fig. 5a). Then, we used these estimated training weights to estimate the relative magnitude of the 15 Hz responses within different subpopulations of neurons (or “channels”) that are tuned to different stimulus orientations on the one trial left out from the training set. This process was repeated until all trials served in both training and test sets. The channel responses estimated from the test trials were then averaged and then converted to power by taking the square of the absolute value of the complex numbers. The resulting mean estimated response profiles are termed “channel tuning functions” (CTFs) expressed in terms of power (µV²).

To first demonstrate the general efficacy of this approach across orientations, we computed CTFs using power at the stimulation frequency (15 Hz) for each of the possible orientations in our stimulus set collapsed across the three exercise conditions. This analysis produced stable tuning curves at each of the nine orientations (Fig. 5b). As a second validation step, we estimated CTFs at each frequency from 4-30 Hz and then shifted the tuning functions to a common reference, resulting in tuning functions centered on 0° (Garcia et al., 2013). Fig. 5c shows the centered CTFs through the tested frequencies (4-30 Hz). There is a clear CTF present at the stimulation frequency of 15 Hz thus confirming that the orientation selective response information is predominantly carried at the stimulation frequency and not in other bands.
Having demonstrated the efficacy of this approach, the key analyses involved testing whether exercise modulated feature-selective response profiles. To do this, we computed separate CTFs using the Fourier coefficients estimated at 15 Hz for each of the three exercise conditions, and then shifted the CTFs to a common center (Fig. 5d). To quantify exercise-induced modulation of the shapes of the respective response profiles, we first evaluated changes in CTF amplitude as a function exercise condition. To increase statistical power, we folded the CTFs from 9 points into 5 points [0°, 20°, 40°, 60°, 80°] (Fig. 5e) and then entered the folded CTFs into a repeated measures ANOVA with exercise intensity [rest, low, high] and channel offset [0°, 20°, 40°, 60°, 80°] as within-participants factors. This analysis revealed a robust effect of channel offset \[F(4,68) = 112.74, p_{\text{null}} < .001, \eta^2 = .87\] and critically, a significant exercise by channel-offset interaction \[F(8,136) = 3.35, p_{\text{null}} < .05, \eta^2 = .16\]. This interaction was driven by increased amplitude of the CTF during low-intensity exercise at the CTF center (0°) and 20° channel offsets \[t(17) = 2.68, p_{\text{null}} < .01, d = .52; t(17) = 2.39, p_{\text{null}} < .01, d = .49\], respectively and decreased amplitude of the CTF during low-intensity exercise at the 40° and 60° channel offsets \[t(17) = -2.14, p_{\text{null}} < .05, d = -.46; t(17) = -2.17, p_{\text{null}} < .05, d = -.44\] respectively, relative to the rest condition. The interaction was also driven by increased CTF amplitude during low-intensity exercise relative to high-intensity exercise at the CTF center (0°) and decreased amplitude at 40° \[t(17) = 2.08, p_{\text{null}} < .05, d = .56; t(17) = -3.04, p_{\text{null}} < .01, d = -.89\], respectively. CTF amplitude did not differ significantly between rest and high-intensity exercise conditions at any of the channel offsets \(p_{\text{null}} > .05\). The selective enhancement around the center of the orientation CTF under low-intensity exercise relative to the other conditions is consistent with the notion that exercise can induce multiplicative gain in feature-selective response profiles.
To further investigate the source of the feature-selective modulations during low-intensity exercise relative to the other conditions, we fit a von Mises distribution to the observed data for each participant and condition (see Methods). One participant was excluded from this analysis due to difficulty fitting the data in the rest condition. The best fitting parameters for bandwidth, gain and baseline were entered into paired-samples t-tests to compare low-intensity exercise vs. rest and low-intensity exercise vs. high-intensity exercise. Gain increased during low-intensity exercise relative to both rest and high intensity exercise \[ t(16) = 2.28, 2.41, p_{\text{null}} < .05, d = .51, .65, \text{respectively} \] (Fig. 5f). Analyses of the bandwidth data revealed an increase in the concentration parameter \( k \) during low-intensity exercise relative to rest, thus indicating reduced tuning profile bandwidth \[ t(16) = 2.53, p_{\text{null}} < .01, d = .69 \]. Bandwidth was not modulated during low-intensity exercise relative to high-intensity exercise \[ t(16) = .49, p_{\text{null}} > .05, d = .17 \] (Fig. 5g). Baseline was not modulated as a function of low-intensity exercise relative to rest or high-intensity exercise \[ t(16) = .26, .53, p_{\text{null}} > .05, d = .07, .18, \text{respectively} \] (Fig. 5h).

**Control analyses.** Six control analyses were carried out in order to check the consistency of the main analyses and rule out any alternative explanations.

First, the IEM approach applied here clearly revealed CTFs that exhibited a graded response, such that the peak response is at the center of the function (i.e., the “preferred” orientation) and the response falls off gradually as the angular deviation of the stimulus increases. However, because our basis set used similarly graded functions, it is possible that the graded CTFs observed here were an artifact of the basis set rather than inherent in the EEG data. To rule out this alternative explanation, we re-ran the analysis replacing the half-wave rectified sinusoid basis set with a set of delta (“stick”) functions at each of the 9 orientations (Fig. 6a). The results of this analysis again revealed graded CTFs modulated by exercise, although the overall
amplitude of the response was attenuated relative to the CTFs extracted using the graded basis set (Fig. 6b).

Second, after artifact rejection, the number of trials in each of the nine orientations in the training set was not exactly the same [mean trials per set: rest 30.94 ± .16, low 30.71 ± .27, high 30.66 ± .24]. We confirmed that there was no systematic bias in the number of training trials per orientation as a function of exercise condition [$F(2, 34) = .94, p_{null} > .05, \eta^2 = .05$], orientation [$F(8, 136) = .27, p_{null} > .05, \eta^2 = .02$] or any interaction between the two factors [$F(16, 272) = .57, p_{null} > .05, \eta^2 = .03$] (Fig. 6c).

Third, we observed a small but significant increase in pedaling cadence in the low- compared to high- intensity exercise condition. The presence of this difference opens the door to the possibility that the modulations in neural activation we observed were more related to cadence than exercise intensity. If true, then there should be reliable correlations between cadence and our various measures of neural activity (i.e. CTF gain, bandwidth and baseline, power at the stimulation frequency and global spectral power). Bootstrapped correlations confirmed that there were no significant correlations (Table 2).

Fourth, consistent with studies in the animal literature (Erisken et al., 2014; McGinley et al., 2015), we report a monotonic increase in pupil size as a function of exercise-intensity. The magnitude of spherical aberrations increase with pupil size, which is known to degrade the optical quality of the retinal image (Lombardo & Lombardo, 2010), thus raising the possibility that exercise-induced pupil dilation may modulate visuocortical gain. To test this, percent change in pupil area and all neural measures was computed as a function of exercise condition. Bootstrapped correlations confirmed there were no significant correlations (Table 3), indicating...
that the exercise-induced changes in neural activity reported here are not confounded by changes in pupil size.

Fifth, our gaze-contingent methodology ensured that any trials where the participant blinked or made eye-movements larger than 1.75° from the central fixation cross were rejected online and re-run. However, small changes in gaze dispersion within the acceptable window could be modulated by exercise condition, which may have contributed towards changes in brain activity. If true, there should be reliable correlations between gaze dispersion scores and measures of neural activity (Figs. 6d). Exercise condition modulated gaze dispersion \([F(2,34) = 14.22, p_{null}<.001, \eta^2 =.46]\), with greater dispersion during low- and high-intensity exercise compared to rest \([t(17)=4.44, 3.67 p_{null}<.001, d = 1.37, 1.30, \text{respectively}]\), but there was no difference between exercise conditions \([t(17) = .42, p_{null}>.05, d = .07]\). To determine if the exercise-induced changes in gaze dispersion tracked with changes in the neural measures, percent change between all exercise conditions (rest vs. low, rest vs. high, low vs. high) was calculated for the gaze dispersion scores and all neural measures, and the percent change values were then correlated (Table. 3). There was no relationship between exercise-induced changes in gaze and changes in CTF characteristics, suggesting that the gain and bandwidth modulation effects we observe as a function of exercise cannot be attributed to changes in changes in gaze position within our acceptable window. Similarly, there was no relationship between gaze and the spectral power measures.

Finally, to assess whether our results were attributable to motion artifacts in the EEG data, we quantified head motion (see Methods) using a dispersion metric similar to the gaze position dispersion metric described above (Fig. 6e). Exercise condition modulated head position dispersion \([F(2,24) = 54.1 p_{null}<.001, \eta^2 = .82]\), with greater dispersion during low- and high-
intensity exercise compared to rest \( t(12) = 7.82, 9.06, p_{\text{null}} < .001, d = 2.48, 2.84 \), but the
exercise conditions were not different \( t(12) = 1.24, p_{\text{null}} > .05, d = .24 \). As with the gaze position
analysis, there were no correlations between head position dispersion and any of the CTF
measures (Table 3). There was, however, a relationship between increased head motion and
increased global spectral power and power at the stimulation frequency as a function of low-
intensity exercise relative to rest.

**DISCUSSION**

Changes in behavioral state induced by physical activity influence the neural correlates of
information processing in humans (Bullock et al., 2015; De Sanctis et al., 2014; Pontifex &
Hillman, 2007), rodents (Ayaz et al., 2013; Fu et al., 2014; Niell & Stryker, 2010; Saleem et al.,
2013) and invertebrates (Chiappe et al., 2010; Maimon et al., 2010). To investigate the effects of
physical activity on feature-selective sensory coding in the human, we applied an IEM approach
to scalp recorded EEG acquired at rest and during acute bouts of low- and high- intensity
exercise. This approach enabled us to estimate feature-selective response profiles from the
spatial distribution of evoked EEG activity across the scalp. Our key finding was that response
profiles exhibited an inverted-U pattern as a function of exercise, with response gain during low-
intensity exercise relative to both rest and high-intensity exercise, and a reduction in estimated
tuning profile bandwidth during low-intensity exercise relative to rest. Critically, the present
data go beyond our previous reports of modulation in human sensory cortex as a function of
exercise (Bullock et al., 2015) by demonstrating the specific nature of exercise effects on
population level feature-selective coding profiles. These findings suggest that evidence for
enhanced sensitivity in single-unit non-human mammalian and invertebrate studies may
generalize to population level responses in human visual cortex
Vision is a fundamentally important sensory domain for successful representation, interaction and navigation of the environment, thus it seems intuitive that physiological arousal induced by acute bouts of activity would induce a high state of gain in visual cortex. The transition to high gain state in visual cortex appears to happen as rapidly as the transition from stationary to running in the mouse (Niell & Stryker, 2010; Saleem et al., 2013) which indicates a close link between visual cortex and motor drive. The present data suggest that behavioral state can also act as a gain modulator in human visual cortex. Furthermore, it is important to note that our IEM technique allows us distinctly different insight to the single unit data, in the sense that we can show effects of behavioral state on the large-scale populations of neurons that are activated during stimulus representations. The unique advantage of this approach is that it provides a holistic understanding of stimulus encoding which cannot be obtained by study single units in isolation (Sprague et al., 2015). Thus, here we extend single-unit observations that behavior state modulates gain, to the population level.

Our observation that physiological arousal can have non-linear effects on neural response gain is also consistent with recent studies of humans and animals (Bullock et al., 2015; McGinley et al., 2015). Gain in the magnitude of the response profile at low-, but not high-intensity exercise or at rest, is consistent with the inverted-U notion that arousal beyond a certain point is detrimental to performance (Yerkes & Dodson, 1908). We observed these inverted-U effects in a previous study that reported non-specific, ERP evidence for sensory response gain in early visual processing during low-intensity exercise when compared to rest and high-intensity exercise (Bullock et al., 2015). Changes in membrane potential may offer an explanation for these effects. There is evidence that membrane potential in cells in visual cortex becomes more depolarized and less variable during locomotion (Polack et al., 2013), and a recent study in
mouse auditory cortex suggests that at medium levels of arousal the membrane potential is low
with minimal levels of spontaneous firing, compared to slow oscillations at low arousal and tonic
depolarization at high levels of arousal (McGinley et al., 2015).

We acknowledge that there is a mismatch between the inverted-U shaped pattern of
effects on CTFs and the drop in task performance as a function of the exercise
conditions when compared to rest. There are several key points to consider. First, the decline in
task performance as a function of exercise is likely due to the use of the method of constant
stimuli to set the orientation task difficulty while participants were at rest (single task) and then
requiring them to perform the task at the same difficulty level while concurrently pedaling to the
beat of a metronome during the exercise conditions (dual task). Second, the neural activity
evoked by the stimulus rotation, which serves as the target-defining feature, is not included in the
model. Instead, consistent with the approach used by Garcia, Srinivasan, & Serences (2013) the
CTFs are reconstructed from the 2s of pre-target activity in order to prevent interference from the
rotation. Furthermore, the reconstructed CTFs are based on neural activity evoked by the steady-
state flickering grating at a fixed orientation, whereas the task performance is based on
discrimination of rotation away from that orientation, which may require a different type of
detector. Hence, it is possible that the increased gain and reduced profile bandwidth during the
low-intensity exercise condition may reflect an enhanced detector relative to rest and high-
intensity exercise conditions, but the behavioral task is not sensitive to these changes. Third,
boosting the signal in local sensory information processing, as appears to be the case in the low-
intensity exercise condition, may not necessarily have effects on downstream processing areas
and thus may not impact upon task performance (Krauzlis, Bollimunta, Arcizet, & Wang, 2014;
Sprague et al., 2015; Zénon & Krauzlis, 2012). Finally, while U-shaped performance curves
have been observed as a function of exercise in mice (McGinley et al., 2015), we have also shown mismatch between sensory recordings and behavior in physically active humans in our previous work. In an ERP study we observed gain modulation in the P1 ERP component during low-intensity exercise but behavioral performance was only enhanced during high intensity exercise. Several behavioral studies provide indirect evidence for enhancement of sensory processing in humans after a bout of acute physical activity (Davranche & Audiffren, 2004; Davranche & Pichon, 2005), thus it is entirely possible that exercise effects on sensory processing may enhance human performance in a task that is better suited to measuring this relationship.

While the IEM results are the primary focus of this discussion, we also acknowledge that the spectral power analyses and classification analysis show different patterns of exercise-induced modulation. The increase in spectral power from 4-30 Hz is consistent with previous recordings of EEG activity in humans (Bailey et al., 2008; Hottenrott et al., 2013), and greater spectral power does not imply greater separability of the neural patterns associated with the different orientations, so it is not surprising that the exercise effects on spectral power do not track with the classification or IEM results. Furthermore, it is not unexpected that the pattern classification and IEM results show different patterns of exercise-induced modulation, given the fundamental differences in the nature of these analytical approaches. Classification techniques essentially determine the separability of the classes (i.e., in this case different stimulus orientations) based on observed patterns of neural activation. Other than assuming that there are different classes (e.g., stimulus orientation), classification approaches do not typically make assumptions about the specific structure of the underlying patterns of neural activation to each class. In contrast, encoding models use a set of a-priori assumptions about the structure of the
feature space (Gur & Snodderly, 2007; Ringach, Bredfeldt, et al., 2002; Ringach, Shapley, et al., 2002) to capture changes in neural codes at the population level, thus enabling stimulus representations to be quantified in terms of native feature space (i.e. gain, bandwidth and baseline) rather than signal space (Garcia et al., 2013; Sprague et al., 2015).

Naturally, there are several limitations to our approach. EEG acquired at the scalp reflects the summed response of dendritic post-synaptic potentials from many different neural populations across the cortex, and thus our conclusions are limited in the sense that we cannot differentiate between cell-types or investigate subcortical function in the human. This is important, as while evidence from the mouse suggests most cortical neurons that respond to visual stimulation are excitatory, broad-spinging neurons that show multiplicative gain without changes in feature selectivity as a function of locomotion, there are subsets of cortical neurons and neurons at earlier, subcortical stages of visual processing that show very different patterns of modulation by locomotion (Erisken et al., 2014; Niell & Stryker, 2010). Another important consideration is that while we do observe large-scale population level modulation of tuning bandwidth as a function of low-intensity exercise relative to rest, this does not imply uniform modulation of tuning profiles across all single units. We also note that the magnitude of the response gain effects observed here in exercising humans are notably smaller than effects observed in locomoting flies and mice (for a summary see Maimon, 2011), although given that we are comparing scalp-recorded EEG in humans with single-cell action potentials in animals and invertebrates, it is not possible to determine whether these inconsistencies are due to fundamental differences in species or techniques. It is also possible that increased perspiration as a function of exercise might influence the neural data by changing electrode impedances across the conditions; however, if this were the case we would predict a monotonic change in the
fidelity of the model as a function of exercise, not the inverted-U curve observed here.

Nevertheless, despite these caveats, the consistencies in overall patterns of locomotion-induced gain represent an important first step in linking behavioral state effects on mouse and human cortex.

In summary, the present study used an IEM approach to investigate the influence of behavioral state on estimated response profiles of neurons in human sensory cortex. Our findings indicate that behavioral state can modulate response gain and tuning profile bandwidth in sensory cortex, and that the effects of increased physiological arousal may be non-linear. Thus, despite profound differences in visual pathways across species, these data suggest that evidence for enhanced sensitivity in intracellular non-human mammalian and invertebrate studies may generalize to population level responses in human sensory cortex. This work provides valuable evidence linking the neural mechanisms of behavior state across species and opens the door to further investigations into the influence of physical activity on the human brain.

REFERENCES


FIGURE CAPTIONS

**Figure 1: Experimental Methods.** (a) Equipment setup. (b) Example of rotation offset detection task. Participants fixated at center for the duration of each trial and indicated at the end whether the target rotation offset was CW or CCW.

**Figure 2: Physiology and Behavior.** (a) Mean heart rate increased as a function of exercise. (b) Normalized pupil area measured over the first 2000 ms of the trial significantly increased as a function of exercise. (c) Target discrimination accuracy decreased slightly as a function of exercise. * $p_{null} < .05$, **$p_{null} < .001$.

**Figure 3: Steady State Responses.** We observed robust responses at 15 Hz across occipital and parieto-occipital electrodes (plots represent spectral power averaged over electrodes Oz, O1, O2, POz, PO3 and PO4 and plotted between 4 and 20 Hz for each exercise condition).

**Figure 4: Classification.** Classification accuracy in rest, low- and high- intensity exercise conditions (chance $p = .11$, represented by the dashed line). Data from artifact-free scalp channels were entered into the classifier. * $p_{null} < .05$.

**Figure 5: Decoding and Encoding.** (a) Graded basis set used in IEM. Nine basis functions spanning $0^\circ$ - $160^\circ$ in $20^\circ$ increments were created from half-sinusoidal functions raised to the seventh power. (b) Channel tuning functions (CTFs) were derived from the model, using the Fourier coefficients at 15 Hz. This plot depicts CTFs collapsed across exercise conditions prior to centering. (c) We ran the IEM on all frequencies between 4 to 30 Hz and collapsed across
exercise conditions and centered the TFs for ease of plotting. This plot confirms a robust tuning function at 15 Hz. (d) Centered CTFs plotted at 15 Hz for rest, low- and high-intensity exercise conditions. (e) Centered CTFs were folded from 9 points into 5 points to increase statistical power. ** Von Mises Fitting.** Mean gain (f), bandwidth (larger k value reflects reduced tuning profile bandwidth; g) and baseline (h) of the best-fitting von Mises function. Errors bars represent SEM. **p_{null} < .01, * p_{null} < .05.**

**Figure 6: Control Analyses.** Delta (Stick) Functions. (a) To rule out the possibility that the CTFs we observed were an artifact of our graded basis set, we re-ran our IEM replacing the original half-sinusoid basis set with a set of Delta (“Stick”) functions. (b) The CTFs that we obtained from this analysis revealed graded CTFs with a similar pattern of modulation by exercise as before. **Trials per Orientation.** (c) Plot shows the mean number of trials present for each orientation in the training set following artifact rejection. **Gaze Dispersion and Head Motion.** (d) Gaze sample dispersion. (e) Head motion sample dispersion. Errors bars represent SEM. **p_{null} < .001.**
Table 1: Mean and standard error values for demographic and cardiovascular data.

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Table 2. Bootstrapped correlations comparing mean cadence with mean neural measures during the low- and high-intensity exercise conditions (-95% CI < mean rho < +95% CI). *p<.05
Table 3. Bootstrapped correlations comparing percent change in pupil area, gaze and head motion with percent change in neural measures (-95% CI < mean rho < +95% CI). *p<.05

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<td>-.76&lt;-.21&lt;.65</td>
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Figure 1: Experimental Methods. (a) Equipment setup. (b) Example of rotation offset detection task. Participants fixated at center for the duration of each trial and indicated at the end whether the target rotation offset was CW or CCW.

Fig. 1
172x74mm (300 x 300 DPI)
Figure 2: Physiology and Behavior. (a) Mean heart rate increased as a function of exercise. (b) Normalized pupil area measured over the first 2000 ms of the trial significantly increased as a function of exercise. (c) Target discrimination accuracy decreased slightly as a function of exercise. * \( p_{null} < .05, \) ** \( p_{null} < .001. \)

Fig. 2

169x53mm (300 x 300 DPI)
Figure 3: Steady State Responses. We observed robust responses at 15 Hz across occipital and parieto-occipital electrodes (plots represent spectral power averaged over electrodes Oz, O1, O2, POz, PO3 and PO4 and plotted between 4 and 20 Hz for each exercise condition).

Fig. 3
179x96mm (300 x 300 DPI)
Figure 4: Classification. Classification accuracy in rest, low- and high- intensity exercise conditions (chance $p=.11$, represented by the dashed line). Data from artifact-free scalp channels were entered into the classifier. * $p_{null} < .05.$
Figure 5: Decoding and Encoding. (a) Graded basis set used in IEM. Nine basis functions spanning 0° - 160° in 20° increments were created from half-sinusoidal functions raised to the seventh power. (b) Channel tuning functions (CTFs) were derived from the model, using the Fourier coefficients at 15 Hz. This plot depicts CTFs collapsed across exercise conditions prior to centering. (c) We ran the IEM on all frequencies between 4 to 30 Hz and collapsed across exercise conditions and centered the TFs for ease of plotting. This plot confirms a robust tuning function at 15 Hz. (d) Centered CTFs plotted at 15 Hz for rest, low- and high- intensity exercise conditions. (e) Centered CTFs were folded from 9 points into 5 points to increase statistical power. Von Mises Fitting. Mean gain (f), bandwidth (larger $k$ value reflects reduced tuning profile bandwidth; g) and baseline (h) of the best-fitting von Mises function. Errors bars represent SEM. ** $p_{null} < .01$, * $p_{null} < .05$. 

Fig. 5
174x188mm (300 x 300 DPI)
Figure 6: Control Analyses. Delta (Stick) Functions. (a) To rule out the possibility that the CTFs we observed were an artifact of our graded basis set, we re-ran our IEM replacing the original half-sinusoid basis set with a set of Delta ("Stick") functions. (b) The CTFs that we obtained from this analysis revealed graded CTFs with a similar pattern of modulation by exercise as before. **Trials per Orientation.** (c) Plot shows the mean number of trials present for each orientation in the training set following artifact rejection. *Gaze Dispersion and Head Motion.** (d) Gaze sample dispersion. (e) Head motion sample dispersion. Errors bars represent SEM. **p_{null} < .001.

Fig.6
174x83mm (300 x 300 DPI)