

Exercise Addiction & Frontal EEG α activity

– a (very) critical review of Gapin, Etnier & Tucker’s 2009 study –

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Research Objective

Trying to elucidate the hypothetical relation between exercise addiction (EA) and asymmetric baseline frontal alpha activity, Gapin, Etnier and Tucker (2009)¹ conducted an EEG study in 28 regularly exercising women. The authors predicted increased right frontal activity associated with negative affect (A-) for addicts. Their results hint at a relation between *left* frontal activity and EA, however.

Many sciences invoke concepts lacking crisp and clear definitions; one may thus grant the notion of EA is sufficiently defined for purposes of the current study. The authors successfully disambiguate their conception (see below) and try to enlighten it by analogy to drug addiction.

In psychology, right prefrontal areas are prominently associated with A- (e.g. Kuhl, 2000). The sources cited by the authors, however, do not support their claim that “negative affect is associated with greater relative right activity” (p. 136). A number of studies (Levesque *et al.*, 2003; Eisenberger, Lieberman, & Williams, 2003) suggest right frontal areas being recruited in A- *regulation* – with these, although not discussed by the authors, the current study lines up nicely.

Materials and Methods

EEG was recorded from one left and one right frontal silver-silver chloride electrode (F3 and F4 on the international 10-20 system) in eight 60s runs of rest; participants (26 caucasian, 2 african-american; age 19-61; all right-handed) were instructed to either open or close their eyes (four runs each; two possible sequences). Offline averages were

¹Citations hereafter refer to the main article if not otherwise indicated.

obtained from left and right mastoids, a middle forehead electrode served as ground. Eye-movements (EOG) were recorded by electrodes above and below subjects' left eyes. All impedances were kept below $5k\Omega$. EEG signals were sampled at 256Hz, bandpass-filtered at 0.1 and 100Hz and amplified 50,000 times. EEG data was transformed using a *Fast Fourier Transform* and averaged over all eight trials. Total alpha band (8-12Hz) power was obtained and normalised via log-transformation. Asymmetry scores were obtained by left frontal activity subtraction from right frontal activity ($\log F4-F3$) where alpha values relate inversely to cortical activity levels.

The experimenters' interest in alpha-band activity makes EEG the method of choice. The results should be taken with a pinch of salt, however: frontal EEG activity does not necessarily originate from frontal brain regions, and comparison of few recording sites only is generally more vulnerable to artefacts that may yield asymmetric EEG signals than comparisons based on multiple channels. This is a serious limitation of the current study. Whole head coverage EEG recordings and possibly – as part of the research question concerns localisation – a combination with fMRI may be enlightening.

The sampling frequency seems sufficient ($\sim 2.5 \times 100\text{Hz}$) but could have been increased to obtain better quality data. Eye-blinks were removed manually but inspection criteria remain unspecified.

To calculate asymmetry scores, all trials, i.e. those with open *and* closed eyes, were averaged. No reasons are given for this procedure which is particularly curious as alpha activity is known to be modulated by opened / closed eyes (Singh, 2006). Separate analysis of conditions would also be of interest to establish independence of, e.g., participants' mental imagery (perhaps of exercise?) and/or environmental stimulation (the laboratory may frighten participants inducing A-).

After EEG recordings, subjects filled in three questionnaires to (i) rule out secondary addictions (Q-EED, Mintz, O'Halloran, Mulholland, & Schneider, 1997), (ii) assess the significance of exercise for their lives (EAI, Terry *et al.*, 2004), and (iii) obtain physical activity levels (Godin & Shepard, 1985) measured in *metabolic equivalent units* (METS).

The authors recognised possible roles of eating disorders in EA and conceptually dissociated the phenomenon of interest, *primary* EA (unrelated to eating disorders), from secondary EA where Q-EED served as a control measure.

To diagnose addiction as defined by the authors in their introduction, EAI is insufficient: ease of use alone cannot justify applying six uniformly scaled questions only over a more complex measure. Crucially, evidence for the test's reliability and/or validity is not provided.

The measure of physical activity levels remains opaque: participants were asked *how often* per week they engage in mild, moderate, and strenuous activity; this information

was transformed into METS. METS do not take individual differences (BMI, nutrition habits, etc.) or the total *duration* of exercise into account. Subjects' personal estimates may further be inaccurate and/or influenced by exceptionally much or little recent exercising. A more reliable assessment could involve detailed records (e.g. diaries) of actual training, where long-term assessment will be crucial to establish genuine addiction.

Participants were asked not to exercise four hours prior to testing. As acknowledged in the discussion, this is insufficient; for subjects may have exercised earlier on testing day or were to do so after testing. Thus, they may have experienced exercise related positive affect (A+) and/or A- regulation throughout the experiment rather than withdrawal.

No affect measures were included in subjects' assessments. This strikes counterintuitive as the experimental hypothesis entertained in this study sets out from addicts' affective states. Standardised questionnaires for affect and affect regulation are available (e.g. Kuhl & Beckmann, 1994). Likewise, participants' motivation to exercise – which likely interacts with affect – is of interest but was not assessed.

The study was limited to females; to the authors' credit, they do not generalise across gender. Possible effects of age and ethnicity on EA are – despite the author's attempt to draw an analogy to drug addiction for which effects of both ethnicity (Wallace *et al.*, 2003) and age (Stansfield & Kirstein, 2005) have been suggested – not considered; neither are data on other addictions (e.g. smoking) reported which may interact with EA.

Results and Interpretation

Q-EED results indicated no eating disorder in any participant. Scores and cut-offs are not reported, however. EAI scores are reported in table 1 (p. 138) but not for each individual subject. Given the rather wide ranges (14-94 for total METS) in the sample, these will be of interest to the critical reader. According to the cut-offs reported, only 9 participants scored within the top category ("at risk" of EA; cut-off 24, mean 25.50, sd = 1.69) while 19 scored in the "symptomatic" range (cut-off 13, mean 18.9, sd 1.99). Throughout the paper, it remains unclear whether these groups are considered as addicts and non-addicts, respectively, or whether the full sample is considered addicted.

METS, the authors claim, predict EA as they found a significant correlation between EAI scores and total METS. Similarly, asymmetric EEG activity is claimed to predict EA as a significant correlation was found between EAI scores and obtained log F4-F3 EEG activity (table 2, p. 139).

Strikingly, though, correlation between log F4-F3 and METS was *not* significant suggesting that EEG asymmetry and METS might be relatively independent measures of

(contributors to) EA in the current sample rather than constituents of a unidirectional causal chain. The authors do not comment on this; but the relation between METS and asymmetry scores may be crucial when comparing addicts to regularly exercising non-addicts.

Regression analyses indicated greater EAI scores in subjects with greater total METS and asymmetry scores, respectively. The authors thus conclude that, contradicting their initial hypothesis, EA is related to increased left frontal activity. Two principal accounts of this main finding are suggested: first, drawing on Tomkin's (1968) *affect regulation hypothesis*, exercise may be employed by addicts as a coping mechanism decreasing A- by means increasing A+ (associated with left frontal activity); second, based on what they call the *motivational directional hypothesis*, left frontal activity may be induced by *approach motivation* which is typically relevant for coping with, e.g., anger and aggression. Addicts may use approach exercise to balance their principal lack of this kind of motivation.

Both these accounts cannot be supported by the acquired data and thus remain merely speculative. To test the proposed explanations, data of subjects' approach and withdrawal behaviour, of their affective states and the effects of A+ and A- on both cortical activity and exercise behaviour would have to be assessed.

No plot for the relation between METS and asymmetry scores is provided. Figure 1 (p. 139) illustrates the reported trend of increasing asymmetry scores with EAI increase; but the scatterplot uncovers a major spread of results. Given the restricted sample size, the reported trend may well be an artefact and may not be replicable in a larger sample better suited for regression analysis.

Conclusions

In summary, Gapin, Etnier and Tucker found increased left frontal alpha baseline EEG activity in individuals with higher EAI scores. Their analysis suggests EAI scores are predicted by both EEG asymmetry and participants' total METS.

Although acknowledging their data do not support conclusions about causal relations, the authors suggest differences in affective states may be responsible for EA. The current study does not justify this conclusion, however. The assumption that some women (those suffering from increased A-) engaging in a critical amount of exercise develop dependence while others do not is not sufficiently justified. No group comparisons between addicts and non-addicts are reported. The relation between alpha band activity and affective states remains another undiscussed supposition.

Overall, the study was not sufficiently controlled. General cognitive functioning and

neuropsychological history were not assessed; the latter is of particular interest in light of Kentgen *et al.*'s (2000) report of increased left frontal activity in depression. The authors controlled for eating disorders and BMI, but not age (subjects ranged from adolescent to elderly) in their analysis. Participants were not screened for possible confounding addictions. It was not controlled for whether or not subjects exercised on testing day. All this may substantially affect the results.

Including a measure of participants' affect would have been crucial to (a) confirm the initial experimental hypothesis, and (b) disambiguate between the proposed interpretations of the results. Additional experimental manipulations (e.g. A- induction) and/or comparison of pre- and post-exercise testing may have been enlightening with respect to both (a) and (b). Finally, comparison between addicts and non-addicts is required to appropriately answer the current paper's research question.

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