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PLACEBO EFFECTS IN SPORT AND EXERCISE

A Meta-Analysis

(Received: 19 September 2010; accepted: 16 February 2011)

The empirical foundation of the ‘placebo effect’ is presented briefly, which is followed by the meta-analysis of the relatively few published reports that have investigated placebo effects in sports performance. Based on the analysis of the fourteen studies included in the meta-analysis, an overall medium effect size (0.4, 95% CI ranged from 0.24 to 0.56) was found. Homogeneity of effect sizes ($\chi^2 (13, N = 196) = 9.35, p = 0.75$) and the feasibility of possible explanation models were also tested. In various sports (e.g. cycling, running, weightlifting) the investigation of the placebo effect on various physiological or performance measures (e.g. muscle power, heart rate, running speed) and psychological attributes (e.g. perceived exertion, post-experiment interviews) yielded significant results. Indeed, the common finding of the reviewed studies was that from the point of view of the athletes there is substantial performance enhancement as a result of different forms of placebos. However, the interpretation of some of the results may be limited by methodological shortcomings. Based on the reviewed articles and further questions emerging from them, methodological recommendations as well as possible research ideas are suggested for further inquiries in the area.

Keywords: placebo effect, meta-analysis, expectation, belief, sport, physical exercise, mechanism, nocebo

Placebo-Effekt im Bereich Sport: Metaanalyse: Die Grundlagen der Erforschung des Placebo-Effekts werden kurz geschildert, sodann werden die Ergebnisse unserer Metaanalyse vorgestellt, die auf Grundlage einiger Studien zur Untersuchung des messbaren Placebo-Effekts bei Sportleistungen durchgeführt wurde. Auf Grundlage der in die Metaanalyse integrierten 14 Studien wurde eine mittlere Wirkungsgröße festgestellt (0.4, mit 95% CI 0.24–0.56). Es wurden die Homogenität der Wirkungsgrößen ($\chi^2 (13, N = 196) = 9.35, p = 0.75$), sowie die Möglichkeit der

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Schlüsselbegriffe: Placebo-Effekt, Metaanalyse, Erwartung, Fehlannahme, Sport, körperlicher Leistungsmechanismus, Nocebo-Effekt

1. Background

1.1. The placebo effect: introduction

The use of placebos is a widely used intervention to investigate non-specific drug effects that are or are believed to be independent of the specific characteristics of the given medicine. The concept of placebo has originally been defined strictly within a medical context, but it has broadened and started to be used beyond the boundaries of medicine: studies appeared on the effects of placebo alcohol, caffeine, etc. (Beedie 2007). The term is used here in this broader sense, according to the definition of Ross & Olson (1981, 408): ‘a placebo is a substance or procedure that is administered with suggestions that it will modify a symptom or sensation but which, unknown to its recipient, has no specific pharmacological impact on the reaction in question’.

The efficacy of a placebo depends on the suggestion (information) received: more powerful effects have usually been reported in the case of deceptive administration (subjects are told that they receive a medication) than in double blind condition (subjects are told that they are to receive either medication or a sugar pill). Participants of clinical drug trials (RCTs) must be informed about the possibility and probability of receiving a placebo treatment. During the therapeutic application of placebos, however, patients are not aware of the possibility of a sham treatment, thus belief, hope, expectancy, and other factors could have a stronger effect on recovery. The aim of using placebos in research is twofold: either to serve as control in RCTs, or for examining its independent therapeutic effects. If there is no additional control group, like a no-treatment group to control for the placebo effect itself, there is no way to conclude that changes in the placebo group may be solely due to the placebo effect (Kienle & Kiene 1996). In this case, the observed change in the placebo group is called ‘perceived placebo effect’ (Ernst & Reschl 1995), and it could stem from multiple sources (regression to the mean, spontaneous recovery, etc.). If some form of control is also included, any observed differences
between changes in the placebo group and this control group are called ‘true placebo effect(s)’ (ERNST & RESCHL 1995). Therefore, in studies investigating the true placebo effect, a no-treatment control group should be included (see Discussion for other possibilities). Inert substances could also evoke harmful effects known as placebo side effects or nocebo effects (BARSKY et al. 2002).

There are many possible mechanisms underlying the true placebo effect, the most often mentioned are response expectancies (KIRSCH 1997) and conditioning (SIEGEL 2002). Placebos have specific effects on both subjective and objective variables, though the two concepts are not completely separable. Placebos are not at all universal therapeutic agents; their effects and effectiveness vary significantly, depending on the target organs, symptoms or illness, and on personal and situational factors (GEERS et al. 2005, 2007).

The most often reported subjective effects of placebos are sedation and stimulation, respectively. In several studies, differences were found in self-rated alertness and/or mood among groups ingesting placebos with different suggestions (WALACH et al. 2002; BRODEUR 1965; KIRSCH 1997; LYERLY et al. 1964; ROSS et al. 1962; ZWYGHIUZEN-DOORENBOS et al. 1990) and placebos with different perceptual characteristics (BLACKWELL et al. 1972). In the study of KIRSCH & WEIXEL (1988), participants experienced significant changes in perceived alertness and tension but not in relaxation after receiving placebos with deceptive stimulant suggestion. In a clinical study, placebos received with stimulative suggestion increased, while placebos with sedative suggestion decreased the sleep onset latency in insomniacs (BOOTZIN et al. 1976). According to a narrative review on placebo effects (BUCKALEW & ROSS 1981), sedative effects of placebos were reported more often and in greater magnitude than stimulative effects.

Research has demonstrated that in addition to perceived changes in internal states, placebos given with proper suggestion could also affect cognitive tasks as well as psychomotor performance either positively (LYERLY et al. 1964; ROSS et al. 1962; ZWYGHIUZEN-DOORENBOS et al. 1990; FRANKENHAUSER et al. 1963; KIRSCH & ROSADINO 1993; LIENERT 1955) or negatively (e.g. WALACH et al. 2002; MOERMAN 2002).

Placebos with perceptual characteristics that evoke stimulative or sedative expectations have caused changes in HR and/or in systolic and diastolic blood pressure (SBP, DBP) (BLACKWELL et al. 1972; FRANKENHAUSER et al. 1963). Placebo interventions that were accompanied by stimulative (but deceptive) suggestions have resulted in elevated HR and/or SBP levels in participants in some studies (BLACKWELL et al. 1972; BOOTZIN et al. 1976). Positive placebo reactions were observed in patients diagnosed with circulatory problems (e.g. heart attack, hypertension, etc.) (LYERLY et al. 1964; ROSS et al. 1962).

The respiratory system could also be influenced by placebos. Inert substances given as bronchodilator/bronchoconstrictor drugs have evoked marked changes in asthmatic subjects’ spirometric parameters (BUTLER & STEPTOE 1986; GODFREY & SILVERMAN 1973; LEIGH et al. 2003; LUPARELLO et al. 1970), and even asthmatic attacks could have been initiated (AL ABSI & ROKKE 1991; COLLOCA &
Benedetti 2007) though the healthy control groups typically showed much weaker responses. These studies highlight the possibility of placebo effects in the respiratory system.

It was postulated that placebos may alleviate pain via the activation of the endogenous opiate system or other mechanisms (Benedetti et al. 2005). The clinical use of placebos was once based on this observation (Spiro 1998), and their efficacy has been proven recently (Flaten et al. 2006; Pollo et al. 2001; Price et al. 2005). Another, closely related and intensively investigated area is the reduction of anxiety and perceived stress by using placebos (Beecher 1960; Spiro 1998). This area of research is very important, because beyond and above the modification of pain levels, the level of anxiety (or stress) heavily influences the functioning of the organism as a whole.

Taking the above-mentioned effects and features into consideration, placebos seem to be ideal candidates for enhancing the physical and mental performance of athletes and thus reducing, complementing, or even preventing the use of performance enhancing agents. Although, as Beedie et al. (2006, 2159) note: ‘sports scientists account for the possibility of a placebo effect in intervention studies by using a placebo control’, to date no significant attention has been paid to the so-called true placebo effect. Beyond speculation, there is empirical evidence about the effects of placebos on sport performance. Unfortunately, since researchers in sports sciences usually do not include a no-treatment control group along with the placebo and/or experimental groups, the placebo-controlled designs in this field usually do not account for the true placebo effect (Ernst & Reschl 1995).

In their review, Beedie & Foad (2009) have summarized the findings of twelve intervention studies in sport performance and simply reported a placebo response range varying from –7.8 to 50.7%. The purpose of the present analysis is to characterize the magnitude of placebo response in intervention studies where the dependent variable was connected to sporting performance. Our method of choice was a meta-analysis to obtain a more objective estimate of the effect sizes by combining the results of various studies.

2. Methods

2.1. Data sources and study selection

In the identification, location and retrieval of research papers we followed the general guidelines of our search included 1) a prior review article in the topic (Beedie & Foad 2009), 2) references in studies, 3) major computerized journal databases (PubMed, PsycINFO, and MEDLINE), 4) published conference programs and proceedings, and 5) doctoral theses available in Dissertation Abstract International, up to May 2010. Database searches included different combinations of terms placebo, sport, sport performance, exercise.
All studies exploring the placebo effect of an intervention in any sporting performance in subjects at all levels of fitness were considered. Exclusion criterion was the placebo group that was used solely as a control group, in which the measurement of placebo effect itself was not addressed. In the case of most articles, identified with the key words mentioned above, this criterion could be identified easily through reading the title and the abstract of the study. We also developed a coding system for the evaluation of the studies and the appraisal was done by one of the authors (FK). In this process only the meta-analytically relevant part of the study was taken into account (see Table 1).

In a meta-analysis one is faced with the question of heterogeneity of the pool of research. For example, is it appropriate to combine studies in which the outcome variables, sampling units, and interventions are not uniform? How could we speak of placebo effects, meta-analytically, when these effects are sometimes evoked by caffeine, carbohydrate, sodium-bicarbonate, etc.? How could we speak of these effects uniformly if they are measured on different variables such as strength, endurance, or time? Could these often heterogeneous features be pooled together in a meta-analysis? The answer to these dilemmas is given, at least in part, by Rosenthal (1991), who states that apples and oranges are good to mix. Since scientists often generalize over subjects unique or specific characteristics in a given study, why couldn’t we do so with studies in a given meta-analysis? It is useful to make general statements about fruits, and there is nothing in meta-analysis that prevents us from doing so, contemplates Rosenthal.

2.2. Trial characteristics

Seventeen possible studies in placebo effect on sport performance were identified. In online databases (PubMed, MEDLINE and PsycINFO) three studies (Meinhardt et al. 2008; Hulston & Jeukendrup 2009), in Beedie & Foad’s review (2009) twelve, in our University library one (Mrňa & Skřivánek 1985), and through personal communication one more study (Wright et al. 2009) was identified. We excluded the study of Meinhardt et al. (2008) as neither a no-treatment group nor a baseline measurement were part of the study design. The study of Mrňa & Skřivánek (1985) was excluded for its poor design and the lack of data provided in it. We have also excluded three studies reviewed by Beedie & Foad (2009): (1) Benedetti’s (2007) research was excluded because although sport performance was measured in this study, the focus was on pain tolerance (by placebo morphine) using time as the dependent measure. We also chose not to include the studies of Foster et al. (2004) and Porcari & Foster (2006) because both were published later in Wright et al. (2009) as ‘Experiment A’. Therefore, we have included their study and counted its three researches as independent ones. Table 1 shows the characteristics of the fourteen studies included in the current meta-analysis.
### Table 1
Summary of the quantitative researches included in the meta-analysis

<table>
<thead>
<tr>
<th>Study ID</th>
<th>First author</th>
<th>Year</th>
<th>Placebo group</th>
<th>Exercise mode</th>
<th>Treatment</th>
<th>Performance and physiological outcome variables</th>
<th>Psychological outcome variables</th>
<th>Design</th>
<th>Coding</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Ariel</td>
<td>1972</td>
<td>6 NR</td>
<td>seated, military and bench press</td>
<td>anabolic steroid (Dianabol)*</td>
<td>maximal lift (lbs)</td>
<td>NR</td>
<td>R</td>
<td>B+</td>
</tr>
<tr>
<td>2</td>
<td>Clark</td>
<td>2000</td>
<td>15 32</td>
<td>cycle ergometer</td>
<td>carbohydrate</td>
<td>power, time</td>
<td>NR</td>
<td>R, balanced repeated measure</td>
<td>B</td>
</tr>
<tr>
<td>3</td>
<td>Maganaris</td>
<td>2000</td>
<td>11 21</td>
<td>bench press, dead lift, and squat</td>
<td>anabolic steroids*</td>
<td>maximum force production (kg)</td>
<td>interview about the experienced influences of the administered substances</td>
<td>B</td>
<td>B</td>
</tr>
<tr>
<td>4</td>
<td>Beedie</td>
<td>2006</td>
<td>6 30</td>
<td>cycle ergometer</td>
<td>caffeine*</td>
<td>mean power, heart rate, oxygen uptake, lactate</td>
<td>post-experiment interview questions about perceived performance, and effects of caffeine</td>
<td>R, B, deceptive administration</td>
<td>B+</td>
</tr>
<tr>
<td>5</td>
<td>Beedie</td>
<td>2007</td>
<td>21 19.6</td>
<td>sprint running</td>
<td>hypothetical 'new ergogenic aid'*</td>
<td>time</td>
<td>post-experiment questionnaire about perceived performance, and effects of caffeine</td>
<td>R, 2B</td>
<td>B</td>
</tr>
<tr>
<td>6</td>
<td>Kalasountas</td>
<td>2007</td>
<td>14 26.6</td>
<td>bench press and seated leg press</td>
<td>amino acids*</td>
<td>maximum force production (lbs)</td>
<td>post-experiment interview about beliefs regarding the pills</td>
<td>pseudo-random</td>
<td>A+</td>
</tr>
<tr>
<td>7</td>
<td>McClung</td>
<td>2007</td>
<td>16 24 40</td>
<td>1-km run</td>
<td>Na-bicarbonate</td>
<td>time, heart rate, blood lactate</td>
<td>RPE</td>
<td>R, 2B, Latin square (deceptive administration)</td>
<td>B+</td>
</tr>
<tr>
<td>8</td>
<td>Foad</td>
<td>2008</td>
<td>14 43</td>
<td>cycle ergometer</td>
<td>caffeine</td>
<td>power, heart rate, max. oxygen uptake, blood lactate</td>
<td>NR</td>
<td>R, balanced placebo design</td>
<td>B+</td>
</tr>
<tr>
<td>Study ID</td>
<td>First author</td>
<td>Year</td>
<td>Placebo group</td>
<td>Exercise mode</td>
<td>Treatment</td>
<td>Performance and physiological outcome variables</td>
<td>Psychological outcome variables</td>
<td>Design</td>
<td>Coding°</td>
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</tr>
<tr>
<td>9</td>
<td>Pollo</td>
<td>2008</td>
<td>11</td>
<td>leg extensor</td>
<td>caffeine*</td>
<td>work performed (kJ)</td>
<td>RPE</td>
<td>R, B</td>
<td>A+</td>
</tr>
<tr>
<td>10</td>
<td>Wright¹</td>
<td>2009</td>
<td>32</td>
<td>5-km running time trial</td>
<td>super-oxygenated water*</td>
<td>running time, heart rate, blood lactate</td>
<td>RPE</td>
<td>B</td>
<td>B</td>
</tr>
<tr>
<td>11</td>
<td>Wright</td>
<td>2009</td>
<td>18</td>
<td>sprint cycling</td>
<td>fast-acting creatine monohydrate*</td>
<td>power</td>
<td>NR</td>
<td>B</td>
<td>B+</td>
</tr>
<tr>
<td>12</td>
<td>Wright</td>
<td>2009</td>
<td>10</td>
<td>6-minute walking test</td>
<td>Peligrino Spa water*</td>
<td>distance</td>
<td>RPE</td>
<td>B</td>
<td>B+</td>
</tr>
<tr>
<td>13</td>
<td>Hulston</td>
<td>2009</td>
<td>10</td>
<td>prolonged cycling</td>
<td>carbohydrate</td>
<td>power, heart rate, oxygen uptake, respiratory-exchange ratio, total carbohydrate oxidation, and total fat oxidation</td>
<td>NR</td>
<td>B</td>
<td>B+</td>
</tr>
<tr>
<td>14</td>
<td>Duncan</td>
<td>2010</td>
<td>12</td>
<td>short-term, high-intensity cycling</td>
<td>caffeine</td>
<td>power, heart rate, fatigue index, peak blood lactate</td>
<td>RPE</td>
<td>balanced placebo</td>
<td>B+</td>
</tr>
</tbody>
</table>

Note: R = random, B = blind, 2B = double blind, RPE = rating of perceived exertion, NR = data not reported

* Placebos were administered in all conditions

¹ 3-part study

² A+: between subject design with baseline assessment following a habituation (run-in) period and/or with assessment of performance after debriefing

A: between subject design with simple baseline assessment

B+: within-subject design with a run-in period and/or with random treatment order to avoid learning effects and/or using different ‘doses’ (suggestions)

B: within-subject design without a run-in period or counterbalanced treatment order
2.3 Data extraction and statistics

The following study characteristics were obtained by one reviewer (MB) and verified by another (FK): ID number, year of publication, author(s), type of exercise, intervention design included in meta-analysis (see the details later), total number of subjects, number of subjects in the baseline trial, number of subjects in the treatment trial, means and SDs of baseline and treatment trials.

Based on Lipsey and Wilson’s (2000) suggestion, since all dependent variables in the included studies are inherently continuous (e.g. time, power, mass) and measured on continuous but different scales (e.g. second or minute, Watt, kg or pound), we had decided to use standardized mean difference effect sizes computed from the given raw values (pre- and posttest means and pre-SD. See the details below). When these data were not given in the original article, they were requested from the authors via email. In cases where the requested data have not been provided, effect size estimations were carried out based on the available probability values. Effect sizes, Hedges’ *c* values, variances weights (w) and CIs for the individual studies are shown in Table 2.

In the case of three studies (Ariel & Saville 1972; Beedie et al. 2007; Clark et al. 2000) we have calculated the effect sizes either based on the approximated

<table>
<thead>
<tr>
<th>Study ID</th>
<th>Author</th>
<th>ES</th>
<th>Hedges’ <em>c</em></th>
<th>Var</th>
<th>w</th>
<th>CI low</th>
<th>CI up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Ariel</td>
<td>1.73</td>
<td>0.71</td>
<td>1.05</td>
<td>0.96</td>
<td>-0.27</td>
<td>3.73</td>
</tr>
<tr>
<td>2</td>
<td>Clark</td>
<td>1.06</td>
<td>0.89</td>
<td>0.13</td>
<td>7.60</td>
<td>0.35</td>
<td>1.77</td>
</tr>
<tr>
<td>3</td>
<td>Maganaris</td>
<td>0.73</td>
<td>0.85</td>
<td>0.15</td>
<td>6.50</td>
<td>0.28</td>
<td>1.17</td>
</tr>
<tr>
<td>4</td>
<td>Beedie</td>
<td>0.24</td>
<td>0.71</td>
<td>0.29</td>
<td>3.42</td>
<td>-0.82</td>
<td>1.30</td>
</tr>
<tr>
<td>5</td>
<td>Beedie</td>
<td>0.02</td>
<td>0.93</td>
<td>0.05</td>
<td>18.90</td>
<td>-0.43</td>
<td>0.47</td>
</tr>
<tr>
<td>6</td>
<td>Kalasountas</td>
<td>0.37</td>
<td>0.89</td>
<td>0.09</td>
<td>10.91</td>
<td>0.07</td>
<td>0.67</td>
</tr>
<tr>
<td>7</td>
<td>McClung</td>
<td>0.13</td>
<td>0.90</td>
<td>0.07</td>
<td>13.74</td>
<td>-0.04</td>
<td>0.65</td>
</tr>
<tr>
<td>8</td>
<td>Foad</td>
<td>0.20</td>
<td>0.89</td>
<td>0.09</td>
<td>11.56</td>
<td>-0.38</td>
<td>0.78</td>
</tr>
<tr>
<td>9</td>
<td>Pollo</td>
<td>0.30</td>
<td>0.85</td>
<td>0.12</td>
<td>8.30</td>
<td>-0.38</td>
<td>0.98</td>
</tr>
<tr>
<td>10</td>
<td>Wright (A)</td>
<td>0.39</td>
<td>0.95</td>
<td>0.04</td>
<td>27.65</td>
<td>0.01</td>
<td>0.76</td>
</tr>
<tr>
<td>11</td>
<td>Wright (B)</td>
<td>-0.12</td>
<td>0.91</td>
<td>0.06</td>
<td>15.75</td>
<td>-0.61</td>
<td>0.38</td>
</tr>
<tr>
<td>12</td>
<td>Wright (C)</td>
<td>0.08</td>
<td>0.84</td>
<td>0.13</td>
<td>7.74</td>
<td>-0.62</td>
<td>0.79</td>
</tr>
<tr>
<td>13</td>
<td>Hulston</td>
<td>0.14</td>
<td>0.84</td>
<td>0.13</td>
<td>7.68</td>
<td>-0.57</td>
<td>0.84</td>
</tr>
<tr>
<td>14</td>
<td>Duncan</td>
<td>0.37</td>
<td>0.87</td>
<td>0.11</td>
<td>9.01</td>
<td>-0.28</td>
<td>1.02</td>
</tr>
</tbody>
</table>

Note: ES = effect size, Var = variance, w = weight, CI low = lower confidence interval, CI up = upper confidence interval.
(smaller than or equal to) or the exact p values. It is obvious that in the former case the effects could be underestimated.

Even though ARIEL and SAVILLE’s study (1972) is a well designed one, the authors only reported the mean differences between training and placebo trials and the approximate significance level of these differences. In the study by CLARK et al. (2000, 1645) ‘full placebo effect’ was computed based on the difference in the power output between the told-carbohydrate and the told-placebo groups. P for the change (given in percent), CI in percent, and peak powers for treatment groups were provided for this effect. For the calculation of the effect size mean baseline data for each group would have been needed. In the abstract mean baseline for all subjects was provided, however, in Table 1 (1645) only peak powers were provided. Based on these data we decided to estimate the effect size based on the significance level (p = 0.06) of the full placebo effect. In BEEDIE et al. (2007) standard deviations for baseline and experimental trials were missing. Therefore, the effect size was estimated on the basis of the reported (exact) probability levels.

Three studies (ARIEL & SAVILLE 1972; KALASOUNTAS et al. 2007; MAGANARIS et al. 2000) examined placebo effect in more than one exercise. Thus we had to consider whether we should count each result in a study as an independent one, or combine them somehow and consider them as a single grand result (i.e. one effect size per study). Following ROSENTHAL’s (1991) and LIPSEY and WILSON’s (2000) recommendations, only one effect size was derived from each study by averaging the standardized mean difference effect sizes.

Study designs of the retrieved researches included between-subject, within-subject, Latin-squared, balanced placebo, and mixed designs. However, the interventions in which placebo responses were measured were all within-subject designs, except for one study (CLARK et al. 2000). There is a controversy regarding whether data obtained from different designs should be mixed in a meta-analysis, and also regarding how to compute effect sizes in repeated measures designs (ARIEL & SAVILLE 1972; KALASOUNTAS et al. 2007; MAGANARIS et al. 2000). In the computation of the effect sizes in case of different study designs, the general outlines of MORRIS & DeSHON (2002) were followed. According to them, a meta-analyst could compare effect size estimates across studies with different designs if (a) effect sizes estimate the same treatment effect, (b) all effect sizes are scaled in the same metric units and (c) meta-analysis uses design-specific estimates of sampling variance. For the first suggestion, it was ascertained that each included study’s effect size estimate represented a placebo effect. Considering the problem of metric units, standardized mean effect sizes were employed, as mentioned earlier, which were computed in accordance to the suggestions of BECKER (1988) and MORRIS & DeSHON (2002). Accordingly, if there was a possibility that variances were not equal over time, i.e. in pre- and posttest scores, then the use of pretest SD was preferred in computing the effect size, because pretest SD is more unaffected by the treatment and therefore more consistent across studies. Uniformly the effect sizes used in this meta-analysis are all in the same metric units and are calculated by
BECKER’s (1988) formula and express the differences between trial and baseline in units of the standard deviation at baseline:

\[ \text{Effect size} = \frac{\text{mean at trial} - \text{mean at baseline}}{\text{standard deviation at baseline}} \]

Sampling variances for the individual effect size and sample bias \(c(df)\) (HEDGES 1982) for the correction of all effect sizes were computed. By the inverse of the variance a weight score \((w)\) was attributed to each effect size, and an overall variance-weighted mean effect size was computed (cf. MORRIS & DESHON 2002).

For the estimation of between-study homogeneity (Q) of effect sizes, a comparison of theoretical and observed variance was made and tested against a chi-square distribution with \(k-1\) degrees of freedom (HEDGES 1982). Since the \(Q\) statistic returned the acceptance of the null-hypothesis of heterogeneity (i.e. effect sizes fitted to a fixed effects model), confidence intervals (CI) for the overall mean and individual effect sizes could have been calculated in a typical way (SÁNCHEZ-MECA & MARÍN-MARTÍNEZ 2008). Beside computing \(Q\) for the overall mean effect size, variables for subgroup analysis were defined \(a\) priori, including the type of exercise (strength vs. endurance), and \(Q\) were computed in these subgroups as well, and between group variance was tested (HEDGES & OLKIN 1985). Effect size calculations based on the above formulas were carried out in Open Office Excel, and effect size estimations were done by WILSON’s macros (2009).

3. Results

3.1. Outliers

The distribution of unweighted effect sizes was examined, and an extreme value (2.39) was found. This is 2 SDs from the second highest, and almost 3 (2.98) SDs from the mean. This effect size refers to ARIEL & SAVILLE’s (1972) study and is the combination of four effect sizes calculated from approximate \(p\) values. After the close examination of these individual effect sizes of this study we have decided not to eliminate the whole study from the meta-analysis, but to trim its effect sizes. Thus out of the four effect sizes (1.94, 1.52, 3.05, and 3.05) the two extremes were eliminated.

3.2. Statistical results

The number of participants enrolled in the placebo arms of the studies was \(N = 196\) (range 6–32; mean sample size of placebo arms = 14). The mean age of people in
the placebo part of the studies was 29.35 years. Four out of the fourteen investigations examined female participants. The size of placebo arms tended to increase in more recent years ($r = 0.399, p = 0.177$). The most commonly examined types of exercises were cycling ($n = 6$), various forms of weight lifting ($n = 4$), and running ($n = 3$).

The overall standardized unweighted mean effect size was 0.4 (95% CI ranged from 0.24 to 0.56), and the variance weighted mean effect size was 0.31. Effect sizes for two subgroups, power and endurance type of exercise, were 0.48 and 0.22 respectively. Applying Cohen’s recommendations for interpreting effect sizes, these can be regarded as moderate and small effect sizes respectively.

The homogeneity ($Q$) of effect sizes for the whole sample was examined. The $Q$ test yielded a non-significant chi-square result $\chi^2(13, N = 196) = 9.35, p = 0.75$, i.e. effect sizes are homogenous. $Q$ tests of effects sizes in the two subgroups of exercise, power and endurance, $\chi^2(3, N = 42) = 2.29, p = 0.51$, and $\chi^2(9, N = 142) = 9.23, p = 0.42$ respectively, also yielded homogeneity.\(^1\) Because of this high homogeneity of variance no models (such as analogue to the ANOVA for categorical or weighted regression analysis for continuous variables) were tested.

4. Discussion and Conclusion

A meta-analysis approach was used in order to obtain effect size estimations of placebo effect in sport performance, based on fourteen different studies published since 1972 to date. The main finding of this meta-analysis was that placebo treatments have a small to moderate effect on sports performance.

According to our results, it can be stated that the placebo effect could play a role in sport performance. Does this effect have a practical implication? If the uniformly positive values of the effect sizes are considered, the answer is clearly ‘yes’. However, there are several other factors to consider. First of all, several studies included in the meta-analysis manifest methodological shortcomings that could affect internal validity. For example, comparing the placebo group to its own baseline may yield learning or habituation effects in the course of the treatment, which could lead to the overestimation of the effect. There are at least three possibilities to assess the placebo effect more properly: 1) comparing a placebo and a no-treatment group, 2) using a balanced placebo design, or 3) comparing the effects of different placebos. Second, results obtained in laboratory settings could have limited external validity in the actual athletic competitions where the environment, importance of the competition, appraisal of the judges, equipment, facilities or opponents’ skills could all be mediating factors of the placebo response. Third, it is well known in the placebo literature that there are marked individual differences in the magni-

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\(^1\) These low and non-significant $Q$ statistics indicated the fit of a fixed effects model. However, a random effects model was also tested, but random effects variance ($v_\theta$) returned a negative value ($-127.26$), consequently it was set to zero, thus subject-level sampling error ($v_i$) remained unchanged.
tude of the placebo responses. While some individuals show remarkable responses to placebo intervention, others might not respond at all. Individual responsivity appears to be a function of certain personal and situational factors, as well as the interaction of the two, and consequently it is difficult to predict.

Placebo treatment in sport may be considered as a way to mobilize psychophysiological reserves from a psychological perspective. The relatively varied and possibly restricted size of these reserves obviously limits the achievable enhancement. The small to moderate effect sizes calculated in this meta-analysis may reflect these limitations, but also the neutralization of the demonstrable placebo effects by the fact that both responders and non-responders were included in the existing studies. Perhaps larger effect sizes could be obtained with athletes who could be identified as placebo-responders. This is the population that would benefit the most from placebo intervention in training and competition. In spite of individual differences in placebo responses, it is worth to keep in perspective that even rather small gains in performance could make a significant difference in competitive situations (Clark et al. 2000).

4.1. Recommendations for further studies

Beyond methodological issues mentioned above, the majority of the studies do not attempt to shed light on underlying psychological factors. Possible mediating factors in placebo research could be personality traits (e.g. dispositional optimism Geers et al. 2005; Geers et al. 2007); social acquiescence (Fischer & Greenberg 1997) and motivation (Aletky & Carlin 1975; Jensen & Karoly 1991). Qualitative and idiographic methods should also be used for a better and deeper understanding of the psychological mechanisms underlying the placebo effects (Beedie 2007).

Furthermore, expectations evoked by the perceptual properties (i.e. colour, size, shape) of placebos, the mode of application (i.e. oral vs. injection), the dosage form and the price of drugs could also be important mediating factors (De Craen et al. 1996, 2000; Moerman 2002). Unfortunately, despite the fact that this issue has been raised in the past (Trojan & Beedie 2008), only one study has been published to date in which these issues have been considered (Bérdi et al. 2010).

Further theoretical and empirical work is needed to determine the possible psychophysiological mechanisms of placebo effects in sports. A role of classical conditioning has been demonstrated at two levels: pharmacological effects of substances and also experiences from muscular exercises may have an effect on the physical performance (Benedetti et al. 2007; Pollo et al. 2008). These effects could partly be the consequences of changes in pain sensation and perception by the conscious and non-conscious regulation of muscular functions (Pollo et al. 2008). However, pain regulation may also be modulated by multiple social and cultural factors (Morris 1997; Moerman & Jonas 2002). Another possible mechanism
may be the reduction of the acute levels of stress or anxiety. The stress- and anxiety-alleviating properties of placebos have been extensively examined in clinical settings (BEECHER 1960; SPIRO 1998) but not in the area of sport sciences.

Pennebaker’s schema-guided selective searching approach offers another possible explanation (PENNEBAKER 1982). According to this theory, conscious perception of internal sensory information is based on active top-down selection mechanisms in which external environmental cues may distract attention from internal stimuli (e.g. signs of fatigue of muscles). Placebos given with proper information, theoretically, could also alter the direction of top-down searching processes: persons having taken a ‘stimulant’ drug can simply ignore or attenuate the signs of fatigue (for the use of a similar rationale, i.e. the possible top-down influences of placebos, see POLO et al. 2008).

The finding that placebos could enhance athletic performance has ethical implications, too. With today’s extreme demands in sporting performance, aimed at the breaking of seemingly unbreakable records, the placebo effect may play a crucial role in athletic success. From this point of view, placebo interventions could be conceptualized as means of legal ‘psychological doping’ (BENEDETTI et al. 2007).

Currently only treatments containing physiologically active substances are regarded as illegal. Based on today’s doping policy, placebo treatment is a legal and undetectable way to enhance sport performance. In this respect, placebo treatment cannot be sharply distinguished from other psychological interventions, e.g. mental training or relaxation techniques. The presently legal state of placebo treatment may or may not change in the future regulations, depending on the judgement of policy makers and the technique of differentiating responders (those who could benefit from placebo intervention) and non-responders (those whose performance would not change in spite of placebo intervention). A better understanding of the placebo phenomenon may certainly open new ways to improve sports performance in at least a proportion of the athletes – on an ethically and morally more acceptable ground than through the use of illicit performance enhancing agents.

References


