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Feeling bad or feeling good, does emotion affect your consumption of food? A meta-analysis of the experimental evidence

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ABSTRACT

Whether emotions affect eating, and in whom, has remained unclear. This meta-analysis assessed the effect of emotions on eating in both healthy and eating disordered individuals. Fifty-six experimental studies investigating the causal effect of emotions on eating behavior were selected including 3670 participants. Separate meta-analyses (random models) were performed for negative and positive emotions. Among healthy people the moderating impact of individual differences in restrained and emotional eating and of being overweight or obese was assessed for negative emotions. Results: Restrained eaters showed increased eating in response to negative emotions. Negative emotions did not affect eating in overweight or obese people, people with eating disorders or in self-assessed emotional eaters. Positive emotion resulted in increased eating across groups. Heterogeneity was high and could be explained by differences in emotion induction procedures, eating measures, and age of participants. These findings indicate that particularly restrained eaters are vulnerable to emotion-induced eating. Additional qualitatively good experiments are called for in combination with studies assessing emotion-eating links in people's naturalistic environment.

1. Feeling bad or feeling good, does emotion affect your consumption of food? A meta-analysis of the experimental evidence

The phenomenon of overeating has become a serious threat for human health. Accordingly, factors that make people vulnerable to overeating have been extensively investigated. An important factor that has been linked to overeating is the experience of negative emotions. This so called 'emotional eating', also referred to as 'comfort eating' (e.g., Gibson, 2012) or 'stress-induced eating' (e.g., Greeno and Wing, 1994) has received an abundant amount of empirical attention in the last decades and the prevalence of self-reported emotional eating is increasing (Gibson, 2012). Despite this attention, the very basic question whether negative emotions affect eating, and more specifically in whom, remains unclear. Therefore, the purpose of the current meta-analysis is to reassess the state of knowledge concerning the effect of negative, but also positive, emotions on eating.

Before detailing on why, how, and in whom emotions may impact eating behavior, it is important to define what can be understood as an emotion and how emotions differ from related constructs. Although different scholars use various definitions (see also Gross, 1998) emotions can be defined as the more fine-grained states that unfold over a relatively short time period and include a mixture of emotional feelings, expressions, and physiology (Gross, 1998). The physiological component typically entails a classic flight-or-flight response and is driven by the hormone adrenaline that shuts down digestion (see also below). There are several discrete emotion types, like sadness, happiness, and anger. Compared to emotion, mood is a more diffuse state, longer in duration, like depression or euphoria (Gross, 1998). Affect can be seen as an umbrella term, tapping the entire affective realm ranging from a discrete negative emotional state like sadness and anger to feeling generally negative or positive. Finally, stress and emotions are highly interdependent as stress is often accompanied by emotions and vice versa (Lazarus, 2006). Particularly a short bout of stress resembles an emotion (that is short in nature as well) and both are physiologically accompanied by the adrenaline driven fight-or-flight response. Prolonged stress, however, is physiologically different: it comes together with the release of the hormone cortisol and carries the potential for bodily harm. As scholars use these terms differently and interchangeably, the term emotion in the present paper is used to refer to short-lived affective states evoked in the laboratory, that are notably different from longer-lasting mood or more chronic stress-related states.

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In the following paragraphs we will revisit theories and previous research related to the relationship between negative emotions, as well as positive emotions, and eating behavior.

1.1. Eating in the emotional case: theoretical perspectives

Emotions are an integral part of people's daily lives. They do not only shape our cognitions, they also give rise to several physiological changes and are important motivators for behavior. However, why emotions would impact eating behavior and in whom has remained a topic of debate. Macht (2008) summarizes the many prevailing theories how both negative and positive emotions affect eating behavior in one model that takes into account both individual differences and emotion features. Different classes of emotion-induced changes of eating are assumed that generally can be summarized as follows.

On the one hand, high-arousal or intense emotions are assumed to suppress eating. This is based on the idea that these emotions activate the autonomic nervous system that gives rise to a set of physiological changes that may be expected to induce satiety, such as slowed gastric emptying, shunting of blood from the gastrointestinal tract to the muscles, and the release of appetite-inhibiting hormones like catecholamine (Torres and Nowson, 2007; Wing et al., 1990).

On the other hand, eating is assumed to be affected by moderate-arousal or moderately-intense emotions. A difference is made between restrained, emotional, and normal eating. *Restrained eating* refers to the intentional and sustained restriction of caloric intake for the purpose of weight loss or weight maintenance (Herman and Mack, 1975). When periods of restraint are difficult to maintain, disinhibition may follow. Emotions typically seem triggers for disinhibition in restrained eaters, as the emotion imposes a more pressing concern than sticking to their self-imposed dietary boundaries (Herman & Polivy, 1984, 1988; Polivy et al., 1994; Ruderman, 1985). Both negative and positive emotions are supposed to increase food intake due to impaired cognitive control. Also, individuals with an eating disorder are typically restrained eaters as they are persistently trying to restrict their eating behavior and even judge themselves largely in terms of their eating habits and their ability to control them (Fairburn et al., 2003). *Emotional eating* refers to overeating or binge eating in response to emotions and is assumed to serve as an attempt to regulate these undesired emotions (Heatherton and Baumeister, 1991; Polivy and Herman, 1993) Who is vulnerable for the phenomenon of emotional eating, however, remains unclear, as also *healthy eaters* sometimes increase their food intake in response to emotions (Macht, 2008).

In sum, it has been postulated that different classes of emotion-induced changes can lead to both increased and decreased eating. In line with the inconsistent views on how emotions affect eating and in whom, empirical results of separate individual studies reflect these same inconsistencies. Important to note is that the emotional eating literature is rich and encompasses cross-sectional, momentary, and longitudinal emotion-eating relationships (see for reviews Greeno and Wing, 1994; Macht, 2008). Although the current meta-analysis will systematically address the empirical evidence in relation to how and in whom emotions affect eating, below we will briefly review the empirical findings in relation to the above described assumptions. In order to assess the true causal nature of the relationship, thus the precise effect of emotion on eating, the present work aims at reviewing laboratory studies only, as they allow for controlling both the emotion and eating environment, have the ability to illuminate extraneous variables, and thus have the advantage of high internal validity.

1.2. Eating in the emotional case: a brief empirical overview

As stated, the empirical findings relating to emotion-induced changes of eating as described in Macht's model (2008) can be summarized as inconsistent. To illustrate, high-arousal or intense emotions are assumed to suppress eating. This was indeed found in one of the earliest studies on this topic (Schachter et al., 1968), but only in normal weight participants, not in obese ones. Most other laboratory studies, however, did not observe decreased eating in response to high-intense emotions, with most studies finding unaffected eating in response to stress (Greeno and Wing, 1994).

In relation to moderately-intense emotions, it is assumed that negative and positive emotions increase food intake in *restrained eaters*. For negative emotions there is indeed evidence for the assumed effect from a systematic review (Greeno and Wing, 1994) and a meta-analysis (Cardi et al., 2015). Positive emotions have been studied more consistently in relation to eating only recently. However, there is some inconsistent evidence as to whether restrained eaters increase food intake in response to positive emotion (e.g., Cools et al., 1992; Yeomans and Coughlan, 2009). Additionally, it is assumed that negative emotions increase food intake in people who use eating as means to regulate their negative emotions. Macht (2008) labels them as *emotional eaters*. It is not clear, however, who qualifies as an emotional eater. A recent review on the predictive validity of self-assessed emotional eating showed that people who assess themselves as emotional eater inconsistently show increased or unaffected eating in response to negative emotions (Bongers and Jansen, 2016). As such findings have not been corroborated by a meta-analysis yet, the present work will do so. Finally, that negative and positive emotions can modulate eating in *healthy eaters* as well, seems common knowledge. It is unclear, however, when emotions result in increased, decreased, or unaffected eating and what the causal triggers of changes in food intake are (Macht, 2008). As the results across studies provide inconsistent evidence relating to the emotion-eating relationships, we deemed a meta-analysis crucial.

Recently, though, a meta-analysis was performed on how emotions, both negative and positive, affect food intake across laboratory settings (Cardi et al., 2015). To sum, the findings revealed that overall negative emotions had a small but significant effect and resulted in increased eating. While in healthy controls and obese people eating was not affected, negative emotions triggered increased food intake in restrained eaters and binge eater subtypes, although the effects were small. Additionally, a small effect was found for positive emotions, in the direction of positive emotions triggering increased food intake. This meta-analysis is a nice start for tackling the causal effect of emotions on eating, but it contains some restrictions that may limit the validity of the obtained effects. Below we will discuss the most important points that may profit from improvement.

First, the search terms in Cardi et al. (2015) were narrow. Of the wide affective realm, only the affect-related term 'mood' was included, while relevant terms like 'emotion', 'affect', 'hedonic', and 'stress' were ignored. This must have left several relevant studies undiscovered and consequently, must have resulted in a lower than possible statistical power. Further, it was examined whether the mood induction was successful *averaged across all included studies*. Although on meta-analytic level (across all included studies), mood was significantly induced, it is a restriction that studies were included that lacked a significant increase of mood in the mood condition compared to the control condition (e.g., Wilkinson et al., 2013). In order to reveal emotion-induced eating in a laboratory setting, which is eating *in response to* emotions, a successfully induced emotion seems a central prerequisite for inclusion.

1.3. Rationale for the current meta-analysis

We considered a novel meta-analysis on the effect of emotion on food intake of valuable importance for numerous reasons. First, given the limitations of the meta-analysis outlined above, we deemed a wider variety of different search terms related to emotions and food intake as crucial. A wider scope in search terminology will result in more studies, and consequently in a qualitatively improved meta-analysis, with more power and stronger point estimates. Additionally, through more stringent inclusion criteria related to the emotion induction (such as including only studies that successfully induced emotions compared to a

control condition), the actual causal effect of emotions on food intake can be tackled, resulting in a more robust answer to the question to what extent emotions trigger increased eating and in whom. Further, on a more general level, additional meta-analyses within a field give valuable merit to the scientific debate relating to the topic under study (see e.g., Peters et al., 2013) and align with the importance of reproducibility and replication (Open Science Collaboration, 2015). Finally, as the theories on emotional eating are diverse, and as it still an unresolved question if emotions trigger eating and in whom, we considered it crucial to do a comprehensive meta-analysis that covers all possible studies that investigated the causal effect of emotions on food intake, thereby including all positively and negatively valenced affect-related phenomena, combined with all possible moderators that have been put forward thus far by scholars, such as having a normal weight vs. being overweight/obese, having an eating disorder vs. having not, and individual differences in both restrained and emotional eating.

2. Method

2.1. Literature search

To find as many relevant studies as possible, an extensive literature search was conducted. In step 1, the databases PubMed, Ovid, Scopus, and Web of Science were examined during February 2016. The search looked for articles that contained at least one of the following terms (emotion*, affect*, hedonic, mood, stress, comfort) in combination with at least one of the following terms (eat*, consume, intake, food) while excluding articles that contained one or more of the following terms (child*, adolesc*, animal, rat, mice, monkey) in their titles, with the asterisks as wild card to find the variations in the respective words. There were no limits set for the year of publication and all titles were included until the present search (February 2016). In step 2 three of the authors searched thoroughly all possibly relevant articles that could meet the inclusion criteria as evaluated by title and citation context. In step 3, a snowball method was used, in which the citations of relevant articles known to the authors were reviewed. Finally, unpublished work was searched, by requesting for unpublished data in professional networks and associations, and by searching the databases Networked Digital Library of Theses and Dissertations (NDLTD) and the European counterpart “DART-Europe”.

2.2. Inclusion criteria

To be included in the meta-analysis, studies were required to meet the following criteria: (a) Studies had to investigate the causal effect of negative and/or positive emotions, so only experimental studies were considered that included: i) random assignment of participants to a (positive and/or negative) emotion induction condition compared to a control condition; ii) both baseline and post-induction emotion measures; and iii) evidence of successful emotion induction as reported in the results section. Induction was considered successful if the negative condition significantly increased negative or decreased positive emotion from pre-induction to post-induction level compared to a neutral control condition; if the positive condition significantly increased positive or decreased negative emotion from pre-induction to post-induction level compared to a neutral control condition; or if difference scores between pre- to post-induction differed significantly between negative and/or positive emotion condition and neutral control condition. Significance scores of p's $< .05$ were considered statistically significant. If multiple emotion measures were included (e.g., blood pressure and various self-reported mood scales like Visual Analogue Scales), emotion induction was considered successful if at least one of the emotion measures reached significance. (b) Consumption had to be registered by objective behavioral eating measures reported in energy, grams, or number of food items consumed after a successful emotion induction. To contrast, studies registering self-reported hunger (Macht, 1999), craving (Gibson and Desmond, 1999), motivation to eat (Macht and Simons, 2000), or food registration in retrospect (Conner et al., 1999) were not eligible. While such measures are important for understanding eating behavior as a whole, they are markedly distinct from actual eating behavior during the experience of negative or positive emotions. (c) An adult population ($\geq$18 years old) of participants with an eating disorder diagnosis, healthy participants, or both types of participants. (d) Only papers written in the English language were considered. (e) Sample size and empirical data (means, standard deviation, standard error) on the amount of food intake had to be reported in order to calculate effect sizes (Lipsey and Wilson, 2001). If the appropriate data were not reported, for example because data were depicted in figures or sample sizes were not adequately specified, authors were contacted to provide data, unless the corresponding paper had been published more than 30 years ago (i.e., before 1986). In total, 15 authors were contacted; 46.7% provided the required data.

2.3. Study selection

The literature search across the four databases identified 9463 articles that could be potentially included in the meta-analysis. An additional 22 potential papers were added based on the snowballing method. All articles were screened for eligibility based on title and abstract, which resulted in 197 records that were further screened. Of these, 69 were rejected because the research did not meet inclusion criteria. Of the remaining 128 records, the full-text articles were screened in detail as the coding process was initiated. During the coding process, 76 articles were excluded: 67 because the described studies did not meet the inclusion criteria, 8 because the appropriate data were not provided, and 1 because it contained duplicate data. Finally, 52 articles (describing 56 independent studies) were included in the database including 3670 participants. Of the 56 included studies, 27 overlapped with Cardi et al.'s meta-analysis (2015) that included 33 studies. See Fig. 1 for a flow diagram of the selection process resulting in the included studies.

2.4. Data coding

All four authors coded the characteristics of included studies and jointly coded the first 15 articles in order to develop the coding scheme. Subsequently the remaining articles were divided among the authors and coded independently. Twelve random articles were assigned to a second coder in order to determine the interrater reliability. One of these was excluded later as it did not fit the inclusion criteria. In addition, eight random studies that were excluded but did belong to the original selection of 120 studies at the start of the coding process were divided across a second coder in order to double check agreement on the inclusion criteria. The coding format included information on the sample, measurement of relevant variables, statistical information, and moderators. More specifically, each study was coded for the following characteristics:

2.4.1. Sample

These included the mean age and BMI, sex distribution (percentage men), true number of participants in the final sample, sample category (healthy, eating disordered, or both), sample type (student or general population), weight type of the sample (normal weight, overweight, obese, or all weight types), and country where the study was conducted. In case of a clinical diagnosis, it was also coded how the diagnosis was assessed. Finally, if anxiety or depression was measured, or restrained, external, or emotional eating was assessed, then this was indicated (if so, also the scale was coded).

2.4.2. Emotion measures

These involved the type of measure (e.g., self-report, physiology, cortisol), the valence and the type of emotion (a discrete negative

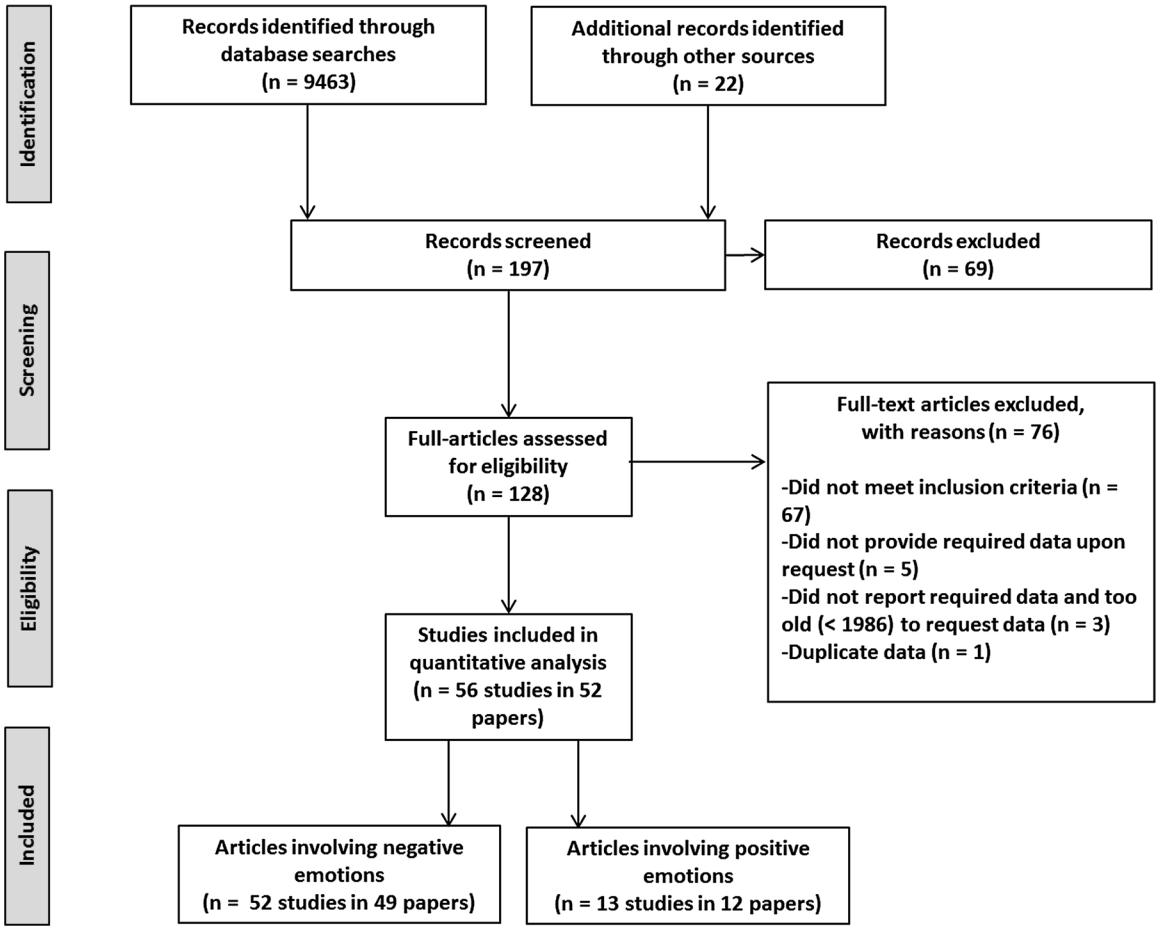


Fig. 1. Flow Diagram Depicting Study Selection.

emotion like sadness or general negative affect like stress or discomfort), whether or not the induction was successful, and the type of induction. This latter was coded as: others' emotional displays (OED) including film excerpts, movies, vignettes or music accompanying an emotion-inducing story; guided emotion induction (GEI) including (autobiographical) recall of emotional events; and social feedback (SF) including false feedback on a performance task, or facing the threat of public speaking like the Trier Social Stress Test (Kirschbaum et al., 1993).

2.4.3. Eating measures

These included the category of the food type offered (sweet, savoury, or both) and a subscription of the offered foods (e.g., meal, popcorn, chocolate). If it was possible to transform measurements into Kcal, this was done.

2.4.4. Statistics

Statistical details to enable the computation of standardized effect sizes were included, entailing the sample size for (sub)groups, whether the emotion induction was based on a between or within-subjects design, and the means and *SD*s for the amount of food consumed. In line with research on different theories of why and in whom emotions cause eating, the following information was coded to enable moderator analysis: healthy sample, eating disordered sample, overweight/obese sample, high or low restrained eating sample, and high or low emotional eating sample. This pertains to studies where participants were classified into groups and defined as any of these moderators. If the relevant statistical information was only available for the overall sample rather than the relevant subset, then only main effects were coded. If studies reported information on subgroups irrelevant for the present study (for example, sleep vs. no-sleep deprived groups), these were coded as separate subgroups and merged at a later phase, before entering the final analyses. If the correlation in studies with paired groups designs was not provided, it was set to 0.70.

2.5. Interrater reliability

In relation to the exclusion of studies, there was agreement for seven out of the eight studies that were excluded initially; for one study the second coder was unsure. After discussion with all authors it was confirmed that the study had been correctly excluded. For the studies that were coded by two raters, kappa was calculated for categorical variables and the intraclass correlation coefficient (ICC) for continuous variables using SPSS. Kappa ranged from 0.70 to 1.00, with all disagreements resolved to reach consensus. There was one outlier for sample type (Kappa of 0.37), which was subsequently recoded as a dichotomous variable by two raters simultaneously indicating whether the sample consisted of students, or a mixed population. The ICC over all continuous variables ranged from 0.67 to 1.00. Subsequently all numeric data necessary for calculating effect sizes and for the meta-regression were rated by at least two coders.

2.6. Meta-analytic strategy

2.6.1. Hedges' g and SE

To estimate the overall effect size and separate effect sizes, Hedges' g was calculated for all studies by means of Comprehensive Meta-Analysis 3.0 (CMA). Effect size calculations were based on a comparison between the control group and the emotion induction group regarding eating behavior. Details regarding the means, standard deviations, and

sample size for emotion induction group and control group were used to calculate Hedges' g (Lipsey and Wilson, 2001).

A common criticism of meta-analysis is the question of whether apples and oranges can be compared. *If* studies actually measure different underlying outcomes, it may not be reasonable to assume that there is a single underlying effect. Determining the extent to which studies are thus comparable is an important consideration, which involves the criteria set in the selection procedure of studies (a-priori), as well as statistical diagnostics after the studies have been selected for inclusion. Ideally, effect sizes found in different studies should differ as would be expected on the basis of random sampling: this is called homogeneity. If effect sizes between studies differ more than would be expected by chance, this has been referred to as (statistical) heterogeneity (Lipsey and Wilson, 2001).

The *Q*-statistic, with a Chi-square distribution, is a heterogeneity test across the studies included in the meta-analysis. A statistically significant *Q*-test ($\alpha = 0.05$) indicates that the null-hypothesis of homogeneity is rejected. As the *Q*-statistic only assesses statistical significance of heterogeneity and has low power with a small number of studies, the I^2 was additionally used to assess heterogeneity; it indicates the percentage of variation across studies that is due to true between-study differences rather than chance.

In the analyses a Random-Effect Model was used to estimate the mean effect size. Separate main-effects for negative and positive emotions on eating behavior were assessed. Subsequently, the mean effect-size for the different moderator subgroups was computed.

2.6.2. Meta-regression

If effects were heterogeneous, meta-regressions were conducted to explore possible reasons for heterogeneity. Each of the following variables was included in separate meta-regressions: age, BMI, sex (coded as female sample only or a mixed sample consisting of males and females), emotion type attempted to induce (coded as 'specific' like anger, sadness or 'general' like negative mood or stress), emotion induction (coded as 'others' emotion display [OED]', 'guided emotion induction [GEI]' or "social feedback [SF]'), and food type offered (coded as 'sweet', 'savoury', or 'sweet and savoury').

3. Results

3.1. Descriptive information

Separate meta-analyses were performed for negative and positive emotions. There were 52 studies for negative emotions (including 3044 participants) and 13 studies for positive emotions (including 768 participants). Nine studies included both positive and negative emotion inductions. Of the 52 studies that induced negative emotions, there were 46 effect sizes for healthy individuals, 8 for samples classified as having an eating disorder, 9 for samples with participants qualified as being overweight or obese, 12 for samples classified as high restrained eaters, 11 for samples classified as low restrained eaters, and 8 effect sizes for samples classified as high and low emotional eaters respectively.

Of the 13 studies that induced positive emotions, there were only four effect sizes for restrained eaters, two for emotional eaters, and one for an eating disordered sample. Given these low numbers, moderator-analyses for positive emotion were not meaningful and only a main effect size was calculated. Table 1 presents the characteristics of all studies included in the database.

3.2. Effect of negative emotions on eating

3.2.1. Main effect

For the main effect of negative emotion on eating the Q-test was statistically significant and the I^2 was high (Deeks et al., 2008), indicating that the variance in the overall main effect was heterogeneous. Therefore, the analysis was conducted as a Random-Effect model. The overall findings illustrated that there was no significant effect for negative emotion on food intake, Hedges's $g = 0.021$, $p = .689$. See Table 2 for details.

To further investigate heterogeneity, meta-regressions were conducted as outlined in section 2.6.2. Emotion induction approached significance in explaining a small part of the variance observed ($n = 52$, $R^2 = 2\%$, $p = .09$), indicating that participants in studies where negative emotion was induced by SF consumed less food than participants where negative emotion was induced by OED, $B = -0.26$, $SE = 0.14$, $p = .06$.

3.2.2. Moderators

For the negative emotions, the mean effect size was calculated for each potential moderator separately (see Table 2). For all analyses, Q-tests were significant and I^2 high, indicating heterogeneity (Deeks et al., 2008). The Random-Effect models showed that there was a medium and statistically significant effect for negative emotion on food intake in restrained eaters, Hedges's $g = 0.740$, $p = .007$, indicating more food intake in the negative emotion condition compared to the neutral condition for restrained eaters (see Fig. 2). To further investigate heterogeneity, meta-regressions illustrated that age explained some of the variance ($n = 8$, $R^2 = 50\%$, $B = 0.26$, $SE = 0.08$, $p = .002$), indicating that older restrained eaters ate more in response to negative emotions than younger restrained eaters.

For all other groups, there were no significant effects of negative emotions on food intake. This applied to healthy controls (Fig. 3), eating disordered individuals (Fig. 4), overweight/obese individuals (Fig. 5), low restrained eaters (Fig. 6), and high vs. low emotional eaters (Fig. 7a vs. b).

To further explore reasons for heterogeneity in these groups, meta-regressions were conducted. Results revealed that in different groups different variables explained some variance. Care should be taken in interpreting these findings, however, as the amount of studies for the subgroups was relatively low.

In the *eating disordered sample*, emotion induction approached significance in explaining some variance ($n = 8$, $R^2 = 19\%$, $p = .06$), indicating that eating disordered participants in studies where negative emotion was induced by SF like a stress test ($B = -0.80$, $SE = 0.38$, $p = .03$) and GEI like recalling an emotional event ($B = -0.84$, $SE = 0.39$, $p = .03$) consumed less food than eating disordered participants where emotions were induced by OED like film excerpts. It is important to note that there were only two studies with OED, and three studies for both SF and GEI. Also food type explained some of the variance ($n = 8$, $B = -0.61$, $SE = 0.17$, $p < .001$, $R^2 = 85\%$), indicating that studies that offered eating disordered samples both sweet and savoury food resulted in less food intake in response to negative emotions compared to studies that offered these samples sweet foods only.

In individuals scoring *high on emotional eating*, age explained some variance ($n = 8$, $R^2 = 35\%$, $B = 0.14$, $SE = 0.06$, $p = .03$), indicating that older emotional eaters ate more in response to negative emotions than younger emotional eaters. Also sex explained some variance ($n = 8$, $R^2 = 64\%$, $B = 0.79$, $SE = 0.31$, $p = .01$), indicating that studies with mixed samples ate more food intake than studies with females only. Please note however, that there were only two studies including mixed, thus male and female, participants. Also in individuals scoring *low on emotion eating*, age explained some variance, indicating that older low emotional eaters ate less in response to negative emotions than younger low emotional eaters, ($n = 8$, $R^2 = 99\%$, $B = -0.12$, $SE = 0.04$, $p = .002$).

In individuals *low in restrained eating*, emotion induction explained some of the variance ($n = 11$, $R^2 = 2\%$, $p = .04$), indicating that emotion induction by GEI resulted in more food intake in low restrained eaters compared to emotion induction by OED ($B = 1.48$, $SE = 0.65$, $p = .02$). It is important to note that there were only two studies with GEI and four studies with OED.

Table 1
Characteristics of the studies including: Author(s) and publication year; Sample with number of participants per study (N), percentage of sex, and type (Students or General Population); the investigated Subgroups; BMI; Emotion induction procedure (others' emotional display, guided emotion induction, or SF); Emotion type induced (specific or general); Valence of the induced emotion; and Food type that was assessed (sweet, savoury, or both).

Author(s)	N (sex % male)	Age (*M*)	Subgroups	BMI (*M*)	Emotion induction procedure	Emotion type induced	Valence induced emotion	Food type
Abrahamson and Wunderlich (1972)	33 (100%), S	20.4	Obese	29.7	SF	General	Negative	Savoury
Agras and Telch (1998)	60 (0%), GP	42.7	ED: BED	36.2	GEI	General	Negative	Sweet and savoury
Appelhans (2010)	34 (0%), GP	33.5	Normal weight vs. obese	27.7	SF	General	Negative	Sweet and savoury
Appelhans et al. (2011)	61 (26%), GP	34.6	Normal weight vs. obese	27.6	GEI	Specific	Negative	Sweet and savoury
Aubie and Jarry (2009) Study 1	88 (0%), S	21.4	ED: BED vs. non-BED	26.2	OED	General	Negative	Sweet
Aubie and Jarry (2009) Study 2	114 (0%), S	22.5	ED: BED vs. non-BED	25.5	OED	General	Negative	Sweet
Bongers et al., 2013a	86 (25%), S	21.6	Healthy	22.5	OED	General	Negative and positive	Sweet and savoury
Bongers et al. (2015)	120 (0%), S	20.0	Healthy	22.3	GEI	Specific	Negative	Sweet
Bongers et al. (2016)	42 (0%), S	20.3	Healthy	21.8	GEI	General	Negative and positive	Sweet and savoury
Born et al. (2010)	9 (0%), S	24.3	Healthy	21.5	SF	General	Negative	Sweet and savoury
Cardi (2018)*	30 (0%), GP	25.8	ED: BED and bulimia nervosa	24.3	GEI	General	Positive	Sweet and savoury
Cavallo and Pinto (2001)	60 (0%), S	19.4	Healthy	23.3	OED	General	Negative	Sweet and savoury
Collins and Stafford (2015)	69 (0%), S	20.3	Healthy	–	OED	General	Positive	Sweet and savoury
Cools et al. (1992)	91 (0%), S	28.6	Healthy: high vs. low RS	23.8	OED	General	Negative and positive	Savoury
Dweck et al. (2014)	64 (0%), S	18.8	Healthy	24.5	SF	General	Negative	Sweet and savoury
Emerson and Papa (2016)*	78 (0%), S	22.4	Healthy: high RS	–	GEI	Specific	Negative and positive	Sweet
Epel et al. (2001)	59 (0%), GP	36	Healthy	25.4	SF	General	Negative	Sweet and savoury
Evers et al. (2009) Study 3	37 (0%), S	22.8	Healthy: high vs. low EE	22.2	GEI	Specific	Negative	Sweet and savoury
Evers et al. (2009) Study 4	57 (0%), S	20.8	Healthy: high vs. low EE	21.9	SF	Specific	Negative and positive	Sweet and savoury
Evers et al. (2010) Study 1	37 (0%), S	22.9	Healthy	23.1	GEI	Specific	Negative	Sweet and savoury
Evers et al. (2013) Study 1	68 (24%), S	21.9	Healthy	21.5	OED	Specific	Positive	Sweet and savoury
Evers et al. (2013) Study 2	84 (0%), S	–	Healthy	22.4	GEI	Specific	Negative and positive	Sweet and savoury
Fay and Finlayson (2011)	30 (0%), S	21.7	Healthy	–	GEI	Specific	Negative	Sweet
Frost et al. (1982)	55 (0%), S	–	Healthy: high vs. low RS	–	GEI	General	Negative and positive	Sweet
Grunberg and Straub (1992)	54 (48%), S	24.1	Healthy	–	OED	General	Negative	Sweet and savoury
Habhab et al. (2009)	40 (0%), S	21.4	Healthy	23.2	SF	General	Negative	Sweet and savoury
Haynes et al. (2003)	80 (0%), S	22.5	Healthy: high vs. low RS	22.6	SF	General	Negative	Sweet and savoury
Heatherton et al. (1991)	75 (0%), S	–	Healthy: high vs. low RS	–	SF	Specific	Negative	Sweet
Heatherton et al. (1998) Study 3	41 (0%), S	–	Healthy: high RS	–	GEI	Specific	Negative	Sweet
Kestenbaum (1992)*	76 (50%), S	20.9	Normal vs. overweight	23.5	SF	General	Negative	Sweet
Kuo (2017)*	97 (0%), S	19.0	Healthy	23.5	SF	General	Negative	Sweet
Laessle and Schulz (2009)	48 (0%), GP	34.1	Obese ED: BED vs. obese non-BED	36.7	SF	General	Negative	Sweet
Lemmens et al. (2011)	42 (29.6%), GP	25.6	Normal vs. overweight	22.4	SF	General	Negative	Sweet and savoury
Levine and Marcus (1997)	40 (0%), S	18.6	ED: bulimic symptomatology vs. non-ED	22.6	SF	General	Negative	Sweet and savoury
Lowe and Maycock (1988)	60 (0%), S	–	Healthy: high vs. low RS	–	GEI	General	Negative	Sweet
Munsch et al. (2008)	69 (0%), GP	36.7	ED: BED	32.8	GEI	General	Negative	Sweet
Oliver et al. (2000)	68 (39.7%), S	26.0	Healthy: high vs. low RS and high vs. low EE	22.1	SF	General	Negative	Sweet and savoury
Polivy et al. (1994)	96 (0%), S	–	Healthy: high vs. low RS	–	SF	Specific	Negative	Sweet
Royal and Kurtz (2010)	52 (9%), S	19.3	Healthy	21.6	SF	General	Negative	Sweet and savoury
Rutters et al. (2009)	129 (50.4%), GP	27.6	Healthy	24.5	SF	General	Negative	Sweet and savoury
Schneider et al. (2010)	61 (26%), GP	34.6	Obese vs. lean	27.6	GEI	Specific	Negative	Sweet and savoury
Schotte et al. (1990)	60 (0%), S	29.6	Healthy: high vs. low RS	23.9	OED	General	Negative	Savoury
Schulz and Laessle (2012)	71 (0%), GP	34.0	Obese ED: BED & obese non-ED	37.0	SF	General	Negative	Sweet
Sheppard-Sawyer et al. (2000)	31 (0%), S	19.4	Healthy: high vs. low RS	–	OED	Specific	Negative	Savoury
Sproesser et al. (2014)	251 (29%), S	24	Healthy: high vs. low EE	23	SF	Specific	Negative and positive	Sweet
Telch and Agras (1996)	60 (0%), GP	44.3	Obese ED: BED vs. obese non-ED	33.4	GEI	General	Negative	Sweet and savoury
Turner et al. (2010)	106 (30%), S	23.5	Healthy	23.0	OED	General	Positive	Sweet
Van Strien and Ouwens (2007)	86 (0%), S	21.1	Healthy	36.7	GEI	General	Negative	Savoury
Van Strien et al. (2012) Study 1	45 (0%), S	21.8	Healthy: high vs. low EE	23.3	OED	Specific	Negative	Sweet and savoury
Van Strien et al. (2012) Study 2	47 (0%), S	19.0	Healthy: high vs. low EE	21.3	SF	Specific	Negative	Sweet
Van Strien et al. (2014)	54 (0%), S	20.1	Healthy: high vs. low EE	20.3	SF	Specific	Negative	Sweet
Wallis and Hetherington (2004)	38 (0%), S	21.7	Healthy: high vs. low EE & high vs. low RS	24.8	SF	General	Negative	Sweet

(*continued on next page*)

Table 1 (*continued*)

Author(s)	N (sex % male)	Age (*M*)	Subgroups	BMI (*M*)	Emotion induction procedure	Emotion type induced	Valence induced emotion	Food type
Wallis and Hetherington (2009)	26 (0%), GP	27.6	Healthy	24.3	SF	Specific	Negative	Sweet
Werthmann et al. (2014)	85 (0%), GP	20.7	Healthy: high vs. low EE	21.9	GEI	General	Negative	Sweet
Wildes et al. (2012)	28 (0%), GP	32.4	Healthy	17.4	OED	General	Negative	Sweet
Yeomans and Coughlan (2009)	96 (0%), S	22.1	Healthy: high vs. low RS	22.5	OED	General	Negative and positive	Sweet and savoury

Note. S = Student sample; GP = General Population sample; ED = Eating Disordered; BED = Binge Eating Disordered; RS = Restrained Eaters; EE = Emotional Eaters; OED = Others' Emotional Display; GEI = Guided Emotion Induction; SF = Social Feedback. 'Healthy' subgroups refer to non-eating disordered and non-obese samples; in some studies these are divided into high vs. low restrained or emotional eaters. Author names followed by * refer to unpublished data.

Table 2
Statistical Data of the Analyses.

			CI 95%			Heterogeneity		
Model	*k*	H'g	Min	Max	*p*	*Q*	*p*	I^2
RE main effect								
Negative emotions	52	0.021	−0.082	0.125	.69	258.26	< .001	80.27
Positive emotions	13	0.237	0.009	0.466	.04	41.73	< .001	71.4
RE Moderators (for negative emotions only)								
Healthy sample	46	0.015	−0.103	0.134	.80	226.27	< .001	80.22
Eating disordered	8	0.112	−0.167	0.392	.43	29.83	< .001	76.53
Obese & overweight	9	0.033	−0.287	0.221	.80	30.37	< .001	73.66
High restrained eaters	12	0.740	0.206	1.273	.01	96.74	< .001	88.63
Low restrained eaters	11	0.247	−0.673	0.178	.26	55.33	< .001	81.93
High emotional eaters	8	0.096	−0.405	0.213	.54	20.10	.005	65.18
Low emotional eaters	8	0.026	−0.285	0.234	.85	15.36	.032	54.44

Note. RE = Random-Effect Model; *k*: number of studies; H'g = Hedges's g.

3.3. Effect of positive emotions on eating

3.3.1. Main effect

For the main effect of positive emotion on eating the Q-test was also statistically significant and the I^2 was high, indicating heterogeneity (Deeks et al., 2008). The Random-Effect model indicated that there was a small, but statistically significant general main effect for positive emotion on food intake, Hedges´s g = 0.237, p = .042, indicating more food intake in the positive emotion condition compared to the neutral condition. See Table 2 for details and see Fig. 8 for the forest plot.

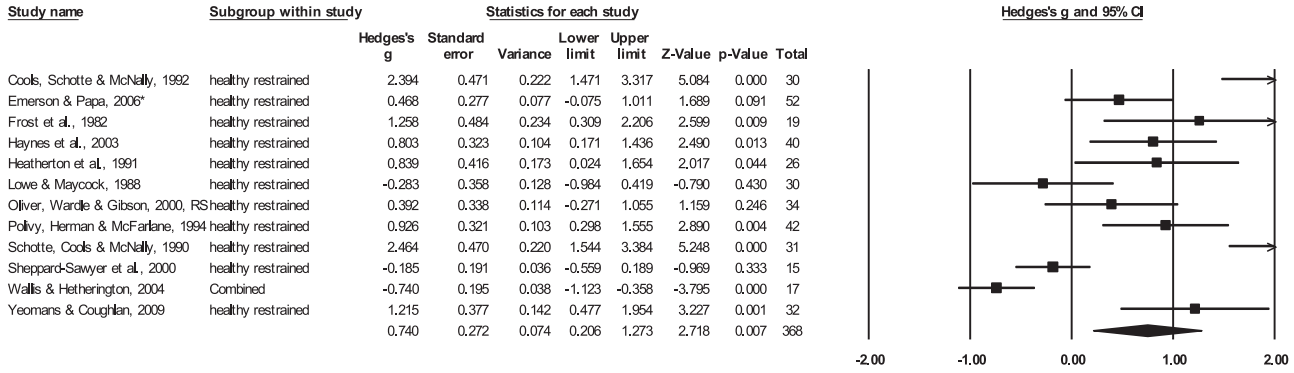
To further investigate heterogeneity, meta-regressions were conducted. The types of food offered explained some variance observed (n = 13, R^2 = 54%, p = .002), indicating that when positive emotion was induced less food consumption was consumed in studies where only sweet foods were offered compared to studies where both sweet and savoury foods were offered (B = −0.70, SE = 0.20, p = .001).

3.4. Publication bias

In order to assess whether the results presented above were subjected to publication bias of the included studies, the effect sizes were visualized in funnel plots by their weight in the analyses (the inverse variance). Panel A of Fig. 9 shows the funnel plot for the main effect of negative emotion on eating behavior; Panel B for positive emotion. Both plots show a variety of studies with a Hedges' g < 0 and > 0, indicating no publication bias with regard to the included studies (Lipsey and Wilson, 2001). Also directly comparing effect sizes between published and unpublished studies revealed that publication status did not significantly explain variance observed: neither for negative emotion (n = 52 [3 unpublished and 49 published studies], R^2 = 0%, B = .06, SE = .24, p = .81) nor for positive emotion (n = 13 [2 unpublished and 11 published studies], R^2 = 0%, B = .07, SE = .33, p = .83).

4. Discussion

Since the late 1950 s, several theories have evolved about the effect that emotions may or may not have on people's eating behavior (Macht, 2008). The present meta-analysis was an attempt to integrate the findings from empirical studies that induced negative and/or positive emotions in the laboratory and subsequently investigated the effect on eating behavior in various groups, ranging from healthy controls and restrained eaters to eating disordered individuals. The meta-analyses found a medium effect of negative emotion on food intake in restrained



Study name	Subgroup within study	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value	Total
Cools, Schotte & McNally, 1992	healthy restrained	2.394	0.471	0.222	1.471	3.317	5.084	0.000	30
Emerson & Papa, 2006*	healthy restrained	0.468	0.277	0.077	-0.075	1.011	1.689	0.091	52
Frost et al., 1982	healthy restrained	1.258	0.484	0.234	0.309	2.206	2.599	0.009	19
Haynes et al., 2003	healthy restrained	0.803	0.323	0.104	0.171	1.436	2.490	0.013	40
Heatherton et al., 1991	healthy restrained	0.839	0.416	0.173	0.024	1.654	2.017	0.044	26
Lowe & Maycock, 1988	healthy restrained	-0.283	0.358	0.128	-0.984	0.419	-0.790	0.430	30
Oliver, Wardle & Gibson, 2000, RS	healthy restrained	0.392	0.338	0.114	-0.271	1.055	1.159	0.246	34
Polivy, Herman & McFarlane, 1994	healthy restrained	0.926	0.321	0.103	0.298	1.555	2.890	0.004	42
Schotte, Cools & McNally, 1990	healthy restrained	2.464	0.470	0.220	1.544	3.384	5.248	0.000	31
Sheppard-Sawyer et al., 2000	healthy restrained	-0.185	0.191	0.036	-0.559	0.189	-0.969	0.333	15
Wallis & Hetherington, 2004	Combined	-0.740	0.195	0.038	-1.123	-0.358	-3.795	0.000	17
Yeomans & Coughlan, 2009	healthy restrained	1.215	0.377	0.142	0.477	1.954	3.227	0.001	32
		0.740	0.272	0.074	0.206	1.273	2.718	0.007	368

Fig. 2. Statistics and forest plot for eating behavior amongst the restrained samples in the neutral vs. negative emotion condition for each study. Effect sizes are reported as Standard error. Total refers to sample size.

eaters, and a small overall effect of positive emotion on food intake. For all other subgroups, no significant emotion-eating relationships were found. Below the findings and their implications specified for each subgroup are considered.

4.1. Negative emotion

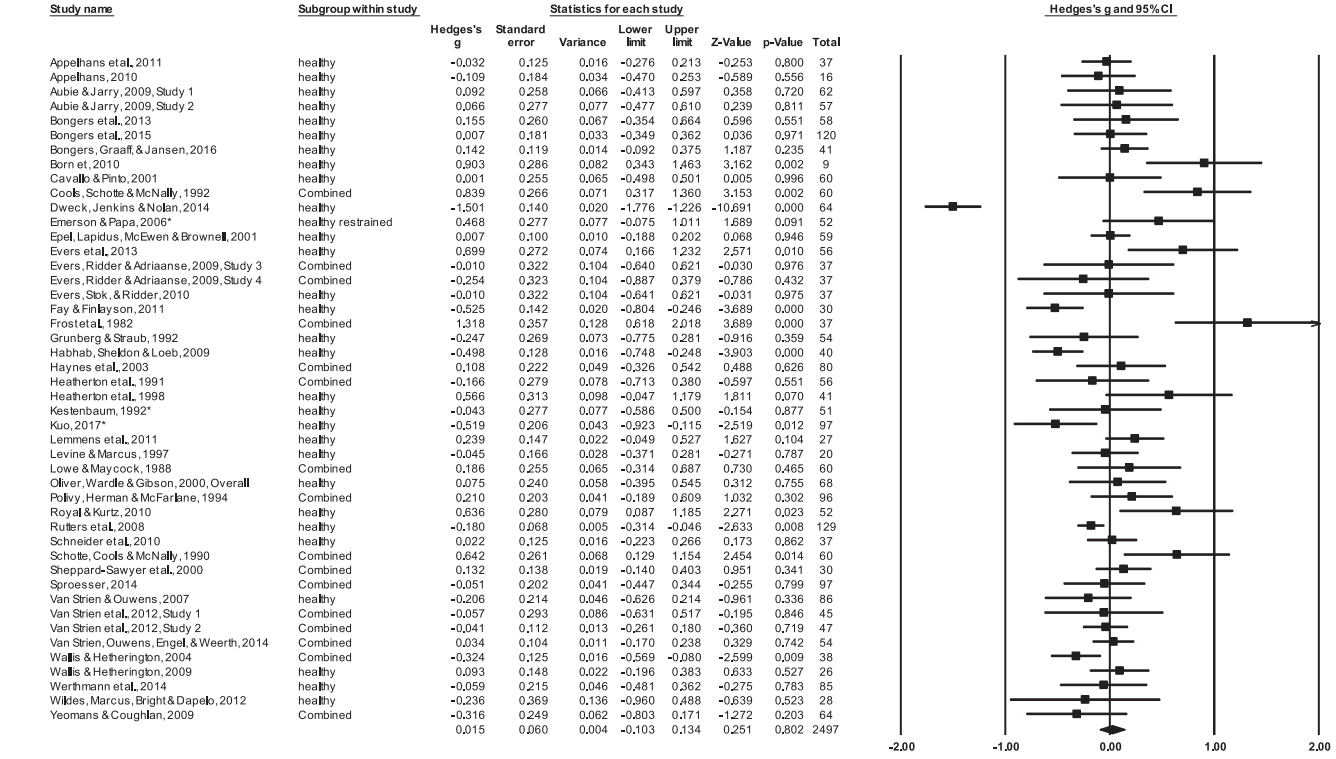
Overall, negative emotions did not cause any changes in eating behavior. Effects were heterogeneous, however, and some of the variance observed was explained by differences in the emotion induction procedures. The absence of an overall effect for negative emotion on eating may not be surprising, however, as it has been postulated that negative emotions particularly affect eating behavior in certain subgroups. The findings for subgroups will be outlined below.

4.1.1. Restrained eaters

In line with the persisting theory that emotions increase food intake in restrained eaters (e.g., Greeno and Wing, 1994; Macht, 2008) our meta-analysis revealed that restrained eaters increased food intake in response to negative emotions. This effect was of medium size, nearly large, and provided additional support for the idea that negative emotions impair the cognitive control of restrained eaters, resulting in disinhibition.

This negative emotion-eating relationship in restrained eating replicated the finding in the meta-analysis of Cardi et al. (2015). Their effect was small rather than medium-to-large, which likely results from the current work including twice as many articles on restrained eating samples, with the current effect size thus being based on a more robust point estimate.

Findings further revealed that the effect sizes for the restrained eater samples were heterogeneous. From a theoretical viewpoint this heterogeneity is not surprising considering the ongoing debate about what restrained eating precisely entails. That is, restrained eating is not always reflected in actual restrictive behavior (e.g., Stice et al., 2004, 2007; Tomiyama et al., 2009). Consequently, it has been questioned what it precisely means when individuals score high on dietary



Study name	Subgroup within study	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value	Total
Appelhans et al., 2011	healthy	-0.032	0.125	0.016	-0.276	0.213	-0.253	0.800	37
Appelhans, 2010	healthy	-0.109	0.184	0.034	-0.470	0.253	-0.589	0.556	16
Aubie & Jarry, 2009, Study 1	healthy	0.092	0.258	0.066	-0.413	0.597	0.358	0.720	62
Aubie & Jarry, 2009, Study 2	healthy	0.066	0.277	0.077	-0.477	0.610	0.239	0.811	57
Bongers et al., 2013	healthy	0.155	0.260	0.067	-0.354	0.664	0.596	0.551	58
Bongers et al., 2015	healthy	0.007	0.181	0.033	-0.349	0.362	0.036	0.971	120
Bongers, Graaff, & Jansen, 2016	healthy	0.142	0.119	0.014	-0.092	0.375	1.187	0.235	41
Born et, 2010	healthy	0.903	0.286	0.082	0.343	1.463	3.162	0.002	9
Cavallo & Pinto, 2001	healthy	0.001	0.255	0.065	-0.498	0.501	0.005	0.996	60
Cools, Schotte & McNally, 1992	Combined	0.839	0.266	0.071	0.317	1.360	3.153	0.002	60
Dweck, Jenkins & Nolan, 2014	healthy	-1.501	0.140	0.020	-1.776	-1.226	-10.691	0.000	64
Emerson & Papa, 2006*	healthy restrained	0.468	0.277	0.077	-0.075	1.011	1.689	0.091	52
Epel, Lapidus, McEwen & Brownell, 2001	healthy	0.007	0.100	0.010	-0.188	0.202	0.068	0.946	59
Evers et al., 2013	healthy	0.699	0.272	0.074	0.166	1.232	2.571	0.010	56
Evers, Ridder & Adriaanse, 2009, Study 3	Combined	-0.010	0.322	0.104	-0.640	0.621	-0.030	0.976	37
Evers, Ridder & Adriaanse, 2009, Study 4	Combined	-0.254	0.323	0.104	-0.887	0.379	-0.786	0.432	37
Evers, Stok, & Ridder, 2010	healthy	-0.010	0.322	0.104	-0.641	0.621	-0.031	0.975	37
Fay & Finlayson, 2011	healthy	-0.525	0.142	0.020	-0.804	-0.246	-3.689	0.000	30
Frost et al., 1982	Combined	1.318	0.357	0.128	0.618	2.018	3.689	0.000	37
Grunberg & Straub, 1992	healthy	-0.247	0.269	0.073	-0.775	0.281	-0.916	0.359	54
Habhab, Sheldon & Loeb, 2009	healthy	-0.498	0.128	0.016	-0.748	-0.248	-3.903	0.000	40
Haynes et al., 2003	Combined	0.108	0.222	0.049	-0.326	0.542	0.488	0.626	80
Heatherton et al., 1991	Combined	-0.166	0.279	0.078	-0.713	0.380	-0.597	0.551	56
Heatherton et al., 1998	healthy	0.566	0.313	0.098	-0.047	1.179	1.811	0.070	41
Kestenbaum, 1992*	healthy	-0.043	0.277	0.077	-0.586	0.500	-0.154	0.877	51
Kuo, 2017*	healthy	-0.519	0.206	0.043	-0.923	-0.115	-2.519	0.012	97
Lemmens et al., 2011	healthy	0.239	0.147	0.022	-0.049	0.527	1.627	0.104	27
Levine & Marcus, 1997	healthy	-0.045	0.166	0.028	-0.371	0.281	-0.271	0.787	20
Lowe & Maycock, 1988	Combined	0.186	0.255	0.065	-0.314	0.687	0.730	0.465	60
Oliver, Wardle & Gibson, 2000, Overall	healthy	0.075	0.240	0.058	-0.395	0.545	0.312	0.755	68
Polivy, Herman & McFarlane, 1994	Combined	0.210	0.203	0.041	-0.189	0.609	1.032	0.302	96
Royal & Kurtz, 2010	healthy	0.636	0.280	0.079	0.087	1.185	2.271	0.023	52
Rutters et al., 2008	healthy	-0.180	0.068	0.005	-0.314	-0.046	-2.633	0.008	129
Schneider et al., 2010	healthy	0.022	0.125	0.016	-0.223	0.266	0.173	0.862	37
Schotte, Cools & McNally, 1990	Combined	0.642	0.261	0.068	0.129	1.154	2.454	0.014	60
Sheppard-Sawyer et al., 2000	Combined	0.132	0.138	0.019	-0.140	0.403	0.951	0.341	30
Sproesser, 2014	Combined	-0.051	0.202	0.041	-0.447	0.344	-0.255	0.799	97
Van Strien & Ouwens, 2007	healthy	-0.206	0.214	0.046	-0.626	0.214	-0.961	0.336	86
Van Strien et al., 2012, Study 1	Combined	-0.057	0.293	0.086	-0.631	0.517	-0.195	0.846	45
Van Strien et al., 2012, Study 2	Combined	-0.041	0.112	0.013	-0.261	0.180	-0.360	0.719	47
Van Strien, Ouwens, Engel, & Weerth, 2014	Combined	0.034	0.104	0.011	-0.170	0.238	0.329	0.742	54
Wallis & Hetherington, 2004	Combined	-0.324	0.125	0.016	-0.569	-0.080	-2.599	0.009	38
Wallis & Hetherington, 2009	healthy	0.093	0.148	0.022	-0.196	0.383	0.633	0.527	26
Werthmann et al., 2014	healthy	-0.059	0.215	0.046	-0.481	0.362	-0.275	0.783	85
Wildes, Marcus, Bright & Dapelo, 2012	healthy	-0.236	0.369	0.136	-0.960	0.488	-0.639	0.523	28
Yeomans & Coughlan, 2009	Combined	-0.316	0.249	0.062	-0.803	0.171	-1.272	0.203	64
		0.015	0.060	0.004	-0.103	0.134	0.251	0.802	2497

Fig. 3. Statistics, sample size and forest plot for eating behavior amongst the healthy samples in the neutral vs. negative emotion condition for each study. Effect sizes are reported as Standard error. Total refers to sample size.

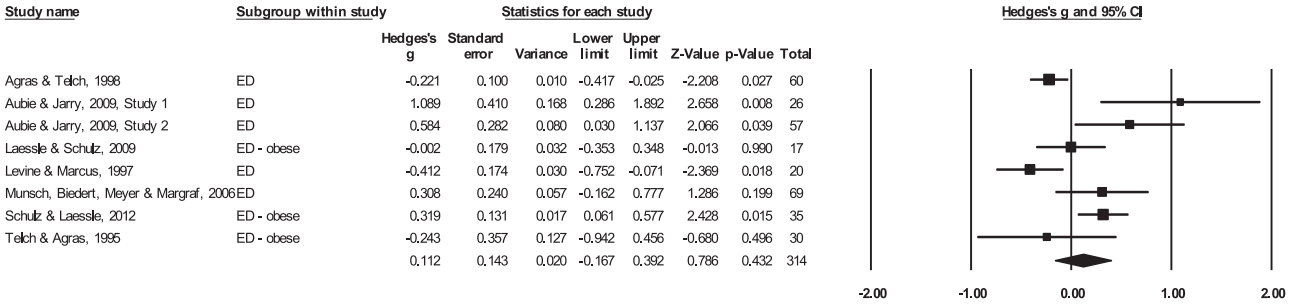


Fig. 4. Statistics, sample size and forest plot for eating behavior amongst the eating disordered samples in the neutral vs. negative emotion condition for each study. Effect sizes are reported as Standard error. Total refers to sample size.

restraint. For example, restrained eaters may cognitively *want* to restrict their food intake or overeating tendencies, yet they may not be successful in doing so (De Witt Huberts et al., 2013). Conversely, dietary restraint has also been linked with positive outcomes (see for a review Schaumberg et al., 2016), implying that for some individuals dietary restraint has been a beneficial strategy to control their weight and food intake. Relatedly, restrained eating is seen as a necessary prerequisite for some individuals, for example for those partaking in weight loss interventions, but also a risk factor for developing eating pathology and obesity over time (Schaumberg et al., 2016).

Results further pointed towards heterogeneity resulting from age differences across the restrained samples, with older restrained eaters showing stronger increased eating in response to negative emotions than younger restrained eaters. Based on the idea that restrained status is relatively stable across time (Neumark-Sztainer et al., 2011), older restrained eaters may already try to cognitively control their eating behavior for a long time, which is difficult and effortful, such that they may be more vulnerable for disinhibitors like negative emotions than their younger counterparts. This assumption is speculative and needs to be corroborated by empirical evidence, but the finding that age may be important in relation to emotion-induced eating in restrained eaters is an interesting finding that warrants future research.

4.1.2. Eating disorders

The current findings revealed that individuals with an eating disorder showed unaffected eating behavior in response to negative emotion in laboratory settings. This finding is unexpected, as several cross-sectional, experimental, and therapy outcome studies suggest an association between negative affect and binge eating behavior (Dingemans et al., 2017). Additionally, Cardi et al.'s meta-analysis (2015) found that negative emotions triggered increased eating in binge eaters. Whereas their findings were based on three studies only, the current findings were derived from eight studies, and thus incorporate a more powerful foundation for the obtained effects. Overall, however, there were relatively few studies with clinical samples that assessed emotion-related eating in the laboratory and the effect sizes were heterogeneous.

Heterogeneity could partly be explained by varieties in emotion inductions and food types offered, indicating that researchers studying emotion-related eating in clinical samples should pay careful attention in selecting their emotion induction and the food types they offer. It is hard, however, to further unravel such findings as these findings were based on a small set of studies. Moreover, the clinical samples that were incorporated in the analysis included mostly people with binge eating disorder (BED: e.g., Aubie and Jarry, 2009), but amongst people with BED there are subtypes that may vary substantially in the emotion-eating relationship and this may thus be an additional source for heterogeneity. For example, individuals with the dietary-depressive BED subtype (as opposed to those with the dietary-only subtype: e.g., Stice et al., 2001) are particularly prone to show an association between depressive symptoms, acute sad mood, and binge eating behavior and research has revealed that higher levels of depression are related to more severe binge eating (see for a review Dingemans et al., 2017). Such nuances in subtypes or depression, however, could unfortunately not be tackled by the present meta-analyses.

4.1.3. Overweight and obesity

The current findings revealed that being overweight or obese was not associated with emotion-related changes in eating behavior. Although obese people were originally assumed to be typical emotional eaters (Schachter et al., 1968), the present findings are in line with Greeno and Wing's (1994) systematic review and Cardi et al.'s meta-analysis (2015) that pointed towards weight status not being indicative for emotions affecting eating behavior. Additionally, across all subgroups BMI did not explain any heterogeneity, which supports the idea that weight status is not related to emotion-induced eating. It is important to note that the amount of studies including overweight or obese individuals was small (n = 8).

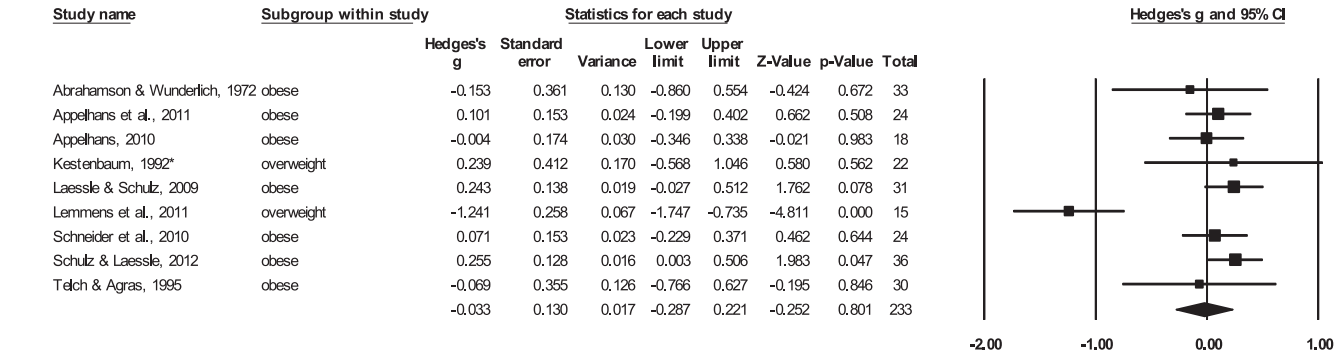


Fig. 5. Statistics, sample size and forest plot for eating behavior amongst the obese and overweight samples (non-eating disordered) in the neutral vs. negative emotion condition for each study. Effect sizes are reported as Standard error. Total refers to sample size.

Study name	Subgroup within study	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value	Total
Cools, Schotte & McNally, 1992	healthy unrestrained	-0.545	0.362	0.131	-1.255	0.165	-1.506	0.132	30
Frost et al., 1982	healthy unrestrained	1.607	0.525	0.276	0.578	2.636	3.060	0.002	18
Haynes et al., 2003	healthy unrestrained	-0.689	0.319	0.102	-1.315	-0.063	-2.157	0.031	40
Heatherton et al., 1991	healthy unrestrained	-1.203	0.408	0.166	-2.002	-0.405	-2.953	0.003	30
Lowe & Maycock, 1988	healthy unrestrained	0.524	0.362	0.131	-0.187	1.234	1.445	0.149	30
Oliver, Wardle & Gibson, 2000, RS	healthy unrestrained	-0.198	0.336	0.113	-0.856	0.460	-0.591	0.555	34
Polivy, Herman & McFarlane, 1994	healthy unrestrained	-0.443	0.272	0.074	-0.976	0.089	-1.631	0.103	54
Schotte, Cools & McNally, 1990	healthy unrestrained	-0.212	0.362	0.131	-0.922	0.498	-0.586	0.558	29
Sheppard-Sawyer et al., 2000	healthy unrestrained	0.483	0.201	0.040	0.089	0.877	2.402	0.016	15
Wallis & Hetherington, 2004	Combined	-0.036	0.162	0.026	-0.354	0.282	-0.221	0.825	21
Yeomans & Coughlan, 2009	healthy unrestrained	-1.964	0.423	0.179	-2.793	-1.134	-4.641	0.000	32
		-0.247	0.217	0.047	-0.673	0.178	-1.139	0.255	333

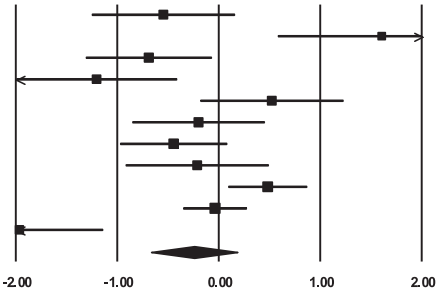


Fig. 6. Statistics, sample size and forest plot for eating behavior amongst the low restrained samples in the neutral vs. negative emotion condition for each study. Effect sizes are reported as Standard error. Total refers to sample size.

4.1.4. Emotional eaters

The individual difference measure 'emotional eating' refers to scores on emotional eater scales that assess the extent to which individuals judge themselves as emotional eaters. An example items is: 'Are you inclined to eat when you feel angry?' (Van Strien et al., 1986). To the best of our knowledge, it is the first time that this measure is part of a meta-analysis on emotion-induced eating. It has commonly been assumed that negative emotions increase food intake in people who use eating as means to regulate their negative emotions, also labeled as emotional eaters (Macht, 2008). However, the current findings revealed that negative emotions did not impact eating behavior amongst self-reported emotional eaters. In other words, assessing oneself as an emotional eater did not translate to increased eating when feeling negative. This finding is in itself unexpected as one would assume that high scores on these emotional eating scales translate into the behavior that is assessed, thus eating when experiencing negative emotions. Previously however, several studies already revealed that emotional eating scales are relatively poor in predicting actual food intake (e.g., Bongers et al., 2013a,b; Evers et al., 2009) as these scales involve multiple sources of recall bias (see also Baumeister et al., 2007) and thus may lack predictive validity (Bongers and Jansen, 2016). The findings of the present meta-analysis are in agreement to this conclusion and collectively demonstrate the urgency for scholars to develop alternative means to assess individual differences in emotional eating (see also Bongers et al., 2013b).

4.2. Positive emotion

In line with Cardi et al.'s meta-analysis (2015) the current findings revealed that positive emotions had a small effect on eating behavior, with positive emotions resulting in increased consumption. This is in line with the observation that across cultures food has been used to highlight the celebration of special occasions like weddings and birthdays that are generally accompanied by positive emotions (Rozin, 1999). Compared to negative emotions, however, positive emotions have hardly been investigated in relation to its impact on eating behavior. Future research is needed to further assess the underlying mechanism and to assess the extent to which certain individuals, like restrained eaters, are particularly vulnerable for increased eating in response to positive emotions.

4.3. Implications, limitations, and future directions

The current findings have several important implications. An asset of the current study being amongst the first meta-analyses on the causal effect of emotions on eating behavior (see also Cardi et al., 2015), is that it gives collective insight into emotion-related eating in both the disordered and non-disordered population, which is a welcome addition

Panel A.

Study name	Subgroup within study	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value	Total
Evers, Ridder & Adriaanse, 2009, Study 3	healthy emo eater	-0.336	0.442	0.196	-1.203	0.531	-0.760	0.447	19
Evers, Ridder & Adriaanse, 2009, Study 4	healthy emo eater	-0.365	0.464	0.216	-1.275	0.545	-0.786	0.432	18
Oliver, Wardle & Gibson, 2000, EE	healthy emo eater	0.438	0.339	0.115	-0.227	1.102	1.291	0.197	34
Sproesser, 2014	healthy emo eater	0.550	0.346	0.120	-0.130	1.229	1.586	0.113	33
Van Strien et al., 2012, Study 1	healthy emo eater	0.769	0.426	0.182	-0.066	1.605	1.804	0.071	22
Van Strien et al., 2012, Study 2	healthy emo eater	-0.373	0.158	0.025	-0.684	-0.062	-2.354	0.019	24
Van Strien, Ouwens, Engel, & Weerth, 2014	healthy emo eater	-0.203	0.141	0.020	-0.480	0.074	-1.435	0.151	29
Wallis & Hetherington, 2004	Combined	-0.630	0.185	0.034	-0.992	-0.268	-3.408	0.001	18
		-0.096	0.158	0.025	-0.405	0.213	-0.610	0.542	197

Panel B.

Study name	Subgroup within study	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value	Total
Evers, Ridder & Adriaanse, 2009, Study 3	healthy low emo eater	0.291	0.452	0.204	-0.594	1.176	0.644	0.520	18
Evers, Ridder & Adriaanse, 2009, Study 4	healthy low emo eater	-0.151	0.445	0.198	-1.023	0.720	-0.340	0.734	19
Oliver, Wardle & Gibson, 2000, EE	healthy low emo eater	-0.168	0.336	0.113	-0.825	0.490	-0.500	0.617	34
Sproesser, 2014	healthy low emo eater	-0.474	0.252	0.063	-0.967	0.019	-1.885	0.059	64
Van Strien et al., 2012, Study 1	healthy low emo eater	-0.820	0.420	0.176	-1.642	0.003	-1.953	0.051	23
Van Strien et al., 2012, Study 2	healthy low emo eater	0.297	0.160	0.025	-0.016	0.609	1.859	0.063	23
Van Strien, Ouwens, Engel, & Weerth, 2014	healthy low emo eater	0.315	0.154	0.024	0.013	0.617	2.047	0.041	25
Wallis & Hetherington, 2004	Combined	-0.068	0.169	0.029	-0.400	0.264	-0.401	0.688	20
		-0.026	0.132	0.017	-0.285	0.234	-0.194	0.846	226

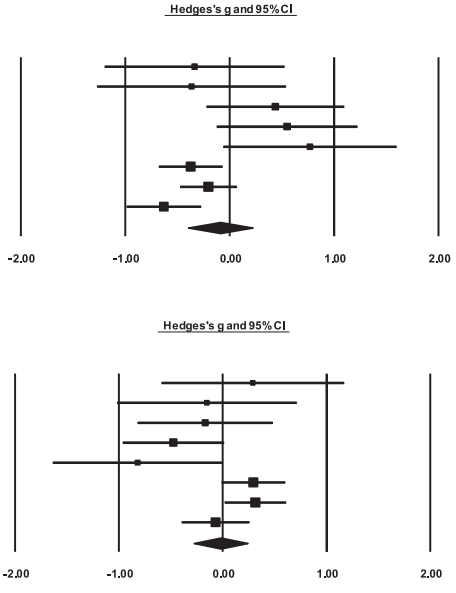
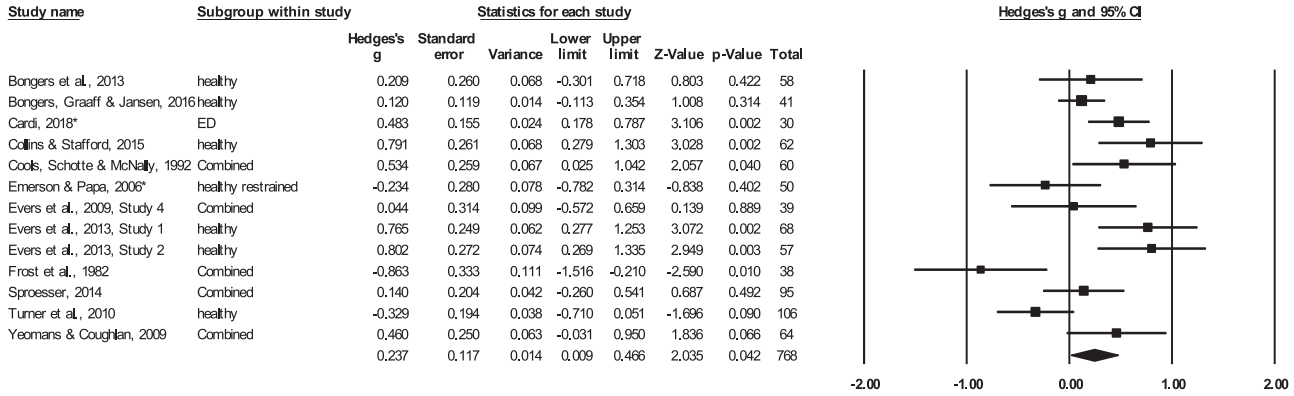


Fig. 7. Statistics, sample size and forest plot for eating behavior amongst the high (Panel A) and low (Panel B) emotional eaters in the neutral vs. negative emotion condition for each study. Effect sizes are reported as Standard error. Total refers to sample size.



Study name	Subgroup within study	Hedges's g	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value	Total
Bongers et al., 2013	healthy	0.209	0.260	0.068	-0.301	0.718	0.803	0.422	58
Bongers, Graaff & Jansen, 2016	healthy	0.120	0.119	0.014	-0.113	0.354	1.008	0.314	41
Cardi, 2018*	ED	0.483	0.155	0.024	0.178	0.787	3.106	0.002	30
Collins & Stafford, 2015	healthy	0.791	0.261	0.068	0.279	1.303	3.028	0.002	62
Cools, Schotte & McNally, 1992	Combined	0.534	0.259	0.067	0.025	1.042	2.057	0.040	60
Emerson & Papa, 2006*	healthy restrained	-0.234	0.280	0.078	-0.782	0.314	-0.838	0.402	50
Evers et al., 2009, Study 4	Combined	0.044	0.314	0.099	-0.572	0.659	0.139	0.889	39
Evers et al., 2013, Study 1	healthy	0.765	0.249	0.062	0.277	1.253	3.072	0.002	68
Evers et al., 2013, Study 2	healthy	0.802	0.272	0.074	0.269	1.335	2.949	0.003	57
Frost et al., 1982	Combined	-0.863	0.333	0.111	-1.516	-0.210	-2.590	0.010	38
Sproesser, 2014	Combined	0.140	0.204	0.042	-0.260	0.541	0.687	0.492	95
Turner et al., 2010	healthy	-0.329	0.194	0.038	-0.710	0.051	-1.696	0.090	106
Yeomans & Coughlan, 2009	Combined	0.460	0.250	0.063	-0.031	0.950	1.836	0.066	64
		0.237	0.117	0.014	0.009	0.466	2.035	0.042	768

Fig. 8. Statistics, sample size and forest plot for eating behavior in the neutral vs. positive emotion condition for each study. Effect sizes are reported as Standard error. Total refers to sample size.

to the many separate studies within this domain. The current work suggests that particularly restrained eaters, despite or due to their cognitive effort to eat less (Schaumberg et al., 2016), are vulnerable to increased eating in response to negative emotions and that positive emotion also triggers increased eating.

For all other subgroups, including healthy controls, obese or overweight people, and individuals assessing themselves as emotional eaters, negative emotions did not affect food intake in laboratory settings. This zero-finding for these subgroups is a relevant finding, as it may signal scholars in the field that the power to detect emotion-eating related patterns in the laboratory across these subgroups may be negligible unless novel moderators are revealed. Hence, it may urge scholars to innovate by looking beyond the existing postulated moderators and to collaborate with scholars from other disciplines.

Despite the implications of these findings, it is also likely that important confounding factors have emerged that affected the present findings. An example is that studies assessing moderators continuously, such as individual differences in restrained or emotional eating as in a regression analysis with interaction terms, could not be captured. On another level, recent research has revealed that it is not necessarily the emotion per se that is responsible for increased eating, but the emotion regulation strategies that people use to deal with these emotions, such as suppression or reappraisal (Evers et al., 2010; Taut et al., 2012; Vandewalle et al., 2014). Such findings illustrate that people may have prejudiced ideas about their emotions triggering eating, while the way of dealing with their emotions may factually be the underlying trigger. Future studies manipulating a broad variety of different emotion regulation strategies would provide important additional evidence.

Another implication of the current meta-analysis is situated in its disclosure that despite the abundant amount of empirical attention on the topic of emotion-related eating, more qualitatively strong experimental studies are still needed to circumvent the shortcomings of the prevailing research on this topic. This need for strong experimental studies is reflected by the finding that the moderators were hindered by heterogeneity; by the fact that of the many articles on this topic only 52 articles could be included in the database because they met the basic inclusion criteria; and by the observation that many studies could not be included because they assessed eating behavior by self-reports or retrospective measures. Self-reported eating behavior is sensitive for bias and there is considerable evidence that in general retrospective measures lead to a serious underreport of food intake (Stice et al., 2004). For this reason, the current meta-analysis only included studies that observed eating behavior in the laboratory. Despite the advantage that such eating measures are relatively more objective, they are limited in their ecological validity.

Accordingly, field studies examining people's daily affective experiences and eating behaviors in their natural environment (Smyth et al., 2001; Wheeler and Reis, 1991) form a valuable addition to laboratory studies in understanding the emotion-eating relationship. The field has lately been increasingly enriched by such daily life studies. To illustrate, a meta-analysis on daily life studies within eating disordered samples, found that negative affect was elevated prior to binge episodes compared to the average levels of negative affect and compared to the level of negative affect prior to regular eating episodes (Haedt-Matt and Keel, 2011). It would be important for future research to conduct a similar meta-analysis on daily life studies, but enriched with healthy, non-clinical samples in order to unravel to what extent such findings align with the present work on laboratory studies.

Studies based on people's daily lives could also be important for other reasons. Lab studies assess emotions, eating behavior, and their interaction at a fixed and relatively restricted point in time. How participants' eating behavior unfolds in time, however, cannot be tackled. For example, people can reveal unaffected or even decreased eating responses to emotions at the single moment of measurement, but then procrastinate a potential overdrive for highly palatable food at another moment outside the scope of the study. Alternatively, when a crisis state has resolved in daily life, there may afterwards be a compensatory increase drive for food intake to attain weight recovery and a likely overshoot in preferably highly palatable food. Thus, lab studies may provide only a snapshot representation of a process that is essentially in constant movement, a limitation that can be prevented by studies tracking valid time patterns in people's daily lives. It needs to be acknowledged though, that also daily-life studies have their limitations: they involve greater participation burden, require significant resources, are difficult to statistically analyze, rely virtually entirely on self-report, and cannot establish causality as well as experiments can (Smyth et al., 2001; Wheeler and Reis, 1991). Therefore evidence based on a combination of both laboratory and field-based studies seems of vital importance.

Notwithstanding the method of measuring the phenomenon of emotion-related eating, the assessment thereof is complex, as a multilayered collection of internal and external factors influences the desire for food and the amount and types of food that will be eaten. Cortisol, for example, which is released in more chronic stress conditions, stimulates the reward system in the brain, suggesting that cortisol may increase the reward value of food (Adam and Epel, 2007). Frequent bits of minor daily stressors may keep the stress arousal system in a chronically activated state, and these low but chronic stress levels may also drive overeating, the need for highly palatable food, or "stress-induced food reward dependence" (Torres and Nowson, 2007). It remains unknown how short-lived emotions triggered in the laboratory relate to chronic stressors or repeated minor daily stressors in daily life. Future studies that also include biological markers of stress and emotions will deepen our understanding of the physiological mechanisms underlying the emotion-eating relationship.

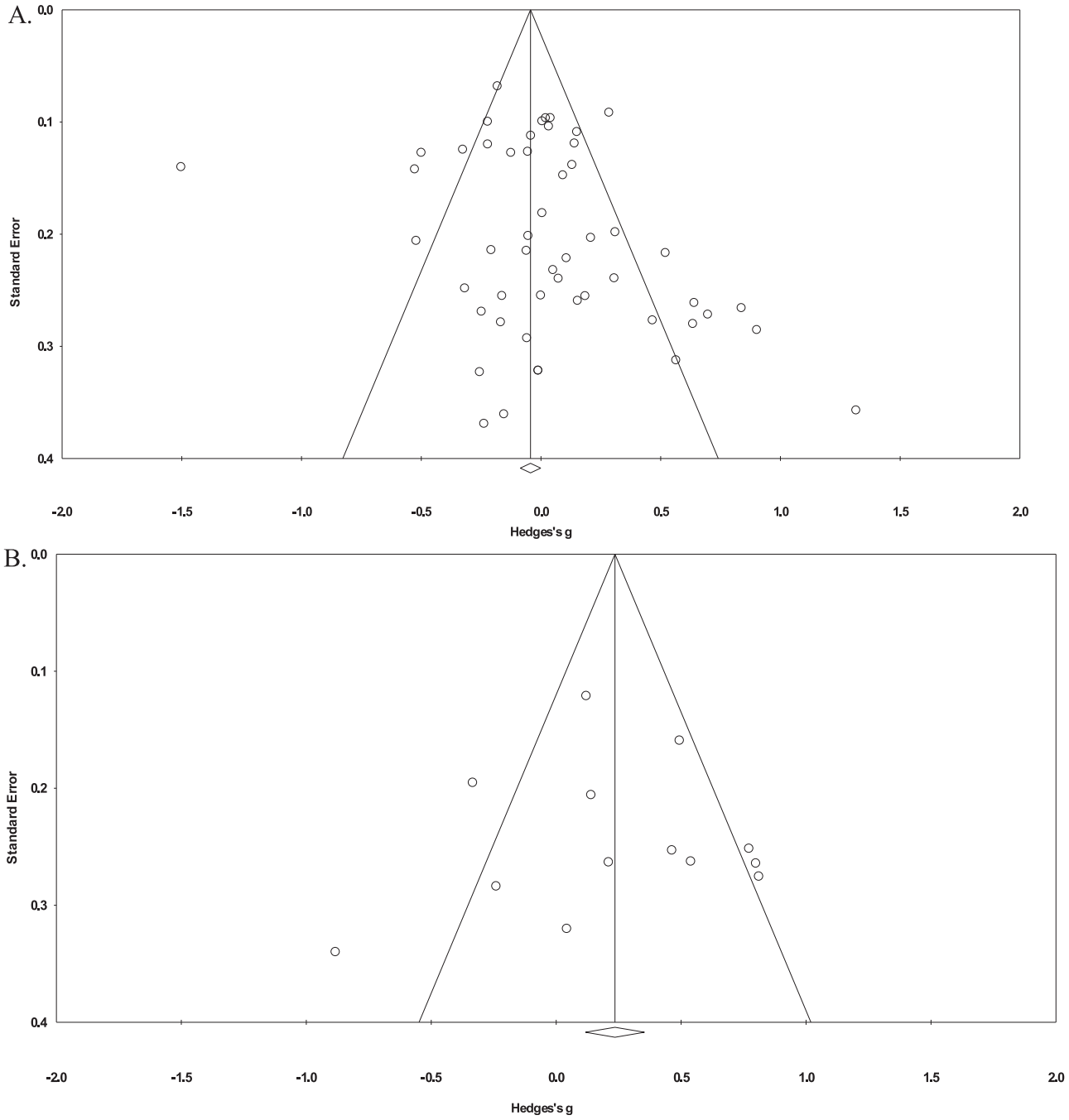


Fig. 9. Funnel plots with inverse variance of effect size plotted against Cohens *d* across the included studies for the main effect for negative emotion (Panel A) and positive emotion (Panel B).

4.4. Concluding comment

The present meta-analysis based on laboratory-based studies found that overall positive emotions resulted in increased eating, and in response to negative emotions only restrained eaters were found to show increased eating. Contrary to the theories about the effect of emotions in eating disordered samples, healthy controls and individuals scoring high on emotional eating measures, results did not reveal emotion-related changes in eating behavior in these subgroups. This meta-analysis points out that despite the empirical attention that has been paid to the topic, relatively many studies investigating the effect of emotions on eating did not meet fundamental quality requirements to be included. This observation calls for more thorough approaches to investigate the widely known phenomenon of emotion-induced eating.

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