Individual Differences in Marketing Placebo Effects: Evidence from Brain Imaging and Behavioral Experiments

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Much recent research has studied whether marketing-based expectancies such as price and brand quality beliefs influence the consumption experience and subsequent behavior, but almost no research has examined individual differences in "marketing placebo effects" (MPE). In this paper, we suggest three moderators of the effect marketing-based expectancies have on the behavioral and neural measures of the consumption experience based on previous findings from the neuroscientific literature investigating traditional clinical pain placebo effects. We used a novel automated structural brain imaging approach to determine individual differences and combined this approach with traditional behavioral experiments. We found that consumers high in reward-seeking, high in need for cognition, and low in somatosensory awareness are more responsive to MPE.

Keywords: individual differences, placebo effects, pricing, labeling, consumption experience, structural brain imaging, reward-seeking, need for cognition, somatosensory awareness

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Multidisciplinary evidence suggests that important factors that have an impact on the value or enjoyment derived from consumption are influenced by psychological associations and cognitive concepts. Effects of brand images, quality or efficacy beliefs about products and treatments, expertise of artists, and nutritional information on food packaging, for example, can occur independent of—and in extreme cases even override—the mere physical sensory consumption experience (Ariely and Norton 2009; Plassmann and Wager 2013). These cognitive concepts are learned by consumers over time and are shaped by everyday experiences with products, services, and social influences.

One important driver of how such cognitive concepts influence consumption is expectancies: beliefs and predictions about future feelings, events, or outcomes. For example, one such belief is that lower-priced goods are of lower quality (Gerstner 1985; Huber and McCann 1982; Rao and Monroe 1988). Hence, prices can serve as an external cue that signals quality and thus generates an expectation about how good the product is. For example, several studies have shown that people enjoy consuming identical products (such as wines or chocolates) more when they have a higher price tag (Goldstein et al. 2008; Plassmann et al. 2008; Wilcox, Roggeveen, and Grewal 2011). The price tag of a painkiller even changes consumers' pain perceptions (Geuter et al. 2013; Waber et al. 2008). Interestingly, these price-based expectancies do not change only *reported measures* of the consumption experience; they also change *neural measures* of consumption enjoyment such as activity in the ventromedial part of the prefrontal cortex (vmPFC) for the case of experienced flavor pleasantness (Plassmann et al. 2008) or the anterior part of the insula for the experienced displeasure of feeling pain (Geuter et al. 2013). That is why such marketing-based expectancy effects have also been referred to as "marketing placebo effects" (MPE) (Shiv, Carmon, and Ariely 2005a, 2005b).

In fact, MPE go way beyond expectancies based on price and quality; a large body of literature in consumer psychology has studied how people's expectancies shape consumption experiences. One of the first papers on this topic showed that brand label information can alter how much people enjoy consuming different beers (Allison and Uhl 1964). Converging evidence for marketing-based expectancy effects has been shown in various follow-up studies for both products and services across a variety of domains (Boulding et al. 1993; Kopalle and Lehmann 2001; Lee, Frederick, and Ariely 2006; Raghunathan, Walker Naylor, and Hoyer 2006; Steenhuis et al. 2010; Wansink and Chandon 2006; Wilcox, Roggeveen, and Grewal 2011; S. A. Wright et al. 2013).

Understanding the brain processes underlying expectancy and valuation during consumption is critical to understanding why expectations have such a powerful influence on consumption. Are these effects mere reporting biases based on post-consumption rationalization and cognitive dissonance, or do expectancies change how the consumption experience is actually encoded in the brain? Complementing the neuroscientific research on price placebo effects mentioned above, a few studies have investigated whether other marketing-based expectancies alter other positive, affective experiences such as taste, flavor, and aesthetic pleasantness in the brain (for a review, see Plassmann and Wager 2013).

For example, de Araujo and colleagues investigated the influence of verbal labels of smells (cheese vs. body odor) on neural signatures of olfactory processing (de Araujo et al. 2005). They found that indeed when subjects smelled identical odors, a positive or negative description altered neural activity in the ventromedial prefrontal cortex (vmPFC) and also in the bilateral amygdala, all linked to olfactory processing. A study by Nitschke and colleagues found that expecting an aversive taste to be less aversive did decrease neural activity in the primary taste cortex, involving taste intensity encoding, although the intensity of the negative taste was kept constant (Nitschke et al. 2006). Finally, Kirk and colleagues (2009) found that the pleasure participants derived from viewing

art pieces, and the accompanying engagement of the vmPFC, was higher when the subjects believed they were created by an expert (i.e., an artist) rather than by a non-expert (i.e., the experimenter).

Together, these findings suggest that across domains and marketing actions, expectancy manipulations are associated with changes in neural activity linked to consumption-related processing in the brain, ruling out the hypothesis that expectancy effects simply reflect demand characteristics or report biases. Expectations truly influence neurobiological responses to the experience of different stimuli showcasing the relevance of expectancy effects for consumer behavior and marketing management. However, almost no research has examined the neural and psychological processes required for such MPE to occur.

Against this background, the goal of this paper is to shed light on individual differences in MPE. To reach this goal, we first draw on neuroscientific evidence about the underlying mechanisms of pain placebo effects to extend Shiv, Carmon, and Ariely's model of MPE and suggest a multidisciplinary model of how marketing-based expectancies alter subjective consumption experiences. We then test the novel aspects of this model with a variety of different MPEs (price, brand labels, claims) and sensory experiences (food and aesthetic consumption) following a two-step procedure: In the first step, we test the neural predictions of our model using a structural imaging approach from neuroscience to study individual trait-related differences (study 1). We find that the volume of gray matter in the striatum, the posterior insula, and the dorsal medial prefrontal cortex moderates the expectancy effects of price and health claims on the experienced taste pleasantness for wine and milkshakes.

In a second step, we rely on existing evidence linking each of these brain areas with personality traits (i.e., the striatum with reward-seeking, the posterior insula with somatosensory awareness, and the dorsal medial prefrontal cortex with need for cognition) to further test the implications of our model for how personality traits moderate the placebo effects of price in behavioral experiments of wine tasting (studies 2a, 2b, and 2c). In the last study, we test the

robustness and generalizability of our effects by studying whether reward responsiveness, somatosensory awareness, and need for cognition also jointly moderate the effects of the perceived expertise of artists on subjective aesthetic experiences (study 3).

We conclude the paper by discussing the implications of our findings for marketing and consumer neuroscience and by highlighting the limitations of our work and calling for future research.

THEORETICAL AND METHODOLOGICAL BACKGROUND

Drawing on existing theories in cognitive neuroscience about pain placebo effects, we first suggest an extended model of processes underlying marketing action–based expectancy effects. We then provide further methodological details about the novel structural brain imaging approach we are applying in this paper.

Behavioral and Brain Mediators of Placebo Effects

A first model of MPE and how they work was suggested by Shiv, Carmon, and Ariely (2005b). In their model they first suggested and then showed empirical evidence for the following effects: External cues such as the price of an energy drink do generate response expectancies about the benefits of the product that in turn change behavioral outcomes such as the number of puzzles solved in a mental-effort task. We have incorporated their findings in the early and later stages of our model, shown in the white boxes in Figure 1. As a process variable, Shiv, Carmon, and Ariely found evidence that the salience of product-specific beliefs mediates the existence of MPE.

Figure 1 about here

Shiv, Carmon, and Ariely's model has been extended by recent research in cognitive neuroscience that sheds light on the underlying neural signatures of MPE. The experiments reviewed above provide evidence that expectancies not only alter reported measures of pleasure or displeasure of consumption but also affect responses in consumption-related brain systems, shown in the light

gray box in Figure 1. Another crucial question, however, is how expectancies actually shape consumption. To shed light on the underlying neural and psychological processes of MPE, we reviewed studies that have examined brain mediators and moderators of expectancy effects and individual differences in such effects. Three important processes emerged from this review to predict anticipatory processes of expectancy effects: (1) dopaminergic functioning related to reward-seeking (i.e., motivation) and learning, (2) processing in the posterior insula cortex and somatosensory cortices that has been linked to sensory processing of bodily states and experiences, and (3) prefrontal processing thought to be involved in cognitive processing, specifically cognitive regulation and appraisal of emotional states.

Dopaminergic functioning related to reward-seeking and learning. Several studies suggest a link between expectancy effects and dopaminergic functioning. A study by Atlas and colleagues (2010) was the first to use formal multilevel mediation analysis to identify the brain regions that link placebo-like expectancy effects on pain-related responses with expectancy effects on subjective pain reports. In their study, cue-based expectations (i.e., an auditory cue thought to predict intensity of pain) and pain reports varied in every trial, and the authors tested whether responses in the brain in a given trial contributed to the link between cue-based expectation of high vs. low pain and changes in the pain experience. After an initial learning phase, the actual level of pain intensity was kept constant. They found that a subset of pain-responsive regions formally mediated trial-by-trial expectancy effects on pain and that expectancy effects on these regions were in turn mediated by expectancy-induced anticipatory responses mostly in the ventral striatum, a region with a relatively high density of dopaminergic neurons linked to reward-seeking and learning behavior.

To complement these findings, several other studies investigated the role of reward responsiveness for pain placebo effects. They found that participants who showed stronger neural markers of reward responsiveness, lower levels of dopamine and opioid binding during pain stimulation (Scott et al. 2007; Wager, Scott, and Zubieta 2007; Zubieta 2005), and larger gray matter

volume in mesolimbic brain regions (e.g., the ventral striatum; Schweinhardt et al. 2009) also showed stronger pain placebo effects. Indeed, while most of the previous studies reviewed in the introduction revealed that expectations alter consumption-related behavioral responses and responses in consumption-related brain regions, some studies on pain placebos found expectancy effects in the striatum, among other regions, to predict expectancy-enhanced placebo analgesia (e.g., Kong et al. 2006).

Also, patient populations that exhibit disorders related to abnormal dopaminergic functioning, such as depression (Kirsch et al. 2008; Rutherford et al. 2010; Sneed et al. 2008) and Parkinson's disease (Benedetti et al. 2004; Lidstone et al. 2010), show relatively high pain placebo response rates. Interestingly, in a study by de la Fuente-Fernández and colleagues, the authors could even show the power of placebo effects by providing in vivo evidence for substantial release of endogenous dopamine in the striatum of Parkinson's disease patients (i.e., a population with a damaged nigrostriatal dopamine system) in response to placebos (de la Fuente-Fernández et al. 2001).

Finally, behavioral studies have revealed correlations between increased pain placebo responsiveness and personality traits linked to increased dopaminergic functioning, such as behavioral activation and optimism (Geers et al. 2005; Morton et al. 2010; Schweinhardt et al. 2009). All of these studies suggest a link between expectancies and dopaminergic processing linked to reward responsiveness. In other words, this part of our MPE model suggests that an external cue such as the price of wine leads to expectations of how good the wine tastes that are linked to a motivational signal of reward-seeking and that people who are more responsive to rewards should exhibit higher MPE.

Processing in the posterior insula cortex and somatosensory cortices that has been linked to awareness of sensory processing and bodily states. Another mechanism can be suggested based on our literature review of pain placebo effects in cognitive neuroscience. In Wager et al.'s pattern classification analysis of individual difference predictors of pain placebo effects, activity in the

somatosensory system (i.e., somatosensory cortices, posterior insula) showed a negative correlation with pain placebo effects (Wager et al. 2011). These areas were also found to formally mediate pain placebo effects in Atlas et al.'s study (2010). From a conceptual standpoint, it makes sense that pain placebo effects should also be altered by brain regions encoding somatosensory or physical aspects of pain processing, because somatosensory pain processing precedes higher-order pain processing to determine the liking/disliking of the pain experience—that is, experienced (dis-) utility.

However, studies investigating expectancy effects on flavor processing have also found that brain activity in somatosensory areas is dampened by expectancy effects (Atlas et al. 2014; Nitschke et al. 2006). On this basis, we suggest a more general role for somatosensory processing to underlie MPE. Somatosensory processing precedes experienced utility processing and is involved in bottom-up processing of expectancy effects. Thus, we suggest that somatosensory processing is another intervening variable in our model of how MPE work and that people who are more aware of their somatosensory states should be less responsive to MPE.

Prefrontal processing thought to be involved in cognitive regulation and appraisal of emotional states. Other potential mechanisms underlying MPE that can be derived from the existing findings about pain placebo effects are linked to cognitive processes involved in emotion regulation and appraisal. For example, Wager et al. found correlations between the magnitude of pain placebo effects on reported pain and the magnitude of heat-evoked responses in pain-processing brain regions (Wager, Scott, and Zubieta 2007). Interestingly, however, pain-processing regions were not the only regions that correlated with pain placebo effects. During pain anticipation, prefrontal brain regions involved in emotional control and emotional appraisal, working memory, and predicted value encoding showed significant positive correlations with pain placebo effects. In fact, a new analysis of Wager and colleagues' data using machine learning and pattern classification techniques to investigate individual differences in pain placebo effects showed that increased anticipatory responses in a frontal (i.e., lateral orbitofrontal, lateral and medial dorsal prefrontal cortex) and

parietal brain system involved in emotion regulation and emotional appraisal had a higher predictive accuracy for placebo effects to occur than activity in the brain's pain-processing regions (Wager et al. 2011).

Similar findings were shown by Atlas et al.'s formal mediation analysis of pain expectancy effects and health label expectancy effects (Atlas et al. 2010; Atlas et al. 2013). Besides pain- and taste-processing regions, Atlas et al. also found the lateral and medial part of the dorsal prefrontal cortex to mediate expectancy effects on pain and taste perception. We suggest in our model that higher-order, top-down cognitive processes of regulating emotional states and emotion generation play a role as an intervening variable for MPE to occur. In turn, people who rely more on such cognitive systems during decision making should be more responsive to MPE.

Taken together, expectancies might affect consumption-related circuitry not only because they simulate the consumption experience and experienced utility prior to consumption, but also because expectancies influence intervening processes such as dopaminergic processing linked to reward-seeking, prefrontal activity linked to cognitive regulation and appraisal of emotional states and experiences (i.e., a top-down cognitive processing), and attention to or away from somatosensory experiences encoded in somatosensory brain areas (i.e., a bottom-up somatosensory processing linked to processing in the posterior insula and somatosensory cortices). These novel process variables of our model are shown in the dark gray boxes in Figure 1 and are the individual differences we are investigating in this paper.

Using Structural Brain Imaging Data to Investigate Brain Moderators Underlying Consumer Behavior

Over the past decade, an increasing number of papers investigating questions related to consumer behavior and marketing have integrated theoretical and methodological approaches from neuroscience (for recent reviews, see Plassmann, Ramsøy, and Milosavljevic 2012; Yoon et al. 2012). The vast majority of studies to date have used functional magnetic resonance imaging (fMRI) to

establish associations between brain processes and consumer behavior (Kable 2011). While fMRI has several important strengths that justify its widespread use, consumer neuroscience research programs would be strengthened by greater inclusion of other neuroscientific techniques that can complement fMRI.

In this paper, we follow this idea and use an approach novel to consumer neuroscience that is more suitable than fMRI to investigate individual differences on a trait level, which is the goal of this paper: automated structural MRI analysis. Differences in brain structures such as gray matter volume can be linked to individual differences in brain function, personality, and behavior. All of these constructs are of crucial importance to understanding the underlying *processes* of marketing-relevant behavior, and thus we believe that automated structural MRI analysis will be an important new method in the toolkit of consumer neuroscience.

MRI-based measures of gray matter have been shown to be related to brain function, in both health and disease (e.g., Newman et al. 2007; Peinemann et al. 2005; Schweinhardt et al. 2009; Tabibnia et al. 2011), possibly because they partly reflect the number and size of neurons and the complexity of their synaptic connections. Likewise, individual anatomical differences—for example, within reward-related dopaminergic pathways—have been linked to significant differences in behavioral effects, including variation of personality traits (Depue and Collins 1999).

Following this idea, in recent years a large amount of literature has emerged showing that individual differences in behavior and personality can be at least partly explained by differences in brain structure (Banissy et al. 2012; M. X. Cohen et al. 2008; DeYoung et al. 2010). The basic assumption underlying such approaches is that regional gray matter volume, as measured by MRI, corresponds to the regional volume and wiring of nerve cell layers in the brain. The most widely used method to investigate large groups of subjects is voxel-based morphometry (VBM). Since its first description (Ashburner and Friston 2000; Wright et al. 1995), VBM has been widely applied to investigate brain structural foundations of pathological processes as well psychological variables and

individual differences in behavior or personality (DeYoung et al. 2010; Schweinhardt et al. 2009; Tabibnia et al. 2011; Yokum, Ng, and Stice 2011).

In essence, VBM is an automated technique that allows the assessment of regional brain volumes (for a recent technical review see Whitwell 2009) using high-resolution structural brain images. Such structural images are usually recorded along with functional brain images during brain imaging experiments. These structural images are then normalized to a common brain template and segmented into different tissue compartments, usually gray matter, white matter, and cerebrospinal fluid (see Figure 2 for the processing flow of the images).

Figure 2 about here

In this paper, we first applied VBM to explore brain regions that showed a variation in gray matter volume (GMV) that predicted individual differences in the magnitude of MPE. We then used the VBM results to inform follow-up behavioral experiments that shed more light on the personality traits linked to the functioning of these brain regions.

STUDY 1: BRAIN MODERATORS OF MARKETING PLACEBO EFFECTS DURING FOOD CONSUMPTION

The goal of study 1 was to test whether individual differences in the size of gray matter volume in (1) the striatum, (2) the posterior insula cortex and somatosensory cortices, and (3) prefrontal areas moderate MPE, as suggested by our model outlined above. To summarize, our model predicted that three different individual differences underlie MPE:

H1a: The *greater* the GMV in the striatum and in prefrontal structures (i.e., lateral and medial dPFC and lateral OFC), the *more responsive* participants are to MPE.

H1b: The *greater* the GMV in the posterior insula and somatosensory cortices, the *less responsive* participants are to MPE.

To test these hypotheses, we used the structural neuroimaging data from three different experiments that have investigated neural correlates of MPE using functional neuroimaging data. We pooled the structural neuroimaging data of three different experiments to ensure an appropriate sample size for such an exploratory analysis (Simmons, Nelson, and Simonsohn 2013). These studies investigated two different types of MPE: prices (high vs. low) and healthfulness claims (light vs. regular; organic vs. regular). Given how similar the underlying neural mechanisms of placebo effects generally seem to be (Atlas and Wager 2013; Plassmann and Wager 2013; S. A. Wright et al. 2013), this seems a reasonable approach. Below, we briefly describe the design of these three studies.

Design and Procedure

The first experiment investigated how the price of wines influenced behavioral and neural measures of experienced utility (Plassmann et al. 2008). The experiment applied a two-factorial within-subjects design with instructed price (high = \$90 and \$45; low = \$10 and \$5) as the first factor and actual retail price (wine 1 = \$90; wine 2 = \$5) as the second factor. A third wine was used as a distractor, with an identical instructed and actual retail price of \$35. During this experiment, 20 participants (11 males, mean age 24.5 years) believed that they would consume five different wines with different retail prices (\$90, \$45, \$35, \$10, \$5) while their brains were scanned using functional magnetic resonance imaging. However, in reality subjects consumed only three different wines; two of the wines were administered with two different prices (wine 1: \$90 and \$10; wine 2: \$45 and \$5) to keep the physical consumption constant. Subjects showed a significant effect of price on experienced utility on a behavioral level (using a Likert scale from 1 = not at all to 6 = very much) and, more important, also on a neural level (see Plassmann et al. 2008 for the details of the results). In the current study, we were interested in whether gray matter volume in specific brain structures—the ones in our model, outlined above—would moderate MPE. Against this background, the behavioral and structural neuroimaging data of the 20 subjects were entered in a novel application of automated, structural brain imaging analysis, the VBM analysis described above.

The second experiment was very similar to the first and served as a neuroimaging pilot study (N = 12, 6 male, mean age 30.3 years) for an extended version of Plassmann et al.'s experiment 1. This extension consisted of two points: First, instead of using wines of very different actual retail price classes, in experiment 2 we used wines of the same price class (€10–€13) and randomly assigned the wines to different instructed price conditions (€3, €16, and €18). Second, we added a condition that varied whether the subjects received the wines for free as in experiment 1 or had to pay for the wine. In this study, we could replicate the behavioral effects of the price condition from experiment 1, but found no significant results of the payment condition and also no significant interaction effect. The results have been reported elsewhere (Skvortsova et al. 2013). As with experiment 1, we used the behavioral and structural neuroimaging data of the 12 subjects for the VBM analysis in the current study.

The third experiment used different types of product labels instead of prices to generate different expectations of the pleasantness of the product. More specifically, we used different types of healthfulness claims for milkshakes that were shown to create either positive expectations about the pleasantness of the taste ("organic"; Lee et al. 2013) or negative expectations about the pleasantness of the taste ("light"; Chandon and Wansink 2012; Raghunathan, Walker Naylor, and Hoyer 2006; Werle et al. 2013); there was also a neutral condition ("regular").[1] In other words, in this experiment, we applied a one-factorial between-subjects design with healthfulness label of a vanilla or chocolate milkshake. A total of 58 subjects participated in this experiment (28 males, M_{age} = 27 years, SEM = 4.25). One group of subjects (N = 29) consumed identical milkshakes but thought they would be either organic or regular; the other group of subjects (N = 29) consumed identical milkshakes but thought they would be either light or regular. While they were drinking, their brains were scanned using fMRI. Subjects showed a significant effect of healthfulness label on experienced

[1] The directions of these effects were pretested in a pre-scanning session and are reported in detail in Atlas et al. (2014).

utility on a behavioral level and, more important, also on a neural level. See Atlas et al. (2014) for the details of the results. In the current study, we were again interested whether gray matter volume in specific brain structures outlined in our model above would moderate MPE. Against this background, we entered the behavioral and structural neuroimaging data of the 58 subjects in our VBM analysis.

To conclude, a total of 90 subjects who participated in one of the three experiments described above were included in the following VBM analysis.

Data Acquisition, Analysis, and Results

All study participants underwent MRI on either a 1.5- or 3-Tesla scanner (Magnetom Trio or Avanto, Siemens, Erlangen, Germany). An eight-channel head coil was used for signal reception. All sequences were performed using a T1-weighted MPRage sequence with a resolution of 1x1x1 mm in sagittal orientation with 160 slices. Specific data acquisition parameters differed slightly in the three experiments of study 1 (3T experiment 1 at Caltech: TR 2200 ms, TE 9.2 ms, flip angle 30°, FOV 256 mm; 3T used for some participants of experiments 2 and 3 at Bonn University: TR 1300 ms, TE 3.9 ms, flip angle 10°, FOV 256 mm; 1.5T used for some participants of experiments 2 and 3 at Bonn University: TR 1520 ms, TE 3.6 ms, flip angle 30°, FOV 256 mm).

Voxel-based morphometry techniques (Ashburner and Friston 2000) were performed in the context of SPM8 (http://www.fil.ion.ucl.ac.uk/spm/software/spm8) using the VBM8 toolbox (http://dbm.neuro.uni-jena.de/vbm/download). Images were automatically segmented and normalized using the high-dimensional DARTEL algorithm as implemented in SPM8. Gray matter datasets were analyzed modulated (nonlinear only), which permits analysis of gray matter volume and controls for individual brain size differences. All gray matter images were smoothed with a Gaussian kernel at half-width full-maximum of 8 mm.

We first performed a whole-brain analysis to investigate which brain regions' gray matter volume varied as a function of responsiveness to MPE, with the goal of providing first evidence for

our model's predictions (see Table 1). To do so, we computed an MPE responsiveness score defined as behavioral experienced utility rating in the high minus low expectancy condition.

Table 1 about here

To address our specific a priori hypotheses outlined above, we then conducted an independent region-of-interest (ROI) analysis and used differences in gray matter volume in the three hypothesized brain regions to predict MPE responsiveness using regression analysis. Importantly, this ROI analysis was independent from the whole-brain analysis because the ROIs were defined based on independent studies in the literature. More specifically, the ROIs were defined as a sphere with a diameter of 8 mm around previously reported coordinates: These coordinates were for the striatal mask a combination of the right and left ventral striatum based on the review paper of Bartra and colleagues (-12/12/-6 and 12/10/-6; Bartra, McGuire, and Kable 2010); for the posterior insula a region based on a review paper by Benedetti and colleagues on placebo effects (44/-15/4; Benedetti et al. 2003); and for the dmPFC a region based on a study by Ochsner and colleagues (-10/50/34; Ochsner et al. 2004) on cognitive regulation and emotion reappraisal. Using MarsBaR (v.0.43; http://marsbar.sourceforge.net) we then extracted individual gray matter volumes of the predefined ROIs and used those ROIs for the regression analyses.

To test whether gray matter volumes in the striatum, the dmPFC, and the posterior insula moderate MPE, we followed the procedure suggested by Judd, Kenny, and McClelland (2001) for within-subjects designs: We entered the MPE responsiveness score as a dependent variable in a regression analysis with gray matter volume in the striatum, the dmPFC, and the posterior insula as predictors, controlling for age and gender (model 1). We entered age and gender in the first regression model because both have been shown to influence gray matter volume (Good et al. 2001; Luders and Toga 2010) and also to be consistent with the model applied in the whole-brain VBM analysis described above. However, to show that the results are not dependent on the inclusion of

these predictors in the model, we also estimated a second model that does not include age and gender. Table 2 lists the results of both models.

We found that the GMV in all three brain structures moderates MPE (see Figures 3a–c). Interestingly, we found a positive relationship for GMV in the striatum (standardized beta coefficient: .30) and the dmPFC (standardized beta coefficient: .37), but a negative relationship for GMV in the posterior insula (standardized beta coefficient: –.30).

Table 2 and Figure 3a-c about here

Discussion

We found three brain regions that are unrelated to neural signatures of experienced utility to correlate with individual differences of MPE: (1) the striatum, (2) the dmPFC, and (3) the posterior insula. WE first would like to acknowledge that there is an ongoing debate about the robustness of structural brain-behavior (SBB) correlations. A recent study failed to replicate SBB correlations in five studies (Boekel et al. (in press)). Even though we cannot exclude that our results may fail to replicate in future studies since we have not conducted such a replication, we think that our results are more robust because of several reasons: i) our analysis is based on three different data sets, which should control for some random effects; ii) we provide additional behavioral evidence from two studies (i.e. studies 2 and 3 of this paper described below) which is actually based on and informed by the SBB correlations, providing some independent evidence for our effects, and iii) according to Simsohn (in press) failures to replicate have to be treated with caution. For example in the case of the Boeckel et al. replication attempts of SBB one could argue that their replications might failed to replicate previous findings because their replication studies were underpowered etc. Note that Simsonhn argues for a sample size of the replication study of 2.5 times the sample size of the orginal study.

A crucial next question from a consumer research standpoint is this: What psychological variables can be linked to changes in GMV in these areas? Answering this question needs to be done with a lot of caution because a given brain region can be involved in several unrelated psychological processes, a problem that has been referred to as "reverse inference" (Ariely and Berns 2010; Plassmann, Ramsøy, and Milosavljevic 2012; Poldrack 2006, 2011). Several solutions have been proposed to test the selectivity but also the specificity using automated brain-mapping frameworks that use text mining, meta-analysis, and/or machine-learning techniques to generate a large database of mappings between neural and psychological functions. Here, we used the following two-step procedure:

We first applied the framework suggested by Yarkoni et al. (2011) to compute measures of how selective and how specific our brain regions of interest are. Their approach allows the computation of a measure of reverse inference indicating how consistently *and* selectively a brain area is involved in a specific psychological process. The activation data used in this framework is based on nearly 200,000 activations from almost 6,000 functional magnetic imaging studies and thus contains a broad set of term-to-activation mappings, which makes this framework well suited for drawing quantitative inferences about mind-brain relationships.

The measures suggested in this framework are as follows: (1) z-scores corresponding to the likelihood that a region will be reported active if a study uses a particular term (i.e., P(Activation|Term)), (2) z-scores corresponding to the likelihood that a term is used in a study given the presence of reported activation (i.e., P(Term|Activation)), and (3) a posterior probability map—the estimated probability of a term being used given the presence of activation (i.e., P(Term|Activation)). The first component can be seen as a measure of forward inference because it indicates consistency. However, a brain area also needs to be *selectively* involved in the psychological process of interest. The second and third components address this point and serve as

measures of such a selectivity—that is, of reverse inference. Note that more details about Yarkoni et al.'s framework are provided in the appendix.

As a second step, we then entered the locations of our peak voxels in the location database of Yarkoni et al.'s (2011) approach and evaluated different options suggested by NeuroSynth based on theoretical considerations drawing on our model. The results are as follows: The subarea of the striatum (i.e., the ventral and dorsal parts) in which the average of the whole-brain VBM analysis fell has forward inference value of $Z_{forward} = 9.05$, signifying consistency, and a reverse inference value of $Z_{reverse} = 6.23$, posterior probability = .78, signifying selectivity of this area to be involved in reward anticipation and motivational processing. Reward processing was the psychological process with the highest reverse inference value; this is consistent with theoretical considerations outlined above.

The subarea of the insula (i.e., the posterior parts) in which the average of the whole-brain VBM analysis fell has forward inference value of $Z_{forward} = 4.75$, signifying consistency, and a reverse inference value of $Z_{reverse} = 4.14$, posterior probability = .78, signifying selectivity of this area to be involved in somatosensory processing, pain-related processing ($Z_{reverse} = 3.73$, posterior probability = .89), and autobiographical processing ($Z_{reverse} = 3.02$, posterior probability = .86). Another potentially relevant process with a higher reverse inference z-score but lower posterior probability was "inhibitory" ($Z_{reverse} = 4.63$, posterior probability = .85). In addition, other studies including a large-scale meta-analysis also found this area to be involved in introspection and somatosensory awareness (Chang et al. 2013; Simmons et al. 2012). These findings are largely consistent with our model outlined above that the neural activity in the posterior part of the insula is linked to somatosensory awareness.

Last, the subarea of the dmPFC in which the average of the whole-brain VBM analysis fell has a forward inference value of $Z_{forward} = 3.53$, signifying consistency, and a reverse inference value of $Z_{reverse} = 3.25$, posterior probability = .91, signifying selectivity of this area to be involved in working memory functions. Other potentially relevant processes were "attribution" (posterior

probability = .91), "decision-making" (posterior probability = .87), "thinking" (posterior probability = .87), and different emotional states (posterior probability = .86–.88). Taken together, based on Yarkoni et al.'s approach, these results show that the dmPFC is involved in a variety of different psychological processes linked to cognitive processing of emotional states and experiences. We further turn here to additional results from studies investigating psychological processes that are in line with our model's theoretical considerations. These are the role of the dmPFC for working memory-based cognitive regulation and appraisal of emotional states—that is, cognitive emotion generation (Kober et al. 2008; Ochsner et al. 2009)—and also more specifically for working memory-based cognitive regulation of responses during value-based decision-making (Venkatraman, Payne, et al. 2009; Venkatraman, Rosati, et al. 2009).

To conclude, the results from study 1 give first evidence that individual differences in MPE are linked to reward processing as signified by differences in GMV in the striatum, somatosensory awareness as signified by differences in GMV in the posterior insula, and cognitive appraisal of emotional experiences as signified by differences in GMV in dmPFC.[2] To provide further evidence for this new model of how MPE work, we tested the individual difference on a personality trait level in studies 2a, 2b, and 2c.

STUDY 2: INDIVIDUAL DIFFERENCES IN REWARD-SEEKING, COGNITIVE, AND SOMATOSENSORY FOCUS FOR THE EFFECTS OF PRICE DURING WINE TASTING

To test the predictions about individual differences of MPE on the psychological personality trait level derived from our conceptual model and also the brain imaging results from study 1, we undertook three experiments that provided further evidence for our model of how MPE work. The logic of these studies is as follows: All three studies (2a, 2b, and 2c) investigated the influence of

[2] Note that more support for the latter two processes is also provided by the other findings of the whole-brain analysis: GMV in other somatosensory areas—that is, somatosensory cortex II—showed negative correlation with MPE responsiveness, and other areas found to be involved in cognitive processing and regulation of emotional states—that is, lateral parts of the ventral PFC—showed a positive correlation with MPE responsiveness (see whole-brain analysis results in Table 1).

high vs. low price tags on food consumption, each testing a different one of the three moderators. The design of these studies is almost identical to the first study by Plassmann et al. (2008) described above. The only differences are that study 2a used price levels that were adapted to the market prices in France and were expressed in euros instead of U.S. dollars (i.e., wine 1: €43 and €5, wine 2 = €30 and €3, distractor wine 3 = €16) and that in all three studies each wine in each condition was sampled only once and experienced utility was sampled using a visual analog scale with anchors "not at all" (coded as 1) to "very much" (coded as 7).

Study 2a: Moderation of Marketing Placebo Effects by Reward-Seeking as a Personality Variable

The purpose of study 2a was to investigate whether participants who are more reward-seeking are also more susceptible to MPE. This prediction was based on our finding in study 1 that higher GMV in the striatum was linked to higher MPE responsiveness and the fact that striatal activity has been linked in overlapping regions to reward-seeking (Beaver et al. 2006; Schweinhardt et al. 2009). We used the reward-seeking subscale of the behavioral activation system (BAS) scale to sample how responsive people are to rewards (Carver and White 1994).

H2a: Marketing placebo effects will be more pronounced the higher subjects score on the reward-seeking subscale.

Design and procedure. Ninety male participants from a French university (M_{age} = 23.0 years, SEM_{age} = 2.44 years) gave experienced utility ratings for each wine after consumption. After the wine-tasting task, we sampled the reward-sensitivity subscale of the behavioral activation scale (Carver and White 1994). The scale has items such as "When I get something I want, I feel excited and energized," and our subjects answered on a 5-point Likert scale ranging from 1 ("do not agree at all") to 5 ("completely agree").

Analysis and results. We first tested whether we could replicate the expectancy effects reported in experiments 1 and 2 of study 1. One subject had to be excluded because he paid no attention to the task. Thus, experienced utility ratings of 89 subjects were entered as a dependent

variable in a one-way within-subjects ANOVA with the high vs. low price condition (pooled over both wines) as predictor. We found a significant effect of price, $F(1, 88) = 57.07$, $p < .001$.

Next, we tested whether differences in reward sensitivity moderated the MPE of price, following the same procedure to test for within-subject design moderators as applied in study 1 (Judd, Kenny, and McClelland 2001). Table 3 lists the results of this analysis; Figure 4a (below) shows the correlation between BAS subscale scores and MPE. We found that the BAS subscale scores indeed moderated the effect of price on experienced utility ratings, $T(1, 87) = 2.98$, $p = .004$.

Table 3 about here

Discussion. In study 2a we showed that a consumer's reward responsiveness as measured by the BAS subscale is indeed positively correlated with the strength of MPE. The more responsive participants were to rewards the more their experienced utility of wine was influenced by price.

Study 2b: Moderation of Marketing Placebo Effects by Somatosensory Awareness as a Personality Variable

Another interesting result from the VBM analysis was a negative correlation between MPE and the gray matter volume in the posterior part of the insula, which as outlined above has been linked to somatosensory processing and introspection. This finding gives first evidence for the idea that a consumer's somatosensory awareness might play an important role for MPE.

More concretely, when MPE are at play, cognitive cues such as the price of or the label on the product generate a signal that affects bottom-up processes of internal somatosensory experiences such as tasting a wine or milkshake. An increased sensitivity of the brain systems encoding somatosensory experience should put more weight on the actual somatosensory experience, allowing less influence of external cognitive cues.

The purpose of study 2b was to investigate whether participants who have high somatosensory awareness are less receptive to MPE. We measured somatic awareness using the private body consciousness (PBC) subscale of the Body Consciousness Questionnaire (BCQ) (Miller, Murphy, and Buss 1981) and predicted:

H2b: The size of MPE decreases the higher subjects score on the private body consciousness subscale.

Design and procedure. Eighty-five alumni of a North American university (45 males, M_{age} = 32.84 years, SEM_{age} = 5.7 years) gave experienced utility ratings for each wine after consumption. After the wine-tasting task, we sampled a scale that measures somatic awareness. This scale was the private body consciousness subscale of the Body Consciousness Questionnaire (Miller, Murphy, and Buss 1981). As its name suggests, PBC is a personality trait that characterizes how attentive (conscious) a person is to his or her internal body signals. The scale has items such as "I'm aware of changes in my body temperature," and subjects responded on a 7-point Likert scale ranging from 1 ("strongly disagree") to 7 ("strongly agree"). Individuals who score high on the PBC scale tend to pay more attention to somatosensory processes. For instance, people high on PBC tend to report more pain than those who are low on this characteristic (Ferguson and Ahles 1998). PBC has also been linked to increased embodied cognition (Häfner 2013).

Analysis and results. We first tested whether we could replicate the expectancy effects reported in studies 1 and 2a. The experienced utility ratings of 85 subjects were entered as a dependent variable in a one-way within-subjects ANOVA with the high vs. low price condition (pooled over both wines) as predictor. We found a significant effect of price, $F(1, 84) = 23.55$, $p < .001$.

Next, we tested whether differences in PBC moderated the MPE of price, following the same procedure to test for within-subject design moderators as applied in study 1 (Judd, Kenny, and McClelland 2001). Table 3 lists the results of this analysis; Figure 4b (below) shows the correlation

between PBC score and MPE. We found that the PBC scores indeed moderated the effect of price on experienced utility ratings, $T(1, 84) = -2.83$, $p = .006$.

Discussion. In study 2b we showed that a consumer's somatosensory awareness as measured by the private body consciousness subscale is indeed negatively correlated with the strength of MPE. The more aware subjects were of their internal bodily signals the less their experienced utility of wine tasting was influenced by price.

Study 2c: Moderation of Marketing Placebo Effects by Need for Cognition as a Personality Variable

The third individual difference found in the VBM analysis was a positive correlation between MPE and the gray matter volume in the dmPFC, which as outlined above has been linked to cognitive processes necessary for the regulation and appraisal of emotional experiences, working memory, and thinking. This finding gives first evidence for the idea that a consumer's need to focus on cognitive cues might play an important role for MPE.

More concretely, when MPE are at play, cognitive cues such as the price of a wine generate a cognitive top-down value signal that affects bottom-up processes of internal somatosensory experiences such as tasting a wine or milkshake. In contrast to the findings about somatosensory focus in study 2b, an increased sensitivity of the brain systems linked to cognitive appraisal of emotional experiences and cognitive thinking should increase how much subjects are influenced by external cognitive cues and in turn increase MPE responsiveness.

The purpose of study 2c was to investigate whether participants who have a higher need for cognition (NFC) are more receptive to MPE. NFC is a personality variable reflecting the extent to which individuals are inclined to engage in cognitive activities (Cacioppo and Petty 1982; Cacioppo, Petty, and Kao 1984). Cohen, Stotland, and Wolfe (1955) defined the NFC as "a need to structure relevant situations in meaningful, integrated ways," "the notion of thinking abstractly is appealing to me," and "a need to understand and make reasonable the experiential world." Cognitive cues based

on marketing actions such as labels or prices can be seen as signals that help to structure experiences, and people who are more responsive to using such cognitive cues to structure their experiences should be more receptive to MPE. Against this background we predict:

H2c: MPE will be more pronounced the higher subjects score on the need for cognition scale.

Design and procedure. The design of this experiment was identical to that of experiment 1 from study 1 and study 2b, using the same price levels and wines (Plassmann et al. 2008). Eighty participants from a North American university population (41 males, M_{age} = 24.01 years, SEM_{age} = 2.8 years) gave experienced utility ratings for each wine after consumption. After the wine-tasting task, we sampled a scale that measured need for cognition. This scale was Cacioppo et al.'s short form of the NFC scale (Cacioppo, Petty, and Kao 1984). Subjects responded on a 7-point Likert scale ranging from 1 ("strongly disagree") to 7 ("strongly agree").

Analysis and results. We first tested whether we could replicate the expectancy effects reported in studies 1, 2a, and 2b. One subject was excluded because he did not pay attention to the task. The experienced utility ratings of 79 subjects were entered as a dependent variable in a one-way within-subjects ANOVA with the high vs. low price condition (pooled over both wines) as predictor. We found a significant effect of price, $F(1, 78) = 44.39$, $p < .001$.

Next, we tested whether individual differences in need for cognition moderated the MPE of price following the same procedure to test for within-subject design moderators as applied in study 1 (Judd, Kenny, and McClelland 2001). Table 3 lists the results of this analysis; Figure 4c shows the correlation between NFC scores and MPE. We found that the NFC scores indeed amplified the effect of price on experienced utility ratings, $T(1, 78) = 2.40$, $p = .019$.

Add Figure 4 about here

Discussion. In study 2c we showed that a consumer's focus on cognitive cues as measured by the need for cognition scale is indeed positively correlated with the strength of MPE. The higher the

participants' need for cognition the more their experienced utility of wine tasting was influenced by price.

Taken together, studies 2a, 2b, and 2c provided further evidence that participants high in reward-seeking and high in need for cognitive processing were more responsive to MPE, whereas subjects high in somatosensory awareness were less responsive to MPE. However, these studies investigated the influence of these three moderators in three separate studies rather than one single study. Also, studies 2a, 2b, and 2c all investigated individual differences in how prices affect food consumption. It remained unclear whether our individual difference effects transfer to other marketing-based expectancy effects, such as different brand labels, on different consumption experiences, such as aesthetic consumption. We addressed these issues in study 3.

STUDY 3: INDIVIDUAL DIFFERENCES IN REWARD-SEEKING, COGNITIVE PROCESSING, AND SOMATOSENSORY AWARENESS FOR THE EFFECTS OF BRANDING DURING AESTHETIC CONSUMPTION

This last study aimed at conceptually replicating our findings from studies 2a, 2b, and 2c for the effect that a different cognitive cue (whether an art piece was generated by an artist or the experimenter on a computer) has on a consumption experience in a different sensory domain (experienced aesthetic pleasantness). In addition, in study 3 we sampled all three personality variables of interest in the same participants to provide increased statistical control of the individual difference effects we found in study 2.

Design and procedure. Subjects were instructed that the goal of the study was to better understand their preferences for different types of art and how their personality influences those preferences. The study was conducted using an online sample (drawn from Amazon's Mechanical Turk) and took on average 7 minutes and 49 seconds to complete. Subjects were paid 50 U.S. cents for their participation.

Subjects were instructed that they would see different pictures and be asked to rate how much they enjoyed looking at each one using a 9-point Likert scale (1 = not at all to 9 = very much). Some of the pictures would show abstract work by artist Wassily Kandinsky; some would show abstract work generated by the experimenter on a computer. Unbeknownst to the participants, all stimuli were unfamiliar abstract art pieces by various artists. This task was adapted from two studies by Kirk and colleagues, and we used the same art stimuli used in these papers (Kirk et al. 2009a,b). Each subject rated 10 pictures, five labeled as crafted by an artist (the high expectancy condition) and five labeled as being generated by the experimenter on his computer (the low expectancy condition). Assignments of pictures to the artist and computer conditions were counterbalanced, and the order in which the pictures were shown was completely randomized. We then sampled in a randomized order the personality scales from studies 2a, 2b, and 2c (i.e., the need for cognition scale, the reward-seeking subscale of the behavioral activation system scale, and the private body consciousness subscale).[3] At the end, subjects were asked about their age, gender, and thoughts about the goal of the study and were then thanked for their participation. The results are shown in Table 3 and Figure 5 (below).

Analysis and results. A total of 581 subjects participated in study 3. We excluded 89 subjects based on three predefined criteria (Simmons, Nelson, and Simonsohn 2011): (1) taking 2 minutes or less or longer than 30 minutes to respond (37 subjects), (2) not passing an instructed manipulation test to measure attention (49 subjects) (Oppenheimer, Meyvis, and Davidenko 2009), and (3) being a self-reported art expert (3 subjects). Therefore, 492 subjects were used for the data analysis.

We first tested whether we could replicate the expectancy effects reported in studies 1 and 2a–c. The experienced utility ratings were entered as a dependent variable in a one-way within-subjects ANOVA with the artist vs. computer condition as predictor. We found a significant effect of

3 Note that although all the scale items used in study 3 were identical to those used in studies 2a–c, the scale anchors differed. In study 3 the reward-seeking subscale uses a 7-point Likert scale and the need for cognition scale uses a 5-point Likert scale.

our expectancy manipulation such that participants showed a higher experienced utility for seeing art pieces created by an artist vs. a computer, $F(1, 491) = 78.91$, $p < .001$.

Next, we tested whether individual differences in reward-seeking, private body consciousness, and need for cognition moderated the MPE of price following the same procedure to test for within-subject design moderators as applied in study 1 (Judd, Kenny, and McClelland 2001). Table 3 lists the results of this analysis; Figure 5 shows the correlation between MPE and (a) reward-seeking, (b) PBC, and (c) NFC scores. We found that the BAS ($T(1, 491) = 6.53$, $p < .001$) and NFC ($T(1, 491) = 2.44$, $p = .015$) scores indeed amplified expectancy effects on experienced utility ratings, whereas PBC moderated expectancy effects on experienced utility ratings ($T(1, 491) = –2.75$, $p = .006$).

Figure 5 about here

Discussion. Study 3 served as an important extension of studies 1 and 2, in that we conceptually replicated our previous results that individual differences in reward-seeking, somatosensory awareness, and cognitive focus moderated marketing expectancy effects on the consumption experience. Importantly, we could show that such effects also hold for a different type of expectancy effects (an artist's expertise) in another sensory domain (aesthetic consumption) and thus are not specific to pricing or health claim effects on food consumption.

GENERAL DISCUSSION

It is widely known that marketers can change how consumers perceive the consumption of their products and subsequent satisfaction, influencing not only purchasing decisions but also usage frequency and recommendation behavior. The existence of marketing placebo effects shows how fundamental the impact of marketing actions can be: Marketing actions change not only consumers' perceptions but also the biological processes underlying their consumption and purchasing decisions. In this paper, we have extended our understanding of the scope of the effects that marketing actions have in important ways: Using a novel application of structural brain imaging in combination with

behavioral experiments, we are among the first to shed light on individual difference variables that affect MPE. Across three studies we found first evidence for three individual differences in MPE on brain and behavioral level. Importantly, we also studied the generalizability of these individual difference effects for different types of marketing-based expectations (price, health claim, brand) and different types of consumption experiences (food and aesthetic consumption).

First, in studies 1, 2a, and 3 we showed that reward-seeking and motivational behavior play an important role in MPE. The more sensitive consumers' neural and behavioral signatures of reward-seeking systems are, the more responsive they are to MPE. Then, in studies 1, 2b, 2c, and 3 we provided first evidence that MPE rely on an interplay between higher-level cognitive top-down processing and lower-level somatosensory bottom-up processing. On one hand, we showed that increased GMV in brain regions involved in cognitive aspects of emotion generation and control, and participants' need for cognition and cognitive focus, favored the existence of MPE. On the other hand, we showed that the more subjects were able to focus on their internal, somatosensory states as compared to external cues and the smaller the GMV in the brain's somatosensory systems, the less responsive they were to MPE.

Understanding the underlying mechanisms of MPE is not only important from an academic perspective; it is also highly relevant for marketers and public policy institutions. Marketers usually aim at increasing consumers' consumption enjoyment, so they need to understand how they can leverage their marketing actions to contribute to greater consumption enjoyment. For example, several studies have shown how marketers are capable of changing their consumers' reward-seeking drive. A mouthwatering smell in a bakery and food samples in a supermarket are triggers for reward-seeking behavior (Wadhwa, Shiv, and Nowlis 2008). Understanding how such actions might interact with MPE is important from the perspective of a marketer.

However, caution is necessary for several reasons. First, an important limitation of the current findings is that they provide correlational and not causal evidence. This calls for future

studies that manipulate reward processing and cognitive and somatic focus rather than measuring individual differences relating to such processes as a personality trait variable. Another interesting extension of this work would be to manipulate neuro-pharmacological processes underlying MPE that relate to our current findings. For example, could the administration of a dopamine blocker attenuate MPE? These questions remain to be answered by future research.

A second point that deserves caution is that MPE might turn into disadvantageous effects on consumers' behavior and well-being. In this case, it is crucial for public policy institutions to understand the mechanisms underlying MPE so they can promote behavior that decreases the existence of MPE. Exploring the impact of other *specific* marketing or public policy actions and their interactions is a worthwhile direction for future research. Are MPE similar across domains, and do they all have the same underlying neurobiological mechanisms? How do different consumption situations affect MPE, and what are the boundary conditions? For example, how are price-based MPE affected by giving out the products for free as compared to having consumers pay for them?

An extreme case for the disadvantageous effects of MPE on consumer well-being that deserves further research is the study of patient populations that show dysfunction of the neurobiological processes underlying MPE. For example, obese patients are thought to have dopamine deficiencies (Volkow, Wang, and Baler 2011). Would our findings imply that they are more or less prone to be biased by healthfulness claims on the packaging? Along those lines, how do MPE affect other patient groups that have impaired dopaminergic pathways, such as addicts and people suffering from severe anxiety, ADHD, and OCD?

This paper can be seen as a showcase of how questions relevant to consumer behavior can benefit from an interdisciplinary consumer neuroscience approach. We first used existing theories in cognitive neuroscience about pain placebo effects to extend Shiv et al.'s 2005 model of how MPE work. This new model gave us novel concrete predictions about brain areas involved in neural processing anteceding MPE, and we then tested individual differences in the structure of these brain

regions in study 1. To do so, we used a brain-imaging tool that is new to consumer neuroscientists' toolkit to determine variability in gray matter volume to identify individual differences in MPE responsiveness. Importantly, consumer neuroscience aims at not only understanding brain structures and function important for a behavior of interest, but also at how brain systems can be linked to psychological variables such as personality traits and psychological states. Thus, rather than merely drawing on reverse inferences about the role of the brain regions showing individual differences in brain structure for MPE, we tested how the predicted brain regions translate into personality traits in studies 2 and 3. Although following such a methodological approach seems very fruitful for future consumer neuroscience studies, it is also important to understand its limitations (Yarkoni 2013).

We would like to highlight two important points: First, it is important to understand how variations in GMV arise. Since the development of modern MRI machines, which allow for the measurements of large numbers of subjects without the need for contrast agents or radiation, literally thousands of studies have been performed investigating the relationship of brain structure to a variety of individual measures. While in the early years of structural brain studies the need for manual volumetry hindered the analysis of large sample sizes, the development of automated techniques such as voxel-based morphometry (Ashburner and Friston 2000; Wright 1995) allowed researchers to increase sample sizes and to investigate individual differences and even longitudinal changes.

One important question that has been investigated is the role of genetic variation in the brain, an approach called imaging genomics (Thompson, Martin, and Wright 2010). These studies have shown that genes contribute to the development and structure of the brain. However, it is important to note that a variety of studies—mainly those performed longitudinally—clearly have shown the influence of our environment and not only of our genetic makeup on regional brain volumes. For example, Draganski and colleagues (2004) showed that extensive juggling training leads to an increase in gray matter volume in areas of the brain related to motion detection and visuomotor integration. These intervention studies have been performed mainly in the motor domain because of

the availability of controlled training programs and the easy assessment of behavioral changes, but some studies suggest the existence of similar mechanisms in other domains, such as learning and memory (Draganski et al. 2006; Maguire et al. 2000). Hence, it is important to note that variability in GMV is *not* due only to genetic predispositions but is also caused by environmental effects such as training and learning; consumers are not born with a specific MPE.

Second, although brain-based measures offer a new way of understanding individual differences in placebo responses, they can be limited because most of the procedures are designed to detect nonzero effects but not to estimate predictive accuracy (Nichols and Poline 2009; Vul et al. 2009; Wager et al. 2011). Thus, post hoc estimates of strengths of brain–MPE correlations might be inflated, and how accurately patterns of brain activity can predict MPE is unknown. In other words, future research is needed to use our extended model to predict activations underlying MPE using machine learning and pattern classification approaches.

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TABLES

TABLE 1

RELATION OF GRAY MATTER VOLUME AND EXPECTANCY EFFECTS ON EXPERIENCED UTILITY

Location	Z-Score / k	MNI Coordinates (Peak)		
		x	y	z
Positive correlation				
R. dmPFC (BA9)*	3.42/65	18	38	32
R. Putamen*	3.26/770	21	12	–1.50
L. lateral OFC	2.83/37	–35	56	–15
R. inf. Occipital	2.98/48	42	–84	–13.5
R. parahippocampal G.	2.93/13	21	–45	–8
L. cuneus	2.88/21	–6	–92	15
L. sup. temp. gyrus	2.90/16	–57	–63	20
Negative correlation				
R. inf. Temporal*	3.75/341	38	6	–48
L. rectal gyrus	2.84/26	–8	14	–24
R. sup. occ.*	3.25/98	32	–72	20
L. sup. occ.*	3.61/194	–23	–75	20
R. posterior insula	3.07/14	45	–12	23
L. precentral gyrus*	3.45/230	–40	–14	32
R. precentral gyrus*	3.67/196	41	5	48

* Also significant at $p < .001$.

Note: The coordinates represent the peak voxel in the respective clusters. Only $p < .005$ with an extend threshold ($k > 10$) are presented. Regions in bold are part of MPE model from Figure 1.

TABLE 2

TESTING FOR MODERATING EFFECTS OF GMV IN dmPFC, STRIATUM, AND POST INSULA: REGRESSIONS PREDICTING THE WITHIN-SUBJECTS MPE (EXPERIENCED UTILITY$_{\text{HIGH EXPECTATION}}$ − EXPERIENCED UTILITY$_{\text{LOW EXPECTATION}}$) FOR EACH SUBJECT IN EXPERIMENT 1, N = 90

Parameter	DV: MPE Model 1	DV: MPE Model 2
Intercept	–.96 (1.71) —	–.85 (1.12) —
GMV dmPFC	5.35 (1.55)*** 1.44	4.75 (1.49)** 1.33
GMV striatum	2.38 (.90)* 1.55	1.87 (.81)$^{\#}$ 1.27
GMV posterior Insula	–10.55 (3.58)** 1.28	–11.68 (3.45)*** 1.19
Gender	.04 (.18) 1.07	—
Age	.03 (.02) 1.55	—
R^2	.315	.298
RMSE	.807	.807
AIC	222.67	220.82

$^{\#}p < .05$. $p < .01$. $^{**}p < .005$. $^{***}p < .001$ (two-tailed p-value used in testing the null hypothesis that the parameter is 0)

Note: For each parameter, the first row shows unstandardized regression coefficients, with standard errors in parentheses; the second row shows the variance inflation factor to quantify multicollinearity issues in this regression.

TABLE 3

TESTING FOR MODERATING EFFECTS OF PERSONALITY SCALES: REGRESSIONS PREDICTING THE WITHIN-SUBJECTS MARKETING PLACEBO MPE (EXPERIENCED UTILITY$_{\text{HIGH EXPECTATION}}$ − EXPERIENCED UTILITY$_{\text{LOW EXPECTATION}}$) FOR EACH SUBJECT

Parameter	Study 2a, N=89 DV: MPE	Study 2b, N=85 DV: MPE	Study 2c, N=79 DV: MPE	Study 3, N=491 DV: MPE
Intercept	–.79 (.96)	–.73 (.42)	.73 (.42)	–.1.58 (.40)
BAS	.58 (1.94)***	—	—	.31 (.05)**** 1.02
PBC	—	–.27 (.09)***	—	–.14 (.05)*** 1.01
NFC	—	—	.22 (.09)*	.21 (.09)** 1.01
R^2	.082	.09	.07	.12
RMSE	1.740	1.0	.87	1.15

Note: For each parameter, the first row shows unstandardized regression coefficients, with standard errors in parentheses. The second row shows the variance inflation factor to quantify multicollinearity issues in this regression.
*$p < .05$ (two-tailed p-value used in testing the null hypothesis that the parameter is 0)
**$p < .01$ (two-tailed p-value used in testing the null hypothesis that the parameter is 0)
***$p < .005$ (two-tailed p-value used in testing the null hypothesis that the parameter is 0)
****$p < .001$ (two-tailed p-value used in testing the null hypothesis that the parameter is 0)

FIGURES

FIGURE 1:

EXTENDED FRAMEWORK OF HOW MARKETING PLACEBO EFFECTS WORK AND FRAMEWORK FOR STUDIES 1–3

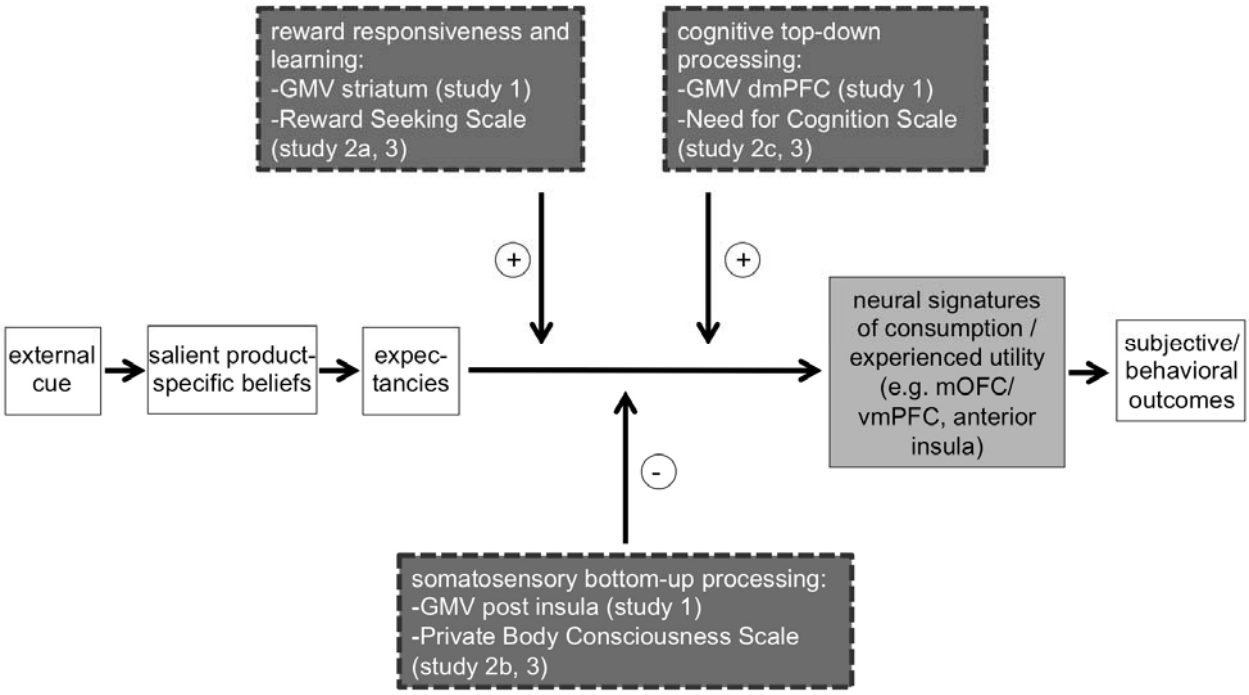


FIGURE 2:

OVERVIEW VOXEL-BASED MORPHOMETRY (VBM) APPROACH

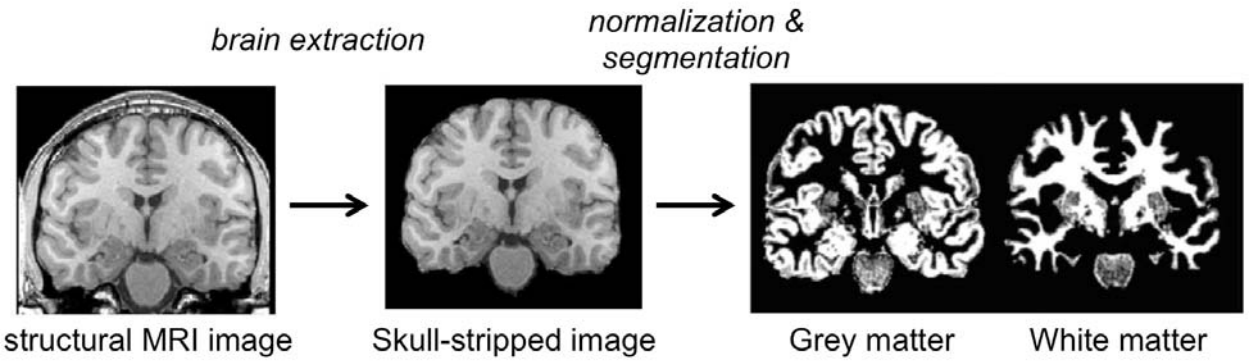


FIGURE 3:

RESULTS OF VBM ANALYSIS: GRAY MATTER VOLUME IN STRIATUM, POSTERIOR INSULA, AND dmPFC CORRELATE WITH SIZE OF MPE

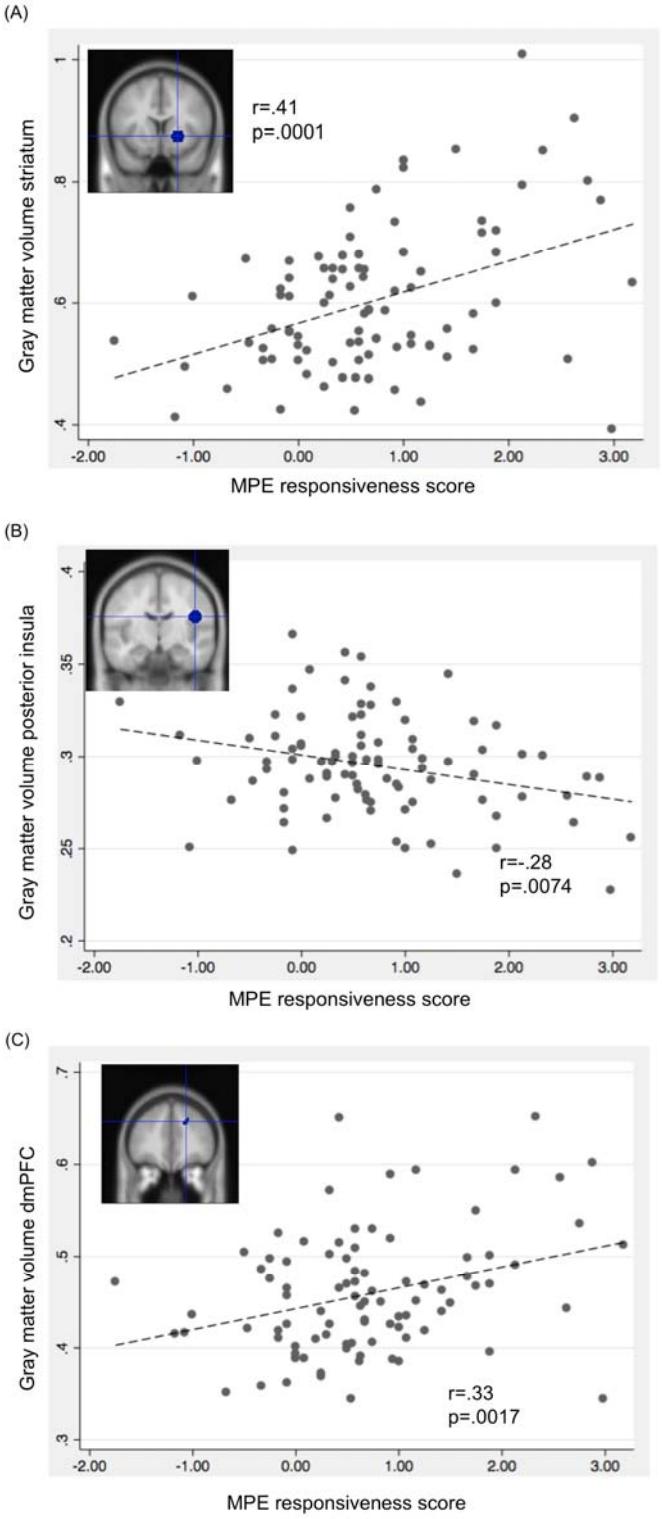


FIGURE 4:

RESULTS FROM STUDIES 2a–c: REWARD-SEEKING, COGNITIVE PROCESSING, AND SOMATOSENSORY AWARENESS MODERATE PRICE EFFECTS ON FOOD PLEASANTNESS

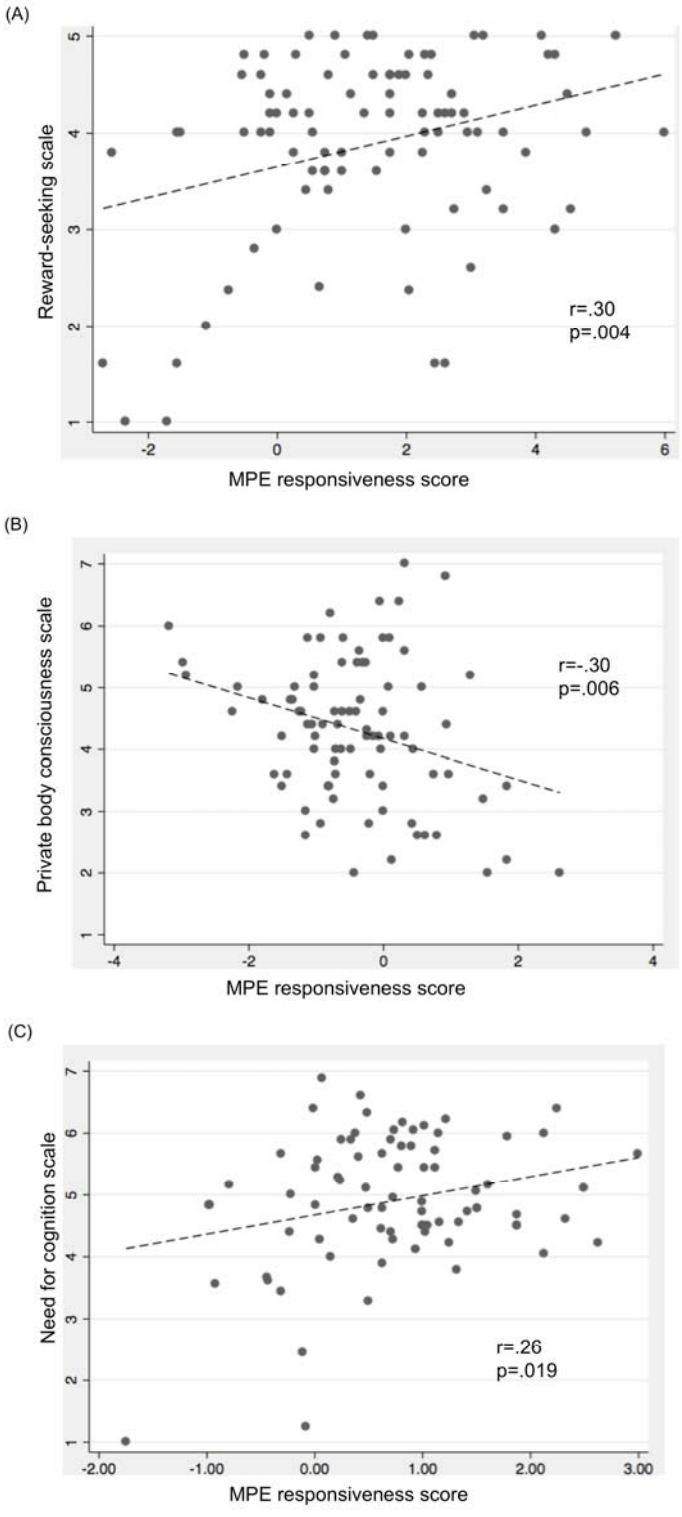
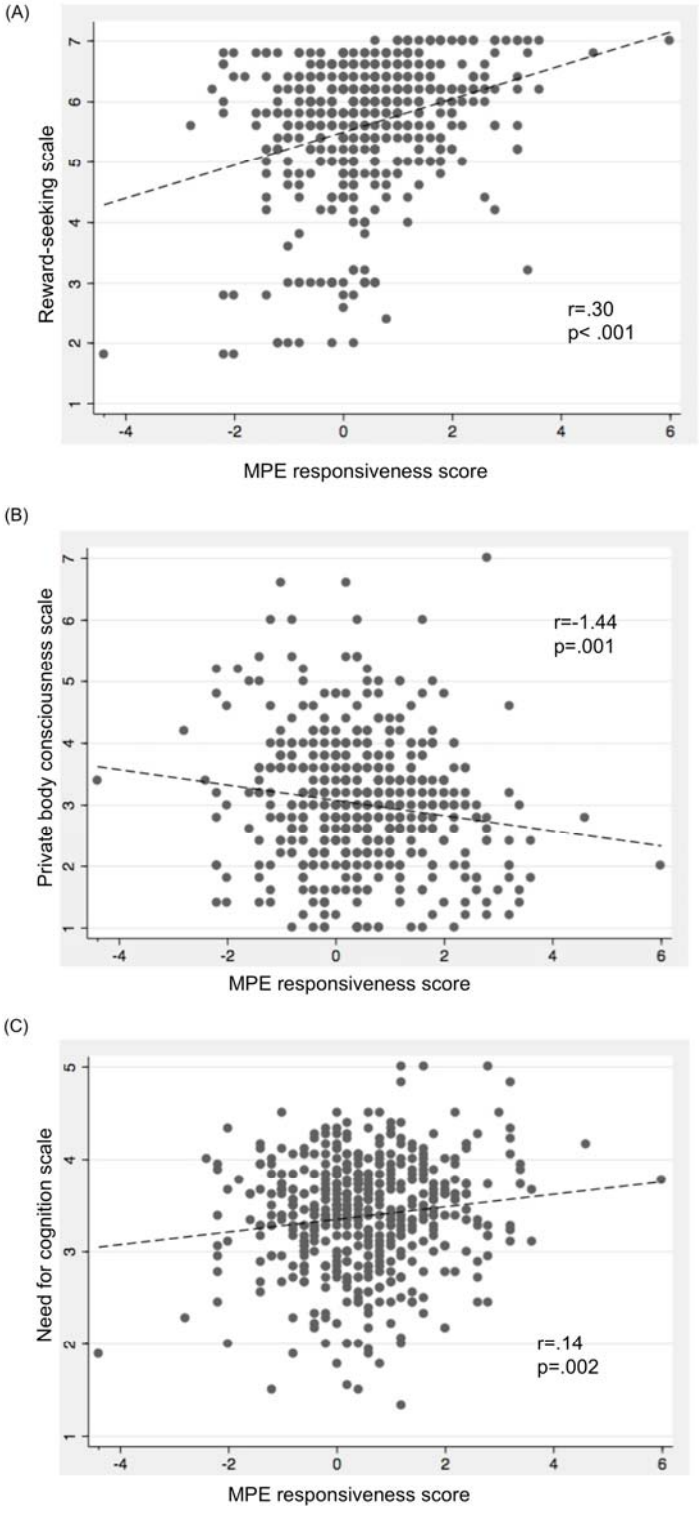


FIGURE 5:

RESULTS FROM STUDY 3: REWARD-SEEKING, COGNITIVE PROCESSING, AND SOMATOSENSORY AWARENESS MODERATE THE EFFECTS OF AN ARTIST'S EXPERTISE ON AESTHETIC PLEASANTNESS



METHODOLOGICAL APPENDIX

In this appendix we detail two methodological aspects of the current paper. We first provide more methodological details about the structural brain analysis that we applied and then give more detail about the methodology and limitations of Yarkoni et al's NeuroSynth framework that has been applied in this paper.

Additional methodological details about voxel-based morphometry (VBM) analysis study

Voxel-based morphometry is an approach that allows comparison of the volume of tissues, in our case brain tissue, between or within groups of subjects (for a detailed review see Ashburner 2009). It is based on the following steps: 1) segmenting the individual brain into different tissue types (usually gray matter, white matter, and cerebrospinal fluid); 2) anatomical normalization of the individual brains to a common template to allow comparison of similar brain areas across subjects; and 3) correlating a factor of interest (e.g., group or behavioral measure) to the individual voxel-wise data.

Here we are applying a more recent and more sophisticated registration method, the DARTEL approach (Ashburner 2007). In contrast to previous normalization procedures it includes a much higher number of parameters for estimating the normalization, which allows for much better between-subject realignment. Also, for the normalization procedure, the DARTEL approach generates a study-specific template. This algorithm has been shown in several studies to provide a very high degree of accuracy compared to the gold standard of manual segmentation (see, for example, a recent proof-of-concept study by Focke et al. 2014).

To execute the VBM analysis we have used the VBM8 toolbox. It integrates the different processing steps into a single toolbox and provides tools for quality control of the data. As suggested in the VBM8 toolbox manual, we used a modulation of the voxel-wise information for nonlinear deformations only, which provides information on the local gray matter volume corrected for individual brain sizes.

Additional details about large-scale automated synthesis of human functional neuroimaging data framework

To add to the methodological details about the NeuroSynth framework from Yarkoni et al. that was briefly outlined in the main text, we here describe how the numbers of the different measures are generated, then provide a brief discussion of the limitations of this approach.

NeuroSynth is a fully automated approach that allows rapid and scalable synthesis of the vast neuroimaging literature. It can be used to "generate large-scale meta-analyses for hundreds of broad psychological concepts; support quantitative inferences about the consistency and specificity with which different cognitive processes elicit regional changes in brain activity; and decode and classify broad cognitive states in new data solely on the basis of observed brain activity" (Yarkoni et al. 2011, p. 665).

In a nutshell, the methodology behind this approach is as follows. For details see Yarkoni et al. (2011).

1. Activation coordinates are extracted from about 6,000 published neuroimaging articles using an automated parser.
2. The full text of all articles is parsed, and each article is tagged with a set of terms that occur at a high frequency in that article. The threshold for "high frequency" is arbitrarily set at .001.

3. A list of several thousand terms that occur at high frequency in 20 or more studies is generated.
4. For each term of interest (e.g., “emotion,” “reward,” etc.), the entire database of coordinates is divided into two sets: those that occur in articles containing the term, and those that don’t.
5. A large-scale meta-analysis is performed comparing the coordinates reported for studies with and without the psychological term of interest. On this basis the different maps described in the main text are generated (i.e., z- and *p*-value maps and posterior probability maps).

Although the NeuroSynth framework is extremely promising, its application requires caution given that the development of the framework is still in its infancy. These are the most important limitations:

1. NeuroSynth is subject to a publication bias of null effects not being published.
2. The current version does not distinguish how specific terms are used because it applies a highly automated approach. For example, a paper stating that the amygdala was not found to correlate with fear processing would still be included in NeuroSynth’s algorithm when linking amygdala to fear. This and related problems of NeuroSynth are similar to sentiment analysis using online data and have been further advanced in that research area in marketing. Thus, such problems might be solved in the future.
3. For technical reasons, NeuroSynth currently includes only a subset of published neuroimaging studies and needs continuous update.

Taken together, NeuroSynth and similar approaches are an excellent first step in the right direction but will need further improvements. That is why it is essential to test the reverse inferences based on NeuroSynth measures in follow-up behavioral experiments, a procedure implemented in this paper.