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A wealth of research has explored 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." In this article, the authors suggest three moderators of the effect of marketing-based expectancies on the behavioral and neural measures of the consumption experience, based on previous findings from neuroscientific literature investigating traditional clinical pain placebo effects. They use a novel automated structural brain imaging approach to determine individual differences and combine this approach with traditional behavioral experiments. The findings show that consumers high in reward seeking, low in somatosensory awareness, and high in need for cognition are more responsive to marketing placebo effects.

Keywords: individual differences, placebo effects, structural brain imaging

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

Multidisciplinary evidence suggests that psychological associations and cognitive concepts influence the value or enjoyment people derive from consumption. For example, effects of brand images, quality or efficacy beliefs about products and treatments, expertise of artists, and nutritional information on food packaging can alter—and in extreme cases even override—the mere physical sensory consumption experience (Ariely and Norton 2009; Plassmann and Wager 2013). Such cognitive concepts are learned over time and shaped by everyday experiences with products, services, and social influences.

One important driver of how such cognitive concepts influence consumption is expectancies, or 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). Therefore, 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 (e.g., wines, 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). These price-based expectancies not only change 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

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anterior part of the insula for the experienced displeasure of feeling pain (Geuter et al. 2013). As such, marketing-based expectancy effects are often referred to as "marketing placebo effects" (MPEs) (Shiv, Carmon, and Ariely 2005a, b).

Indeed, MPEs go far beyond expectancies based on price and quality; a large body of literature in consumer psychology has investigated how people's expectancies shape consumption experiences. One of the first articles on this topic showed that brand label information can alter how much people enjoy consuming different beers (Allison and Uhl 1964). Converging evidence also exists for marketing-based expectancy effects 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, Naylor, and Hoyer 2006; Steenhuis et al. 2010; Wansink and Chandon 2006; Wilcox, Roggeveen, and Grewal 2011; 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 postconsumption 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, 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 et al. (2005) investigated the influence of verbal labels of smells (cheese vs. body odor) on neural signatures of olfactory processing. They found that when participants smelled identical odors, a positive or negative description altered neural activity in the vmPFC and also in the bilateral amygdala, both of which are linked to olfactory processing. Nitschke et al. (2006) found that expecting an aversive taste to be less aversive decreased neural activity in the primary taste cortex, which involves taste intensity encoding, though they kept intensity of the negative taste constant. Finally, Kirk, Skov, Hulme, et al. (2009) found that the pleasure participants derived from viewing art pieces, and the accompanying engagement of the vmPFC, was higher when the participants believed that they were created by an expert (i.e., an artist) rather than by a nonexpert (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 MPEs to occur.

Against this background, the goal of this article is to shed light on individual differences in MPEs. To reach this goal, we first draw on neuroscientific evidence for the underlying mechanisms of pain placebo effects to extend Shiv, Carmon, and Ariely's (2005a, b) model of MPEs and suggest a multidisciplinary model of how marketing-based expectancies alter subjective consumption experiences. We then test the novel aspects of this model with various MPEs (e.g., 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 evaluate individual trait-related differences (Study 1). We find that the volume of gray matter in the striatum, the posterior insula, and the dorsomedial prefrontal cortex (dmPFC) 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 that links each of these brain areas with personality traits (i.e., the striatum with reward seeking, the posterior insula with somatosensory awareness, and the dmPFC 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–2c). In Study 3, we test the robustness and generalizability of our effects by examining whether reward responsiveness, somatosensory awareness, and need for cognition also jointly moderate the effects of the perceived expertise of artists on subjective aesthetic experiences. We conclude by discussing the implications of our findings for marketing and consumer neuroscience, highlighting the limitations of our work, and calling for further research.

THEORETICAL AND METHODOLOGICAL BACKGROUND

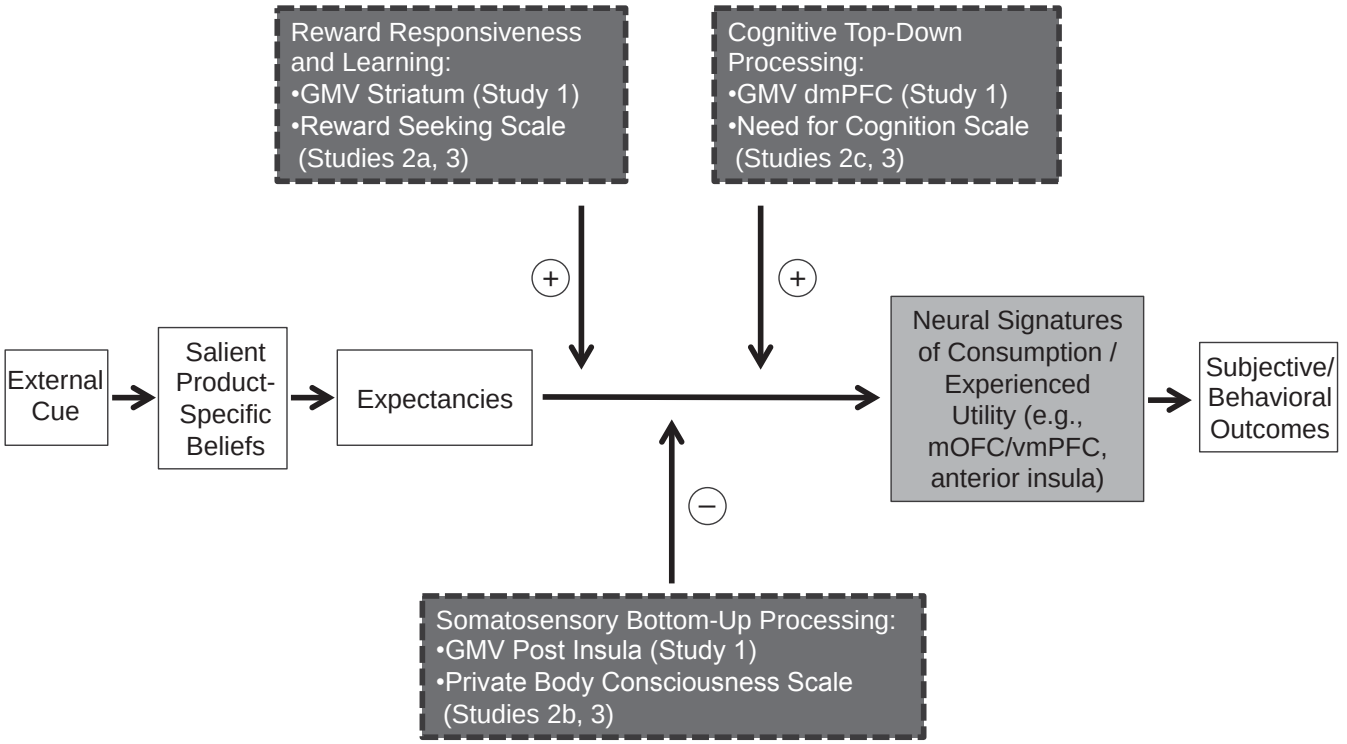
Drawing from existing theories in cognitive neuroscience on pain placebo effects, we first suggest an extended model of processes underlying marketing-based expectancy effects. We then provide further methodological details about the novel automated structural brain imaging approach we apply herein.

Behavioral and Brain Mediators of Placebo Effects

Shiv, Carmon, and Ariely (2005b) were the first to suggest a model of MPEs and how they work. 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, generate response expectancies of the benefits of the energy drink that, in turn, change behavioral outcomes, such as the number of puzzles solved in a mental-effort task. We incorporate 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 MPEs.

Recent research in cognitive neuroscience has extended Shiv, Carmon, and Ariely's (2005b) model to shed light on the underlying neural signatures of MPEs. The experiments reviewed previously 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 MPEs, we reviewed

Figure 1
EXTENDED FRAMEWORK OF HOW MPEs WORK AND FRAMEWORK FOR STUDIES 1–3



Notes: GMV = gray matter volume; mOFC = medial orbitofrontal cortex; vmPFC = ventromedial prefrontal cortex; dmPFC = dorsomedial prefrontal cortex.

studies in the domain of traditional pain placebo effects that examine 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 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 have suggested a link between expectancy effects and dopaminergic functioning. Atlas et al.'s (2010) study 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 versus low pain and changes in the pain experience. After an initial learning phase, they kept the actual level of pain intensity constant. They found that a subset of pain-responsive regions formally mediated trial-by-trial expectancy effects on pain and that, in turn, expectancy effects in these regions were 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 have investigated the role of reward responsiveness for pain placebo effects, finding 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 studies reviewed in the introduction reveal that expectations alter consumption-related behavioral responses and responses in consumption-related brain regions, some studies on pain placebos have found that expectancy effects in the striatum, among other regions, predict expectancy-enhanced placebo analgesia (e.g., Kong et al. 2006).

In addition, 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. In their study, De la Fuente-Fernández et al. (2001) even showed the power of placebo effects by providing in vivo evidence for the 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.

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 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 exhibit higher MPEs.

Processing in the posterior insula cortex and somatosensory cortices linked to awareness of sensory processing and bodily states. We can suggest another mechanism from our literature review of pain placebo effects in cognitive neuroscience. In Wager et al.'s (2011) pattern classification analysis of individual difference predictors of pain placebo effects, activity in the somatosensory system (i.e., somatosensory cortices and posterior insula) showed a negative correlation with pain placebo effects. Atlas et al. (2010) also found that these areas formally mediate pain placebo effects. 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 (dis)liking of the pain experience—that is, experienced (dis)utility.

However, studies investigating expectancy effects on flavor processing have also found that expectancy effects dampen brain activity in somatosensory areas (Atlas et al. 2014; Nitschke et al. 2006). On this basis, we suggest a more general role for somatosensory processing to underlie MPEs. 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 MPEs work and that people who are more aware of their somatosensory states are less responsive to MPEs.

Prefrontal processing thought to be involved in cognitive regulation and appraisal of emotional states. Other potential mechanisms underlying MPEs that we can derive from the existing findings on pain placebo effects are linked to cognitive processes involved in emotion regulation and appraisal. For example, Wager, Scott, and Zubieta (2007) found a correlation between the magnitude of pain placebo effects on reported pain and the magnitude of heat-evoked responses in pain-processing brain regions. 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. Indeed, in a new analysis of their data using machine-learning and pattern classification techniques to investigate individual differences in pain placebo effects, Wager et al. (2011) showed that increased anticipatory responses in a frontal (i.e., lateral orbitofrontal, lateral prefrontal, and dmPFC) 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.

Atlas et al. (2010) and Atlas et al. (2014) showed similar findings in their formal mediation analyses of pain expectancy effects and health label expectancy effects, respectively. In addition to pain- and taste-processing regions, Atlas and colleagues found that the lateral prefrontal cortex and the dmPFC mediated expectancy effects on pain and taste perceptions. In our model, we suggest that higher-order, top-down cognitive processes of regulating emotional states and emotion generation play a role as an intervening variable for MPEs to occur. In turn, people who rely more on such cognitive systems (i.e., have a cognitive focus) during decision making should be more responsive to MPEs.

Taken together, expectancies might affect consumption-related circuitry not only because they simulate the consumption experience and experienced utility before 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 appear in the dark gray boxes in Figure 1 and are the individual differences we investigate herein.

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

In the past decade, an increasing number of studies 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 these studies have used functional magnetic resonance imaging (fMRI) to establish associations between brain processes and consumer behavior (Kable 2011). Although 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 article, we follow this idea and use an approach novel to consumer neuroscience that is more suitable than fMRI for investigating individual differences on a trait level: automated structural magnetic resonance imaging (MRI) analysis. Differences in brain structures, such as gray matter volume, can be linked to individual differences in brain function, personality, and behavior. All 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 become an important new method in the tool kit of consumer neuroscience.

Research has shown that MRI-based measures of gray matter are 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,

research has linked individual anatomical differences—for example, within reward-related dopaminergic pathways—to significant differences in behavioral effects, including variation of personality traits (Depue and Collins 1999).

In line with this idea, in recent years a wealth 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; 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 participants 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 as 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 for 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 (for the processing flow of the images, see Figure 2).

We first applied VBM to explore brain regions that showed a variation in gray matter volume 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 MPES DURING FOOD CONSUMPTION

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

H_{1a}: The greater the gray matter volume in the striatum and prefrontal structures (i.e., lateral orbitofrontal, lateral prefrontal, and dmPFC), the more responsive participants are to MPEs.

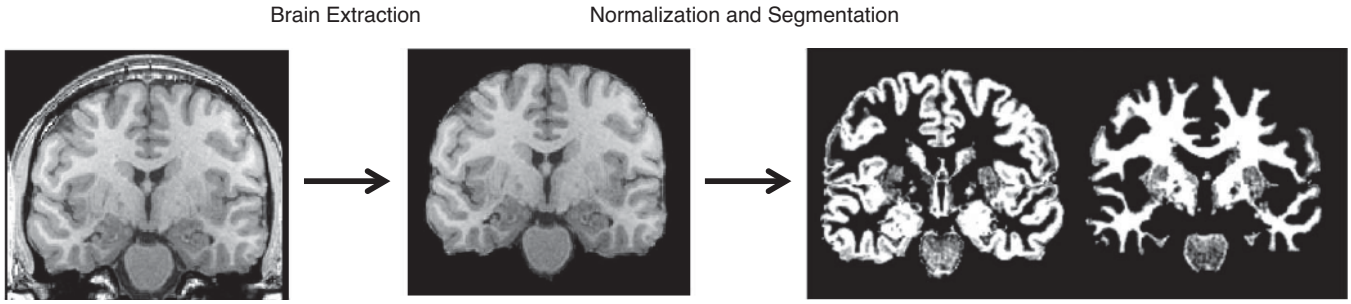
H_{1b}: The greater the gray matter volume in the posterior insula and somatosensory cortices, the less responsive participants are to MPEs.

To test these hypotheses, we used the structural neuroimaging data from three experiments that investigated neural correlates of MPE using functional neuroimaging data. We pooled the structural neuroimaging data of the three experiments to ensure an appropriate sample size for such an exploratory analysis (Simmons, Nelson, and Simonsohn 2013). These studies investigated two types of MPEs: prices (high vs. low) and healthfulness claims (light vs. regular and organic vs. regular). Considering the seemingly similar underlying neural mechanisms of placebo effects (Atlas and Wager 2013; Plassmann and Wager 2013; Wright et al. 2013), this seems a reasonable approach. Next, we briefly describe the design of the 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-subject 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 served as a distractor, with an identical instructed and actual retail price of \$35. During the experiment, 20 participants (11 men, mean age 24.5 years) believed that they would consume five different wines with different retail prices (\$90, \$45, \$35, \$10, and \$5) while their brains were scanned using fMRI. However, in reality they consumed only three different wines, two of which were administered with two sets of prices (wine 1: \$90 and \$10; wine 2: \$45 and \$5) to keep the physical consumption constant. Participants showed a significant effect of price on experienced utility on a behavioral level (six-point Likert scale; 1 = "not at all," and 6 = "very much") and, more

Figure 2
OVERVIEW OF VBM APPROACH



importantly, also on a neural level (for the details of the results, see Plassmann et al. 2008). In the current study, we were interested in whether gray matter volume in specific brain structures (i.e., those in our model) would moderate MPEs. Against this background, we entered the behavioral and structural neuroimaging data of the 20 participants in a novel application of automated, structural brain imaging analysis—namely, the VBM analysis.

The second experiment was similar to the first and served as a neuroimaging pilot study (N = 12, 6 men, mean age of 30.3 years) for an extended version of Plassmann et al.'s (2008) Experiment 1. This extension consisted of two points: First, instead of using wines of different actual retail price classes, Experiment 2 used wines of the same price class (€10–€13) and randomly assigned the wines to different instructed price conditions (€3, €16, and €18). Second, a condition that varied whether the participants received the wines for free as in Experiment 1 or had to pay for the wine was added. In this second experiment, the behavioral effects of the price condition from the first experiment could be replicated, but the experiment found no significant results of the payment condition or significant interaction effect (for the reported results, see Skvortsova et al. 2013). In the current study, we used the behavioral and structural neuroimaging data of the 12 participants for the VBM analysis.

The third experiment used different types of product labels instead of prices to generate different expectations of the pleasantness of the product. More specifically, different types of healthfulness claims were used for milkshakes that were shown to create either positive expectations of the pleasantness of the taste ("organic"; Lee et al. 2013) or negative expectations of the pleasantness of the taste ("light"; Chandon and Wansink 2012; Raghunathan, Naylor, and Hoyer 2006; Werle, Trendal, and Ardito 2013); there was also a neutral condition ("regular").[1] In other words, this experiment applied a one-factorial between-subjects design with a healthfulness label of a vanilla or chocolate milkshake. In total, 58 participants took part in this experiment (28 men, M_{age} = 27 years, SE = 4.25). One group of participants (N = 29) consumed identical milkshakes but believed that they were either organic or regular; the other group (N = 29) consumed identical milkshakes but believed that they were either light or regular. While participants were drinking, their brains were scanned using fMRI. Participants showed a significant effect of healthfulness label on experienced utility on a behavioral level and, more importantly, also on a neural level (for details of the results, see Atlas et al. 2014). In the current study, we were again interested in whether gray matter volume in specific brain structures outlined in our model would moderate MPEs. Against this background, we entered the behavioral and structural neuroimaging data of the 58 participants in our VBM analysis. Thus, we included 90 participants who took part in one of these three experiments in our 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). An eight-channel head coil was used for signal reception. All sequences were performed using a T1-weighted MP-RAGE sequence with a resolution of 1 × 1 × 1 millimeter (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).

We performed VBM techniques (Ashburner and Friston 2000) 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 with the high-dimensional DARTEL algorithm as implemented in SPM8. Gray matter data sets 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 MPEs, 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.

To address our specific a priori hypotheses, 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

Table 1
GRAY MATTER VOLUME AND EXPECTANCY EFFECTS ON EXPERIENCED UTILITY

		Montreal Neurological Institute Coordinates (Peak)		
Location	*Z-Score/k*	*x*	*y*	*z*
Positive Correlation				
*R. dmPFC (BA9)**	3.42/65	18	38	32
*R. putamen**	3.26/770	21	12	–1.5
L. lateral orbitofrontal cortex	2.83/37	–35	56	–15
R. inferior 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. superior temporal 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. superior occipital*	3.25/98	32	–72	20
L. superior occipital*	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 $p < .001$.

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

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

regression analysis. Importantly, this ROI analysis was independent of the whole-brain analysis because we defined the ROIs according to independent studies in the literature. More specifically, we defined the ROIs 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 striata, based on the work of Bartra, McGuire, and Kable (2010) (–12/12/–6 and 12/10/–6); for the posterior insula, a region based on the work of Benedetti et al. (2003) on placebo effects (44/–15/4); and for the dmPFC, a region based on a study by Ochsner et al. (2004) on cognitive regulation and emotion reappraisal (–10/50/34). 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 posterior insula, and the dmPFC moderate MPEs, we followed Judd, Kenny, and McClelland's (2001) suggested procedure for within-subject designs: we entered the MPE responsiveness score as a dependent variable in a regression analysis with gray matter volumes in the striatum, the posterior insula, and the dmPFC as predictors, controlling for age and gender (Model 1). We entered age and gender in the first regression model because both can 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. 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 gray matter volume in all three brain structures moderates MPEs (see Figure 3). We found a positive relationship for gray matter volume in the striatum (standardized beta coefficient: .30) and the dmPFC (standardized beta coefficient: .37) but a negative relationship in the posterior insula (standardized beta coefficient: –.30).

Table 2

TESTING FOR MODERATING EFFECTS OF GRAY MATTER VOLUME IN dmPFC, STRIATUM, AND POSTERIOR INSULA: REGRESSIONS PREDICTING THE WITHIN-SUBJECT MPEs (EXPERIENCED UTILITY$_{\text{HIGH EXPECTATION}}$ − EXPERIENCED UTILITY$_{\text{LOW EXPECTATION}}$) FOR EACH PARTICIPANT IN STUDY 1

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

†$p < .05$.
*$p < .01$.
**$p < .005$.
***$p < .001$.

Notes: N = 90. We used the two-tailed p-value used in testing the null hypothesis that the parameter is 0. 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. GMV = gray matter volume; DV = dependent variable; RMSE = root mean square error; AIC = Akaike information criterion.

Discussion

We found three brain regions unrelated to neural signatures of experienced utility to correlate with individual differences of MPEs: the striatum, the posterior insula, and the dmPFC. We want to acknowledge that there is ongoing debate about the robustness of structural brain behavior (SBB) correlations. A recent study failed to replicate SBB correlations in five experiments (Boekel et al. 2015). Although we cannot exclude that our results may fail to replicate in future studies, as we have not conducted such a replication, we believe that our results are more robust for three reasons: (1) our analysis is based on three different data sets, which should control for some random effects; (2) we provide additional behavioral evidence from two studies (i.e., Studies 2 and 3, described subsequently), which is actually based on and informed by the SBB correlations, providing some independent evidence for our effects; and (3) according to Simonsohn (2015), failures to replicate need to be treated with caution. For example, in the case of Boekel et al.'s (2015) replication attempts of SBB, we might argue that their replications failed to reproduce previous findings because their studies were underpowered. Note that Simonsohn argues for a sample size of the replication study of 2.5 times the sample size of the original study.

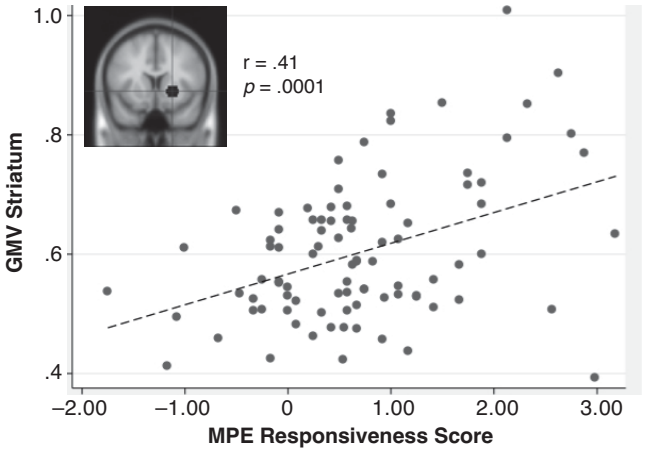
A crucial next question from a consumer research standpoint is: What psychological variables can be linked to changes in gray matter volume in these areas? This question should be answered with caution because a given brain region can be involved in several unrelated psychological processes, a problem 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 Yarkoni et al.'s (2011) suggested framework to compute measures of how selective and how specific our brain regions of interest are. Their approach allows for 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 are based on nearly 200,000 activations from almost 6,000 fMRI studies and thus contain a broad set of term-to-activation mappings, which makes this framework well suited for drawing quantitative inferences about mind–brain relationships.

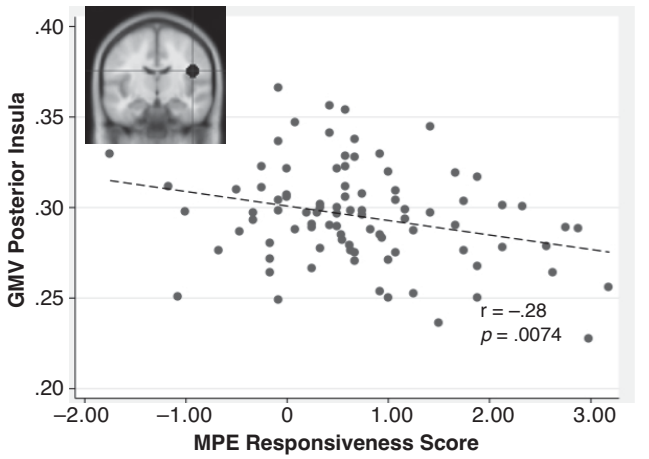
The measures suggested in this framework are as follows: (1) z-scores corresponding to the likelihood that a region

Figure 3
RESULTS OF VBM ANALYSIS: GRAY MATTER VOLUMES IN STRIATUM, POSTERIOR INSULA, AND dmPFC CORRELATE WITH SIZE OF MPEs

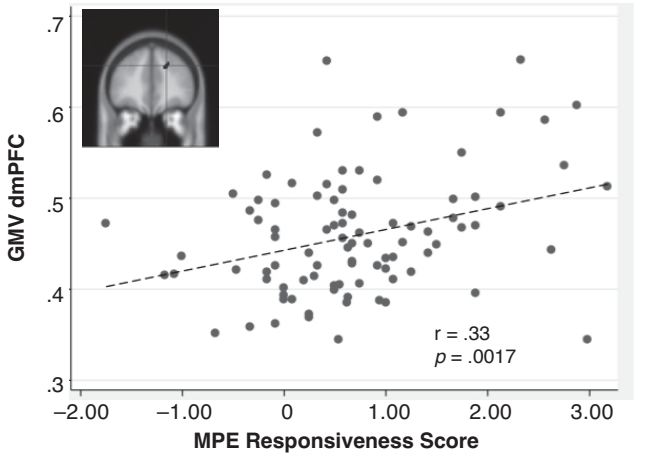
A: Striatum



B: Posterior Insula



C: dmPFC



Notes: GMV = gray matter volume.

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 viewed 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 selectivity—that is, of reverse inference. The Methodological Appendix provides more details on Yarkoni et al.'s (2011) framework.

As a second step, we 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 according to theoretical considerations of 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 a forward inference value of $Z_{forward} = 9.05$, signifying consistency, and a reverse inference value of $Z_{reverse} = 6.23$ (posterior probability = .78), signifying that selectivity of this area is 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 previously.

The subarea of the insula (i.e., the posterior parts) in which the average of the whole-brain VBM analysis fell has a forward inference value of $Z_{forward} = 4.75$, signifying consistency, and a reverse inference value of $Z_{reverse} = 4.14$ (posterior probability = .78), signifying that selectivity of this area is 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). Other large-scale meta-analyses have 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 that the neural activity in the posterior part of the insula is linked to somatosensory awareness.

Finally, 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 that selectivity of this area is 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, and based on Yarkoni et al.'s (2011) approach, these results show that the dmPFC is involved in various psychological processes linked to cognitive processing of emotional states and experiences. Here, we also note additional results from other meta-analyses investigating psychological processes that are in line with our model's theoretical considerations: 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 provide first evidence that individual differences in MPEs are linked to reward processing as signified by differences in gray matter volume in the striatum, somatosensory awareness as signified by differences in gray matter volume in the posterior insula, and cognitive appraisal of emotional experiences as signified by differences in gray matter volume in dmPFC.[2] To provide further evidence for this new model of how MPEs work, we tested the individual difference on a personality trait level in Studies 2a–2c.

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

To test the predictions about individual differences of MPEs on the psychological personality trait level derived from our conceptual model and also the brain imaging results from Study 1, we undertook three experiments to offer further evidence for our model of how MPEs work. The logic of these studies is as follows: All three studies (2a, 2b, and 2c) investigate the influence of high versus low price tags on food consumption, each testing a different one of the three moderators. The design of these studies is almost identical to Experiment 1 by Plassmann et al. (2008) described previously. The only differences are that Study 2a uses price levels that were adapted to 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; and 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) and "very much" (coded as 7).

Study 2a: Moderation of MPEs 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 MPEs. This prediction was based on our finding in Study 1 that higher gray matter volume in the striatum was linked to higher MPE responsiveness and also 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).

H_{2a}: MPEs are more pronounced the higher participants 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 BAS scale (Carver and White 1994). The scale has items such as "When I get something I want, I feel excited and energized," and participants answered them on a five-point Likert scale (1 = "do not agree at all," and 5 = "completely agree").

Analysis and results. We first tested whether we could replicate the expectancy effects reported in Plassmann et al.'s (2008) Experiments 1 and 2 in Study 1. We excluded one participant because he paid no attention to the task. Thus, we entered experienced utility ratings of 89 participants as a dependent variable in a one-way within-subject analysis of variance (ANOVA) with the high versus low price condition (pooled over both wines) as a predictor. We found a significant effect of price ($F(1, 88) = 57.07$, $p < .001$).

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

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

Study 2b: Moderation of MPEs by Somatosensory Awareness as a Personality Variable

Another noteworthy result from the VBM analysis was a negative correlation between MPEs and the gray matter volume in the posterior part of the insula, which, as we outlined previously, 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 MPEs.

More concretely, when MPEs 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 wine or a 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 MPEs. We measured somatic awareness using the private body consciousness (PBC) subscale of the Body Consciousness Questionnaire (Miller, Murphy, and Buss 1981). We predicted the following:

H_{2b}: MPEs are less pronounced the higher participants score on the PBC subscale.

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

Table 3

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

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)	–1.56 (.41)
BAS	.58 (1.94)**	—	—	.31 (.05)***
				1.02
PBC	—	–.27 (.09)***	—	–.14 (.05)**
				1.01
Need for cognition	—	—	.22 (.09)†	.21 (.09)*
				1.01
R^2	.082	.09	.07	.12
RMSE	1.740	1.0	.87	1.15

†$p < .05$.
*$p < .01$.
**$p < .005$.
***$p < .001$.

Notes: We used the two-tailed p-value in testing the null hypothesis that the parameter is 0. 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. DV = dependent variable; RMSE = root mean square error.

Design and procedure. Eighty-five alumni of a North American university (45 men, 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 PBC 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 participants responded on a seven-point Likert scale (1 = "strongly disagree," and 7 = "strongly agree"). People who score high on the PBC scale tend to pay more attention to somatosensory processes. For example, people high in PBC tend to report more pain than those low in this characteristic (Ferguson and Ahles 1998). Research has also linked PBC 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. We entered the experienced utility ratings of 85 participants as a dependent variable in a one-way within-subject ANOVA with the high versus low price condition (pooled over both wines) as a predictor. We found a significant effect of price ($F(1, 84) = 23.55$, $p < .001$).

We then tested whether differences in PBC moderated the MPE of price, following the same procedure to test for within-subject design moderators as in Study 1 (Judd, Kenny, and McClelland 2001). Table 3 lists the results of this analysis, and Figure 4, Panel B, shows the correlation between the PBC scores and MPEs. 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 show that a consumer's somatosensory awareness as measured by the PBC subscale is indeed negatively correlated with the strength of MPE. The more aware participants were of their internal bodily signals, the less price influenced their experienced utility of wine tasting.

Study 2c: Moderation of MPEs by Need for Cognition as a Personality Variable

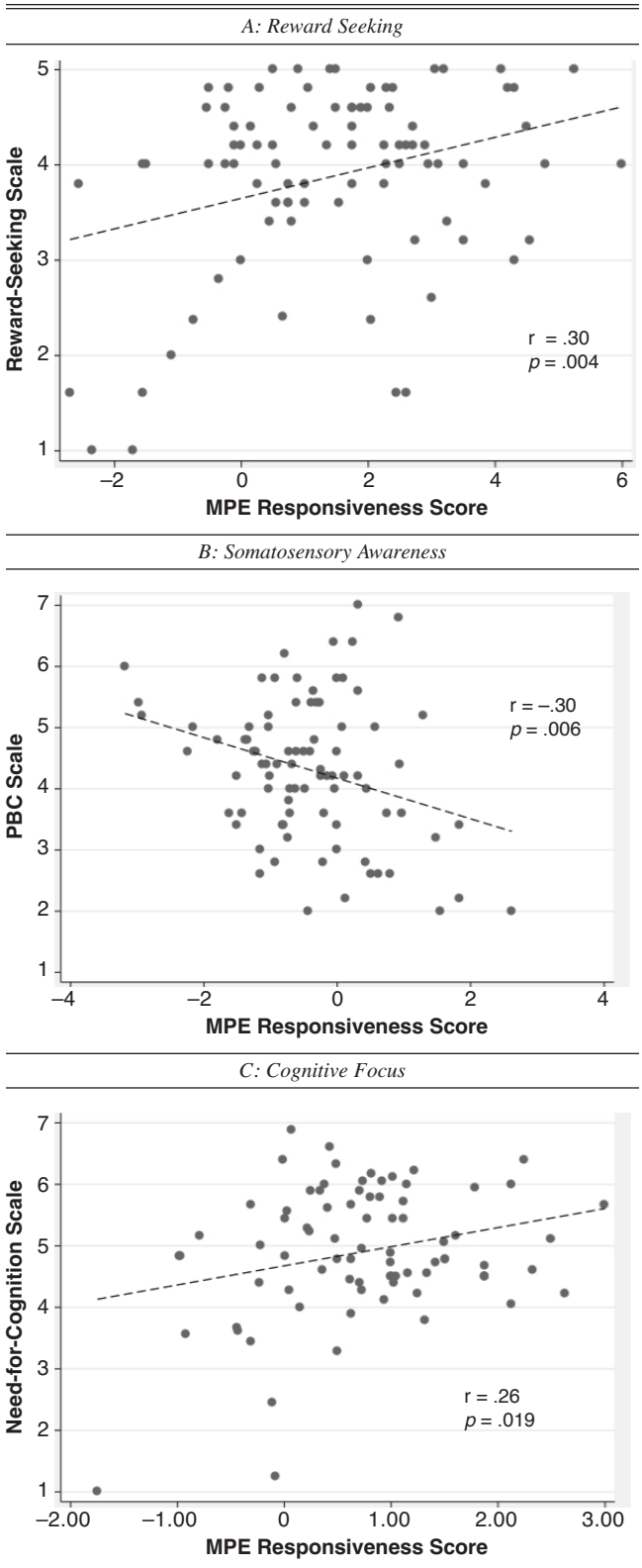
The third individual difference found in the VBM analysis was a positive correlation between MPEs and the gray matter volume in the dmPFC, which, as we outlined previously, has been linked to cognitive processes necessary for the regulation and appraisal of emotional experiences, working memory, and 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 MPEs.

More concretely, when MPEs 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 wine or a milkshake. In contrast with the findings on 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 participants 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 high need for cognition are more receptive to MPEs. Need for cognition is a personality variable reflecting the extent to which people are inclined to engage in cognitive activities (Cacioppo and Petty 1982; Cacioppo, Petty, and Kao 1984). Cohen, Stotland, and Wolfe (1955, p. 292) define need for cognition as "a need to structure relevant situations in meaningful, integrated ways" and "a need to understand and make reasonable the experiential world." Cognitive cues based on marketing actions, such as labels or prices, represent signals that help structure experiences, and people who are more responsive to using such cognitive cues to structure their experiences should be more receptive to MPEs. Against this background, we predict the following:

H_{2c}: MPEs will be more pronounced the higher participants score on the need-for-cognition scale.

Figure 4
RESULTS FROM STUDIES 2a–2c: REWARD SEEKING, SOMATOSENSORY AWARENESS, AND COGNITIVE PROCESSING MODERATE PRICE EFFECTS ON FOOD PLEASANTNESS



Notes: GMV = gray matter volume.

Design and procedure. The design was identical to that of Plassmann et al.'s (2008) Experiment 1 in Study 1 and Study 2b, using the same price levels and wines. Eighty participants from a North American university population (41 men, 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 (Cacioppo, Petty, and Kao's [1984] short form of the need-for-cognition scale). Participants responded on a seven-point Likert scale (1 = "strongly disagree," and 7 = "strongly agree").

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

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

Discussion. In Study 2c, we show that a consumer's focus on cognitive cues as measured by the need-for-cognition scale is indeed positively correlated with the strength of MPEs. The higher participants' need for cognition score, the more price influenced their experienced utility of wine tasting.

Taken together, Studies 2a–2c provide further evidence that participants high in reward seeking and need for cognitive processing were more responsive to MPEs, whereas those high in somatosensory awareness were less responsive to MPEs. However, these studies investigate the influence of these three moderators in three separate studies rather than a single study. In addition, they all investigate individual differences in how prices affect food consumption. It remains unclear whether our individual difference effects will transfer to other marketing-based expectancy effects, such as different brand labels, or different consumption experiences, such as aesthetic consumption. We address these issues in Study 3.

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

The goal of Study 3 was to conceptually replicate our findings from Studies 2a–2c for the effect of a different cognitive cue (whether an art piece was generated by an artist or the experimenter on a computer) on a consumption experience in a different sensory domain (experienced aesthetic pleasantness). In addition, 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

We informed participants that the goal of the study was to better understand their preferences for different types of art and how their personality influences those preferences. We conducted the study using an online sample (drawn from Amazon.com's Mechanical Turk), and the study took, on average, 7 minutes and 49 seconds to complete. Participants were paid US$.50 for their participation.

We told participants that they would see different pictures and would be asked to rate how much they enjoyed looking at each one on a nine-point Likert scale (1 = "not at all," and 9 = "very much"). Some of the pictures would show abstract work by artist Wassily Kandinsky, and 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. We adapted this task from Kirk, Skov, Christensen, et al. (2009) and Kirk, Skov, Hulme, et al. (2009) and used the same art stimuli. Each participant rated ten pictures, five labeled as crafted by an artist (the high expectancy condition) and five labeled as generated by the experimenter on his computer (the low expectancy condition). We counterbalanced assignments of pictures to the artist and computer conditions, 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 reward-seeking subscale of the BAS scale, the PBC subscale, and the need-for-cognition scale, respectively).[3] At the end, participants answered questions about their age, gender, and thoughts about the goal of the study and were thanked for their participation. The results appear in Table 3 and Figure 5.

Analysis and Results

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

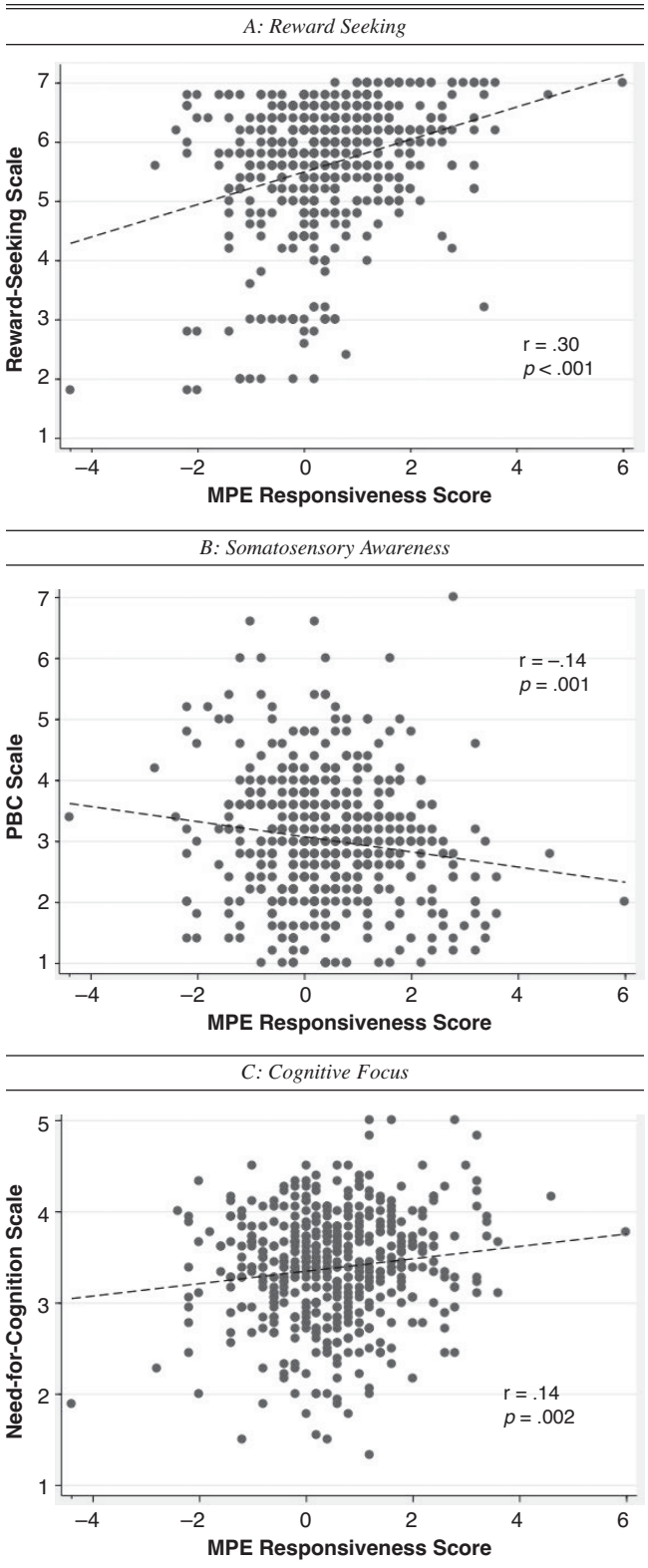
We first tested whether we could replicate the expectancy effects reported in Studies 1 and 2a–2c. We entered the experienced utility ratings as a dependent variable in a one-way within-subject ANOVA with the artist versus computer condition as a predictor. We found a significant effect of our expectancy manipulation, in that participants showed a higher experienced utility for seeing art pieces created by an artist than a computer ($F(1, 491) = 78.91$, $p < .001$).

We then tested whether individual differences in reward-seeking, PBC, and need for cognition moderated the MPEs of price following the same procedure to test for within-subject design moderators as in Study 1 (Judd, Kenny, and McClelland 2001). Table 3 lists the results of this analysis,

[3]Note that though all the scale items used in Study 3 were identical to those used in Studies 2a–2c, the scale anchors differed. In Study 3, the reward-seeking subscale uses a seven-point Likert scale, and the need-for-cognition scale uses a five-point Likert scale.

Figure 5

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



Notes: GMV = gray matter volume.

and Figure 5 shows the correlations between MPEs and (1) reward seeking, (2) PBC, and (3) need-for-cognition scores. We found that the BAS ($t(1, 491) = 6.53$, $p < .001$) and need-for-cognition ($t(1, 491) = 2.44$, $p = .015$) scores indeed amplified expectancy effects on experienced utility ratings, whereas PBC moderated expectancy effects on experienced utility ratings ($t(1, 491) = –2.75$, $p = .006$).

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 processing moderated marketing-based expectancy effects on the consumption experience. Importantly, we 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 MPEs 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 article, we extend understanding of the scope of the effects of marketing actions 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 MPEs. Across three studies, we found first evidence for three individual differences in MPEs on brain and behavioral levels. Importantly, we also examined the generalizability of these individual difference effects for different types of marketing-based expectations (i.e., price, health claim, and brand) and different types of consumption experiences (i.e., food and aesthetic consumption).

In Studies 1, 2a, and 3, we show that reward-seeking and motivational behavior play an important role in MPEs. The more sensitive consumers' neural and behavioral signatures of reward-seeking systems are, the more responsive they are to MPEs. Then, in Studies 1, 2b, 2c, and 3, we provide evidence that MPEs rely on an interplay between higher-level cognitive top-down processing and lower-level somatosensory bottom-up processing. On the one hand, we show that (1) increased gray matter volume in brain regions involved in cognitive aspects of emotion generation and control and (2) participants' cognitive focus and need for cognition favored the existence of MPEs. On the other hand, we show that (3) the more participants were able to focus on their internal, somatosensory states, as compared with external cues, and (4) the larger the gray matter volume in the brain's somatosensory systems, the less responsive they were to MPEs.

Not only is understanding the underlying mechanisms of MPEs important from an academic perspective, it is also highly relevant for marketers and public policy institutions. Marketers usually aim to increase 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 that marketers are capable of changing their customers' reward-seeking drives. A mouthwatering smell in a bakery and food samples in a supermarket are triggers for reward-seeking behavior (Wadhwa, Shiv, and Nowlis 2008). Thus, understanding how such actions might interact with MPEs 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, cognitive, and somatic focus rather than measure individual differences related to such processes as a personality trait variable. Another important extension of this work would be to manipulate neuropharmacological processes underlying MPEs related to our current findings. For example, does the administration of a dopamine blocker attenuate MPEs?

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

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

This article showcases how questions relevant to consumer behavior can benefit from an interdisciplinary consumer neuroscience approach. We used existing theories in cognitive neuroscience about pain placebo effects to extend Shiv, Carmon, and Ariely's (2005a, b) model of how MPEs work. This new model gave us novel concrete predictions about brain areas involved in neural processing anteceding MPEs, and we 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' tool kit to determine variability in gray matter volume to identify individual differences in MPE responsiveness. Importantly, consumer neuroscience aims not only to understand brain structures and functions important for a behavior of interest but also to determine 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 MPEs, we tested how the predicted brain regions translate into personality traits in Studies 2 and 3. Although following such a methodological approach seems fruitful for future consumer neuroscience studies, it is also important to understand its limitations (Yarkoni 2015).

We also want to highlight two important points: First, it is important to understand how variations in gray matter volume arise. Since the development of modern MRI machines, which allow for the measurements of a large number of people without the need for contrast agents or radiation, literally thousands of studies have investigated the relationship between brain structure and a variety of individual measures. Although 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 VBM (Ashburner and Friston 2000; Wright et al. 1995) allowed researchers to increase sample sizes and to investigate individual differences and even longitudinal changes.

One important question that has been investigated often 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 various studies—mainly those performed longitudinally—have shown the influence of people's environment and not only of their genetic makeup on regional brain volumes. For example, Draganski et al. (2004) show 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). Thus, variability in gray matter volume not only is due to genetic predispositions but also is mostly 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 the strength of brain–MPE correlations might be inflated, and how accurately patterns of brain activity can predict MPEs is unknown. In other words, further research is necessary to use our extended model to predict activations underlying MPEs using machine-learning and pattern classification approaches.

METHODOLOGICAL APPENDIX

In this Appendix, we detail two methodological aspects of the article. First, we provide more methodological details on the structural brain analysis that we applied, and second, we give more detail on the methodology and limitations of Yarkoni et al.'s (2011) NeuroSynth framework that we applied herein.

Additional Methodological Details About the VBM Analysis Study

The VBM is an approach that enables 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 enable comparison of similar brain areas across participants, 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 with previous normalization procedures, it includes a higher number of parameters for estimating the normalization, which allows for much better between-subject realignment. In addition, for the normalization procedure, the DARTEL approach generates a study-specific template. This algorithm can provide a higher degree of accuracy than the gold standard of manual segmentation (see, e.g., Focke et al.'s [2014] proof-of-concept study).

To execute the VBM analysis, we used the VBM8 toolbox, which 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 on the NeuroSynth framework from Yarkoni et al. (2011), we describe how the numbers of the different measures are generated and then provide a brief discussion of the limitations of this approach. NeuroSynth is a fully automated approach that allows for 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 approximately 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"), the entire database of coordinates is divided into two sets: those that occur in articles containing the term, and those that do not.
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. The following 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 study 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 updating.

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 we implement in this article.

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