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Neural Predictors of Message Effectiveness during Counterarguing in Antidrug Campaigns

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A substantial amount of research has focused on predicting the effectiveness of persuasive messages. However, characteristics of both the message itself and its receiver can impact theoretically predicted effects. For example, recent work published in this journal demonstrated that issue involvement modulates the relationship between message sensation value (MSV) and argument strength (AS). When exposed to anti-cannabis public service announcements (PSAs), high-drug-risk individuals rate these messages as having low effectiveness regardless of variation in MSV and AS. Accordingly, for high-risk individuals, MSV and AS lose their predictive power in message design; moreover, the all too common use of high MSV, high AS PSAs to dissuade drug use may be ineffective, as high-risk viewers are more likely to engage in counterarguing. In this paper, we use functional magnetic resonance imaging to investigate the neural correlates of counterarguing. Subsequently, we employ a brain-as-predictor approach using neural activation and self-report data to predict message effectiveness in two independent samples. We demonstrate that by adding two neural predictors within the middle frontal gyrus and superior temporal gyrus to self-report data, the prediction accuracy of message effectiveness in high-drug-risk individuals during counterarguing can reach, and even surpass, the prediction accuracy for low-drug-risk individuals.

Keywords: Counterarguing; Persuasion; Health Communication; Antidrug Public Service Announcements; Brain Imaging; Functional Magnetic Resonance Imaging

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Three influential media persuasion theories—the elaboration likelihood model (ELM; Petty & Cacioppo, 1986), the activation model of information exposure (Donohew, Palmgreen, & Duncan, 1980), and the limited capacity model of motivated mediated message processing (Lang, 2009)—have produced consistent results regarding the cognitive, emotional, and behavioral effects of public service announcements (PSAs). However, several recent studies have found interactions where the theoretically predicted effects of a given message variable—such as the strength of an argument or the degree to which a message is physiologically arousing—are conditional upon other variables related to the message or to the audience members (e.g., Kang, Cappella, & Fishbein, 2006; Lang & Yegiyan, 2008; Langleben et al., 2009; Stephenson & Palmgreen, 2001).

In a recent article in this journal, our research group reported on multilevel predictors of message effectiveness for anti-cannabis PSAs in an effort to test some of these interactions (Weber, Westcott-Baker, & Anderson, 2013). Specifically, we examined two important message features: (1) *argument strength* (AS; also argument quality, claim strength, etc.), which ostensibly refers to an objective feature of the argument that determines its persuasiveness, but is nearly always operationalized using receiver ratings (O'Keefe, 2006) and (2) *message arousingness*, which is often operationalized as *message sensation value* (MSV; Morgan, Palmgreen, Stephenson, Hoyle, & Lorch, 2003), a content-analytical measure of message features thought to elicit physiological arousal. Additionally, we examined the cross-level interaction between these message features and the degree to which audience members were involved in the issue topic (in this case, the degree of risk or involvement with cannabis use).

Our group demonstrated that issue involvement strongly modulates the relationship between MSV and AS. Participants with low drug risk showed an interaction between MSV and AS, whereby the most information-dense messages (high MSV and high AS) were of low effectiveness. High-drug-risk participants, on the other hand, rated anti-cannabis PSAs as having low effectiveness regardless of variation in MSV and AS. Both of these effects are consistent with the theories referenced above. For low-risk participants, high-information messages are over-stimulating and perceived as noxious. At the same time, high-risk participants are more likely to engage in biased central processing to defend their beliefs from counterattitudinal information, resulting in greater likelihood of counterarguing. Accordingly, for high-risk individuals, MSV and AS lose their predictive power in message design, and the main targets of antidrug campaigns will remain unpersuaded.

In the present study, we explore the neural correlates of resistance to persuasion in high-risk individuals. Specifically, we seek to show that the universally low ratings that high-risk individuals report for antidrug messages stem from a tendency to engage in active counterarguing with the message. By adopting a brain-as-predictor approach (Berkman & Falk, 2013), we demonstrate that when adding neural predictors to traditional self-report data, the prediction accuracy of message effectiveness in independent samples of high-drug-risk (counterarguing) individuals can reach and even surpass the prediction accuracy of low-drug-risk (non-counterarguing)

individuals. The following sections present reviews on the persuasion and campaign literature related to counterarguing, a background on the current state of the field with regard to understanding mental states and predicting behavior from neuroimaging data, and a review of neuroimaging findings related to persuasion generally and health/antidrug messages specifically.

Counterarguing as a Mechanism of Resistance to Persuasion

Counterarguing is one of many processes by which an individual can resist persuasion (Wegener, Petty, Smoak, & Fabrigar, 2004). Resistance processes vary in the degree to which they are conscious and effortful; for example, selective inattention to counter-attitudinal messages is relatively effortless, whereas reactance—rejecting messages based on a perceived threat to personal freedom (Brehm, 1966)—involves slightly more cognitive effort and awareness. Biased central processing and generating counter-arguments require a relatively high degree of conscious, effortful cognition. Because counterarguing is effortful, distraction or other reductions in ability to elaborate reduce this type of resistance (Buller, 1986; Petty & Cacioppo, 1986).

Relevant to the current research, Petty and Cacioppo (1979, 1986) noted that when issue involvement is high, participants are motivated to defend their prior knowledge and beliefs in the face of a counterattitudinal message by arguing with the message. While biased in favor of prior belief, such elaboration involves cognitive effort and, like relatively objective central processing, theoretically results in stable attitudes. Research has further indicated that the more important the individual deems the attitude to be, the greater the likelihood of counterargument (Zuwerink & Devine, 1996).

The story of counterarguing and its effects is not simple. People are not always successful in their attempts to resist persuasion, and resulting attitude valence, stability, and confidence have been shown to depend on a number of additional factors (Petty, Tormala, & Rucker, 2004). For example, individuals who successfully resist strong arguments against their existing attitude show increased confidence in their original attitude, though those exposed to weaker attacks show no change in attitude confidence (Tormala & Petty, 2002). Follow-up research demonstrated that this effect was moderated by negative elaboration—that is, counterarguing (Tormala & Petty, 2004; see also Ahluwalia, Burnkrant, & Unnava, 2000). So, for individuals motivated and able to defend their existing attitudes, AS seems to have a *negative* impact on persuasive outcomes: individuals who successfully counterargue with strong arguments are all the more committed to their original belief. As with the interactions between AS and other variables reviewed above, this effect calls into question the utility of strong arguments in health messages aimed at high-involvement populations.

Exposure to persuasive messages in general and attitude-inconsistent messages in particular results in self-reported physiological arousal and negative affect (e.g., Palmgreen et al., 1991; Raju & Unnava, 2006; Stephenson & Palmgreen, 2001; Stephenson & Southwell, 2006; Yzer, Vohs, Luciana, Cuthbert, & MacDonald, 2011).

Raju and Unnava (2006) demonstrated that individuals who are more committed to their prior belief are more likely to counterargue with a message as a means of reducing negative arousal, whereas less committed individuals are more likely to reduce arousal by changing their attitudes in the direction of the persuasive message. Moreover, they found that physiological arousal serves as the motivating factor in engaging in effortful elaboration. When individuals were given an opportunity to counterargue with the message, the reported arousal levels were decreased relative to those who were not given the opportunity.

In addition to general cognitive ability and motivation, the capacity to counterargue also seems to draw upon self-regulatory abilities. Specifically, counterarguing involves integrating and synthesizing information to avoid adopting a counterattitudinal position. Importantly, self-regulatory abilities have been shown to have a limited capacity (Muraven, Tice, & Baumeister, 1998). Wheeler, Briñol, and Hermann (2007) showed that individuals who had engaged in a prior self-regulatory task generated fewer counterarguments and showed reduced subsequent resistance to counter-attitudinal persuasion than individuals who had not been ego-depleted, despite the fact that ability to elaborate was otherwise high. The self-regulatory processes that facilitate counterarguing are of particular importance for this study, since this conceptualization suggests that neural activity in regions associated with self-related executive processing should be more strongly activated during counterarguing. We review several previous neuroimaging studies supporting this view below.

In summary, counterarguing seems to be one potential response to the physiological arousal and negative affect that exposure to a counterattitudinal message engenders, and is more likely (relative to changing one's attitude) when involvement is high and prior attitude is strong. Factors affecting ability (such as distraction) and motivation (such as perceived self-relevance) to process centrally affect the likelihood to counterargue, as does self-regulatory capacity. Our prior paper (Weber et al., 2013) demonstrated that the interaction between AS and MSV works as predicted: PSAs that combine high MSV with low AS were most effective among low-risk participants, but this strategy falls apart among high-risk participants, who merely report low perceived effectiveness for messages of all types. If this interaction can be consistently demonstrated, then it is not only problematic to use traditional high MSV, high AS ads to target to high-risk individuals but also difficult for researchers and message designers to use variations in these constructs as predictors of counterarguing and persuasive outcomes in high-risk participants in order to study the mechanism of this process or to create more effective messages. However, in this paper, we construct a method for using this contrast to detect neural correlates and predictors associated with this mechanism, allowing an alternative means of exploring this interaction. The next section introduces the current research paradigms for studying neural correlates of relatively complex mental processes, as well as a new approach for predicting real-world outcomes from neuroimaging data in both individuals and in larger populations.

Predicting Behavior from Neuroimaging Data

For the majority of the last two decades, researchers using functional neuroimaging for social scientific research have concentrated upon brain-mapping studies where the goal is to pinpoint brain areas, networks, and patterns of connectivity that are involved in a given cognitive function. These types of studies involve using very simple, well-controlled stimuli or manipulations to elicit a given cognitive function reliably and, to the extent possible, without confound. Recently, researchers across many fields have built on the body of knowledge generated by these brain-mapping studies and have branched out to explore more complex and naturalistic stimuli.

Such studies, however, are not without their potential flaws and criticisms. One major issue in moving away from the traditional paradigm is the *reverse-inference* problem (Poldrack, 2006). In the traditional *forward inference* brain-mapping paradigm, it is assumed that the study's task or manipulation invokes only one psychological process compared with the control condition. Thus, the presence of the resulting brain activation (when "subtracting" the control condition) must be a result of the psychological process invoked by that task. However, the reverse path is not one-to-one. No brain area (or even network) is uniquely diagnostic for a given cognitive or affective process. That is, any given area is likely to be involved in many different psychological processes. *Reverse inference* occurs when a researcher uses findings from prior neuroimaging studies to interpret the meaning of brain activation in response to a different task in which the cognitive mechanisms are unknown or under study. In a well-known example of faulty reverse inference, in a *New York Times* op-ed during the 2008 Presidential primary elections (Iacoboni et al., 2007), a group of researchers interpreted functional magnetic resonance imaging (fMRI) scans of swing voters viewing candidate speeches, claiming that activation in certain limbic areas indicated specific feelings about the candidates in response to the campaign messages. In the presence of a task or stimulus with unknown resulting psychological processes, brain activation in a specific area cannot be used as evidence of what process takes place during the task (such as what feelings swing voters experience in response to campaign messages based on limbic activity). Because of this issue, making behavioral predictions and inferring cognitive states based on brain responses ("brain-decoding") has historically been considered difficult, or even impossible.

Recent developments, however, have suggested that useful information about cognitive states and consequent behavior can be extracted from brain-imaging data. For instance, sophisticated research paradigms and analysis methods have allowed researchers to not only map neural correlates of more complicated and higher-order processes such as interpreting real-world experiences (Spiers & Maguire, 2007) but also actually predict real-world behavioral outcomes such as music purchases (Berns & Moore, 2012) and smoking-cessation intervention success (Falk, Berkman, & Lieberman, 2012; Falk, Berkman, Whalen, & Lieberman, 2011) from brain-imaging data. Researchers have even begun to "decode" brain activation such as reconstructing crude versions of what the participants "saw" in a stimulus image or video from functional imaging data (Nishimoto et al., 2011).

Many of these methods involve data mining or the use of trained virtual neural networks to identify the most likely interpretations of given patterns of activation (for a review of current avenues of research in this direction, see Poldrack, 2011). Another avenue—the *brain-as-predictor* approach (Berkman & Falk, 2013)—takes a slightly different tack than the traditional brain-mapping paradigm. Rather than investigating stimulus–response to infer the neural mechanisms of the mediating cognitive state, the brain-as-predictor approach uses a multi-step method to pinpoint relevant brain-activation patterns and uses these patterns to predict real-world outcomes such as intelligence (Choi et al., 2008), language acquisition (Tan et al., 2011), and consumer choices both at the individual level (Levy, Lazzaro, Rutledge, & Glimcher, 2011; Tusche, Bode, & Haynes, 2010) and the population level (Berns & Moore, 2012). Importantly, this paradigm involves a path of analysis that is not a part of the traditional forward-inference technique—brain-imaging data are used to predict self-report and behavioral outcomes.

In brain-as-predictor studies, brain maps related to the cognitive task of interest are identified in one of a few ways, each of which can be thought of as falling under traditional forward inference. For instance, in the *test-validation approach*—similar in rationale to the process of performing exploratory and confirmatory factor analysis on survey data—a task involving a known process may be tested on one sample to identify the relevant activation pattern, and then the identified pattern is confirmed and used as a predictor for behavioral outcomes in a separate sample (Falk, Berkman, Mann, Harrison, & Lieberman, 2010; Falk et al., 2011). Another method involves having participants (either in the same or a separate sample) perform a *localizer task*—a task that engages the cognitive process of interest but is a separate task from the manipulation under investigation (Chua et al., 2011)—in order to identify the pattern of activation for the hypothesized cognitive process before testing that pattern in response to the task of interest as a predictor for behavioral and self-report measures. A final approach (adopted in this study) uses neural activity in one sample to predict outcomes in an independent sample, thereby ensuring that regions of interest (ROIs) are defined independently of the variable of interest (Berns & Moore, 2012).

Importantly, none of the brain-as-predictor methods involve making the fallacious logical leap inherent in reverse-inference: researchers are not merely "interpreting" a pattern of activation as involving a given cognitive process based only on a casual (and potentially cherry-picked) review of past findings involving those same brain areas. Instead, a theoretical model of cognitive processes and their associated brain activation patterns leading to behavioral outcomes is built and tested using traditional, deductive hypothesis-testing methods.

The benefits of this approach are two-fold. First, neurophysiological correlates of stimuli once considered to be too "complex" to study can be identified—that is, mapping of processes involved in more complex cognitive and affective tasks can be accomplished with the multi-step approach. Second, and perhaps more importantly for fields like communication, real-world outcomes—and even population-level predictions—can be explained with greater accuracy than is possible using traditional

experimental and survey data (Falk et al., 2012), providing valuable insight into the cognitive and affective mechanisms of psychological and social processes. This allows researchers to test the formerly "black-box" intervening states involved in communication and other social processes—such as the cognitive and affective states involved in the persuasion process—allowing scholars to refine theory and practitioners to design better interventions based on these findings.

Neuroimaging Investigations of Persuasion and Health-message Effectiveness

One benefit to neuroimaging is the ability to bypass shortcomings in people's ability to accurately self-report attitudes and behaviors. An increasing number of neuroimaging studies attempt to tackle unanswered questions in persuasion research without requiring participants to engage in conscious introspection and self-reporting. The following sections review the current research in neuroimaging and persuasion/health-message effectiveness, identifying neural correlates associated with persuasive outcomes and reviewing work using neuroimaging to predict actual health-message persuasion outcomes in individuals and populations.

Neural Correlates of Persuasion and Health-message Effectiveness

As a complex, higher-order mental process, persuasion—or resisting persuasion—is expected to involve a number of different brain areas and networks. Because (as reviewed above) persuasion theory predicts both affective and cognitive processes in response to persuasive messages, investigators have explored the neural underpinnings of both of these subprocesses.

Regarding affective responses, a recent meta-analysis of 24 fMRI studies showed significant overlap in activation of brain areas associated with the processing of affective and social information across different persuasion tasks (Cascio, Shumaker, Beard, Albarracin, & Falk, 2014). Specifically, neural regions associated with reward/positive valuation (ventral medial prefrontal cortex and ventral striatum), social pain/negative valuation (anterior insula, dorsal anterior cingulate cortex, and sub-anterior cingulate cortex), and salience detection (amygdala) were involved when participants underwent influence and/or changed their attitudes and resulting behaviors. The authors suggest that these commonalities in neural involvement across persuasion tasks indicate that, rather than merely reporting socially desirable outcomes in response to persuasion tasks (a possible explanation for outcomes in self-report data alone), influenced individuals engaged affective processing systems and altered neural responses to the attitude objects related to the persuasive messages.

Similarly, in a set of three studies examining the neural correlates of persuasion across participant cultures (American and Korean) and across media types (text and video), Falk and colleagues found that, across all three studies, feeling persuaded was associated with increased activity in areas involved in auditory and language processing (posterior superior temporal sulcus), in social and mentalizing tasks (dorsal medial prefrontal cortex [DMPFC] and the temporal pole) and in selecting

among competing beliefs and memory representations (left ventral lateral prefrontal cortex; Falk, Rameson, Kang, Ingaki, & Lieberman, 2009).

As predicted by persuasion theory and research, areas associated with self-referential processing have also been implicated in persuasive outcomes. Specific to health messages, Chua, Liberzon, Welsh, and Strecher (2009) varied the degree to which smoking-cessation messages were tailored to the participant (high vs. low tailoring within participants, as well as a "generic"/no-tailoring condition) for 24 smokers. Since all participants were smokers, clearly smoking-cessation messages would be highly personally relevant; however, the degree of tailoring manipulates the relevance of the individual message and the degree to which it increases involvement—indeed, in post-scan measures, participants rated high-tailored messages as more personally relevant than low-tailored messages. In the tailored condition relative to the generic condition, activation was observed in areas associated with self-referential processing such as autobiographical memories and evaluating self-traits (rostral medial prefrontal cortex [rostral MPFC]/Brodmann Area 9 [BA9]; precunus; and posterior cingulate cortex); these areas showed even greater activation in the high-tailored condition relative to the low-tailored. In a follow-up study Chua et al. (2011) found that activation in the DMPFC (and to a marginal extent the precuneus) in response to high-tailored messages predicted smoking cessation in a 4-month period. While these findings suggest that tailoring messages to individual subjects can influence cessation behavior, they do not clarify how other message features (e.g., MSV or AS) influence neural activation, or the extent to which such neural activation is predictive (or serves as a correlate) of either increased effectiveness or counterarguing in high-involvement groups. Tailoring to increase self-relevance has long been assumed to increase persuasion by increasing AS and/or motivation to process, but (as reviewed above) in populations with established beliefs, both of these variables have the potential to increase counterarguing.

Also particularly relevant to the current study, in a recent article Ramsay and colleagues pointed out that although much persuasion theory posits the involvement of explicit cognition (i.e., executive functions), most persuasion neuroimaging studies to date have focused on networks associated with affective and social information (Ramsay, Yzer, Luciana, Vohs, & MacDonald, 2013). To investigate the role of executive function and its interactions with affective networks in antidrug PSA message processing, the authors showed 70 adolescents ads during fMRI scanning. Stimulus ads were 20 antidrug PSAs—the 10 strongest and 10 weakest PSAs as measured by self-report of perceived message effectiveness (PME) in a separate study—as well as 10 product advertisements that were unrelated to drugs; thus, stimulus conditions included strong antidrug PSAs, weak antidrug PSAs, and non-drug-related ads (within participants).

In contrasts between the non-drug ads and antidrug PSAs, differences in arousal-related activity were found in socioemotional network areas—specifically the bilateral amygdala (increased activation for antidrug PSAs but not for non-drug ads), medial orbital frontal cortex, and paracingulate gyrus (deactivation across all ads, with more for nondrug ads than antidrug PSAs), with additional differences reported in the bilateral hippocampus and superior temporal gyrus (STG). Within antidrug ads,

contrasts between strong and weak conditions revealed differences in arousal-related activity in executive-functioning areas—the lateral prefrontal cortex (PFC), especially the bilateral middle frontal gyrus (MFG; deactivation that was stronger for weak than for strong PSAs) and left inferior frontal gyrus (IFG; increased activation for strong PSAs and deactivation for weak PSAs), with additional activation differences in socioemotional and language-processing regions (bilateral hippocampal gyrus, lingual gyrus, occipital lobe, and precuneus). Moreover, individual differences in PME outcomes for the antidrug PSAs were positively correlated with arousal-related activation changes in lateral executive regions (left IFG and left MFG), indicating that arousal-related activity changes were greater in these areas when participants reported the PSAs to be more convincing, whereas arousal-related activation was not significantly correlated with PME for socioemotional ROIs.

Importantly, the authors point out that areas of the subgenual and DMPFC that have been identified in other work as associated with persuasion and behavior change were, in their study, significantly associated with arousal-related activity when contrasting non-drug ads with antidrug PSAs, but were not significant in analyses comparing strong to weak antidrug ads. Ramsay et al. (2013, p. 1145) suggest that "the socioemotional network is necessary, but not sufficient, for persuasive message processing," whereas "activity in executive control regions likely relies on and integrates information from socioemotional brain areas to make judgments about incoming persuasive information."

One significant drawback of their study, as in other persuasion-related neurophysiological studies, is that Ramsay et al. (2013) did not examine the role of issue involvement in the persuasion process. As reviewed above, involvement has been shown in experiments and surveys to be a critical factor in health-message processing, as it moderates the effects of AS, MSV, and other message-related variables (and their interactions) on persuasion-related outcomes. Thus, it is unclear whether there are differential effects in activation patterns associated with message processing for high- vs. low-risk individuals (the authors did not report whether prior drug use predicted differences in PME or in any fMRI outcomes). Additionally, the outcomes measured in the Ramsay et al. (2013) study, like in most other studies, were restricted to the neural correlates of PME broadly—not correlates related to the process of counterarguing specifically. Our study is an attempt to close this gap in the literature. To our knowledge, our study is the first to specifically address the neurophysiological *correlates* of counterarguing in high- vs. low-drug-risk individuals and to use these correlates in a brain-as-predictor approach.

Predicting Real-world Persuasion Outcomes Using Neuroimaging

The brain-as-predictor approach (Cascio, Dal Cin, & Falk, 2013) has demonstrated that it is possible to use functional neuroimaging data not only to identify regions involved in various mental tasks but also as *predictors* for behavioral outcomes. A number of studies by Falk and colleagues have focused on identifying patterns of brain activation associated with persuasive processes and using those patterns to

predict persuasive self-report and behavioral outcomes at both an the individual and population level. In line with associations between self-referential processing and persuasion, Falk et al. (2010) found that activation changes in the MPFC in response to persuasive messages about the risks of sun exposure predicted participants' changes in sunscreen use, explaining more variance in subsequent behavior with fMRI data than did post-message self-report measures about attitudes and behavioral intentions. In a follow-up study, the researchers found that smokers' MPFC responses to antismoking PSAs explained more variance in exhaled CO, a marker of recent tobacco smoking, than post-message self-report (Falk et al., 2011).

At the population level, Falk et al. (2012) showed that activity in the MPFC in a group of smokers viewing antismoking campaign ads predicted the rank ordering of ads with regard to their future campaign success as measured by subsequent calls to a smoking-cessation hotline when the campaign aired. Moreover, both the participant self-report measures of perceived effectiveness *and* industry expert rank orderings produced different, incorrect predictions. Similarly, the same research group (Falk et al., 2014) found that MPFC activity in smokers in response to graphics with textual messages about quitting predicted the click-through rate for a subsequent population-level email campaign, but only for messages that contained graphic images rated by another sample as giving strong reasons to quit as opposed to neutral images. This suggests that the ability for MPFC-related activity to serve as a predictor for population-level behavior is specific to stimuli that are likely to invoke self-related processing (i.e., when the audience perceives the message to be personally relevant as well as convincing). Falk et al. (2014) also demonstrated that neurological activation associated with self-related processing predicts population-level responses to antismoking PSAs above and beyond self-report data.

As with the research into neural correlates of behavior, Falk and colleagues' work does not address potential differences in outcomes related to issue involvement and counterarguing as a barrier to persuasion. This study is the first to specifically address the neurophysiological *predictors* of counterarguing in high- vs. low-drug-risk individuals. In other words, by applying the logic and findings from Falk et al. (2010, 2011, 2012, 2014) to independent anti-cannabis campaign data, we seek evidence of neural predictors for the theoretically well-established effects of AS-MSV interactions on message effectiveness as detailed in Weber et al. (2013).

Method

A total of five independent data sources were utilized in this study (Figure 1). The first two data sources provided information about the AS and MSV of the PSAs used as stimuli in the present experiment (see samples 2 and 3 in Kang et al., 2006). The third (see sample 2 in Weber et al., 2013) and fourth (see sample one in Kang et al., 2006) data sources provided independent ratings of PME for each PSA. Finally, the present study exposed participants to a subset of these video PSAs while measuring neural activity in a brain-imaging environment. Consistent with prior brain-as-predictor approaches (see Berkman & Falk, 2013), percent signal change and

self-report ratings in the brain-imaging sample were used to predict ratings of PME in data sources 3 and 4.

Participants

Twenty-eight right-handed healthy participants aged 18–25 (M = 20.3, standard deviation [SD] = 1.5) with no contraindication to fMRI scanning completed the brain-imaging portion of this study. This sample was drawn from the undergraduate research pool at a large California university. Participants received course credit and were paid $50 for participation. Of these participants, 14 were high-risk for cannabis use and the remaining 14 were low-risk, as measured using Cappella, Yzer, and Fishbein's (2003) risk for marijuana use scale. This scale, which has been used in several studies similar to this one (Kang et al., 2006; Weber et al., 2013), measures both the frequency of cannabis use and other items known to correlate strongly with use, such as the frequency with which the participant is offered cannabis, how many friends of the participant use cannabis, and the participant's proclivity for sensation seeking.

Study Materials and Measures

Experimental stimuli. Participants were exposed to 32 unique anti-cannabis video PSAs retrieved from the antidrug PSA archive at the University of Pennsylvania Annenberg School for Communication (see Kang et al., 2006). The PSAs were part of a national anti-cannabis drug campaign and were selected according to previously

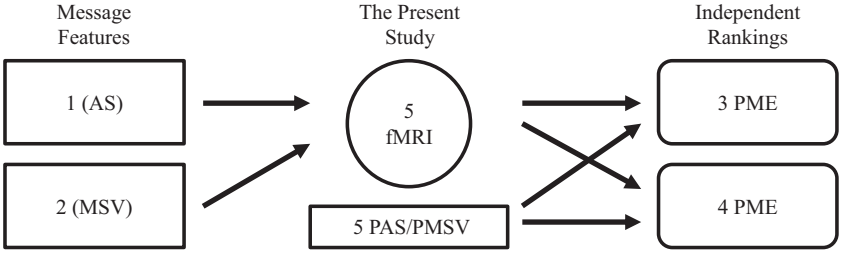

Figure 1 Five independent samples used in the present study.
Note: The first data source provided information about the AS of PSAs used as stimuli in the present experiment (see sample 2 in Kang et al., 2006). This dataset is comprised of 322 adolescents (M_{age} = 15.4, SD_{age} = 1.95, female = 50.3%) recruited from 15 US cities. The second data source provided a measure of MSV for the same PSAs (see sample 3 in Kang et al., 2006). Here, trained coders content analyzed MSV features of each PSA (κ = .79). For the third data source, 599 freshmen students (M_{age} = 19.65, SD_{age} = 2.12, female = 73.8%) rated the message effectiveness of each PSA (see sample 2 in Weber et al., 2013). In the fourth data source, 601 adolescents (M_{age} = 15.3, female = 49.9%) rated the same PSAs on perceived message effectiveness (see sample 1 in Kang et al., 2006). The fifth data source is comprised of 28 female undergraduate students (M_{age} = 20.3, SD_{age} = 1.5) who participated in the neuroimaging component of the present study.

and independently established MSV (high/low) and AS (high/low) scores (see data sources 1 and 2). PSAs were slightly trimmed so that each was exactly 30 s in duration.

Perceived message effectiveness. PME is a self-report measure that strongly correlates with the actual effectiveness (AE) of a persuasive message (Bigsby, Cappella, & Seitz, 2013; Dillard, Shen, & Vail, 2007; Dillard, Weber, & Vail, 2007). Participants used a 5-point Likert scale to rate each PSA according to the extent that it: is convincing, is important, helps them/friends stay away from marijuana, and makes them feel confident in their ability to resist marijuana use (Cronbach's $\alpha = .87$).

Perceived message sensation value (PMSV). PMSV is a 17-item, 7-point Likert scale that measures three dimensions of a message: (1) emotional arousal, (2) dramatic impact, and (3) novelty (Palmgreen, Stephenson, Everett, Baseheart, & Francies, 2002). As participants needed to evaluate a set of 32 PSAs (see below), it was important to use a short form version of PMSV. In the data sources we adopted from Kang et al. (2006), we found a three-item, 7-point Likert scale short form measure of PMSV that asked participants to rate the extent to which each PSA was creative, fast-paced, and dramatic. Two of the three items correlated highly (fast-paced, $r = .73$, $p < 0.001$; creative, $r =. 65$, $p < 0.001$) with the content analytical variable MSV in data sources 1 and 2. Thus, we used these two items for our short form PMSV measure. The correlation between the short version PMSV measure in our prediction sample 5 and the full-item PMSV measure in data sources 1 and 2 was $r = .80$ ($p < 0.001$).

Perceived argument strength (PAS). Participants used the standard nine-item, 5-point Likert scale for evaluations of PAS (Zhao, Strasser, Cappella, Lerman, & Fishbein, 2011). This measure is used as an alternative to thought-listing measures when the topic is sensitive in nature.

Procedure

Upon arrival at the brain-imaging facility, participants provided informed consent and filled out an fMRI screening form. Participants then completed a pre-test questionnaire that measured basic demographics (age, gender, and ethnicity), handedness, and cannabis-use risk. Participants were then briefed on the brain-imaging portion of the study, asked to change into a medical patient gown, and positioned within the brain-imaging scanner.

In two functional runs (16 m per run), participants viewed 32 unique PSAs (16 PSAs per run) that differed in MSV and AS. Each PSA lasted 30 s, was separated by a 10 s baseline, and each functional run included four control clips (30 s duration) where the video/audio sequence associated with different antidrug PSAs was reversed and scrambled so that message meaning was removed but visual and auditory intensity remained constant. Finally, each functional run included four rest blocks (30 s duration), in which participants were presented with a black screen and instructed to close their eyes (Figure 2 for a block design).

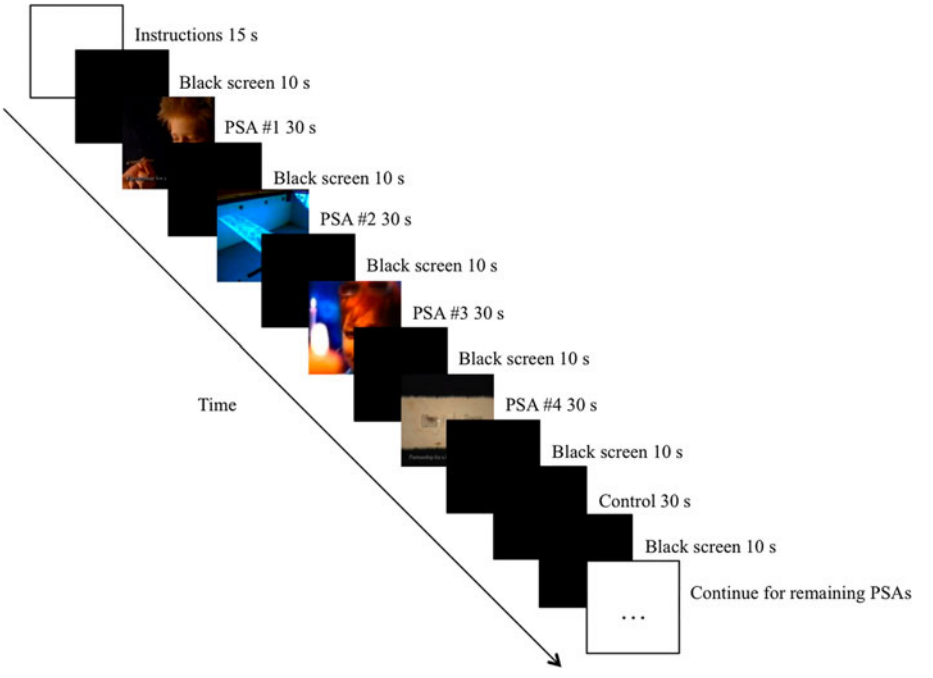

Figure 2 Block design for each functional run. In each of the two functional runs, participants watched 16 antidrug PSAs and were exposed to eight control conditions (four scrambled videos, four black screens). Each video varied in MSV (high/low) and AS (high/low) and video order was counterbalanced along these dimensions.

After completing the two functional runs, participants then viewed all 32 PSAs outside of the scanner. At this time, participants completed PME ratings for each PSA. Once complete, participants were debriefed, compensated, and thanked for their time. The total procedure took roughly 90 minutes.

fMRI acquisition. Data were acquired on a 3-tesla Siemens Magnetom TIM Trio system with an 8-channel phased-array head coil. A T2-weighted single-shot echo planar gradient sequence measured blood oxygenated level dependent contrasts (Repetition time [TR] = 2000 ms, echo time [TE] = 27.2 ms, flip angle [FA] = 77 degrees, field of view [FOV] = 22 × 22 cm^2). Each volume consisted of 40 interleaved slices acquired parallel to the AC-PC plane (3 mm slice thickness, 0 mm gap, 64 × 64 matrix). A high-resolution T1-weighted weighted sagittal sequence image of the whole brain (TR = 1620 ms, TE = 3.87 ms, FOV = 250 mm, voxel resolution 1 × 1 x 1 mm, FOV = 250 mm) was collected prior to each functional run.

Data Analysis

fMRI preprocessing. Data preprocessing and analysis were performed using FEAT (fMRI Expert Analysis Tool v6.0) from the Oxford Center for Functional MRI of the

Brain (FMRIB) Software Library (FSL v5.0; http://www.fmrib.ox.ac.uk/fsl). First, the data were motion corrected using FSL's Motion Correction FMRIB Linear Registration Tool (Jenkinson, Bannister, Brady, & Smith, 2002), which aligns all brain volumes to a common coordinate system. The data were then brain extracted using FSL's BET (Brain Extraction Tool; Smith, 2002), which removes extra-brain matters such as skull, meninges, venous and arterial processes, and cerebral spinal fluid. Next, the data were grand-mean intensity normalized, which improves combining results across participants. The data were then high-pass temporal filtered (σ = 59.5 s), which removes low-frequency components of the signal by removing a Gaussian-weighted running average of the time series. The data were additionally normalized to standard space prior to subsequent analysis using FSL's FLIRT utility (Jenkinson et al., 2002; Jenkinson & Smith, 2001) to align the participant's functional and structural data—that is, to bring their functional space into alignment with their high-resolution structural scan. Subsequently, FMRIB's Nonlinear Registration Tool (Andersson, Jenkinson, & Smith, 2007a, 2007b) was used to register participant structural data to the Montreal Neurological Institute [MNI] 152 standard template—that is, to bring both the structural and functional data into alignment with a standard template using a nonlinear transformation. Finally, the data were resliced to 5 mm isotropic voxels using FLIRT with nearest-neighbor interpolation.

Functionally defined regions of interest (fROIs) analysis. In a series of first-level general linear model (GLM) analyses, externally derived experimental conditions (MSV and AS) for each run within each participant were modeled as explanatory variables (EVs). Control and rest conditions were also modeled as EVs and all EVs were convolved with the hemodynamic response function (Gamma convolution = 6 s, SD = 3). Covariates included temporal derivatives for each EV. In a second-level analysis, runs were combined for each participant using a fixed-effects model. Finally, these lower-level analyses were combined in a third-level FLAME 1 analysis (FMRIB Local Analysis of Mixed Effects; Beckmann & Smith, 2004; Woolrich, Behrens, Beckmann, Jenkinson, & Smith, 2004) that modeled main effects for AS, MSV, and the interaction term. In this analysis, cannabis risk (high/low) was modeled as an EV and contrasts were calculated for mean activation (high/low risk), high risk > low risk, and low risk > high risk. Voxels were considered to be significant if they survived cluster-based thresholding, corrected for multiple comparisons ($Z > 2.3$, $p < .05$; Worsley, 2001). The resulting contrast images were used as functionally defined regions of interest (fROIs) in subsequent signal change analyses.

fMRI messages analysis. This analysis observed group-level signal change for each of the 32 antidrug PSAs within the fROIs identified in the aforementioned analysis. In the first-level GLM analysis, each video, the control conditions, and rest conditions was modeled as an EV. PSA EVs were contrasted against scrambled video and control EVs. These EVs were convolved with the hemodynamic response function (Gamma convolution = 6 s, SD = 3) and temporal derivatives for each EV were included as covariates. We then conducted a higher-level analysis for each run

order (16 videos each) using a fixed-effects model with cannabis risk groups (high/low) as EVs. Replicating the procedure utilized by Falk et al. (2014), a signal change analysis on the group-level data[1] was then conducted for each of the previously identified fROIs using Featquery in FSL. Percent signal change from baseline (control) for each PSA was averaged within a 5 mm sphere around the peak voxel identified for each fROI.

Brain-as-predictor analysis. In this analysis, we used the signal change estimates for each fROI in the AS × MSV contrast (Table 1) for each PSA and the corresponding self-report data from our small fMRI sample (source 5) to predict PME in two large, independent samples: freshmen students at a large California university (source 3) and US adolescents (source 4). Following the procedures in Falk et al. (2012, 2014), we converted signal change estimates within our fROIs into ranks—that is, we obtained a ranked list of PSAs for each risk group (from 1 = lowest to 32 = highest). Using rank transformations is a preferred strategy when combining data from multiple data sources. Likewise, the self-report data in our fMRI sample and the message effectiveness evaluations for each PSA were converted into ranks from 1 (lowest) to 32 (highest) for each risk group in both independent samples.

Resembling the analytical procedure in Falk et al. (2012, 2014), we used a stepwise rank regression analysis (Cuzick, 2005; McCullagh, 1980) for testing the prediction models. In a first step, we used rank-ordered self-reported PAS and PMSV in our small fMRI sample to establish a prediction reference point that is based on traditional persuasion theory. In a second step, we added the signal change information of select neural predictors (fROIs) to the rank regression.

Results

Behavioral Data

Message effectiveness. PME was distributed largely symmetrically and approximately normally in all data sources with comparable means and SDs—source 3 sample (Weber et al., 2013): $M = -.07$, SD = 1.08, min/max = –2/+2; source 4 sample (Kang et al., 2006): $M = -.31$, SD = 1.21, min/max = –2.6/+1.6; source 5, fMRI sample: $M = -.04$, SD = 1.13, min/max = –2/+2.

Experimental conditions. For both the high- and low-risk group participants in our fMRI sample, the mean of PMSV of messages in the high-MSV condition was significantly higher than in the low-MSV condition (high risk: $\Delta M = .46$, $p < 0.001$; low risk: $\Delta M = .44$, $p < 0.001$). Likewise, for both high- and low-risk participants, the mean of PAS of messages in the high-AS condition was significantly higher than in the low-AS condition (high risk: $\Delta M = .22$, $p < 0.001$; low risk: $\Delta M = .31$, $p < 0.001$) indicating that our experimental manipulation, which was based on content analyses (MSV) and an evaluation of extracted arguments by an independent sample (AS), was successful.

Table 1 Activations by risk group.

Structure (fROI)	Maximum *Z*-score	Cluster size	Max *Z*-score coordinates (mm)
MSV × AS			
High risk > low risk			
Superior LOC (left)	3.33	1477	(−28, −76, 18)
Fusiform gyrus	2.85		(−30, −86, −18)
High-risk group			
Cerebellum	4.55	3997	(−26, −76, −42)
Middle temporal gyrus	4.45	4295	(−44, −60, 6)
Inferior LOC (left)	4.15		(−58, −68, 0)
Superior lateral occipital cortex (left)	3.79		(−38, −84, 20)
STG	4.26	5624	(56, 4, −18)
Precuneus	4.23	2707	(6, −52, 48)
FP	4.13	1140	(8, 56, 38)
MFG	3.32	1210	(48, 24, 28)
Precentral gyrus	3.15		(46, −2, 28)
Low-risk group			
Inferior LOC (right)	4.86	2583	(48, −64, 6)
Middle temporal gyrus	3.98		(44, −56, 12)
Inferior LOC (left)	4.24	1381	(−48, −72, 14)
MSV high > low			
High-risk group			
Temporal occipital fusiform cortex	4.62	12,342	(30, −56, −12)
Temporal fusiform cortex	4.55		(−30, −44, −20)
Fusiform gyrus	4.32		(20, −82, −8)
Low-risk group			
Occipital pole	5.75	17,523	(14, −92, 32)
Temporal occipital fusiform cortex	5.66		(28, −48, −16)
Fusiform gyrus	5.37		(30, −70, −16)
AS high > low			
High-risk group			
Lingual gyrus	4.18	1843	(−4, −84, −6)
Occipital pole	3.78		(14, −94, 8)
Low-risk group			
Lingual gyrus	5.14	11,241	(4, −88, −6)
Superior LOC	4.52		(−30, −82, 22)
Occipital pole	4.41		(10, −96, 6)

Note: Reported activations are cluster corrected ($Z > 2.3$, cluster $p < .05$). Coordinates are in MNI 152 space in units of millimeters. If the *x*-coordinate is positive, the activation is in the right hemisphere; if the *x*-coordinate is negative, the activation is in the left hemisphere. The *y*-axis spans from posterior (negative values) to anterior (positive values), while the *z*-axis is from inferior (negative values) to superior (positive values). Structures without voxel counts represent local maxima within a given cluster.

The self-report data in our fMRI experiment confirm that we were able to re-create the counterarguing conditions detailed in Weber et al. (2013). We estimated a multi-level model with videos as a repeated measures variable nested within participants, accounting for the non-independent evaluations of 32 videos by each

participant. For low-drug-risk participants and PME as an outcome variable, effects for AS ($F = 13.8$, $p < .001$), MSV ($F = 7.3$, $p < .007$), and the interaction between AS and MSV ($F = 8.4$, $p < .004$) were significant. In contrast, for high-drug-risk participants, only a negative AS effect was significant ($F = 7.5$, $p < .006$), while the effects for MSV ($F = .89$, $p < .35$) and the interaction AS × MSV did not reach statistical significance ($F = 1.49$, $p = .22$), indicating that counterarguing primarily occurred in high-drug-risk participants (see Weber et al., 2013).

As expected, high-risk participants overall evaluated the stimulus PSAs as less effective than low-risk participants (PME, $\Delta M = .19$, $F = 6.58$, $p < .01$). Notably, the effects in our fMRI sample resembled the counterarguing conditions in Weber et al., 2013, but not in Kang et al. (2006). Assuming that high-risk adolescents would be more involved in the issue and thus motivated to attend to and process drug PSAs, Kang et al. (2006, p. 358) hypothesized and showed that the interaction between MSV and AS "is more likely to occur among high-risk than low-risk adolescents." As our self-report data in two samples and the activation pattern in the next section demonstrate, we concur with the assumption that high-risk participants are characterized by higher issue involvement and thus are more motivated to attend and process the messages. However, consistent with the ELM, our self-report data indicate that this interaction served to increase biased processing in high-risk participants rather than to draw their attention to arguments that they would find convincing and would result in increased persuasion, as Kang et al. (and other authors) have assumed.

The Neural Correlates of Counterarguing

We report key results for selected brain structures in this section. Generally speaking, we find commonalities in activation across both risk groups in posterior structures (e.g., the occipital lobe). However, we find additional activation exclusively among high-risk participants in more anterior regions under conditions of both high MSV and high AS. Thus, in high-risk participants, we see overall more brain activation in response to antidrug messages compared with low-risk participants (Figure 3). All activation reported here is cluster-corrected for multiple comparisons ($Z > 2.3$, $p < .05$). Table 1 specifies structures, maximum Z-scores, and coordinates by group and contrast.

Regions active in multiple contrasts and groups. Three main structures show commonalities in activation across both risk groups: the lateral occipital cortex (LOC), and the lingual and fusiform gyri. As explained in the discussion section, these findings are largely consistent with prior research on media persuasion. We find significant activation resultant from the MSV × AS interaction in the LOC for both risk groups (high-risk max $Z = 3.76$; low-risk max $Z = 4.86$), with significantly stronger activation in the superior LOC for high-risk participants than low-risk (high risk > low risk max $Z = 3.33$). Furthermore, the interaction of MSV and AS yields significantly greater activation in the fusiform gyrus for high-risk participants than

their low-risk counterparts (max $Z = 2.85$). We also find fusiform gyrus activation among both groups of participants for the MSV high > low contrast (high-risk max $Z = 4.32$; low-risk max $Z = 5.37$), but for neither group in the AS high > low contrast. Finally, our data reveal significant activation across risk groups in the lingual gyrus for the high > low AS contrast (high-risk max $Z = 4.18$; low-risk max $Z = 5.14$). However, no significant activation is present in this region for the MSV high > low contrast or the MSV × AS interaction.

Regions active exclusively for high-risk participants in the MSV × AS interaction. We find activation in the MSV × AS interaction among high-risk participants, but no significant activation among low-risk participants, in several structures; this suggests that these regions are likely to be especially useful for explaining differences in message processing between the risk groups (Figure 2). Most notably, we see activation exclusively among high-risk participants in the precuneus (PRE, max $Z = 4.23$), the frontal pole (FP, max $Z = 4.13$), the MFG (max $Z = 3.32$), and the STG (max $Z = 4.26$).

Brain-as-Predictor

As explained in the method section, we used both self-report and brain activation data from our fMRI sample (source 5) to predict message effectiveness in two independent, large samples (sources 3 and 4). As a reminder, following the procedures in Falk et al. (2012, 2014), we converted our data into ranks from 1 (lowest) to 32 (highest) for each risk group in both prediction samples.

For both prediction samples, we found similar results largely following theoretical expectations. For low-risk individuals in source 3, we see a significant effect for PMSV ($\beta = -.27$, $p < .05$) in our rank regression, a non-significant effect for PAS ($\beta = -.22$, $p = .70$), and a marginally significant interaction between PMSV and PAS

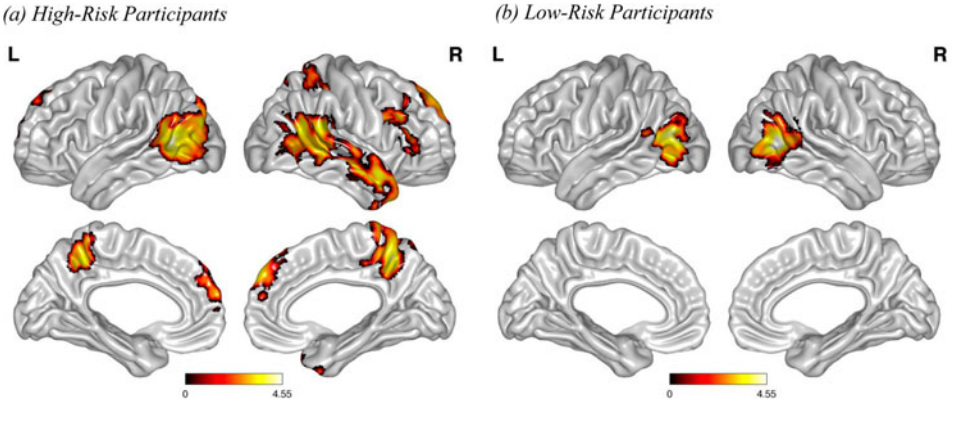

Figure 3 Lateral and medial view of each hemisphere (Xia, Wang, & He, 2013) showing activation for the MSV × AS interaction in (a) high-risk participants and (b) low-risk participants. All activation is cluster corrected ($Z > 2.3$, cluster $p < .05$); red indicates lower significant Z-scores, while yellow indicates higher significant Z-scores.

($\beta = 1.1$, $p < .07$). For low-risk individuals in source 4, we see a significant effect for PMSV ($\beta = -.21$, $p < .05$), a non-significant effect for PAS ($\beta = -.72$, $p = .19$), and a significant effect for the interaction between PMSV and PAS ($\beta = 1.52$, $p < .01$). The strong interaction between PMSV and PAS is of disordinal nature, suggesting that the significant interaction is meaningful even without significant main effects (Leigh & Kinnear, 1980).

In contrast, for the high-risk individuals, none of these effects reach significance in either sample. Accordingly, the models' prediction accuracy is high and significant for low-risk individuals (source 3: adjusted $R^2 = .53$, $p < .001$; source 4: adjusted $R^2 = .57$, $p < .001$), but less than half of this magnitude and non-significant for high-risk individuals (source 3 adjusted $R^2 = .23$, $p = .16$; source 4: adjusted $R^2 = .36$, $p < .10$). Essentially, while the prediction based on only self-report data of a small sample produced decent predictions of effectiveness among independent samples of low-risk individuals, the model failed to predict PME in independent samples of high-risk individuals.

Next, we added our neural correlates in Table 1 for the AS × MSV contrast (actual AS and MSV as defined by our conditions, *not* perceived AS and MSV) to our regression model. Two structures emerged as powerful predictors for PME in independent samples: the STG (56, 4, –18; Montreal Neurological Institute [MNI] 152 space) and the MFG (48, 24, 28) partly reaching into BA9. All other structures did not reach statistical significance. For the high-risk group in source 3, we find significant effects for both the self-report data and the neural predictors in the regression (PMSV, $\beta = .30$, $p < .05$; PAS, $\beta = -.57$, $p < .31$; PMSV × PAS, $\beta = 1.13$, $p < .05$; STG, $\beta = .40$, $p < .02$; MFG, $\beta = -.58$, $p < .001$) indicating effects of our neural predictors that go above and beyond self-report data. The same effect pattern holds when applied to source 4 (PMSV, $\beta = .26$, $p < .07$; PAS, $\beta = -.23$, $p < .63$; PMSV × PAS, $\beta = .86$, $p < .09$; STG, $\beta = .54$, $p < .001$; MFG, $\beta = -.45$, $p < .01$) with a slight emphasis on the neural predictors over the self-report data. In contrast, for the low-risk group in source 3, only MFG ($\beta = .40$, $p < .002$) reached statistical significance in addition to the self-report effects.

Further, compared to the high-risk group, the MFG effect has a positive direction; that is, signal change increases in MFG predict *higher* PSA ranks in low-risk individuals, but *lower* PSA ranks in high-risk individuals. For participants in source 4, none of the neural predictors significantly improved the predictions for low-risk individuals (although STG with $p < .09$ and MFG with $p < .09$ came close). With the two neural predictors included in the models, the prediction accuracy for high-risk individuals now almost reaches the level for low-risk individuals in source 3, and even surpasses the prediction accuracy in source 4 (source 3: high risk, adjusted $R^2 = .46$, $p < .001$; low risk, adjusted $R^2 = .65$, $p < .001$; source 4: high risk, adjusted $R^2 = .59$, $p < .001$; low risk, adjusted $R^2 = .57$, $p < .001$).

In a final step, we investigated the predictive power of previously and independently identified neural predictors for the persuasiveness of antidrug messages. Specifically, we considered one fROI (left BA9, max-z location: –9, 54, 33, MNI-space) from Chua et al. (2009), two fROIs (IFG; max-z: –46, 28, 12, MFG;

max-*z*: –30, 12, 54) from Ramsay et al. (2013), and one fROI (MPFC; center of gravity: –4.2, 56.8, –3.6)[2] from Falk et al. (2014).

Both Ramsay's MFG and Chua's left BA9 significantly improved the predictions for high-risk participants but *not* for low-risk participants in both samples (source 3: Ramsay's MFG, $\beta = .64$, $p < .0001$; Chua left BA9, $\beta = -.34$, $p < .02$, STG, $\beta = .67$, $p < .0001$, MFG, $\beta = -.78$, $p < .0001$; source 4: Ramsay's MFG, $\beta = .59$, $p < .0001$; Chua left BA9, $\beta = -.36$, $p < .02$; STG, $\beta = .80$, $p < .0001$, MFG, $\beta = -.63$, $p < .0001$). For the low-risk participants, none of the previously identified predictors significantly improved the predictions. With the addition of the two external neural predictors, the prediction accuracy in high-risk individuals matched and surpassed the prediction accuracy in low-risk individuals (source 3: high risk, adjusted $R^2 = .66$, $p < .001$; low risk, adjusted $R^2 = .67$, $p < .001$; source 4: high risk, adjusted $R^2 = .75$, $p < .001$; low risk, adjusted $R^2 = .61$, $p < .001$).

When we removed our neural predictors from the model (while keeping the self-report data) and replaced them with the external neural predictors, the only external predictor that contributed significantly was Falk's MPFC, and only for low-risk individuals. This suggests that although close in location to our neural predictors, the predictors previously identified by Ramsay and Chua cover a distinct aspect in the counterarguing process in high-risk individuals that spans across all four neural predictors.

Discussion

Overall, our findings suggest an interpretation consistent with prior research on the neural mechanisms of media persuasion (e.g., Chua et al., 2009; Falk et al., 2010; Ramsay et al., 2013). While both high- and low-risk participants are more attentive to high MSV and high AS messages, the interaction of MSV and AS among high-risk participants yields activity suggesting an increase in self-referencing as well as higher-order executive processing and cognitive effort that is absent among low-risk participants. This is consistent with counterarguing by high-drug-risk participants when faced with an antidrug message.

Visual Processing and Attention

For both groups, we find strong activation in LOC in all contrasts, as well as activation in the lingual and fusiform gyri for the AS and MSV contrasts, respectively. Broadly speaking, these areas are thought to be involved in visual processing and top-down control of attention (Hopfinger, Buonocore, & Mangun, 2000). This suggests that the combination of high MSV and high AS yields greater attention to the message for both risk groups, with a particularly pronounced effect for high-risk participants compared with their low-risk counterparts. Given the different patterns of activation across the MSV and AS contrasts, it seems that MSV is driving fusiform activation, whereas AS is driving activation in the lingual gyrus. Our results replicate Ramsay et al.'s (2013) finding that stronger arguments produce

greater activation in the lingual gyrus and LOC than weak ones. The results also accord with Chua et al.'s (2009) finding that tailored persuasive messages produce greater activation in the lingual gyrus than generic messages, and Falk et al.'s (2010) finding that activity in the fusiform gyrus is associated with successful persuasion to change health-related behavior.

Executive Processing and Self-referencing

Unlike the low-risk group, our high-risk participants showed patterns of activation in the MSV × AS interaction that are consistent with greater self-referencing, semantic processing, and weighing between different outcomes. Four structures in particular lend credence to this claim. First, the precuneus has been associated with self-referencing (e.g., Cavanna & Trimble, 2006) and integrating new information with prior knowledge (Wilson, Molnar-Szakacs, & Iacoboni, 2008). It appears to be more active in response to high-tailored messages than low-tailored ones (Chua et al., 2009) and stronger messages than weaker ones (Ramsay et al., 2013). Furthermore, Falk et al. (2010) found the precuneus to be associated with both intention and actual behavioral change in response to a health-related PSA. Second, the frontal pole (FP) is important to executive decision-making, and seems to enable cognitive branching to weigh between competing choices (Koechlin & Hyafil, 2007). Third, the MFG has been associated with language comprehension, semantic processing, and related high-order cognitive functions (Wilson et al., 2008). Falk et al. (2010) found the MFG to be associated with successful persuasion to change health-related behavior, Ramsay et al. (2013) found it to be more strongly activated by strong messages than weak ones, and Chua et al. (2009) found it to be more active in high-tailored messages than low-tailored ones. Finally, the STG is known to play an important role in language processing, narrative comprehension (Hasson, Furman, Clark, Dudai, & Davachi, 2008), as well as monitoring of social behavior and the mental states of others (Adolphs, 2006). Taken together, the activation of these regions among high-risk, but not low-risk, participants suggests greater executive and effortful processing by high-risk individuals to weigh the antidrug PSA message against existing conflicting beliefs. Notably, greater executive and effortful processing is difficult to conciliate with the assumption that high-risk participants simply ignored the messages or found the PSAs less convincing.

Brain-as-Predictor

The brain-as-predictor results reported in this study are particularly compelling for two reasons. First, we see that the inclusion of fROIs of counterarguing dramatically improves the prediction of message effectiveness in independent samples, especially among high-risk individuals. It is important to note here that, unlike previous persuasion neuroscience studies, we identified our fROIs of counterarguing by means of an experimental manipulation of message features that is firmly rooted in persuasion theory. Within this frame and consistent with previous studies (Weber

et al., 2013), we find that using self-report data in a small sample to predict message effectiveness in a larger sample works among low-risk participants but fails to do so for high-risk participants. However, once only two of our fROIs of counterarguing are included, the prediction accuracy among counterarguing high-drug-risk individuals in independent samples can reach and even surpass the prediction accuracy of non-counterarguing low-drug-risk individuals. Notably, like Falk et al. (2012), we accomplished this result with only a small sample of participants in an fMRI experiment. This provides further evidence for the notion that "neural focus groups" can indeed predict population-level media effects.

Secondly, we see that previously identified fROIs associated with processing persuasive messages do not independently predict PME in our paradigm. Still, it should be noted that these fROIs do improve model fit when included with fROIs identified in Table 1. Considered together, the results of our persuasion-theory-based fROI analysis combined with the brain-as-predictor paradigm provide evidence consistent with counterarguing processes that go beyond simply disregarding or finding PSAs less convincing. Moreover, our results demonstrate that these fROIs predict significant additional variance in PME in similar groups within new samples.

Limitations

A potential limitation of our study stems from a reliance on self-report measures of PME instead of a measurement of AE or behavior. While we acknowledge that PME is an indirect measure of AE, empirical results consistently show a robust relationship between PME and AE (Bigsby et al., 2013; Dillard, Shen, et al., 2007; Dillard, Weber, et al., 2007). Relatedly, the present study does not utilize a behavioral measure as an outcome variable. The extension of commonly used message effectiveness measures to behavioral outcomes has been demonstrated in other brain-as-predictor studies (e.g., Falk et al., 2010, 2011, 2014). Nevertheless, our results demonstrate the utility of self-report measures in a brain-as-predictor approach and provide a theoretically grounded extension that contextualizes previous research findings. For instance, Falk et al. (2014) found that neural activity in the MPFC associated with processing high AS PSAs predicts outcomes in an independent sample. In our study, we see similar results, but only for low-risk participants. These findings correspond with recent developments in the persuasion literature (see Weber et al., 2013) and underscore the importance of accounting for the interaction between individual factors and message features.

Another limitation relates to the nature of conducting experimental research in an fMRI environment. We argue here that issue involvement in high-risk participants leads to biased processing. However, some of our effects may be dampened by the fact that we required low-risk participants to pay attention to the messages while being in the scanner. At the same time, high-risk participants had no chance to disengage or *not* pay attention to the message, as all were told to watch and rate each PSA. It is important to note, however, that we indeed required both participant groups to watch and be at least attentive enough to rate PSAs. We may believe that

counterarguing is an inevitable outcome of that situation for our participants, but we did not *force* counterarguing. In addition, if counterarguing was an inevitable outcome in both groups, we should not see the clear group differences evident in our results.

A final limitation refers to the depth of our fMRI analyses. For this paper, we conducted a standard GLM analysis for our imaging data, which primarily identifies neural correlates of the experimental task (brain maps). This type of analysis ignores the fact that brain activation always spans across multiple networks in a complex, dynamic process. Thus, subsequent analyses should investigate functional connectivity among the neural correlates of counterarguing. For instance, in a recent psychophysiological interaction analysis, Ramsay et al. (2013) found that, within antidrug ads, contrasts between strong and weak conditions revealed differences in arousal-related activity in executive-functioning areas—the lateral PFC, especially the bilateral MFG (deactivation that was stronger for weak than for strong PSAs) and left IFG (increased activation for strong PSAs and deactivation for weak PSAs), with additional activation differences in socioemotional and language-processing regions (bilateral hippocampal gyrus, lingual gyrus, occipital lobe, and precuneus). As a next step, we call on our fellow researchers to join us and investigate if the dynamic interaction between individual characteristics and message features as persuasion theory dictates modulates moment-by-moment functional connectivity in message receivers' brains.

Conclusion

The present study is among a growing body of literature that uses neural activity in a small sample to predict outcomes in a larger independent sample. This brain-as-predictor approach (Berkman & Falk, 2013) not only explains considerable variance but also seems to do so even when there is limited demographic correspondence between samples. This is a clear advantage in that this physiological approach seems to bypass many of the limitations, such as people's inability to accurately self-report attitudes and behavior, inherent to more traditional methods. The findings reported in this study also provide confirmatory support for the structural correlates of persuasion identified in previous research (e.g., Chua et al., 2009; Falk et al., 2010; Ramsay et al., 2013). Moreover, the present study contextualizes these earlier findings by offering a theoretically grounded investigation of the neural correlates of counterarguing. We hope that our findings assist researchers and practitioners as they work to design messages that are more likely to persuade high-drug-risk target audiences.

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Notes

[1] Unlike Falk et al. (2014), we derived signal change of PSA contrasts on the group (second) level and not on the subject (first) level. This procedure avoids using average PSA ranks per subject for a second ranking procedure in which PSA are ranked against each other. We found this procedure less constraining for the variance of ranks and thus more conservative. In other words, with the ranking procedure used in Falk et al., the stability and fit of our prediction models reported here would increase rather than decrease.

[2] Compared to the MPFC mask generated by Falk et al. (2014), our MFG activation is more superior, posterior, and lateral. We find MFG activation in the right hemisphere, whereas Falk and colleagues found more medial MPFC activation tending toward the left hemisphere. However, Chua's fROI is comparably close to Falk's MPFC.

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