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Baseline physical activity moderates brain-behaviour relationships in response to framed health messages

Jeesung Ahn 101,2,3,4, Nicole Cooper², Yoona Kang^{2,4}, Matthew Brook O'Donnell², Mikella A Green⁵, Nanna Notthoff^{6,7}, Laura L. Carstensen 106, Gregory R Samanez-Larkin 105, Emily B. Falk 1,2,8,9,4

E-mail: jeesung@sas.upenn.edu; Emily B Falk, Annenberg School for Communication, University of Pennsylvania, 3620 Walnut St., Philadelphia, PA 19104, USA. E-mail: emily.falk@asc.upenn.edu

Abstract

Health messaging often employs gain-framing (highlighting behaviour benefits) or loss-framing (emphasizing nonengagement risks) to promote behaviour change. This study examined how neural responses to gain- and loss-framed messages predict changes in physical activity. We conducted a *mega-analysis* of raw fMRI and pedometer/accelerometer data from four studies (N = 240) that tracked brain activity during message exposure and real-world physical activity longitudinally. Focusing on brain regions theorized by the Affect–Integration–Motivation framework—the anterior insula, ventral striatum, ventromedial prefrontal cortex, dorsal striatum, and presupplementary motor area—we found that baseline physical activity levels moderated brain–behaviour relationships in response to message framing. More active individuals increased physical activity post-intervention when these brain regions responded more strongly to loss-framed messages, suggesting that neural sensitivity to inactivity risks may reinforce behaviour maintenance in this group. Conversely, less active individuals increased physical activity when brain responses were stronger to gain-framed messages, indicating that sensitivity to activity benefits may facilitate action initiation in this group. These findings suggest that message effectiveness depends on the interaction between framing, neural processing, and pre-existing behavioural patterns. By linking neurocognitive mechanisms with real-world outcomes, we highlight the importance of personalized, neuroscience-informed health interventions tailored to individual neural and behavioural characteristics to optimize behaviour change strategies.

Keywords: health communication; message framing; physical activity; brain-behaviour relationships; fMRI; individual differences

Introduction

Physical inactivity increases the risk for a wide range of health problems, including diabetes (Hu 2003, Healy et al. 2008, Venables and Jeukendrup 2009), cardiovascular disease (de Rooij et al. 2016), cancer mortality (Seguin et al. 2014), premature death (Wilmot et al. 2012), and all-cause mortality (Biswas et al., 2015, Carlson et al. 2018, Mokdad et al., 2004). To address this public health challenge (Lee et al. 2012), efforts to promote activity often involve persuasive messaging campaigns. However, important questions remain about how specific message features influence

behaviour change and how these effects vary across different individuals.

Message framing: gain vs. loss

Health messages can be framed to emphasize either the *benefits* of adopting behaviour (gain-framed) or the *risks* of not engaging in the behaviour (loss-framed). The framing of health messages can play an important role in their persuasive effectiveness (Gallagher & Updegraff, 2011, Rothman and Salovey 1997, Updegraff and Rothman 2013).

¹Department of Psychology, University of Pennsylvania, Philadelphia, PA 19104, United States

²Annenberg School for Communication, University of Pennsylvania, Philadelphia, PA 19104, United States

³Department of Psychiatry and Behavioral Sciences, Stanford University, Palo Alto, CA 94304, United States

 $^{^4}$ Department of Psychology, Rutgers, The State University of New Jersey, Camden, NJ 08102, United States

⁵Department of Psychology and Neuroscience, Duke University, Durham, NC 27708, United States

⁶Department of Psychology, Stanford University, Stanford, CA 94305, United States

⁷Faculty of Sport Science, Leipzig University, Leipzig 04109, Germany

⁸Wharton Marketing Department, University of Pennsylvania, Philadelphia, PA 19104, United States

⁹Wharton Operations, Information and Decisions Department, University of Pennsylvania, Philadelphia, PA 19104, United State

^{*}Corresponding author. Jeesung Ahn, Department of Psychiatry and Behavioral Sciences, Stanford University, 401 Quarry Rd, Palo Alto, CA 94304, USA.

The relative effectiveness of gain- vs. loss-framed messages may depend on the type of health behaviour being targeted. For instance, gain-framed messages are typically more effective than loss-framed messages for disease prevention behaviours, such as sunscreen use (Detweiler et al. 1999), smoking cessation (Toll et al. 2007), and flossing (OKeefe and Jensen 2007). In contrast, loss-framed messages tend to be more effective for illness detection behaviours, including breast self-examination (Meyerowitz and Chaiken 1987, Meyerowitz et al. 1991) and HIV testing (Kalichman and Coley 1995).

Some evidence suggests that physical activity, as disease prevention behaviour, may be more effectively promoted through gain-framed messaging (Gallagher and Updegraff, 2011, Latimer et al. 2008). However, empirical evidence remains inconclusive. Although some studies report greater effectiveness for gain-framed appeals (McCall and Ginis 2004), meta-analyses and reviews highlight substantial variability in framing effects across contexts (O'Keefe and Jensen 2007, van't Riet et al. 2010, Williams et al. 2019). This inconsistency suggests that individual differences—such as baseline engagement in physical activity—may moderate responses to framed messages (Jones et al. 2004, Berry and Carson 2010).

Individual differences in health persuasion: baseline engagement

Mixed findings related to message framing effects in physical activity promotion may partly be attributable to individual-level characteristics of message recipients (Latimer et al. 2008). Studies across various health domains (e.g. smoking, Fucito et al. 2010; skin cancer self-examination, van't Riet et al. 2012) have shown that the effectiveness of gain- and loss-framed messages can vary depending on recipients' baseline engagement in the target behaviour. In the context of physical activity, loss-framed messages emphasizing inactivity risks (e.g. obesity, chronic disease) have increased resolve to exercise among physically inactive individuals (de Bruijn et al. 2014). Conversely, gain-framed messages highlighting the benefits of being active (e.g. improved cardiovascular health, enhanced mood) have increased motivation among already active populations (Hevel et al. 2019).

These findings suggest that individual differences in past behaviour and experiences can influence how message framing affects motivation. However, framing effects have been inconsistently observed across different outcome measures, even within individual studies, with effects detected on some metrics (e.g. resolve to exercise) but not on others (e.g. exercise intention) (de Bruijn et al. 2014). Many studies also rely heavily on self-reported data (Williams et al. 2019), which may not fully capture realworld behaviour change (Sheeran 2002), leaving open the possibility that interactions between baseline behaviour and framing effects may or may not operate in the same way with objectively logged behaviour change. To address these questions, we focus on the use of objective measures, such as accelerometers, which provide ecologically valid assessments of physical activity (Dunton 2017).

Furthermore, prior research on physical activity has largely focused on between-group comparisons, categorizing participants by baseline characteristics (e.g. active vs. inactive) and exposing them to either gain- or loss-framed messages. As a result, little is known about how baseline behaviours relate to individuals' relative processing of differently framed messages and how this processing translates into behavioural outcomes.

Understanding within-person variability in the neurocognitive processing of framed health messages is critical for identifying mechanisms underlying intervention success.

Individual differences in neural responses to message framing

The Affect-Integration-Motivation (AIM) framework (Samanez-Larkin and Knutson 2015) proposes that individual variability in neural responses to framed health messages may play a critical role in motivating behaviour change. It provides a theoretical lens to explore how affective salience (e.g. emotional responses and detection of salient health outcomes), value integration (e.g. weighing the benefits and risks of behaviour change), and motivation (e.g. translating evaluations into action) contribute to individual differences in processing framed health messages. The framework hypothesizes that activity in specific brain regions underpins these processes: the anterior insula and ventral striatum (affective salience), the ventromedial prefrontal cortex (vmPFC; value integration), and the presupplementary motor area (pre-SMA) and dorsal striatum (motivation). These regions are theorized to form a functional pathway that predicts behaviour and decisionmaking across various contexts.

Within this pathway, the vmPFC and ventral striatum constitute core components of the brain's valuation system, integrating subjective value signals for both rewarding and aversive outcomes (Levy and Glimcher 2012, Bartra et al. 2013). Health messaging research highlights the vmPFC's role in predicting behaviour change (Falk et al. 2010, 2011, 2012, 2015, Kang et al. 2018). For example, greater vmPFC activation during exposure to antismoking messages has been associated with subsequent reductions in smoking outside the laboratory (Falk et al. 2011, Cooper et al. 2015), although some studies have reported null associations between vmPFC activation and message effectiveness (e.g. Schmälzle et al. 2020).

Research on gain- vs. loss-framing provides further evidence for the role of valuation regions in driving health behaviour change. For example, one study found that greater vmPFC activation in response to gain-framed messages, compared to loss-framed messages, was associated with increased sunscreen use (Vezich et al. 2017). This finding aligns with neuroeconomic research demonstrating heightened vmPFC and ventral striatal activity during the processing of economic gains relative to losses (Tom et al. 2007). Such differential neural responses within valuation regions suggest that gain- and loss-framed messages may engage distinct processes to motivate behaviour change.

While valuation processes are central to understanding message effectiveness, the AIM framework proposes that valuation is intertwined with affective and motivational processes (Samanez-Larkin and Knutson 2015). That is, individual differences in health behaviour change may also reflect variability in how individuals process affective salience and integrate perceived rewards or risks to guide health decisions (Keller et al. 2003, Notthoff and Carstensen 2014, Mather 2016, Raposo et al. 2021). Specifically, emotional salience encoded by the anterior insula and ventral striatum may inform value computations in the vmPFC, which integrates these signals with other factors, such as self-relevance (Rameson et al. 2010). These integrated valuations may ultimately drive behaviour through motivational circuits involving the dorsal striatum and pre-SMA (Samanez-Larkin and Knutson 2015). A more comprehensive understanding of how neural systems supporting affect, valuation, and motivation contribute to individual differences in processing gain- and loss-framed message could

Table 1. Participant demographics and message intervention characteristics.

| Study | Participants | | | Messages | |
|-----------------------------------|-----------------------------|--------------------|---------------------|-------------------------------|---------------------------|
| | Recruited N (Included N) | Mean age (s.d.) | Gender (Women %) | Count | Message Duration (sec) |
| Study 1 | 66 | 33.69 | 66.67 | Gain: 18 | 10 |
| (Falk et al. 2015) | (44) | (14.90) | | Loss: 15 | |
| | | | | Control: 23 Non-health: 20 | |
| Study 2 | 220 | 33.30 | 61.54 | Gain: 10 | 8 |
| (Kang et al. 2018) | (131) | (11.15) | | Loss: 10 | |
| | | | | Control: 10 | |
| | | | | Non-health: 30 | |
| Study 3 | 37 | 73.11 | 67.86 | Gain: 30 | 16 |
| (Carstensen and Goldstein et al., | (30) | (6.51) | | Loss: 30 | |
| unpublished manuscript) | | | | Control: 24 | |
| | | | | Non-health: 6 | |
| Study 4 | 43 | 50.06 | 48.39 | Gain: 15 | 16 |
| (Carstensen and Goldstein et al., | (35) | (23.50) | | Loss: 15 | |
| unpublished manuscript) | | | | Control: 13 | |
| | | | | Non-health: 2 | |
| Total | 366 | 40.35 | 63.25 | Gain: 73 (58+) | 12.5 |
| | (240) | (19.03) | | Loss: 70 (55 ⁺) | |
| | , , | , , | | Control: 70 (57+) | |
| | | | | Non-health: 58 (56+) | |

 $N = number; Included \ N = number \ of participants included \ in the current investigation \ after applying \ rigorous \ exclusion \ criteria; s.d. = standard \ deviation; leaves the current investigation of the properties of participants included in the current investigation after applying \ rigorous \ exclusion \ criteria; s.d. = standard \ deviation; leaves \ rigorous \ exclusion \ rigorous \ rigorous \ exclusion \ rigorous \ exclusion \ rigorous \ rigorous \ exclusion \ rigorous \ exclusion \ rigorous \ exclusion \ rigorous \ rigoro$ gender = percentage of participants identifying as women (the remainder identified as male; studies did not provide an option for participants to identify as other categories); + = total number of distinct messages after removing duplicates used in both Studies 3 and 4; message duration = length of time each message was presented; all messages were presented for the same duration within each study.

help clarify why people vary in their responsiveness to framed health messaging interventions.

The current study

This study investigates how health message framing is associated with neural activity in regions of interest (ROIs) proposed by the AIM framework. Specifically, we examine whether neural responses to gain- and loss-framed messages are related to post-intervention physical activity behaviour, and whether these brain-behaviour relationships are moderated by baseline physical activity levels. By linking neural responses to objectively measured real-world physical activity over time, we aim to better understand how individual differences in message processing contribute to variation in intervention outcomes.

To achieve this, we use functional magnetic resonance imaging (fMRI) to monitor neural responses to gain- and loss-framed messages promoting physical activity. This study addresses whether (i) gain- and loss-framed messages elicit different levels of activation in ROIs associated with affective salience, value integration, and motivation; (ii) neural responses in these ROIs are related to post-intervention physical activity behaviour outside the lab; and (iii) participants with varying baseline levels of physical activity exhibit different relationships between neural responses and post-intervention behaviour. To comprehensively answer these questions, we conduct a mega-analysis by pooling and re-analyzing raw fMRI and pedometer/accelerometer data from four independent datasets with similar study designs.

Methods

Study inclusion and procedures

We aggregated four datasets collected by laboratories at the University of Michigan (Falk et al. 2015), the University of Pennsylvania (Kang et al. 2018), and Stanford University (P.I. Laura Carstensen, P.I. Mary K. Goldstein; unpublished). We refer to these datasets as Study 1 (Falk et al. 2015), Study 2 (Kang et al. 2018), and Studies 3 and 4 (Carstensen and Goldstein et al., unpublished manuscript; see Table 1). Studies 1 and 2 examined neural responses to health messages and subsequent changes in physical activity. Studies 3 and 4 focused on age-related differences in neural reactivity to positive and negative health messages (see Mather and Carstensen 2005, Carstensen and DeLiema 2018).

All datasets followed a three-session design (Fig. 1). During the baseline appointment (T1), participants visited the lab and were fitted with accelerometers (Studies 1 and 2) or pedometers (Studies 3 and 4). Each participant's baseline physical activity was measured starting from T1 until their T2 visit. The fMRI intervention appointment (T2) occurred ~1 week (Studies 1, 3, and 4) or up to 10 days (Study 2) after T1. At T2, participants underwent fMRI scanning while viewing and listening to health messages designed to promote physical activity. Post-intervention physical activity was tracked for up to 4 weeks (Studies 1 and 2) or 1 week (Studies 3 and 4). Participants then returned to the lab for the final session (T3), where endpoint data were collected. Detailed experimental procedures for each study are provided in Supplementary Material 1 [S1].

Study characteristics and participants

A total of 240 participants from an original sample of 366 were included in our mega-analysis. For overall participant demographics, see Table 1. The large sample size allowed us to apply rigorous exclusion criteria: excessive motion during fMRI scanning (n = 33), technical errors during fMRI scanning (n=16), fMRI ineligibility discovered during scanning (n=24)e.g. claustrophobia), withdrawal before the T2 fMRI session (n=12), withdrawal before the T3 endpoint session (n=4), and missing physical activity data despite completing all sessions

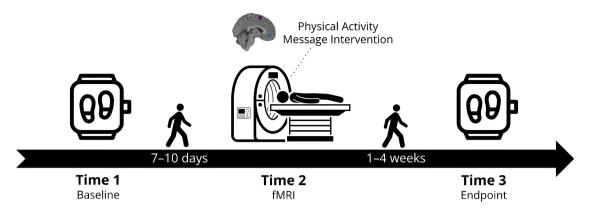


Figure 1. Three-session study design and physical activity monitoring timeline. All studies followed a three-session design. At the baseline session (T1), participants were fitted with accelerometers (Studies 1 and 2) or pedometers (Studies 3 and 4) to measure baseline physical activity until the fMRI intervention session (T2). T2 occurred ~1 week after T1 (or up to 10 days later in Study 2), during which participants underwent fMRI scanning while viewing and listening to physical activity-promoting health messages. Post-intervention physical activity was monitored for up to 4 weeks (Studies 1 and 2) or 1 week (Studies 3 and 4). Participants returned for the endpoint session (T3) to complete behavioural assessments.

(n=37; e.g. accelerometer/pedometer malfunctions). Full exclusion criteria for individual studies are detailed in Supplementary Table 1 [S2].

Study 3 adopted a between-person design, where participants were exposed to either gain- or loss-framed messages but not both. Therefore, Study 3 was excluded from analyses focusing on within-person comparisons of gain vs. loss framing. For more detailed descriptions of each study, see Supplementary Material 1 [S1]. Since most participants in Study 3 were older adults (i.e. >65 years), who typically exhibit different baseline physical activity levels compared to younger adults (Chodzko-Zajko et al. 2009), we also tested whether age moderated the current results. No significant age effects were found (Supplementary Tables 6 and 7 [S7]).

Health messages

All physical activity messages were presented in text and audio format inside the fMRI scanner. For the purpose of the current investigation, two researchers independently coded each message as gain-framed, loss-framed, control, or non-health-related. Gain-framed messages were defined as those describing positive or desirable outcomes associated with physical activity, including direct benefits (e.g. "Being active feels good, gives you more energy, and makes you feel better about yourself.") or the prevention of negative outcomes (e.g. "When people are stronger and more active, they have better posture, which is the best way to manage and prevent back pain."). Loss-framed messages were defined as those describing negative or undesirable outcomes associated with physical inactivity, including negative health consequences (e.g. "A sedentary lifestyle increases the risk of developing diabetes, hypertension, colon cancer, depression, anxiety, obesity, and weak muscles and bones.") or missed opportunities (e.g. "You are more likely to die early if you stay inactive. You will miss out on the people and things that mean most to you in life.").

Messages that did not describe physical activity outcomes and instead focused on behavioural instructions were coded as control messages (e.g. "Find a time to go for a 15–20 minute walk 3–4 times a week. Try casual walks to and from nearby places."). All studies also included non-health-related stimuli unrelated to physical activity (e.g. "Balance your checkbook regularly. Check your bank account balance to avoid errors."), which were excluded from the analyses.

Across the 4 datasets, 73 gain-framed messages, 70 loss-framed messages, 70 control messages, and 58 non-health-related stimuli were identified. Intercoder reliability was 91.53%. Discrepancies in coding were resolved through discussions between the coders and the larger research team. Table 1 breaks down the number of messages included in the analyses. Table 2 provides examples of gain-, loss-framed, and control messages used in each study. Studies 3 and 4 utilized the same pool of health messages. The full list of stimuli is provided in Supplementary Material 10 [S10].

Physical activity measures

Physical activity was tracked using either wrist-worn GENEA triaxial accelerometers (Studies 1 and 2) or Yamax Digi-walker SW200 pedometers (Studies 3 and 4). Accelerometers recorded continuous physical activity levels throughout the measurement period, while pedometers tracked daily step counts. Participants in Studies 3 and 4 were instructed to manually record their pedometer-measured steps in a paper log at the end of each day. To minimize between-study discrepancies that might be introduced by the use of different devices (i.e. accelerometer vs. pedometer), we converted accelerometer outputs from Studies 1 and 2 into daily step counts using Activinsights GENEActiv software (https://www.activinsights.com/expertise/geneactiv/ downloads-software/). To further account for potential discrepancies between accelerometer- vs. pedometer-based measurements, we standardized step count data within each study and used each participant's standardized z-scores as the final measure of physical activity. Sensitivity analyses confirmed that the results were robust to both raw and standardized metrics (Supplementary Table 5 [S6]).

fMRI data acquisition and preprocessing

To incorporate raw neuroimaging data acquired from four different projects, we organized brain images using the standardized Brain Imaging Data Structure (BIDS) format (Gorgolewski, 2016) and developed an in-house Nipype-based pipeline for all image processing and analyses (Gorgolewski et al. 2011, http://nipype.readthedocs.io/en/latest). fMRI data were preprocessed using the standard workflow of fMRIPrep software version 20.0.6 (Esteban et al. 2019; RRID:SCR_016216), which was based on Nipype 1.4.2 (Gorgolewski et al. 2011; RRID:SCR_002502). All functional images were resampled to 3 × 3 × 3 mm voxels in a common template

Table 2. Example messages from the health message interventions.

| Study | Example Health Messages Gain-framed health message: Sitting less over time might mean fewer pills. When you sit less, your body is better at making good cholesterol, and lowering bad cholesterol, blood pressure, and blood sugar. Loss-framed health message: The more you sit the more damage it does to your body. When you sit for long periods of time, your body can't handle sugar and fat—this can mean higher risk for disease. Control health message: After an hour of sitting, try standing for 5 min. Stand up while you read, watch TV, talk on the phone, fold laundry, or write an email. Non-health-related stimulus: Use more coupons when you shop. Sign up for email lists from your favorite stores—that way, coupons will be sent to you. | | | |
|---|---|--|--|--|
| Study 1 (Falk et al. 2015) | | | | |
| Study 2 (Kang et al. 2018) | Gain-framed health message: As you become more active, your bones will grow stronger. Stronger bones will help you stay pain-free. Loss-framed health message: If you continue to be sedentary, your bones will weaken faster as you get older. This makes it more difficult to do things you like. Control health message: Find a time every day when you can get out and walk around for at least 15 min. For example, maybe you can walk to and from your job every day. Non-health-related stimulus: Balance your checkbook on a regular basis. Look up your bank account balance to avoid errors. | | | |
| Study 3 (Carstensen and Goldstein et al., unpublished manuscript) | Gain-framed health message: Walking regularly improves people's ability to cope with stressful events in their lives. Loss-framed health message: Not walking regularly decreases people's ability to cope with their lives' stressful events. | | | |
| Study 4 (Carstensen & Goldstein et al., unpublished manuscript) | Control health message: People can walk in my places, for example at home and in the workplace. Non-health-related stimulus: Many impressionist painters were inspired by pedestrians. | | | |

space of $53 \times 63 \times 46$ voxels. Full descriptions of the preprocessing pipelines are available in Supplementary Material 1 [S1].

General linear model analysis

To extract neural responses to gain- and loss-framed messages, we conducted first-level general linear model (GLM) analyses using Nipype-based SPM 12 (Wellcome Department of Cognitive Neurology, London, UK). fMRI volumes were treated as temporally correlated time series and modeled by convolving a canonical haemodynamic response function with a boxcar function marking the onset and duration of each trial. The resulting haemodynamic functions were entered as covariates in a GLM, along with covariates representing session effects and realignment parameters.

First-level models included separate regressors for each message trial type—gain-framed, loss-framed, and control—as well as for non-health-related stimuli and response periods. Least squares parameter estimates of the best-fitting synthetic haemodynamic response function for each regressor of interest (averaged across scans) were calculated and stored as contrast images for each participant.

Specifically, we conducted pairwise contrasts comparing neural responses to (i) gain- vs. loss-framed messages; (ii) gainframed vs. control messages; and (iii) loss-framed vs. control messages. Our primary analysis focused on neural responses to gain- vs. loss-framed messages. To further isolate the effects of framed messages, we conducted supplementary analyses comparing both gain-framed and loss-framed messages to control messages that did not describe the health outcomes of physical activity.

To preserve spatial specificity in subcortical regions while ensuring adequate smoothing in cortical areas, we applied a 6 mm full-width at half-maximum (FWHM) smoothing kernel at the first-level GLM stage. This kernel size was chosen based on recommendations for studies involving both cortical and subcortical regions, optimizing the balance between sensitivity and spatial resolution (Mikl et al. 2008).

ROI analysis

The regions of interest (ROI) masks, selected based on the AIM framework, were derived meta-analytically using the Neurosynth online database (Yarkoni et al. 2011). The following search terms were used to generate association maps: "anterior insula", "ventral striatum", "vmPFC", "dorsal striatum", and "pre SMA". The association maps were thresholded at p < .01, FDR corrected (Fig. 2). To address the large spatial extent of the resulting maps, we identified the peak coordinate with the highest z-score within each ROI and created a spherical mask extending up to 10 mm from the peak, constrained to overlap with the ROI boundaries from the association map. The masks were further refined by restricting them to grey matter, as defined by the MNI ICBM152 template provided by Nilearn. All masks were visually inspected to confirm alignment with the intended ROIs. Further details on each ROI mask can be found in Supplementary Table 2 [S3] and additional results using the full, unrestricted masks are included in Supplementary Table 8 and Supplementary Figure 4 [S8].

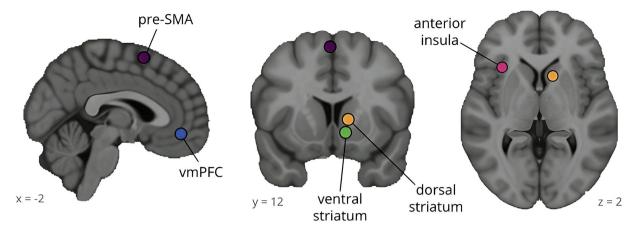


Figure 2. Affect-value integration-motivation ROIs. Masks for the anterior insula, ventral striatum, vmPFC, dorsal striatum, and pre-SMA were meta-analytically generated from Neurosynth association maps (p < .01, FDR corrected; Yarkoni et al. 2011). Within each ROI map, a spherical mask with a radius of up to 10 mm was created centering on the coordinate with the highest z-score. The spherical masks were further constrained to grey matter using the MNI ICBM152 template. For illustration purposes, each mask is displayed as a complete circle in the figure.

Parameter estimates of activity within each ROI were extracted from first-level GLM contrasts comparing (i) gain- vs. loss-framed messages; (ii) gain-framed vs. control messages; and (iii) lossframed vs. control messages. We conducted robust linear regression analyses to test whether neural activity within each ROI was associated with post-intervention step counts. We also examined whether the relationship between ROI activity during health message exposure and post-intervention step counts was moderated by baseline physical activity, measured between the T1 and T2 visits. All regression models included covariates to control for the effects of age, study, and experimental conditions not central to this investigation. A dummy variable for each study was included to control for potential study-level differences, such as participant characteristics, data collection methods, and other study-specific factors that were not the focus of this investigation. Lastly, we performed follow-up simple slopes analyses to further explore whether the relationship between ROI activity and postintervention physical activity varied across three levels of baseline physical activity engagement: 1 s.d.) below the mean step count across participants, the mean step counts, and 1 s.d. above the mean. We report results that remained robust to FDR correction at p < .05.

Results

Physical activity behaviours

At baseline, participants walked an average of 9394 steps per day (s.d. = 3628) across the four studies (see Table 3 for details). After the message intervention, participants walked an average of 9506 steps per day (s.d. = 3899). On average, the intervention did not result in a significant change in step counts (paired t-test comparing pre- vs. post-intervention: t(239) = 0.72, p = .47, mean increase = 112.45 steps/day). The non-significant increase in step counts was primarily driven by participants in Studies 1 and 2, who exhibited minimal change in mean step counts, though substantial individual variability was present. In contrast, participants in Studies 3 and 4 demonstrated significant increases in step counts following the intervention (Study 3: t(29) = 2.43, p = .02, mean increase = 661 steps/day; Study 4: t(34) = 2.69, p = .01, mean increase = 850 steps/day). Given the within-person designs of Studies 1, 2, and 4, the current investigation focused on within-person relationships between brain

Table 3. Objectively measured physical activity behaviours at baseline and post-intervention.

| | Daily step counts (s.d.) | | | | |
|--------------------|--------------------------|-----------------------|-------------------------|--|--|
| Study | Baseline | Post- intervention | Change | | |
| Study 1 | 10073 | 10210 | +137 (2554) | | |
| (Falk et al. 2015) | (3258) | (4121) | t(43) = 0.36, p = .72 | | |
| Study 2 | 10473 | 10254 | -218 (2621) | | |
| (Kang et al. 2018) | (3172) | (3530) | t(130) = -0.95, p = .34 | | |
| Study 3 | 5867 | 6527 | +661 (1490) | | |
| (unpublished) | (3534) | (4023) | t(29) = 2.43, p = .02 | | |
| Study 4 | 7256 | 8377 | +850 (1869) | | |
| (unpublished) | (3214) | (3460) | t(34) = 2.69, p = .01 | | |
| Total | 9394 | 9506 | +112 (2419) | | |
| | (3628) | (3899) | t(239) = 0.72, p = .47 | | |

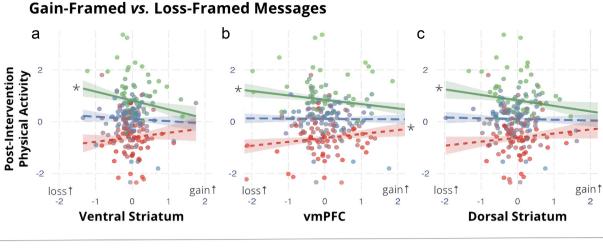
Note: Average daily step counts during baseline and post-intervention periods, as well as the change in step counts (post-baseline). Substantial individual variability was observed, as indicated by the s.d.

activity and behaviour rather than between-person main effects of gain vs. loss framing on behaviour change. Also, the analysis emphasized individual-level differences in how brain responses and physical activity interacted, rather than study-level effects.

Baseline behaviour moderates neural responses to gain vs. loss framing and physical activity outcomes

We first examined whether neural responses to gain- vs. lossframed messages differed in our a priori-defined ROIs associated with affective salience, value integration, and motivation. On average, we did not observe any significant main effects of message framing in these core ROIs (p > .05, Supplementary Table 3 [S4]). Moreover, robust linear regression models showed that ROI activity during gain- vs. loss-framed messages was not significantly associated with post-intervention physical activity across the overall sample (FDR-corrected p > .05).

However, robust regression analyses indicated that the relationship between ROI responses and post-intervention physical activity varied depending on participants' baseline physical activity. Neural responses to gain- vs. loss-framed messages in the ventral striatum, vmPFC, and dorsal striatum showed negative



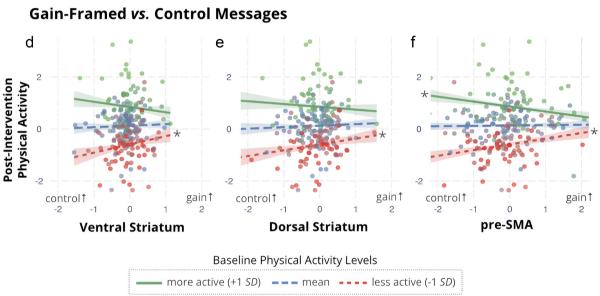


Figure 3. Brain-behaviour relationships explaining post-intervention physical activity outcomes. Upper panel: Simple slopes analyses showed that more active participants (+1 s.d., solid line) exhibited greater post-message physical activity when their (a) ventral striatum, (b) vmPFC, and (c) dorsal striatum responded more strongly to loss- vs. gain-framed health messages. Less active participants (-1 s.d., dotted line) showed increases in physical activity when their (b) vmPFC responses were stronger to gain- us. loss-framed messages. Positive x-axis values indicate greater neural responses to gain- vs. loss-framed messages and negative values indicate greater responses to loss- vs. gain-framed messages. Lower panel: Less active participants (-1 s.d., dotted line) showed greater post-message physical activity when their (d) ventral striatum, (e) dorsal striatum, and (f) pre-SMA responded more strongly to gain-framed messages compared to control messages. More active participants (+1 s.d., solid line) showed greater post-intervention activity when their (f) pre-SMA responded more strongly to control vs. gain-framed messages. Positive x-axis values indicate greater neural responses to gain- vs. control messages and negative values indicate greater responses to control vs. gain-framed messages. Note: All analyses controlled for age, study, and experimental conditions. Asterisks (*) indicate significance in simple slopes analyses at p < .05.

interactions with baseline physical activity in explaining step counts after message exposure (Fig. 3, Table 4). Although similar negative interaction effects were observed in the anterior insula and pre-SMA, these did not reach statistical significance at p < .05(Table 4).

Post hoc simple slopes analyses revealed that the negative interaction effects were primarily driven by participants with higher baseline physical activity. Participants who were 1 s.d. more active than average at baseline walked more after message exposure when their neural responses to loss-framed messages, relative to gain-framed messages, were greater in the ventral striatum ($\beta = -0.34$, p = .008), vmPFC ($\beta = -0.17$, p = .01), and dorsal striatum ($\beta = -0.21$, p = 0.04; Fig. 3, upper panel; significance indicated with asterisks).

Participants who were 1 s.d. less active than average at baseline also showed significant effects in the vmPFC ($\beta = 0.15$, p = .04; Fig. 3, upper panel). Specifically, stronger vmPFC responses to gain-framed messages, compared to loss-framed messages, were associated with increased physical activity post-intervention for this group of participants.

Baseline behaviour moderates neural responses to gain-framed vs. control messages and physical activity outcomes

To further understand the unique contributions of each framing type to post-intervention behaviours, we compared ROI activity in response to gain-framed and loss-framed messages against nonframed control messages. These control messages provided

Table 4. Interaction of ROI responses and baseline physical activity in predicting post-intervention physical activity.

| ROI | β | t (df) | p (uncorrected) | p (FDR corrected) | | | | |
|---|-------|-------------|--------------------|----------------------|--|--|--|--|
| Gain-framed messages vs. loss-framed messages | | | | | | | | |
| Anterior insula | -0.04 | -0.66 (197) | 0.54 | 0.54 | | | | |
| Ventral striatum | -0.27 | -2.69 (197) | 0.007* | 0.02* | | | | |
| vmPFC | -0.17 | -3.52 (197) | 0.001* | 0.005* | | | | |
| Dorsal striatum | -0.19 | -2.32 (197) | 0.02* | 0.04* | | | | |
| pre-SMA | -0.04 | -0.92 (197) | 0.39 | 0.48 | | | | |
| Gain-framed messages vs. control messages | | | | | | | | |
| Anterior insula | -0.07 | -0.91 (209) | 0.41 | 0.41 | | | | |
| Ventral striatum | -0.27 | -2.72 (209) | 0.009* | 0.02* | | | | |
| vmPFC | -0.06 | -1.71 (209) | 0.12 | 0.15 | | | | |
| Dorsal striatum | -0.17 | -2.33 (209) | 0.03* | 0.04* | | | | |
| pre-SMA | -0.21 | -4.57 (209) | 0.001* | 0.001* | | | | |
| Loss-framed messages vs. control messages | | | | | | | | |
| Anterior insula | 0.02 | 0.37 (215) | 0.72 | 0.78 | | | | |
| Ventral striatum | 0.03 | 0.30 (215) | 0.78 | 0.78 | | | | |
| vmPFC | 0.04 | 0.87 (215) | 0.39 | 0.66 | | | | |
| Dorsal striatum | 0.10 | 1.13 (215) | 0.25 | 0.64 | | | | |
| pre-SMA | -0.06 | -1.57 (215) | 0.12 | 0.60 | | | | |

Note: Robust regression analyses controlled for the effects of age, study, and experimental conditions that were not the focus of this investigation. *Statistical significance at p < .05.

general how-to guidance on physical activity without referencing specific health outcomes. When comparing neural responses to gain-framed vs. control messages, we observed negative interactions between baseline physical activity and activity in the ventral striatum, dorsal striatum, and pre-SMA in explaining physical activity after message exposure (Table 4). In contrast, no significant interactions between ROI responses to loss-framed vs. control messages and baseline physical activity were observed to explain post-intervention physical activity (p > .05, Table 4).

Post hoc simple slopes analyses revealed that participants with lower baseline physical activity walked more after message exposure when their ROI responses showed increased activity to gainframed messages compared to control messages (simple slopes for participants 1 s.d. below the mean in baseline physical activity: ventral striatum $\beta = 0.32$, p = .02; dorsal striatum $\beta = 0.22$, p = .02; pre-SMA $\beta = 0.22$, p = .001; Fig. 3, lower panel). Conversely, participants with higher baseline physical activity walked more after message exposure when their pre-SMA responses were stronger to control messages than to gain-framed messages (simple slopes for participants 1 s.d. above the mean in baseline physical activity: pre-SMA $\beta = -0.19$, p = .002; Fig. 3, lower panel).

Discussion

This study examined how neural responses to gain- and lossframed persuasive health messages are associated with physical activity outcomes following message exposure. We focused on brain regions involved in the affect-value integration-motivation pathway—the anterior insula, ventral striatum, vmPFC, dorsal striatum, and pre-SMA. The results demonstrated that baseline engagement in physical activity moderated the relationship between neural responses to framed messages and postintervention behaviour. Specifically, individuals who were more physically active at baseline exhibited greater increases in physical activity when their neural responses to loss-framed messages were stronger than to gain-framed messages. In contrast,

less active participants showed the largest increases in physical activity when their neural activity was more responsive to gain-framed messages. These findings suggest that behaviour change may be influenced by individual differences in baseline behavioural engagement and in the neural processing of value and motivational salience related to framed health messages.

Previous research on physical activity promotion has primarily employed between-person designs to assess message framing effects and suggested that loss-framed messages may benefit less active individuals, whereas gain-framed messages may motivate more active individuals (de Bruijn et al. 2014, Hevel et al. 2019). Our within-person findings, however, revealed divergent patterns of framing effects: more active individuals increased their activity more when their neural responses to loss-framed messages were stronger than to gain-framed messages, and less active individuals exhibited the opposite pattern. One potential explanation for the discrepancy with prior literature is the measure-dependent effects observed in earlier between-person studies, where framing effects were evident for some outcomes (e.g. resolve to exercise) but not for others (e.g. exercise intention) (de Bruijn et al. 2014). Building on these foundational insights, our study utilized real-world behavioural data to capture objective variance in physical activity (as opposed to subjective measures) and combined this with neural data. This incorporation allowed us to disentangle both group-level differences in physical activity levels across participants and within-person differences in the neural processing of framed health messages. Our findings align with broader research on framing, which suggests that responses to framed messages are influenced by contextual and personal factors rather than being universally stronger for one frame over the other (Kahneman and Tversky 1984, O'Keefe and Jensen 2007).

We found that all ROIs exhibited interaction effects with baseline behaviour in the same direction, a result that is only partially consistent with our theoretical affective salience, value integration, and motivation (AIM) framework. The AIM regions have been hypothesized to be key neural predictors of value-based choice and behaviour (Samanez-Larkin and Knutson 2015). Our findings empirically extend this hypothesis by suggesting that their role in predicting behaviour change may vary depending on both contextual factors (e.g. message framing) and individual differences (e.g. baseline behavioural engagement). However, the AIM framework has proposed that the anterior insula and ventral striatum play opposing roles in value-based decision-making, with the anterior insula encoding negative valence (Lindquist et al. 2012, Palminteri et al. 2012) and the ventral striatum tracking positive valence (Bartra et al. 2013, Samanez-Larkin and Knutson 2015). Although we did not observe significant effects in the anterior insula, the consistent directional interactions across all ROIs—such as the trend of both anterior insula and ventral striatum exhibiting increased responses to gain-framed messages being associated with higher physical activity in less active individuals—suggests that differentiating affective valence may not be the primary factor driving message-consistent behavioural outcomes at the within-person level. Instead, these regions may function collectively as part of an excitatory corticostriatal circuit, encoding motivational salience that subsequently influences behaviour change after message exposure (Bartra et al. 2013, Balleine and O'Doherty 2010, Haber 2016, Hunnicutt et al. 2016, Rangel and Clithero 2014).

The ventral striatum was initially conceptualized within the AIM framework as playing a central role in processing gains and positive affect, serving as a precursor to value integration and motivation (Haber and Knutson 2010, Samanez-Larkin and Knutson 2015). Among less active individuals, greater ventral striatal activity in response to gain-framed compared to control messages was associated with the largest increases in physical activity following message exposure. This finding aligns with the ventral striatum's predicted role in encoding reward-related stimuli to guide behaviour (Bartra et al. 2013, Knutson and Genevsky 2018). Unexpectedly, this pattern reversed in more active individuals, where higher ventral striatal responses to loss-framed compared to gain-framed messages were associated with greater behaviour change. This is consistent with the possibility that loss-framed messages emphasizing the risks or negative consequences of inactivity may hold greater salience for individuals already engaged in physical activity, reinforcing their existing goals to maintain an active lifestyle. These results point to the ventral striatum's role that extends beyond encoding reward to encompass the processing of aversive cues and negative outcomes when relevant to behavioural goals. The ventral striatum can be sensitive to motivationally salient stimuli regardless of valence, responding to both positive and negative signals (Delgado et al. 2008, Levita et al. 2009).

The vmPFC is a critical hub for integrating value signals to guide adaptive decision-making and behaviour (Bartra et al. 2013, Rangel and Clithero 2014, Haber 2016). It has often been implicated in health message receptivity, with increased vmPFC activity during message exposure predicting real-world behaviour change (Cooper et al. 2015, Falk et al. 2010, 2011, 2012, 2015, Kang et al. 2018, Vezich et al. 2017). Our findings extend this evidence by demonstrating that pre-existing behavioural engagement moderates the relationship between vmPFC activity and message framing, offering new insights into brain-behaviour associations not captured by prior aggregate analyses. Specifically, for participants who were more active at baseline, greater vmPFC responses to loss- vs. gain-framed messages were associated with higher physical activity following message exposure. The vmPFC's role in integrating value signals from salient inputs (e.g. ventral striatal projections) may support this process by assigning greater value to avoiding negative outcomes (Tversky and Kahneman 1992, Falk et al. 2010, Bartra et al. 2013). In contrast, among less active individuals, stronger vmPFC responses to gain- vs. loss-framed messages were associated with the largest increases in physical activity. For these individuals, persuasive messages may become more effective when the vmPFC is more involved in integrating reward-related salience from ventral striatal activity.

More active individuals exhibited greater physical activity following the intervention when their dorsal striatum responded more strongly to loss-framed compared to gain-framed messages. The dorsal striatum facilitates goal-directed behaviour and habit formation by integrating motivational signals (Balleine et al. 2007, Liljeholm and O'Doherty 2012, Haber 2016). It receives valuerelated inputs from regions such as the vmPFC and communicates with motor planning areas, including the pre-SMA, to translate motivational states into action (Mogenson et al. 1980, Nachev et al. 2008, Liljeholm and O'Doherty 2012, Samanez-Larkin and Knutson 2015, Knutson and Genevsky 2018). Among individuals already engaged in physical activity, heightened dorsal striatal sensitivity to risk-related information may promote the translation of perceived negative outcomes into goal-directed behaviour that sustains or increases activity levels.

Among more active individuals, lower pre-SMA activity in response to gain-framed messages compared to control messages was associated with greater increases in physical activity. The pre-SMA integrates cognitive and motor processes, facilitating the selection, preparation, and execution of goal-directed actions (Nachev et al. 2008, Liljeholm and O'Doherty 2012). Control messages, which emphasize instructional "how-to" content, may be

more effective for this group than gain-framed messages that focus on health outcomes. However, the specific role of pre-SMA activity in distinguishing instructional from motivational framing remains unclear, particularly given the absence of significant effects in the gain vs. loss contrast. In contrast, the dorsal striatum demonstrated sensitivity to gain vs. loss framing in relation to behavioural outcomes, suggesting that these "motivation" regions may contribute differently to processing message content and influencing future behaviour. Further research is needed to clarify their distinct roles in supporting behaviour change.

Less active individuals exhibited opposite brain-behaviour relationships in the dorsal striatum and pre-SMA compared to their more active counterparts. Increased activity in these regions in response to gain-framed messages was significantly associated with positive intervention outcomes. This pattern aligns with previous framing research suggesting that emphasizing immediate benefits and positive outcomes can be particularly effective for promoting disease prevention behaviours such as physical activity (Gallagher and Updegraff, 2011, Rothman et al. 2006, Latimer et al. 2010). Our findings extend this line of work by showing that the benefits of positive framing can be especially pronounced among less active individuals, who may require stronger motivational cues to overcome lower baseline engagement. Gain-framed messages may therefore be particularly effective for this population when they successfully engage the neurocognitive circuits necessary to translate motivational cues into meaningful behaviour change.

Notably, among less active individuals, effects in the dorsal striatum and pre-SMA were specific to the gain vs. control contrast, with no significant findings for the gain vs. loss or loss vs. control comparisons. This pattern again suggests that emphasizing benefits may be more effective than providing instructional content for promoting behaviour change in this low- engagement population. However, further research (e.g. whole-brain functional connectivity analyses) is needed to clarify the distinct contributions of the dorsal striatum and pre-SMA, particularly whether they are differentially engaged in tracking motivational values from rewards- and risks-related information

Strengths and limitations

The present study provides new neurocognitive insight into message framing effects in physical activity interventions. Our megaanalysis demonstrated relationships between theory-driven ROI responses to health messages and physical activity behaviours across a diverse group of participants, ranging from young to older adults recruited from various communities. We incorporated a large volume of raw behavioural and fMRI data, linking fMRI responses to persuasive health messages with objectively measured real-world physical activity. This mega-analytic approach represents one of the major strengths of this investigation, as it allowed for a larger sample size and more robust handling of outliers and missing data without sacrificing statistical power. However, a limitation of this approach is the potential confound introduced by differences in scanning protocols, such as the use of multiband imaging in only one of the four studies (Study 2; Srirangarajan et al. 2021). Although we statistically controlled for study effects in our analyses, such variability may still influence the results and limit full comparability across datasets. As the field moves toward open access to larger neuroimaging datasets, future research may prioritize developing streamlined methods for processing mega-analytic data to improve harmonization and minimize potential confounds.

Our findings should be interpreted with several caveats. First, three of the four datasets included in our mega-analysis used a within-person design in which participants saw both gainand loss-framed messages in the scanner. Thus, it was beyond the scope of this study to determine which message frame was more effective than the other at causally eliciting subsequent behaviour change. Instead, we focused on within-person associations between neural activity and physical activity behaviours.

We analysed standardized average step counts by combining accelerometer data (Studies 1 and 2) and pedometer data (Studies 3 and 4), using one data point per participant for baseline and another for post-intervention physical activity. However, accelerometers are generally more sensitive and may overestimate step counts compared to pedometers, particularly in less active individuals (Tudor-Locke et al. 2002), which could partly explain the lower baseline activity observed in Studies 3 and 4. To address this limitation, we controlled for study-level differences in all models. In addition, we focused on total step counts as our main outcome and did not examine different levels (light, moderate, or vigorous) of walking behaviour. This decision was made because pedometers captured only daily step totals, whereas accelerometers could have provided additional information on activity intensity; total step counts were the common metric available across data sources. Future studies may test whether the current findings differ across device types and modes of physical activity.

Our selection of ROIs was theoretically guided by the AIM framework, which informed our understanding of the brain regions involved in processing framed health messages. However, we rely on reverse inference to interpret the psychological mechanisms at play, and the overall pattern of results being similar across ROIs is inconsistent with the AIM framework. Future research should incorporate interventions that specifically target these psychological mechanisms (e.g. affect, motivation) and include measures to directly assess them.

Finally, we primarily focused on baseline step counts as the key individual-level factor in our analyses. Although baseline physical activity is a critical marker of variability across participants, other important characteristics, such as body mass index (Young et al. 2016) and psychological constructs like self-efficacy (Williams and French 2011, Olander et al. 2013) could also influence responses to health messaging. Future research should aim to integrate a broader range of individual difference measures to provide a more comprehensive understanding of the factors that moderate the effectiveness of health messaging and brain-behaviour relationships. By doing so, we can better identify subgroups of individuals who may be more or less responsive to different types of framed messages, ultimately leading to more personalized and effective health interventions.

Conclusion

Our mega-analytic investigation provides novel insights into the neurocognitive mechanisms that underlie why certain health messages are more effective for different individuals. We show that message effectiveness depends not only on how it is framed, but also on how recipients engage with the promoted behaviour in daily life and process message content at the neural level. Among physically active individuals, stronger neural responses in regions involved in salience detection and the motivational valuation of inactivity risks were associated with greater adherence to exercise recommendations. In contrast, less active individuals were more likely to increase physical activity when they exhibited greater

neural activity in regions tracking the salience and motivational value of exercise benefits.

Together, these findings provide a neuroscience-informed framework for understanding how health messaging interacts with individual differences in neural processing and baseline behaviours to maximize intervention effectiveness. By linking real-world physical activity behaviours with neural responses to gain- and loss-framed messages, our study underscores the importance of considering individual variability in neurocognitive processes when designing interventions. Future research should investigate how a broader range of individual traits, message features, and neural activity patterns interact to better identify the conditions under which specific messages effectively drive behaviour change across diverse populations.

Supplementary data

Supplementary data is available at SCAN online.

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Data availability

The data underlying this article are available from the corresponding authors upon reasonable request.

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