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FlashReport

Personality traits function as causal concepts

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ABSTRACT

Traits can be either descriptive or causal, summarizing people and behavior, or explaining behavior. We hypothesized that isolated traits are primarily causal. Participants made rapid judgments of causal relations between 128 word pairs, including 32 trait-action pairs. Fenker, Waldmann, and Holyoak (2005) showed that causal relations are identified faster when words appear in a predictive sequence (cause → effect) than in a diagnostic sequence (effect → cause). They hypothesized that this occurs because causes always occur *before* effects. However traits are always inferred *after* behavioral observations. Nevertheless, if they are causally linked, participants may identify predictive sequences (clumsy → stumble) faster than diagnostic sequences (blush → shy). Participants did just that, and just as strongly as for non-social causes, suggesting that traits are primarily causal concepts.

Personality traits are used in several different ways (Newman & Uleman, 1993; Uleman, 2005). Traits can describe one behavior (Alice did a friendly thing) or behavioral regularities (Alice does friendly things). Traits can describe someone at one time (Alice is friendly) or over time (Alice is usually friendly). Traits can also describe causes for a person's behavior (Alice is friendly which causes her to smile). The distinction between the descriptive and explanatory functions of traits is important. Explanations enable inferences and predictions about future behavior in unrelated situations. Descriptions confer less inferential power. The context in which a trait term is used typically allows us to determine its intended functional meaning (Murphy & Andrews, 1993; Uleman, 2005). However in the absence of a social or epistemic context, as in spontaneously activated concepts (Uleman, Newman, & Moskowitz, 1996), do traits function primarily as descriptions or as causes?

Until recently, traits' explanatory function was assumed to operate only in the domain of explicit causal judgments, as described by classic attribution theories (Heider, 1944; Jones & Davis, 1965; Kelley, 1973). The realization that people make trait inferences spontaneously—without intention or awareness—did not change this assumption. Spontaneous trait inferences (STIs) were often conceptualized as descriptive rather than causal (Hamilton, 1988; Reeder 1985; Uleman et al. 1996). Although these inferences might inform subsequent causal attributions (e.g., Gilbert, 1998, p. 113), there was no evidence that STIs were causal inferences, per se.

Carlston, Skowronski and colleagues demonstrated that the likelihood of making spontaneous trait inferences (STIs) from behaviors depends on many of the same factors that affect intentional dispositional inferences, whereas spontaneous trait transference (STT) does not. (STT occurs when a communicator who describes an actor's behavior becomes associated with that behavior's trait implication.) For example, intentional trait attributions and STIs are more likely to form from negative than positive behaviors, because the former are more diagnostic, but STTs are unaffected by valence (Carlston & Skowronski, 2005). And doubts about whether the behavior describes the actor disrupt STI but not STT (Crawford, Skowronski, Stiff, & Scherer, 2007). Based on such evidence, Carlston and Skowronski (2005) characterized the processes producing STIs as attributional and those producing STTs as associative. One hallmark of attributional processes is "the degree to which the formed linkage is labeled to indicate that the trait is a property of, and not merely associated with, an individual" (p. 885).

However, this dichotomy between attributions and associations may be too simple, and even misleading. A central feature of intentional trait attributions is that they are causal. Indeed, "attribution" is often synonymous with "causal attribution" in social psychology. But does the causal meaning of traits arise from the "attributional processes" that produce them, spontaneously or otherwise, or is it inherent in the meaning of traits themselves? Notwithstanding the fact that traits are used several ways, might the basic meaning of traits include causing (explaining) behaviors, in the same way that acid causes corrosion or sunlight causes freckles? Might traits be theory-based concepts (Murphy & Medin, 1985), so that however they are activated, theories about their role in causing behaviors are also activated?

The current experiment examined the representational link between personality traits and behaviors, to test that hypothesis that the primary function of traits is causal explanation of behaviors. If

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traits are purely descriptive, summarizing behavior, traits and behaviors should be associatively linked in semantic memory. But if the primary function of traits is explanation, traits and behaviors should be causally linked.

We adopted a relation recognition paradigm that Fenker, Waldmann, and Holyoak (2005) used to demonstrate causal links in semantic memory. They hypothesized asymmetric reaction times for detecting causal relationships, depending on the temporal sequence in which 'cause' and 'effect' are presented to participants (see Supplementary material). They theorized that not all cognitive relationships (links) are the same (e.g., sparrow "is a" bird, spark "causes" fire, cold "is the opposite of" hot) and that recognizing relationships requires mapping concepts to roles ('cause' and 'effect'). Thus detecting causal relationships would be faster when cause-effect concepts appear in the predictive temporal order that matches their canonical sequence in experience. Recognizing causal relationships between the same concepts presented in a diagnostic sequence requires "re-mapping" them to their cause-effect order, and hence would be slower. This is what they found. People identified causally related concepts faster in predictive sequences (cause → effect) than diagnostic sequences (effect → cause).

Note that this RT asymmetry is characteristic of causal relations, and is not due to differences in "associative link strength" in each direction. Concept pairs had equal associative strength in the predictive and diagnostic directions. But not all links (relations) are associative.

Participants completed two different relation recognition tasks: the nonsocial task of Fenker and colleagues, and a modified social version that used trait-behavior word pairs. If traits and behaviors are causally linked, participants should recognize causal relationships more quickly when words are presented in a predictive sequence (trait → behavior). But if traits and behaviors are merely associatively linked, reaction times should be unaffected by temporal order. Finally, if traits and behaviors are usually experienced in a diagnostic sequence, as the impossibility of observing traits and the ubiquity of STI suggest, reaction times for behavior-trait pairs should be faster.

Method

Participants

Participants were 107 undergraduates at NYU who completed the study for course credit.

Stimuli

For the social task, 32 trait-behaviors word pairs (e.g., dumb-fail, silly-giggle) were selected from the University of Southern Florida word association norms (Nelson, McEvoy, & Schreiber, 1998).[1] Following Fenker et al. (2005), all pairs were of low associative strength (likelihood of free association in both predictive and diagnostic directions).[2] Thirty-two filler adjective–verb word pairs (e.g., gentle-touch, bitter-taste, slow-move) were also selected from the USF list. These were also of low associative strength in both directions.[3] See the Supplementary material.

The 32 trait-behavior pairs were also pretested to ensure equal *statistical contingency* (in addition to association likelihood) in both directions, so this could not produce an RT asymmetry. Pretest participants ($N = 60$) estimated conditional frequencies on 100-point scales. In the diagnostic direction, they responded to, "If 100 people [*action*], how many are [*trait*]?" In the predictive direction, they responded to, "If 100 people are [*trait*], how many [action]?" Pairs were the same in diagnostic ($M = 62.05$, $SD = 14.7$) and predictive ($M = 62.78$, $SD = 14.3$) directions, $t(29)$[4] $= .212$, $p = .83$.

Explicit causal ratings were also obtained to ensure that trait-behavior pairs were causally related. The same pilot participants rated each trait-action pair on "How likely is it that being [*trait*] causes someone to [*action*]?" from 0 (extremely unlikely) to 100 (extremely likely). The mean rating was greater than 50, $t(31) = 10.2$, $p < .01$.

For the nonsocial task, we selected 32 causally related word pairs (e.g., spark-fire, moon-tide, acid-corrosion) and 32 associated word pairs (e.g., shrimp-ocean, soup-cracker, elevator-floor) from the original task (Fenker et al., 2005). These pairs were also of equal associative strength in the predictive and diagnostic directions, in terms of free associations and statistical contingencies. See Supplementary material.

Procedure

Participants completed the nonsocial and then the social relation recognition tasks on computers with DirectRT software. They made speeded causal judgments for each of 64 word pairs. Following Fenker et al. (2005), we asked participants to "determine whether the concepts described by each word pair are causally related". If a causal relationship was detected ("either because the concept described by the first word *causes* or is *caused by* the concept described by the second word"), participants pressed the C-key. If they were not, participants pressed the N-key.

Nonsocial stimuli consisted of 32 cause-effect word pairs and 32 associated word pairs. Social stimuli consisted of 32 trait-behavior word pairs (causal) and 32 associated adjective–verb pairs. In both tasks, half the pairs were presented in each order. The correct response to causal nonsocial word pairs and trait-behavior pairs is 'yes' regardless of order of presentation. The correct response to associated word pairs is 'no'. Assignment of pairs to each order was counterbalanced across two versions of the experiment. Across both tasks, word pairs were blocked in groups of four so that four predictive, four diagnostic, and eight associative pairs were randomly presented within each block. Thus there were equal numbers of trials for which the correct answer was 'yes' and 'no'. Participants responded to all 64 word pairs in sequence without a break. There was a 1-min break between the nonsocial and social tasks.

Each trial began with a fixation cross (500 ms), followed by the first word of a pair (1000 ms), a second fixation cross (500 ms), and the second word of a pair (Fig. 1). Each screen replaced the one before it so there was never more than one stimulus on screen. All stimuli were centered. Response times were recorded from the time of the final stimulus onset until a response was made. Eight

[1] These norms measure pre-existing associations between words serving as cues and targets in cued recall memory tests. Strengths of association were the proportion of participants who generated the target word when free associating from the cue word (Forward Strength of Association), and the proportion who generated the cue word when free associating from the target word (Backward Strength of Association), producing two indices of association—FSA and BSA.

[2] Likelihood of free association in the predictive direction was the proportion of participants who generated the verb when presented with the trait. Likelihood in the diagnostic direction was the proportion who generated the trait term when presented with the verb. Averaged across word pairs, the mean likelihood was greater in the diagnostic direction ($M = .03$, $SD = .037$) than the predictive direction ($M = .01$, $SD = .018$), $t(31) = 4.56$, $p < .01$. This difference could only reduce reaction times for judging diagnostic pairs, which would mask our hypothesized RT advantage for predictive pairs.

[3] Averaged across word pairs, the mean likelihood was greater in the verb → adjective direction ($M = .035$, $SD = .038$) than the adjective → verb direction ($M = .009$, $SD = .025$), $t(31) = 3.05$, $p < .01$. This difference is irrelevant because these are filler trials.

[4] Statistical contingency ratings were not obtained for two of the 32 word pairs due to a photocopying error.

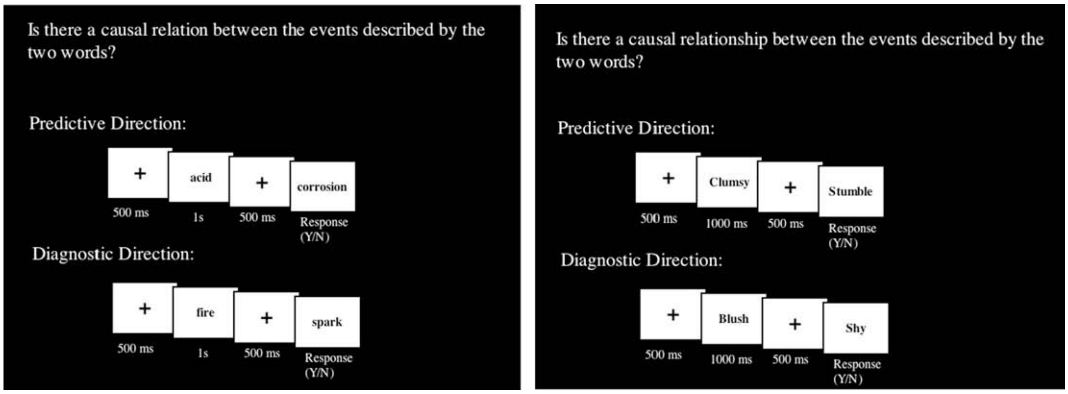

Fig. 1. Sample trials from nonsocial and social relation recognition tasks.

practice trials (with feedback) preceded the social and nonsocial tasks (without feedback).

Results

Nonsocial relation recognition task

Following Fenker et al. (2005), we analyzed only causal trials that were answered correctly and excluded outliers more than two *SD*s from the participant's mean RT for each trial type (predictive or diagnostic). Additionally, we dropped the 7% of participants with error rates above 50% on either the nonsocial causal trials or associated trials. Seven participants failed to detect causal relationships on over half of the 32 causal trials, and 1 responded "causal" to 28 of the 32 associated trials. (Fenker et al. dropped the 11% of *P*s, over three studies, with error rates above 20%.) The remaining 99 participants were correct on 89.3% of the trials, and 4.6% of their responses were excluded as outliers.

Participants detected causal relationships 77.04 ms faster for causal pairs in predictive ($M = 1223.42$, $SD = 391.92$) than diagnostic sequences ($M = 1300.46$, $SD = 451.37$), $t(98) = 3.11$, $p < .01$. Importantly, this facilitation was not due to a speed–accuracy trade-off. Participants made more errors across diagnostic trials ($M = 2.00$, $SD = 1.63$) than predictive trials ($M = 1.44$, $SD = 1.35$), $t(98) = 3.2$, $p < .01$. Results replicated Fenker et al. (2005).

Social relation recognition task

As before, we analyzed only correct trials, and excluded outliers over two *SD*s from participants' means. The same 99 participants (as above) were correct on 86.2% of the social trials, and 4.6% of these were excluded as outliers.

Participants detected causal relationships 72.22 ms faster when trait-behavior pairs appeared in predictive ($M = 1242.46$, $SD = 466.40$) than diagnostic sequences ($M = 1314.68$, $SD = 520.62$), $t(98) = 2.14$, $p < .05$. Once again, there was no speed–accuracy trade-off. Participants made slightly more errors across diagnostic trials ($M = 2.33$, $SD = 2.15$) than predictive trials ($M = 2.11$, $SD = 2.07$), $t(98) = 1.09$, $p > .85$.

An ANOVA that included both the nonsocial and social tasks showed no significant difference between them, $F(1, 98) = .017$. The RT asymmetry was as strong on the social task as the nonsocial task (see Fig. 2).

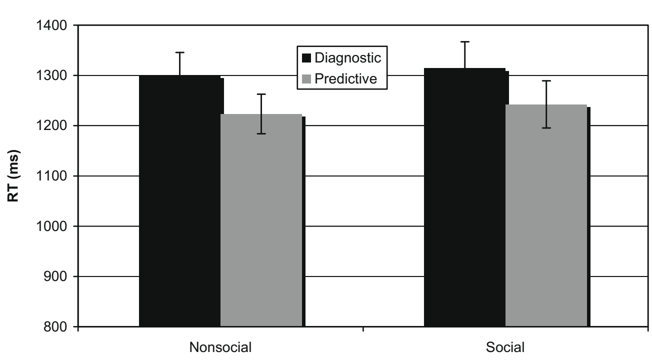

Fig. 2. Response times to causal word pairs in nonsocial and social relation recognition tasks as a function of temporal sequence of presentation (diagnostic or predictive).

Discussion

This research used a relation recognition paradigm, novel to social psychology, to examine whether personality traits and behaviors are causally (not just associatively) linked in semantic memory. Fenker et al. (2005) demonstrated that causally linked concepts produce asymmetric reaction times for detecting causal relationships, with predictive word orders faster than diagnostic orders. Our results replicated Fenker et al., and also showed the same degree of asymmetry with trait-behavior pairs. These were identified as causally related more quickly when in predictive (trait → behavior) than in diagnostic sequences (behavior → trait). This was not due to an asymmetry in associative strength or statistical contingency. This suggests that traits and behaviors are mentally represented as causally linked, and that isolated traits are inherently causes of behaviors.

One might object that traits functioned as causes in this task only because the task instructions primed causality. We cannot rule out this possibility (which presumes that traits have causal meanings to be primed in the first place). But we think it unlikely because the RT asymmetry was just as strong for the social as the nonsocial word pairs, where causal relations are indisputable. Furthermore, the associated noncausal pairs showed no asymmetry.

These results are surprising in light of how expert people are at making diagnostic STIs (Uleman et al., 1996). In addition, Maass and her colleagues demonstrated that people are more likely to infer traits from behaviors than behaviors from traits—even when the number of behaviors implied by traits equals the number of traits implied by behaviors (Maass, Cadinu, Taroni, & Masserini, 2006; Maass, Colombo, Colombo, & Sherman, 2001). People are *so* expert at making diagnostic inferences from behaviors, both unintentionally and intentionally, that one would expect an RT advantage for the diagnostic order, behavior → trait. But precisely the opposite occurred.

Our results also suggest this asymmetry does not depend on *observing* cause and effect in a particular order, because traits cannot be observed.

Apparently traits and behaviors are causally related in semantic memory, regardless of how these concepts are activated. So it may be misleading to label the processes that produce STI and STT as attributional and associative, respectively, if only attributional processes are thought to have causal implications. In the case of traits and behaviors, at least, causing behaviors may be inherent in the meaning of traits. Using theory-based concepts may entail activating those theories, even if they remain implicit (e.g., Uleman, Saribay, & Gonzalez, 2008).

Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.jesp.2009.08.018.

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