Delay and Death-thought Accessibility: A Meta-analysis

Christopher T. Steinman & John A. Updegraff

Kent State University

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Abstract

The dual-process component of Terror Management Theory (TMT) proposes that different types of threats lead to increases in death-thought accessibility (DTA) after different delay intervals. Experimental studies of terror management threats' effect on DTA were collected and coded for their use of explicitly death-related (vs. not explicitly death-related) threats, and for their use of delay and task-switching during the delay. Results reveal that studies using death-related threats achieved larger DTA effect-sizes when they included more task-switching or a longer delay between the threat and the DTA measurement. In contrast, studies using threats that were not explicitly death-related achieved smaller DTA effect-sizes when they included more task-switching between the threat and the DTA measurement. These findings provide partial support for the dual-process component's predictions regarding delay and DTA. Limitations and future directions are discussed.

Keywords: terror management, death-thought accessibility, delay, dual process, meta-analysis

Delay and Death-thought Accessibility: A Meta-analysis

Under what conditions do individuals most readily think of death? The question of *death-thought accessibility* (DTA), or when individuals most readily think of death, has been a growing subject of research, most notably within *Terror Management Theory* (TMT; Greenberg, Pyszczynski, & Solomon, 1986; Solomon, Greenberg, & Pyszczynski, 1991a). TMT proposes that humans are faced with a unique psychological conflict: we have an instinctual desire to survive, yet also the self-awareness to recognize that we will inevitably cease to exist. This conflict is thought to produce a profound sense of terror. Individuals are compelled to manage this terror by embracing *cultural worldviews*, social value systems that promote perceptions of life as being coherent and meaningful, and that provide individuals with a sense of symbolic immortality (Dechesne, et al. 2003). These cultural values thus function to thwart concerns regarding mortality. Consequently, TMT implies a social and cognitive link between cultural values and concerns regarding mortality or death.

In support of this perspective, early TMT studies showed that invoking explicit thoughts of death leads individuals to more strongly defend their cultural values (Rosenblatt, Greenberg, Solomon, Pyszczynski, & Lyon, 1989; Greenberg et al. 1990). In these studies, participants were assigned to either a *Mortality Salience* (MS) condition, where they were asked to reflect on their own death, or a control condition. Following exposure to MS, participants were more willing to punish and derogate "worldview violators," those who expressed beliefs or behaviors that were inconsistent with the participants' cultural values. Inversely, MS also increased participants' willingness to reward and positively evaluate others who confirmed or upheld participants' cultural values. These first studies were critical in establishing a link between explicit thoughts

of death and desire to uphold cultural values, a finding which has been confirmed by later studies (e.g. Greenberg, Simon, Pyszczynski, Solomon, & Chatel, 1992; McGregor, et al. 1998).

However, some early studies also presented conflicting results (Greenberg, Pyszczysnki, Solomon, Simon, & Breus, 1994). Individuals seemed less willing to defend cultural values while they were still experiencing strongly explicit thoughts of death. Rather, it seemed individuals defended cultural values after explicit thoughts of death had subsided or weakened, becoming implicit: that is, cognitions related to death that are accessible, but reside outside of full conscious awareness. The implications of this are two-fold. First, stronger explicit reminders of death should require more time to pass or more distraction before affecting cultural value defense. Second, cultural value defense should only be expected to occur when thoughts of death have become implicit. Exploring this second idea required a means to quantify implicit thoughts of death. Accordingly, Greenberg et al. (1994) developed a measure of *death-thought accessibility* (DTA), which is now widely used.

The classic measure of DTA takes the form of a word fragment completion task (Greenberg, et al. 1994). Participants are asked to complete a series of word fragments (e.g. C O F F _ _) with the first word that comes to mind. Unbeknownst to the participants, several of these fragments can be completed with either a death-related or neutral word (e.g. *coffin* or *coffee*, respectively). Thus, completing a greater number of fragments with death-related words indicates heightened DTA, or activation of implicit thoughts of death. Alternative methods for measuring DTA have also been used, including the measurement of reaction times to death-related stimuli (e.g. Firtsche, Jonas, & Fankhanel, 2008; Vail, Arndt, Motyl, & Pyzczynski, 2012), and the number of references to death in free writing tasks (e.g. Gailliot, Schmeichel, & Maner, 2007; Trafimow & Hughes, 2012).

Greenberg and colleagues' (1994) first study of DTA found that immediately following explicit thoughts of death, DTA was suppressed; however, following explicit thoughts of death and a delay, DTA increased. This effect has been replicated (e.g. Harmon-Jones, et al. 1997), though some researchers have recently found the opposite effect of delay (Trafimow & Hughes, 2012). Additionally, Greenberg and colleagues' (1994) findings suggested that individuals tend to defend cultural values only when DTA is high, that is, when thoughts of death are implicitly rather than explicitly active. This provided the first direct evidence that implicit thoughts of death play a role in predicting defense of cultural values, and laid the foundation for future use of DTA in investigations of TMT processes.

This finding, combined with other evidence regarding the effects of cognitive load and subliminal death primes on DTA (Arndt, Greenberg, Solomon, Pyszczynski, & Simon, 1997; Arndt, Greenberg, Pyszczynski, & Solomon, 1997) ultimately led to the articulation of the *dual-process component* of TMT (Pyszczynski, Solomon, & Greenberg, 1999; Greenberg, Arndt, Simon, Pyszczynski, & Solomon, 2000). The dual-process component of TMT theorizes that there are distinct psychological defenses in response to implicit and explicit processing of death-related information, and also indicates when implicit thoughts of death (heightened DTA) should be expected to occur. Specifically, the dual-process component argues that explicit processing of death-related information, such as that produced by mortality salience (MS) manipulations, first leads to a set of *proximal defenses* (Pyszczynski, Greenberg & Solomon, 1999). These proximal defenses include pseudo-rational distortions of one's vulnerability to death (e.g. underestimating one's risk of death), and also active suppression of death-related thoughts, including suppression of DTA. However, once an individual has become distracted from explicit thoughts of death, such as after completing neutral delay tasks, DTA resurges and proximal defenses subside. This

resurgence of DTA following suppression is thought to be the result of cognitive mechanisms similar to those that drive the classic paradoxical effect of thought-suppression (i.e. attempting 'not to think of a white bear' actually makes it more likely that one will think of a white bear; Wegner, Schneider, Carter, & White, 1987). Along with resurgence in DTA, individuals begin to engage in *distal defenses*, which include defending and affirming cultural values, and attempts to enhance one's self-esteem (Pyszczynski, Solomon, & Greenberg, 1999).

Additionally, the dual-process component describes what is expected to happen in response to threats that *do not* invoke explicit processing of death-related information, such as subliminal priming of death-related constructs (Pyszczynski, Solomon, & Greenberg, 1999). In this case, proximal defenses are 'skipped.' Instead, individuals begin to engage in distal defenses, such as affirmation of values, immediately. Importantly, because proximal defenses are skipped, DTA is also not suppressed and therefore becomes heightened immediately following these threats. Thus the dual-process component predicts that threats involving explicit thoughts of death lead only to delayed increases in DTA, while threats that do not involve explicit thoughts of death lead to immediate increases in DTA. Some additional evidence implies that, in the case of threats that are not explicitly death-related, the immediate increase in DTA might taper off following a delay (see Figure 1 for a hypothetical model of DTA's associations with delay; Schimel, Hayes, Williams, & Jahrig, 2007; Martens, Burke, Schimel, & Faucher, 2011). Thus the dual-process component carries important implications for terror management studies' procedural designs, as inclusion of a delay may either enhance or diminish DTA or particular defenses (i.e. proximal or distal) in response to terror management threats.

Providing insight into the dual-process component of TMT is not the only major contribution of DTA to TMT, however. While early studies revealed that thoughts of death

predict concerns related to cultural values, DTA enabled researchers to test the opposite hypothesis: that concerns regarding cultural values affect thoughts of death. If cultural values buffer thoughts of death, as TMT suggests, then challenging cultural values may instigate thoughts of death or increases in DTA. Indeed, several studies have supported this. Among others, threats to relationship solidarity (e.g. Florian, Mikulincer, & Hirschberger, 2002; Mikulincer, Florian, Birnbaum, & Malishkevich, 2002; Taubman-Ben-Ari & Katz-Ben-Ami, 2008), self-esteem (e.g. Hayes, Schimel, Faucher, & Williams, 2008), and cultural identity or worldview (e.g. Schimel, Hayes, Williams, & Jahrig, 2007) have all been shown to increase DTA. Critically, many of these studies suggest that cultural value threats have an immediate effect on DTA. This is consistent with the dual-process component's predictions; since these threats do not produce explicit thoughts of death, they should be expected to result in immediate increases in DTA.

Testing the Dual-process Components Predictions Regarding Delay and DTA

Three prior reviews have addressed the dual-process component of TMT, but none have quantitatively synthesized the role of delay on DTA. The closest review to address this question was a qualitative review of the literature from 1997 to 2009, in which Hayes and colleagues concluded that DTA is suppressed immediately following MS, but resurges following a delay or distraction (Hayes, Schimel, Arndt & Faucher, 2010). Martens, Burke, Schimel & Faucher (2011) meta-analytically showed that relative to meaning threats, MS manipulations are associated with larger effect-sizes following a delay, but this review only compared MS manipulations to meaning threats so that delay's unique association with each particular type of threat could not be isolated. Burke, Martens, & Faucher's (2010) meta-analysis primarily examined the role of death-related threats on worldview- and esteem-related outcomes rather

than DTA, and found stronger effects of mortality salience on worldview and esteem outcomes after a delay compared with after no delay. Thus, these prior reviews provide indirect support for the notion that DTA is suppressed immediately following MS but resurges afterwards.

Direct support for the role of delay on DTA, however, is mixed. Although some studies experimentally test the dual-process component's predictions regarding delay and DTA, these studies are few and most of them were conducted early in the history of the development of TMT (e.g. Harmon-Jones et al. 1997; Arndt, Greenberg, Solomon, Pyszczynski, & Simon, 1997; Schimel, Hayes, Williams, & Jahrig, 2007). More recently, a series of 6 studies failed to find evidence of delayed increases in DTA following mortality salience (Trafimow & Hughes, 2012), thus casting some doubt on this element of the dual-process component of TMT.

Present Study

The literature on DTA is now sufficiently large to meta-analytically evaluate the influence of delay on DTA. Across this literature, there exists significant variability in studies' use of delay when measuring DTA. This presents a unique opportunity to meta-analyze whether studies' use of delay is associated with differences in DTA, and whether this influence of delay differs according to type of threat. Thus, the primary aim of the present study is to test the dual-process component's predictions regarding delay and DTA. Specifically, we tested the following hypotheses:

- There will be no effect of death-reminders or mortality salience on DTA in studies that do not use a delay, but DTA effect-sizes will be larger in studies that use longer delays.
- There will be a moderate or strong effect of threats that are not explicitly death-related on DTA in studies that do not include a delay, but the effect will be diminished in studies that use longer delays.

Additionally, we examined whether MS—reflection on one's personal mortality—results in distinct effects as compared to less direct reminders of death (e.g. thinking about war; i.e. death-reminders). MS may result in unique effects since it is a stronger, more personal reminder of death (Greenberg et al. 1994; Hayes, Schimel, Arndt, & Faucher, 2010).

Method

In order to analyze these hypotheses, we computed a mean difference indicator of effect-size, Hedges' *g*, from experimental studies of threats' effect on DTA. Hedges' *g* is similar to Cohen's *d* in that it reflects a standardized difference in means, though it also corrects for inflated effect-sizes among studies with small samples. Thus, a value of $g = 1$ reflects a mean difference that is approximate to the value of one standard deviation. Hedges' *g* can also be interpreted following Cohen's (1988) suggestion for Cohen's *d*, that .2 indicates a small effect-size, .5 indicates a medium effect-size, and .8 indicates a large effect-size. All effect-sizes were computed using the standard formulas contained in Comprehensive Meta-analysis (CMA) software (Borenstein, Hedges, Higgins, & Rothstein, 2005), and all analyses were conducted using meta-analysis packages in STATA 12.

Study Search and Criteria for Inclusion

Studies were identified from Google Scholar and PsycINFO using combinations of the following search terms: *death-thought*, *accessibility, death-related, thoughts, death-construct,* and *constructs*. Studies were also identified using citations from a previous review of DTA (Hayes, Schimel, Arndt, & Faucher, 2010). This search returned a total of 556 unique works. From these, 121 empirical papers that measured DTA were identified. Several papers, studies, or effects were excluded from this analysis because their methods conflicted with the present study's primary aim and analytic strategy, or because there was insufficient data for estimating

an effect-size. This resulted in a total of 78 papers and 99 effect-sizes included in the present analysis. Explanations for other exclusions are as follows.

In order to best capture the causal effects of threat manipulations on DTA, and in order to regress effects on delay, we used only between-subjects experimental studies in our analysis. Thus, some studies were excluded for being non-experimental (e.g. Arndt, Cook, Goldenberg, & Cox, 2007, study 4; Dunkel, 2009, study 3; Ves, Arndt, & Cox, 2012, study 1; Scharf & Cohen, 2013, study 1). In a similar vein, studies were excluded for their use of the DTA measure as an independent variable (Zhou, Liu, Chen, & Yu, 2008, study 1; Cooper, Goldenberg, & Arndt, 2011, study 1), or for use of within-subjects manipulations of delay following threats (Simon, et al. 1997, study 4; Mikulincer & Florian, 2000, study 1; Ulrich & Cohrs, 2008, study 3; Trafimow & Hughes, 2012, studies 1, 2, and 6). Within-subjects manipulations were excluded because it is possible that completing one DTA measure could affect subsequent DTA measures, whether in terms of activation or suppression of DTA, and therefore we reasoned that between-subjects manipulations would more clearly capture the effects of a single experimental manipulation of threat. Studies were also excluded if they did not manipulate threat in a clear fashion or for not including a theoretically neutral control condition regarding the question at hand (Arndt, Greenberg, Solomon, Pyszczynski, & Simon, 1997, study 3; Arndt, Greenberg, Simon, Pyszczynski, & Solomon, 1998, study 2; Silvia, 2001, study 1; Mikulincer & Florian, 2002, study 3; Martens, Greenberg, Schimel, & Landau, 2004, study 1; Schmeichel & Martens, 2005; King, Hicks, & Abdelkhalik, 2009, studies 1, 2, and 3; Schmeichel, et al. 2009, study 3; Chatard & Selimbegovic, 2011, studies 1 and 2; O'Connor & McFadden, 2012, study 1).

Studies were excluded if they allowed for the possibility of defense of values between the threat exposure and the measurement of DTA (Harmon-Jones, et al. 1997, study 3; Goldenberg,

Pyszczynski, McCoy, Greenberg, & Solomon, 1999, study 3; Mikulincer & Florian, 2000, study 1; Goldenberg, Cox, Pyszczynski, Greenberg, & Soloman, 2002, study 1; Mikulincer & Florian, 2002, study 3; Schimel, Hayes, Williams, & Jahrig, 2007, study 2; Hayes, Schimel & Williams, 2008, study 1; Hayes, Schimel, Faucher, & Williams, 2008, study 3; Cox, et al., 2008, study 1; Norenzayan, Dar-Nimrod, Hansen, & Proulx, 2009, study 3; Vaes, Heflick, & Goldenberg, 2010, study 3; Williams, Schimel, Hayes, & Martens, 2010, study 3; Wojtkowiak & Rutjens, 2011, study 1; Davis, Juhl, & Routledge, 2011, study 1; Edmondson, et al. 2011, study 2; Cohen, Sullivan, Solomon, Greenberg, & Ogilvie, 2011, study 5; Cohen, Soenke, Solomon, & Greenberg, 2013, study 4). This exclusion criterion was used because defense of values and self-affirmation have been shown to prevent increases in DTA (Greenberg, et al. 1990; Davis, Juhl, & Routledge, 2011), and so reflect theoretically-predicted reduced DTA effect-sizes. Indeed, supporting this rationale, random-effects meta-regression revealed that studies permitting defense between threat and DTA measurement ($k = 17$) achieved a significantly smaller DTA effect-size ($g = .27$, 95% C.I. = 0.05, 0.5; $p < 0.001$) than those in the primary sample ($t(119) = -2.41$, $p = .02$).

Because a major aim of this study was to investigate the impact of delay on DTA, studies were excluded for use of cognitive load instead of delay for two reasons (Arndt, Cook, Goldenberg, & Cox, 2007, study 2). First, it is difficult to map cognitive load onto the same dimension as delay. Second, there is evidence that ego-depletion can have a causal effect on TMT processes (Gailliot, Schmeichel, & Baumeister, 2006), which may suggest that cognitive load directly affects rather than moderates DTA. Thus, because we have reason to believe that cognitive load may function differently than delay, and focused only on delay in the present study.

Studies were excluded if they used explicit measures of DTA, such as the Death-anxiety Scale (Gailliot, Schmeichel, & Baumeister, 2006, study 3; Trafimow & Hughes, 2012, study 5). These were excluded because TMT makes a critical distinction between explicit and implicit thoughts of death, and the former is opposed to theoretical perspectives on DTA (Pyszczynski, Greenberg, & Solomon, 1999). Studies were also excluded if they involved interactions that could not be broken down into a pure main effect (i.e. the manipulation was unavoidably either a double-threat or a threat+affirmation; e.g., Goldenberg, Cox, Pyszczynski, Greenberg, & Solomon, 2002, study 1; Koole & Van den Berg, 2005, study 4; Hirschberger, 2006, study 4; Cox, et al. 2008, study 1; Martin & Kamins, 2010, study 1 pretest; Agroskin & Jonas, 2013, study 3), as well as for use of unusually long-term delay (Cox, Reid-Arndt, Arndt, & Moser, 2012, study 1).

Finally, in the case of studies that met criteria but did not include sufficient information for calculating effect-sizes, authors were emailed. In some cases, the needed data was obtained. If sufficient data was not obtained, the study was excluded. The only exception to this rule was for studies that did not provide condition-level *n*'s, but reported a study-level *n* and also the total number of conditions in the study. In this case, condition-level *n*'s were estimated by dividing the study-level *n* by the number of conditions.

Coding of Studies

Studies were coded on a variety of factors. First, independent variables were coded as being either MS, death-reminders, or not explicitly related to death (e.g. subliminal primes, cultural value threats). Studies using a manipulation that focused on personal mortality, such as the classic MS manipulation, were coded in the MS category (e.g. Florian, Mikulincer, & Hirschberger, 2001; Arndt, Cook, Goldenberg, & Cox, 2007). Studies that made a simple

reference to death or death-related violence were coded as death-reminders. Importantly, some of these may have been intended to threaten cultural values, but did so by means of exposure to some death-related concept (e.g. terrorism in one's own country; Das, Bushman, Bezemer, Kerkhof, & Vermeulen, 2009). Finally, all other threats were classified as not explicitly death-related (e.g. Schimel, Hayes, Williams, & Jahrig, 2007).

If studies included more than one of these types of threats, an effect-size was calculated for each threat type.[1] If studies included multiple of the same type of threat, the threat condition that was most consistent with theory and/or other research was selected for use (e.g. standard MS instead of suicide-related MS; Fritsche, Jonas, & Fankhanel, 2008). The exception to this was when a study used the same threat type twice, but with different delays and control conditions with respective delays. Our reason for selecting between similar threats rather than pooling them was to avoid ambiguity and to maintain theoretical consistency.

Similarly, when studies utilized multiple control conditions, the most conservative control condition was selected for use in two steps. First, when possible, we avoided control conditions that were likely to affirm personal values, which could reduce DTA in the control condition and thus inflate the DTA effect-size. For example, in a sample of Americans, we assumed that reading about a local synagogue was less affirming than reading about a church, given that Americans are predominantly Christian (Cohen, Soenke, Solomon, & Greenberg, 2013), and that pictures of intact buildings were less affirming than pictures of buildings being constructed (as construction denotes human progress; Vail, Arndt, Motyl, & Pyszczynski, 2012). There were few studies that required this decision, and they generally tested the effect of value threats on DTA. Second, when possible, we selected the control condition that was most likely to be related to pain (e.g. dental pain salience rather than television salience) or negative emotion

(e.g. exam anxiety over watching television; anger, Chatard & Selimbigovic, 2011), except in the case of body-related disgust (e.g. dust mites cause less body-related disgust than bed bugs; Burris & Rempel, 2004). This is because general negative emotion has been argued to be an important control, although evidence suggests it does not affect TMT processes (Greenberg et al. 1995), except in the case of fear of death (e.g. Mikulincer & Florian, 2000), and sometimes disgust (see Goldenberg et al. 2001; but cf. Fessler & Navarrete, 2005).

Second, studies were also coded on the type of DTA operationalization they had used (word fragment, reaction time, or free writing). Word fragment studies were additionally coded for the number of death-related word fragments that they included, and the total number of neutral word fragments they included.

Finally, studies were also coded on their use of delay. Previous TMT meta-analyses have used two different methods for coding delay, each involving unique limitations. First, the number of tasks that participants completed during the delay has been operationalized as delay length (Burke, Martens, & Faucher, 2010). The potential problem with this method is that it is impossible to be certain that all delay tasks are equivalent in length. As a result, it is perhaps more accurate to describe this coding method as *task-switching* than delay length. Alternatively, previous studies have used dichotomous coding of whether the study included some delay or no delay (Martens, Burke, Schimel, & Facher, 2011). This does address the issue of potentially non-ordinal data. However, dichotomizing a continuous variable naturally results in a loss of statistical power, which might mask effects. Because of the unique limitations of each of these methods, both were used in the present study.

Additionally, the present study introduces a third method for coding delay: estimated delay length (seconds). To estimate delay length, studies were coded on the types of delay tasks

they used, and authors' estimations of total delay length. If any of the delay tasks were Likert-style questionnaires, the studies were also coded on the number of questionnaire items included during the delay. We then estimated length data for each delay task type using data from an independent sample (n = 43), in which we timed participants on typical TMT delay tasks (i.e. the PANAS and reading an excerpt from "The Growing Stone" by Albert Camus), as well as author's reports of delay length (see Table 1 for descriptive statistics regarding delay task length estimates). We used this information to impute total delay length estimates for each study. Specifically, authors' reports were used to impute delay estimates for use of puzzles as delay (3.5 min; Maxfield et al., 2007; Carey & Sarma, 2011). Data from the independent sample was used to impute values for the average questionnaire item length (to be multiplied by the number of questionnaire items that were used during the study's delay), and also for the use of written passages as a delay task. Any other type of delay task (k = 5 of total k = 99) was given the average value of all "other" delay task types (217.34 seconds). For studies that used multiple delay tasks, the length estimates for each delay task were summed.

One limitation to operationalizing delay length in this manner is that imputing mean delay length by task type cannot account for variability within each delay task type (as indicated by the standard deviations in Table 1; Cooper, Hedges, & Valentine, 2009). However, because of studies' varied use of delay task types and combinations of different delay tasks, this method still results in noticeable variability in delay length estimates across studies (m = 127.5 seconds, SD = 157.19). Thus, this technique should significantly increase both the likelihood that our delay variable is ordinal, and increases statistical power to detect associations with delay. Because each of the three methods of operationalizing delay is associated with its own

limitations, using all three methods (delay vs. no delay, task-switching, and estimated delay length) should provide the clearest picture of how delay relates to effect-sizes across studies.

A summary of studies and their codes can be found in Table 2.

Publication Bias and Small-study Effects

The present analysis only included published studies, partly as a means of quality control (e.g. peer review), and partly because our coding of delay required specific procedural information that is relatively incidental, and so unlikely to be included with unpublished data. Publication bias and small-study effects were explored using funnel plots (Sterne & Harbord, 2004) and p-curve analysis (Simonsohn, Nelson, & Simmons, 2014). These analyses were conducted on the full sample of effect sizes, but also broken down by threat type, and by delay within each threat type. The funnel plots revealed some small-study effects. However, the p-curve analyses suggest that these small-study effects were not driven by a preferential publishing of findings near the cutoff for statistical significance. Moreover, 43% of the effects in the present study were published even though they were not statistically significant, which can perhaps be explained by the fact that DTA was often of secondary rather than primary interest in the publications. Importantly, small-study effects did not appear to be meaningfully associated with studies' use of delay.[2]

Results

Overall Effect-size and Comparison of Threats

Because the studies to be analyzed involve diversity in sample characteristics and method, we used a random-effects model for effect-size estimates (DerSimonian & Laird, 1986; Cooper, Hedges, & Valentine, 2009). This decision was supported by a test revealing that effect-sizes were not homogeneous across all studies ($k = 99$; $Q_T\,(98) = 274.1$, $p < 0.001$), with other

statistics indicating a notable degree of heterogeneity in the effect-sizes (T^2 = .13; I^2 = 64.2%). Besides justifying the use of a random-effects model, the existence of heterogeneity in effect-sizes also confirms an important auxiliary hypothesis— that variance in effect-sizes exists— to test for potential moderation across studies. Overall, there was a moderate effect of all experimental manipulations on death-thought accessibility (DTA) effect-size (g = .57, 95% C.I. = (.48, .66), $p < 0.001$).

Next, we analyzed studies by type of threat and compared them using the Analog to the ANOVA (Q-test), which revealed that mortality salience (MS; k = 31), death-reminders (k = 20), and other types of threats (k = 48) significantly differed in their effect on DTA (Q_B (2) = 10.55, $p < 0.05$; see Table 3 for effect-size estimates). Direct group comparisons using random-effects meta-regression revealed that MS studies resulted in marginally larger effect-sizes on DTA than death-reminder studies (β = .25, SE = .13 ; t (50) = 1.97, p = .054). However, MS studies did not display larger effect-sizes than studies of threats that were not explicitly related to death (β = .16, SE = .11; t (78) = 1.51, p = .14), and effect-sizes did not significantly differ between death-reminders and studies without explicitly death-related threats (β = -.09, SE = . 12; t (67) = -0.71, p = .48).

Effect-size by Study Characteristics

Analyses were conducted in order to determine if differences in studies' methodological or procedural characteristics were associated with the effect-sizes achieved, including differences operationalization of DTA, delay length, and in a subsample of MS studies, the control condition used.

DTA Measurement. Studies operationalized DTA in three different ways: word fragment completion tasks (k = 91), reaction time to recognize death-related words (k =5), and

the number of death-related references in free writing-type tasks (k =3). Because the sample of studies was small for the free-writing group, we excluded it from comparative analysis. Analysis revealed that studies using the word fragment task did not differ in effect-size from those that used a reaction time paradigm (β = .19, SE = .23; $t(95) = 0.81$, p = .42).

Specifically regarding the word fragment completion task, studies tended to include different numbers of death-related word fragments in the measure. Measures with more death-related word fragments may be more or less likely to capture DTA effects, especially if the early death-related items prime or suppress identification of later ones. As well, having more neutral word fragments—space between the death-related fragments—could enhance or diminish this effect. Thus, hierarchical meta-regression was used to, first, regress effect-sizes on the number of death-related word fragments, and second, regress effect-sizes on death-related fragments while controlling for the number of neutral word fragments. The number of death-related word fragments did not predict DTA effect-size either by itself (β = -.005, SE = .02; t (83) = -0.29, p = .77), or when the number of neutral fragments was entered as a covariate (b = -.03, SE = .03; t (77) = -0.97, p = .33).

Delay between Threats and DTA Measurement. Having shown that DTA was not influenced by the type of DTA measure used, we collapsed across DTA measure type to investigate the role of delay on DTA. Three different operationalizations of delay were used, as described earlier: whether studies included a delay task or not (Martens, Burke, Schimel, & Faucher, 2011), the number of delay tasks that were included in the study, representing *task-switching* (Burke, Martens, & Faucher, 2010), and estimated delay length (seconds). Importantly, only a few studies included three delay tasks (the maximum), which might serve as outliers. However, forcing a coding cap of two delay tasks did not change any results in terms of

statistical significance, except for changing one result from significant to marginal significance, and so we present all findings using the original coding of up to three delay tasks.

As a preliminary step, analyses of the general effects of delay on DTA, regardless of threat type, were conducted ($k = 99$). There was no significant effect of delay on DTA, whether delay was assessed as inclusion of any delay ($\beta = -.04$, SE = .09; $t = -0.43$, $p = .67$), task-switching ($\beta = -.007$, SE = .06; $t = 0.12$, $p = .9$), or estimated delay length ($\beta = .0003$, SE = .0003; $t = 0.92$, $p = .36$). This finding is consistent with TMT, which suggests that delay should not have a main-effect on DTA independently of the type of threat.

Next, analyses were conducted to examine whether delay predicts DTA following specific types of threats. Because in the present study we found effect-size differences between mortality salience (MS) and death-reminders, we ran analyses at three levels to examine this relationship. Specifically, we sought to determine whether delay was positively associated with DTA following any type of death-related threat, following mortality salience threats only, and following death-reminders only. Finally, we tested for the influence of delay on DTA following threats that were not explicitly death-related.

All death-related threats. Regressing DTA effect-sizes on delay for all types of death-related threats (both MS and death-reminders; $k = 51$) revealed marginally significant, positive effects of delay for studies with no delay tasks vs. studies with delay tasks ($\beta = .25$, SE = .13; $t = 1.92$, $p = .06$). Analysis also revealed a significant positive effect of task-switching ($\beta = .17$, SE = .07; $t = 2.41$, $p = .02$) and estimated delay length ($\beta = .001$, SE = .0004; $t = 3.05$, $p = .004$; see Figure 2) in predicting the effect of all death-related threats on DTA. A summary of these findings can be found in Table 4. These findings are generally consistent with the dual-process component of TMT. In contrast, the effect-size at the intercept (no delay) was significantly

larger than $g = 0$ ($k = 16$, $g = .43$, 95% C.I. = .29, .57, $p < 0.001$), suggesting that immediate DTA suppression may not be occurring, which is inconsistent with the dual-process component.

Mortality salience. Conducting similar analyses on only mortality salience (MS) studies ($k = 31$) provided similar results. MS studies' inclusion of a delay versus no delay did not significantly predict DTA effect-size ($\beta = .34$, SE = .19; $t = 1.79$, $p = .08$). However, DTA effect-size was significantly predicted by both task-switching ($\beta = .25$, SE = .1; $t = 2.65$, $p = .01$) and estimated delay length ($\beta = .002$, SE = .0004; $t = 4.06$, $p < 0.001$; see Figure 3). Although the sample was small ($k = 6$), observing effect-size of MS on DTA at the intercept (no delay) revealed an effect that was significantly larger than $g = 0$ ($g = .42$, 95% C.I. = .18, .66, $p = 0.001$).

Death-reminders. Finally, regressing effect-sizes on delay for only death-reminders ($k = 20$) did not reveal any significant effects (all p's > .4; see Table 4 for estimates). This may imply that there is something unique about personal reflections on mortality (MS) as compared to other reminders of death, but the limitations of this interpretation will be highlighted in the discussion. Again, the estimated effect-size at the intercept (no delay) was significantly larger than $g = 0$ ($k = 10$, $g = .43$, 95% C.I. = .24, .62, $p < 0.001$).

Not explicitly death-related threats. Besides death-related threats, TMT also proposes that threats that are not explicit related to death, such as cultural value threats, can increase DTA. In addition, some research indicates that the effect of these threats on DTA decreases following a delay (Schimel, Hayes, Williams, & Jahrig, 2007; Martens, Burke, Schimel, & Faucher, 2011). Thus, DTA effect-sizes were regressed on studies' use of delay in order to test for the effect of delay following threats that are not explicitly related to death ($k = 48$). Results indicate that studies including a delay vs. no delay ($\beta = -.4$, SE = .13; $t = -2.99$, $p = .004$) and more task-

switching (β = -.19, SE = .08; t = -2.45, p = .02; see Figure 4) tended to achieve a smaller DTA effect-size following threats that are not explicitly related to death (see Table 4 for estimates). However, estimated length of delay did not predict significant differences in DTA (β = -.0007; SE = .0004; t = -1.57, p = .12).

Control Conditions. In many cases, studies used unique control conditions, making meta-analytic comparison of control conditions difficult. The exception to this is mortality salience studies, which often used either *dental pain salience* (k = 21) or *television salience* (k = 5) as a control (i.e. think about dental pain or watching television). Traditionally, the distinction between these two control conditions is important, as it is expected that pain can control for general negative experience but television watching cannot (Greenberg et al. 1995). Comparison of MS studies with these two types of control conditions revealed that the control conditions did not predict differences in DTA (β = -.29, SE = .25; t = -1.14, p = .26), even when estimated delay length was entered as a covariate (b = -.32, SE = .2; t = -1.59, p = .13). This suggests that, at least in the case of studies of MS's effect on DTA, general negative emotional experience may not be a crucial control.

Discussion

The present analysis sought to test two aspects of the dual-process component of TMT, and a third related hypothesis. First, the dual-process component of TMT argues that following explicitly death-related threats (a) DTA should be immediately suppressed, but that (b) after a delay, this suppression should subside and DTA differences should emerge. In contrast, we additionally hypothesized that delay would be negatively associated with DTA effect-size in the case of threats that are not explicitly related to death.

Before accounting for delay, studies of the effect of TMT threats on DTA seem to have resulted in a moderately strong effect of TMT threats on DTA (g = .57). Analysis also revealed that mortality salience (MS) manipulations seem to lead to greater DTA than death-reminders. This seems to suggest that reflecting on one's *personal* mortality leads to particularly high levels of DTA. One critical limitation to this interpretation of the present data, however, is that these studies are independent and used a variety of control conditions. It is possible that different types of control conditions in each class of threat may drive the difference in effect-size. This perspective is supported by the fact that MS studies tend to employ relatively similar control conditions (e.g. two questions about dental pain; two questions about watching television), while death-reminder and other threat studies tend to use control conditions that are unique to the study. While we present some evidence that neutral versus negative controls do not influence effects in the case of MS and DTA, it is unclear that this should be extrapolated to, for example, death-reminders. We do acknowledge that we computed effect sizes using the most conservative control conditions available, and so attempted to minimize the likelihood that the effects of threat would be confounded with the nature of the control conditions.

The present study also observed whether there were differences in DTA effect-size as a function of DTA's operationalization. Interestingly, the word fragment completion task and reaction-time measures of DTA did not predict differences in DTA effect-size. As well, the number of word fragments in the word fragment completion task was not associated with different effect-sizes. This latter finding in particular may be conflated with culture, lab, or language, however. For example, the frequency at which certain death-related words or concepts are commonly used may vary by language, which might affect priming sensitivity.

In terms of the immediate suppression of DTA following death-related threats, there seem to be several instances where DTA was not immediately suppressed. Although the dual-process component does not articulate that there will be *no effect* of DTA immediately following death-related threats, DTA suppression does imply this. One possible explanation for immediate, but smaller DTA effect-size following death-related threats is that only strongly death-related concepts are immediately suppressed, while loosely death-related ones are not. Alternatively, it may be that only strongly death-related concepts are immediately primed, but that a spreading activation to loosely death-related concepts is temporarily stifled.

As for delay effects, the present study found that studies including more task-switching or a longer delay following MS tended to achieve larger DTA effects. In the case of threats that were not explicitly related to death, including any delay or more task-switching during the delay was associated with smaller DTA effects. These findings are fairly consistent with the dual-process component's predictions regarding the effect of delay (Pyszczynski, Greenberg & Solomon, 1999), as well as with studies that imply a negative association between delay and DTA following threats that are not explicitly death-related (Martens, Burke, Schimel, & Faucher, 2011).

Limitations

There are several limitations to the present study. First, the present analysis only included a subset of all death-thought accessibility (DTA) studies. Specifically, only studies that were both experimental and met strict theoretical design criteria (aside from delay) for increasing DTA were included. Because we used strict criteria for excluding studies based on theoretical design, the present study offers a rigorous test of TMT's predictions regarding DTA, but does not account for other manipulations that could influence DTA. Additionally, because some DTA

studies were excluded for having designs that should lead to theoretically predicted reductions in DTA effect-size, it is unclear whether these studies might have detected differences in DTA, or the extent to which they reduced DTA effects. (Notably, reductions in DTA were the objective of some of these studies; e.g. Cohen, Sullivan, Solomon, Greenberg, & Ogilvie, 2011.)

All comparative analyses in the present study are strictly correlational. These tests may suggest conditions under which threats are most likely to lead to causal increases in DTA. However, the present tests comparing different types of threats, or studies' methodological and procedural characteristics do not imply causal influence of these factors on DTA. Similarly, because these comparisons are correlational, neither do these results imply that causal relationships are *not* underlying these relationships, even when the correlation appears null. Lastly, the analyses were based only on published studies. While it is unclear whether biases may have favored the publication of results consistent with the TMT perspective of delay on DTA, it is important to recognize that nearly all of these studies were not explicitly testing the role of delay on DTA (cf. Schimel, Hayes, Williams, & Jahrig, 2007; Trafimow & Hughes, 2012). Rather the majority of studies were simply testing DTA responses to threats, but happened to contain information useful for testing the overall relationship between delay length and the magnitude of DTA. As well, we did not find any evidence that studies' use of delay was associated with small-study effects.

Finally, it is important to note that the present data cannot control for the possibility that particular *types* of delay tasks (e.g. PANAS) influence DTA independently of delay length. However, we emphasize that studies used a variety of different delay tasks (different questionnaires, passages, puzzles, and other tasks), and that many studies included no delay task

or multiple types of delay tasks, which should minimize the influence of any particular type of delay task.

Future Directions

The present study provides evidence that not all threats are the same in terms of DTA. They may lead to different effect-sizes, and also may interact with delay differently (positive vs. negative effect on DTA). Especially interesting is that there might be a distinction between the effects of MS and that of death-reminders, both in terms of effect-size and delay. This could suggest that participants who are less engaged in MS express DTA in a way that is more similar to the effects of death-reminders (smaller effect, no association with delay). It would be worthwhile for future research to explore how impersonal death-reminders are different than moderate to deep elaboration on personal mortality. This may provide the opportunity to expand TMT's predictive power, but is also practically useful since death-reminders are quite common in the context of daily living.

Second, the present study presents strong evidence that, across a wide range of studies, delay seems to share a positive relationship with death-thought accessibility (DTA) following mortality salience (MS). Thus it is striking that researchers have sometimes consistently found the opposite effect of delay following MS (Trafimow & Hughes, 2012). Most of the studies conducted by Trafimow & Hughes (2012) did not meet the design and methodological specifications of the current study (e.g. some were not between-subjects), and thus could be considered anomalies within the larger context of DTA research.

However, the current results indicate that DTA is not truly or fully suppressed immediately following MS. While this cannot explain the delayed reductions in DTA observed by Trafimow & Hughes (2012), it does agree with their finding immediate effects of MS on

DTA. The present finding that DTA is not suppressed may reflect chance, but highlights the need for closer inspection of DTA activation and suppression immediately following MS. One possible explanation for these findings is that DTA is only partially suppressed. For example, perhaps only the most strongly or weakly death-related words are initially suppressed. If this is the case, it could lead to unique predictions and warrant some re-specifications of TMT. For example, partial DTA suppression could imply that proximal defenses are actually associated with activation of strongly death-related concepts in the absence of loosely death-related concepts, whereas distal defenses are associated with a spreading activation of loosely death-related concepts. Thus, TMT may benefit from greater specification and more precise measurement of death-thought accessibility.

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Footnotes

[1] In some cases, this required calculating two effect-sizes, one for each type of threat (e.g. MS vs. value threat), using the same control condition (12 controls used to draw 24 effects). These are the only cases in which samples were dependent rather than independent, and so this dependency may only influence analyses that include multiple threat types (e.g. overall DTA effect-size), but not any analyses by threat type. To determine whether it would be inappropriate to include these studies as independent in any analyses, we meta-analytically compared the weighted averages of the dependent effects to the effects considered as independent. The analysis revealed that accounting for the effects as either dependent or independent did not significantly influence the effect-size estimate ($t = 0.03$, $p = .97$), and the sets did not express a large difference in heterogeneity (weighted averages $T^2 = .1$, independent $T^2 = .17$). For this reason, and for the sake of parsimony, these effects were included as independent in all analyses.

[2] Funnel plots, p-curves, and associated analyses are available as supplementary materials.

Tables

Table 1.

Delay Length Estimates by Task Type

Delay Task Type	Derived from	Mean/Est. Seconds	SD
Passage	Independent Sample (n = 43)	162.32	57.95
One Questionnaire Item	Independent Sample (n = 43)	3.76	0.35
PANAS (20 item)	Independent Sample (n = 43)	75.2	20.71
PANAS-X (60 item)	Length of Item x Number of Items	225.6	--
Puzzle	Authors' Reports (3.5 min)	210	--
Other	Average Task Length	217.34	--

Table 2.

DTA Effect-size, Threat Type, and Delay Codes for DTA Studies

Authorship	Year	Study	Threat Code	Delay Tasks	Delay Length (seconds)	Sample Size	*g*	*S.E.*
Arndt, Cook, Goldenberg, & Cox	2007	1	MS	2	420	33	.88	.36
Arndt, Cook, Goldenberg, & Cox	2007	1	DR	2	420	32	.01	.34
Arndt, Cook, Goldenberg, & Cox	2007	2	DR	2	420	20	-.4	.43
Arndt, Greenberg, Pyszczynski, & Solomon	1997	1	NEDR	0	0	22	1.07	.44
Arndt, Greenberg, Pyszczynski, & Solomon	1997	3	NEDR	0	0	25	.94	.41
Burris & Rempel	2004	6	DR	0	0	67	.56	.25
Burris & Rempel	2004	6 F	DR	0	0	29	.69	.37
Burris & Rempel	2004	6 F	DR	1	214.82	28	.61	.38
Carey & Sarma	2011	1	DR	1	210	80	1.16	.24
Chatard & Selimbegovic	2011	5	NEDR	0	0	28	-.07	.37
Chatard, Pyszczynski, & Arndt	2012	1	MS	0	0	105	.01	.19
Cohen, Soenke, Solomon, & Greenberg	2013	2	MS	2	387.92	67	.167	.28
Cohen, Soenke, Solomon, & Greenberg	2013	2	NEDR	2	387.92	67	1.92	.29
Cohen, Soenke, Solomon, & Greenberg	2013	3	MS	2	387.92	28	1.02	.39
Cohen, Soenke, Solomon, & Greenberg	2013	3	NEDR	2	387.92	26	1.57	.44
Cohen, Soenke, Solomon, & Greenberg	2013	4	MS	2	387.92	41	1.07	.33
Cox, et al.	2008	1	MS	2	285.2	114	.87	.34
Das, Bushman, Bezemer, Kerkhof, & Vermeulen	2009	1	DR	0	0	44	.35	.28
Das, Bushman, Bezemer, Kerkhof, & Vermeulen	2009	1	DR	0	0	56	.22	.28
Das, Bushman, Bezemer, Kerkhof, & Vermeulen	2009	2	DR	0	0	101	.5	.2
Echebarria-Echabe	2013	1	MS	2	365.22	45	1	.31
Echebarria-Echabe	2013	1	NEDR	2	365.22	45	.23	.3
Echebarria-Echabe	2013	2	MS	2	365.22	45	1.3	.32
Echebarria-Echabe	2013	2	NEDR	2	365.22	45	.15	.29
Edmondson, et al.	2011	1	MS	0	0	125	.76	.18
Edmondson, et al.	2011	1	MS	2	420	179	.68	.15
Edmondson, et al.	2011	1	NEDR	0	0	127	.18	.18

Edmondson, et al.	2011	1	NEDR	2	420	174	.21	.15
Edmondson, et al.	2011	2	MS	0	0	142	.42	.17
Florian, Mikulincer, & Hirschberger	2001	2	MS	1	71.44	120	.52	.18
Florian, Mikulincer, & Hirschberger	2002	3	NEDR	0	0	66	.71	.22
Fritsche, Jonas, & Fankhanel	2008	4	MS	2	116.56	57	.43	.26
Fritsche, et al.	2007	2	MS	1	75.2	140	.28	.17
Gailliot, Schmeichel, & Baumeister	2006	2	NEDR	1	86.48	19	.94	.46
Gailliot, Schmeichel, & Maner	2007	1	NEDR	1	60.16	46	1.06	.48
Goldenberg, Arndt, Hart, & Routledge	2008	2 P	NEDR	0	0	34	.65	.34
Goldenberg, Pyszczynski, McCoy, Greenberg, & Solomon	1999	2	NEDR	0	0	38	.32	.23
Goldenberg, Pyszczynski, McCoy, Greenberg, & Solomon	1999	3	NEDR	0	0	26	.57	.28
Golec de Zavala, Cichocka, Orehek, & Abdollahi	2012	1	MS	0	0	158	.32	.16
Grover, Miller, Solomon, Webster, & Saucier	2010	1	MS	2	169.8	103	.43	.2
Grover, Miller, Solomon, Webster, & Saucier	2010	1	DR	2	169.8	103	.43	.2
Harmon-Jones, et al.	1997	3	MS	1	225.6	26	.39	.38
Harmon-Jones, et al.	1997	3	MS	2	387.9	25	2.35	.5
Hayes, Schimel, & Williams	2008	1	NEDR	0	0	50	.62	.29
Hayes, Schimel, Faucher, & Williams	2008	1	NEDR	0	0	38	.46	.32
Hayes, Schimel, Faucher, & Williams	2008	2	NEDR	0	0	30	.88	.37
Hayes, Schimel, Faucher, & Williams	2008	3	NEDR	0	0	65	.36	.25
Hirschberger, Ein-Dor, & Almakias	2008	1 P	NEDR	1	75.2	27	.96	.4
Hirschberger, Ein-Dor, & Almakias	2008	2 P	DR	1	75.2	40	.72	.32
Hirschberger, Florian, & Mikulincer	2005	3	NEDR	0	0	31	.15	.18
Jessop & Wade	2008	1	MS	1	210	39	.67	.32
Jessop & Wade	2008	1	DR	1	210	39	.92	.33
Jessop, Albery, Rutter, & Garrod	2008	4	DR	1	210	61	.46	.26
Jonas & Fischer	2006	3	MS	1	75.2	50	.1	.28
Landau, et al.	2004	2	NEDR	0	0	31	.01	.28
Landau, et al.	2004	2	NEDR	0	0	31	.96	.37
Ma-Kellams & Blascovich	2012	1	MS	1	75.2	63	.35	.25
Maxfield, et al.	2007	1	MS	1	210	116	-.21	.26
Mikulincer & Florian	2000	2	MS	0	0	87	.15	.17
Mikulincer & Florian	2000	3	MS	1	82.7	83	1.38	.19

Mikulincer, Florian, Birnbaum, & Malishkevich	2002	1	NEDR	0	0	72	.65	.24
Mikulincer, Florian, Birnbaum, & Malishkevich	2002	1	DR	0	0	72	.8	.24
Mikulincer, Florian, Birnbaum, & Malishkevich	2002	2	NEDR	0	0	60	.85	.27
Mikulincer, Florian, Birnbaum, & Malishkevich	2002	3	NEDR	0	0	58	.84	.23
Motyl, et al.	2012	1	NEDR	1	90	36	.16	.24
Motyl, et al.	2012	3	NEDR	1	75.2	62	-.4	.33
Navarrete, Kurzban, Fessler, & Kirkpatrick	2004	2	MS	1	225.6	50	.62	.29
Navarrete, Kurzban, Fessler, & Kirkpatrick	2004	2	NEDR	1	225.6	51	.27	.28
Proulx & Heine	2008	1b	NEDR	1	75.2	40	-.15	.31
Proulx & Heine	2009	1	NEDR	1	75.2	40	-.37	.31
Proulx & Heine	2009	2	NEDR	1	75.2	53	-.11	.27
Routledge, Arndt, Sedikides, Wildschut	2008	3	MS	1	162.32	38	.69	.33
Rutjens & Loseman	2010	1	MS	1	75.2	52	1.21	.3
Rutjens & Loseman	2010	1	NEDR	1	75.2	52	.3	.27
Rutjens, van der Pligt, & Harreveld	2009	1	MS	2	285.2	53	.29	.28
Rutjens, van der Pligt, & Harreveld	2009	2	NEDR	0	0	43	.8	.31
Rutjens, van der Pligt, & Harreveld	2009	3	MS	1	75.2	45	.72	.3
Schimel, Hayes, Williams, & Jahrig	2007	1	NEDR	2	435.6	31	.22	.35
Schimel, Hayes, Williams, & Jahrig	2007	1	NEDR	0	0	30	1.08	.38
Schimel, Hayes, Williams, & Jahrig	2007	3	NEDR	0	0	38	1.03	.34
Schimel, Hayes, Williams, & Jahrig	2007	4	NEDR	0	0	35	1.07	.35
Schimel, Hayes, Williams, & Jahrig	2007	5	NEDR	0	0	40	.88	.33
Shehryar & Hunt	2005	1 P	DR	0	0	49	.83	.29
Silvia	2001	1	NEDR	1	3.76	20	.38	.43
Taubman-Ben-Ari	2004	2	NEDR	0	0	68	.98	.25
Taubman-Ben-Ari	2004	3	NEDR	0	0	74	.74	.24
Taubman-Ben-Ari	2011	2	MS	0	0	46	.89	.3
Taubman-Ben-Ari	2011	2	NEDR	0	0	45	.97	.31
Taubman-Ben-Ari	2011	3	NEDR	0	0	51	.82	.29
Taubman-Ben-Ari & Katz-Ben-Ami	2008	1	NEDR	0	0	60	.62	.26
Trafimow & Hughes	2012	3	MS	2	162.32	60	0	.25
Trafimow & Hughes	2012	3	MS	0	0	60	.31	.26

Ullrich & Cohrs	2008	3	DR	0	0	26	-.1	.26
Vail, Arndt, Motyl, & Pyszczynski	2012	1	NEDR	2	285.2	25	.86	.41
Vail, Arndt, Motyl, & Pyszczynski	2012	4	NEDR	0	0	26	1.15	.41
Van Beest, Williams, & Van Dijk	2011	2 P	DR	0	0	50	.63	.29
Van Tongeren & Green	2010	1	NEDR	3	225.6	101	-.13	.2
Van Tongeren & Green	2010	2	NEDR	2	319.6	122	-.22	.18
Wojtkowiak & Rutjens	2011	1	MS	1	210	36	.93	.35

Note: "MS" = mortality salience, "DR" = death-reminder, "NEDR" = not explicitly death-related threat. "P" = pretest/pilot study, "F" = follow up study.

Table 3.

DTA Effect-size Estimates by Threat Type

Threat	*k*	*g*	95% C.I.	T^2
All Threat Types	99	.57	(.48, .66)	.12
Death-related	51	.6	(.48, .72)	.11
Mortality Salience	31	.7	(.54,.85)	.12
Death-reminders	20	.44	(.28, .61)	.07
Not Explicitly Death-related	48	.54	(.4, .67)	.14

$\boldsymbol{T^2}$ represents tau-squared, with the square root of this number reflecting the estimated standard deviation of underlying effects across studies.

Table 4.

Independent Analyses of Delay as a Moderator of DTA Effect-size, by Threat Type

Type of Threat	*k*	*β*	S.E.	*t*	*p*
All Death-related Threats					
No Delay vs. Delay	51	.25	.13	1.92	.06
Task-switching	51	.17	.07	2.41	.02
Estimated Delay Length (seconds)	51	.001	.0004	3.05	.004
Mortality Salience Only					
No Delay vs. Delay	31	.34	.19	1.79	.08
Task-switching	31	.25	.1	2.65	.01
Estimated Delay Length (seconds)	31	.002	.0005	4.06	<.001
Death-reminders Only					
No Delay vs. Delay	20	.002	.18	0.01	.99
Task-switching	20	-.05	.12	-0.44	.67
Estimated Delay Length (seconds)	20	-.0006	.0007	-0.82	.42
Not Explicitly Death-related Threats					
No Delay vs. Delay	48	-.4	.13	-2.99	.004
Task-switching	48	-.19	.08	-2.45	.02
Estimated Delay Length (seconds)	48	-.0007	.0004	-1.57	.12

Figures

Figure 1. Hypothetical Model of Threat Type and Delay Predicting DTA

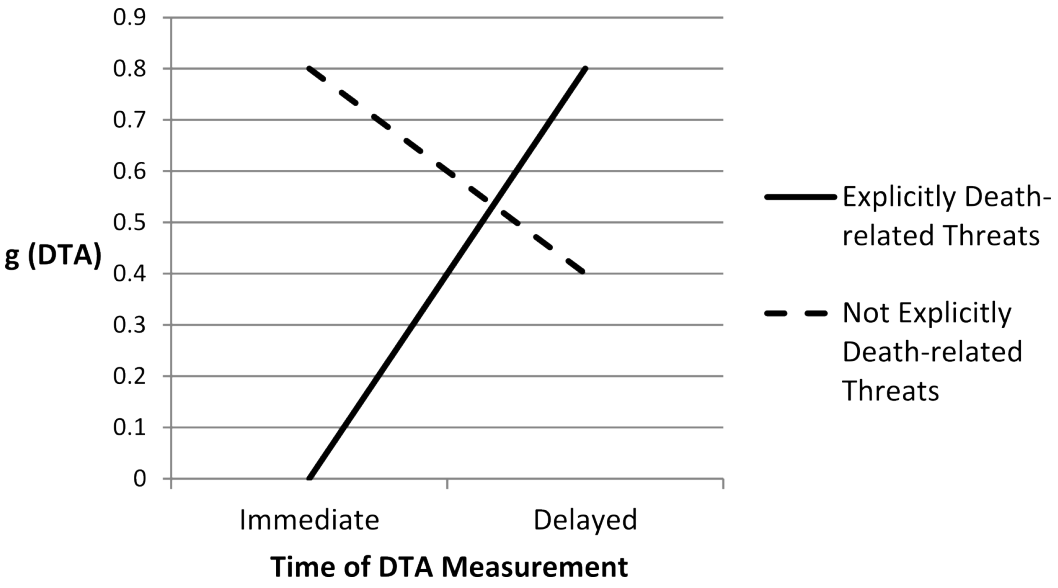

Figure 2. Effect-size by Estimated Delay Length (seconds) for All Death-related Threats

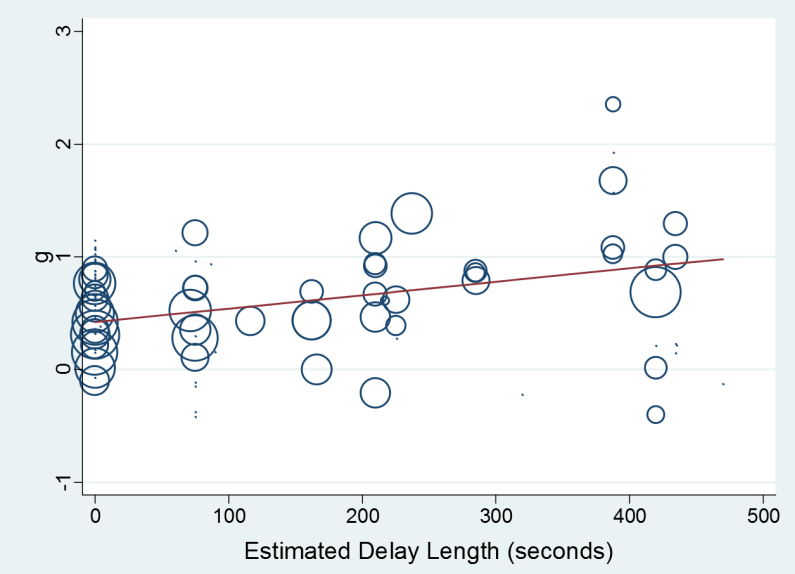

Figure 3. Effect-size by Estimated Delay Length for Mortality Salience Threats

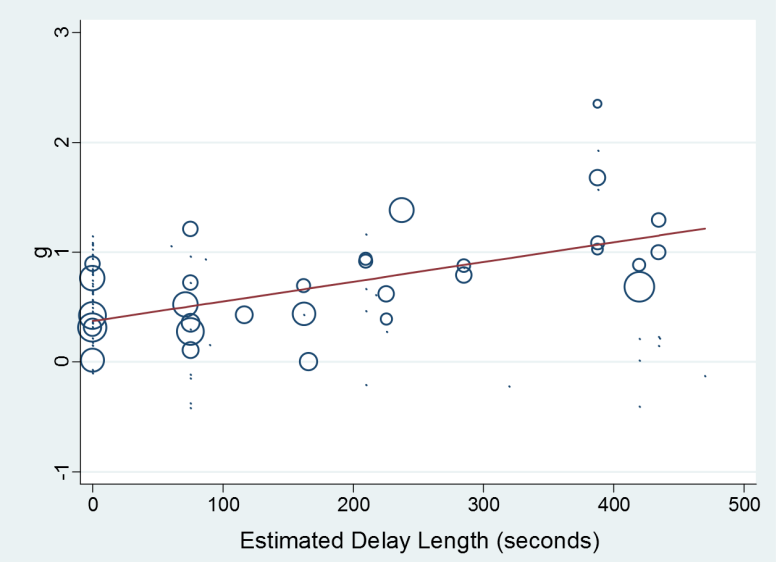

Figure 4. Effect-size by Task Switching for Not Explicitly Death-related Threats