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Gender Differences in Alexithymia

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The alexithymia literature was meta-analyzed to determine whether there was empirical support for gender differences. Our a priori theoretical motivation for expecting higher mean levels of alexithymia in men than in women was based on Levant's (1992) "Normative Male Alexithymia" hypothesis, which suggests a pattern of restrictive emotionality in traditionally reared men. Some previous works have questioned whether there is a detectable gender difference in alexithymia (i.e., Heesacker et al., 1999; Wester, Vogel, Pressly, & Heesacker, 2002), but they have not comprehensively or empirically cumulated results across studies, although Levant et al.'s (2006) narrative review suggests men tend to score higher than women on average, at least in nonclinical samples. An effect size estimate based on 41 existing samples found consistent, although expectedly small, differences in mean alexithymia between women and men (*Hedges' d* = .22). Men exhibited higher levels of alexithymia. There were no significant moderator effects for clinical versus nonclinical populations or alexithymia measure used, although there were relatively few clinical samples and non-TAS measures. Implications for theory and practice are discussed.

Keywords: alexithymia, restrictive emotionality, traditional masculinity ideology

Literally, alexithymia means "without words for emotions." Sifneos (1967, 1972) originally used the term to describe the extreme difficulty certain psychiatric patients had in identifying and describing their feelings. In addition to these emotional difficulties, the patients had very concrete cognitive styles, used little symbolism and fantasy, and reported impoverished dream states (Campos, Chiva, & Moreau, 2000; Nemiah & Sifneos, 1970). Further, they often demonstrated difficulty both in discerning the emotional states of others (Berenbaum & Prince, 1994; Parker, Taylor, & Bagby, 1993) and displaying empathy in interpersonal relationships (Krystal, 1979).

This pattern was particularly evident in patients with psychosomatic illnesses (Sayar, Kirmayar, & Taillefer, 2003; Sifneos, 1967; Taylor, 1984). Similar characteristics have also been noted in patients with posttraumatic stress disorder (PTSD) (Henry et al., 1992; Krystal, 1979), substance dependence (Henry et al., 1992; Krystal, 1979), and chronic pain disorders (Mendelson, 1982). However, alexithymia appears not to be a substantive feature of any of these syndromes, but instead a related, comorbid condition (Taylor, 1984). In addition to the appearance of alexithymia in clinical populations, variability along a continuum of alexithymia symptoms has also been observed in nonclinical populations. That variability in nonclinical populations is the primary focus of the current study (although clinical samples are included for comparison purposes), which consists of a meta-analytic investigation of mean gender differences in alexithymia.

Etiology of Alexithymia

A variety of theories seek to explain the etiology of alexithymia; explanations have ranged from the biological to the intrapsychic to the interpersonal (see Taylor, 1984 for a review). For example, some research has connected alexithymia to difficulties originating in the interhemispheric transfer of information within the brain or abnormalities in hemispheric specialization (Sifneos, 1988; Zeitlin, Lane, O'Leary, & Schrift, 1989). Specifically, right

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hemispheric deficits have been linked to higher levels of alexithymia (Lumley & Sielky, 2000; Spalletta et al., 2001). Another biologically based explanation focuses on increased noradrenergic activity and decreased basal activity within the hypothalamic-pituitary-adrenal (HPA) axis, which relates to higher levels of alexithymia (Henry et al., 1992; Spitzer, Brandl, Rose, Nauch, & Freyberger, 2005). However, the samples in these studies were derived from various clinical populations (e.g., stroke patients, patients with Major Depressive Disorder), thus, it is unclear the extent to which these biological mechanisms generalize as explanations of variability in alexithymia in nonclinical populations. For example, in nonclinical populations, social learning processes may make an important contribution to the development of alexithymia (Borens, Grosse-Schultze, Jaensch, & Kortemme, 1977; Cremerius, 1977). We focus on a social learning theory explanation as a basis for expecting gender differences in alexithymia, as described further in the following paragraphs.

Alexithymia as the Product of Gender Role Socialization

Levant (1992) proposed the "Normative Male Alexithymia" (NMA) hypothesis to account for a socialized pattern of restrictive emotionality influenced by traditional masculinity ideology that he observed in many men. Both working with research participants in the Boston University Fatherhood Project, and with clients in his clinical practice, Levant observed that only with great difficulty and practice could many of the men find the words to describe their emotional states (Levant, 1992). He theorized that those men had been discouraged as boys from expressing and talking about their emotions by parents, peers, school teachers, or sports coaches, and some were even punished for doing so. Hence, they did not develop a vocabulary for, nor an awareness of, many of their emotions (Levant, 1992).

In particular, these men showed the greatest deficits in identifying and expressing emotions that induce a sense of vulnerability (like sadness or fear) or that express attachment (like fondness or caring). Although restricted emotionality may be adaptive in some ways, particularly in highly competitive environments, Levant's clients often reported significant difficulties in their personal lives and presented with a variety of potentially related problems, including marital difficulties, estrangement from their children, substance abuse, domestic violence, and sexual addiction (Levant & Kopecky, 1995).

Levant's clinical observations are consistent with the dominant theoretical perspective in the psychology of men and masculinity, the Gender Role Strain Paradigm (Pleck, 1981, 1995). This perspective posits societal forces differentially shape men according to the degree to which they have been reared as boys to adopt the norms of traditional masculinity (Levant, 1992; Thompson & Pleck, 1995; Unger, 1990). Boys reared under the influence of traditional masculinity ideology are theorized to develop into men who endorse and conform to more traditional masculine gender roles, as can be measured by instruments such as the Male Role Norms Inventory–Revised (MRNI–R; Levant, Smalley, Aupont, House, Richmond, & Noronha, 2007) and the Conformity to Masculine Norms Inventory (CMNI; Mahalik, Locke, Ludlow, Diemer, Scott, Gottfried, et al., 2003).

One normative masculine role requirement is the restriction of emotional expression. This norm is theorized to establish and maintain power and to hide vulnerability in a patriarchal social system (Levant, 1992). Levant (1992, 1995, 1998) drew on the Gender Role Strain Paradigm to theorize that mild to moderate forms of alexithymia would occur more frequently among men whose socialization as boys was influenced to greater degrees by traditional masculinity ideology. Indeed, empirical research finds a relationship between the endorsement of traditional masculinity ideology and alexithymia in men. Levant et al. (2003) indicated that even after controlling for demographic differences, traditional masculinity ideology accounts for unique variance in alexithymia in men.

The view that socialization plays a role in the development of restricted emotionality confronts the conventional view in our society that boys and men are essentially "hardwired" to be less emotional and more logical than are girls and women. This more conventional view derives from presumed biologically based gender differences in the experience and expression of emotion (see Wester, Vogel, Pressly, & Heesacker, 2002 for a review). Levant's (1998)

review of relevant developmental psychology research literature on masculine emotion socialization concluded that the essentialistic, conventional perspective that men, by nature, are less emotional was not supported by the existing evidence. Indeed, evidence suggests boys start life with greater emotional reactivity and expressiveness than girls (Levant, 1998). However, they become less verbally expressive than girls at about the age of 2 years and less facially expressive by 6 years. This developmental change suggests that socialization shapes gender-appropriate emotional behavior and may account for gender differences in emotional awareness and expressivity (Levant, 1998).

Counterarguments to the Normative Male Alexithymia Hypothesis

Levant's (1992, 1995, 1998) NMA hypothesis, with its strong implication of observable gender differences in levels of alexithymia, has been challenged both by Heesacker et al. (1999) and Wester et al. (2002). Specifically, the authors have debated the existence of gender differences in alexithymia and cautioned readers against seeking to find gender differences where none are found. In general, these authors do raise several important points. For example, we agree that the public (and sometimes counselors and psychologists) stereotypes men as unable to express or even experience emotion. Too often, this stereotype is held as an immutable difference that should be accepted instead of explored. We also agree that, contrary to traditional masculine norms, many men do in fact have very full emotional experiences, and we do not claim that those who do not are unable to.

However, these two papers also claim that Levant's theory is challenged by the results of empirical studies of alexithymia. Heesacker et al.'s (1999) research article on gender-based emotional stereotyping states ". . . despite a number of men's psychotherapy articles focusing on alexithymia . . . as an emotional deficit of men . . . recent empirical research on alexithymia suggests there are no gender differences . . ." (p. 484). Similarly, Wester et al.'s (2002) review of the literature on gender differences in emotions commented "empirical research on the verbal expression of emotions in general . . . and alexithymia specifically, do *not* demonstrate a consistent gender-based pattern of results" (p. 635). Yet, neither Heesacker et al. nor Wester et al. systematically reviewed the extant literature on gender differences in alexithymia. Heesacker et al. (1999) refers to a single nonconfirming study on gender differences in alexithymia, and Wester et al. (2002) refers to only two such studies. This raises the potential that their counterarguments are based on an unrepresentative sampling of studies of gender differences in alexithymia.

Need for More Comprehensive Review of Gender Differences in Alexithymia

To begin to address this issue, Levant et al. (2006) reviewed 45 published studies that examined gender differences in alexithymia, either as a focus, or as part of a larger research question. The 13 studies using a psychiatric or medical clinic sample were examined separately from those that used a nonclinical sample. Levant et al. (2006) noted that few studies using clinical samples found gender differences (two found males more alexithymic than females, one found females more alexithymic than males, and 10 found no differences between males and females). However, the 32 studies using nonclinical samples presented a very different picture: 17 of these studies found males more alexithymic than females, one found females more alexithymic than males, and 14 found no differences between males and females. Yet, this narrative review still leaves open the issue of the magnitude of the gender difference in alexithymia, as well as the extent of the distinction between clinical and nonclinical samples, if any.

Overview of Current Study

This study addresses whether gender differences in alexithymia are evidenced in the literature, such that men score higher on measures of alexithymia than do women. Of course, the examination of demographic differences in any psychological trait must be undertaken with care. For example, Quintana, Troyano, and Taylor (2001) caution that demographic variables may merely serve as proxies for psychological variables in multicultural research. Additionally, special care must be taken when examining gender differences in psychological variables, because findings of such differences have all too often been used to

the detriment of women (Eagley, 1995; Enns, 2000; Hyde & Plant, 1995).

Furthermore, for many psychological variables, it is important to recall that even when there are identified mean differences, the genders have overlapping distributions with more similarities than differences (Hyde, 2005). However, such examinations of gender differences may prove important if they provide support for a more general theoretical perspective or help providers identify needs that can more effectively be addressed when acknowledged.

Additionally, even if gender differences are found, this study addresses only whether they exist, and not why. We frame this study as the first step in supporting Levant's (1992) NMA hypothesis, seeking first to observe whether gender differences exist before seeking to causally link gender role socialization to the existence of these differences.

In summary, the purpose of this study is to further investigate gender differences in alexithymia, and to determine the magnitude of those differences. To do so, we conducted a meta-analysis of the existing empirical studies. Our samples included those considered in the Levant et al. (2006) narrative review, as well as additional unpublished and newer studies. Although we hypothesized that gender differences indeed would be found, we did not expect them to be large, as gender differences in general are usually small (Hyde, 2005). Further contributing to expectations of a relatively small overall gender effect, our theoretical reason for expecting differences, namely, the NMA hypothesis, suggests that only the subpopulation of men who have received strong traditionally masculine gender role socialization would exhibit a tendency for higher levels of alexithymia. However, because the studies available for meta-analysis do not directly measure socialization, the meta-analysis simply reflects the influence of including this subpopulation in the greater pool of all adult men.

Method

Identification of Primary Studies

The Levant et al. (2006) narrative review identified 45 studies (32 nonclinical and 13 clinical) that were included in the initial meta-analytic sample. However, a close inspection of these studies suggested there were two pairs of studies that, although they addressed different global research questions, appeared to rely on the same samples. The pairs were: (a) Lane, Sechrest, Riedel, Weldon, Kaszniak, and Schwartz (1996) and Lane, Sechrest, and Riedel (1998); and (b) Kokkonen, Veijola, Karvonen, Läksy, Jokelainen, Järvelin, and Joukamaa (2003) and Joukamaa, Kokkonen, Veijola, Läksy, Karvonen, Jokelainen, and Järvelin (2003). Thus, for each of these two pairs of studies, only one effect size was entered into the meta-analysis. (Both pairs involved nonclinical samples.) In addition, two other nonclinical studies in the Levant et al. (2006) review reported results from multiple independent samples. Specifically, Parker, Bagby, Taylor, Ender, and Schmitz (1993) reported results from three samples, and Bagby, Parker, and Taylor (1994) reported results from two samples. Those samples were analyzed separately in the meta-analysis.

Of the remaining original studies identified in the Levant et al. review, eight did not provide enough statistical information to be included in the effect size estimation. Most typically, these excluded studies either did not report alexithymia means and standard deviations by gender, or did not report an r or t statistic for the test of the gender-alexithymia relationship that could be transformed to a d effect size estimate. (Often the excluded studies simply stated that statistical tests had yielded no between-groups differences for men and women on the measure of alexithymia.) The excluded studies were Bach, Bach, Bohmer, and Nutzinger (1994); Cohen, Auld, and Brooker (1994); Dion (1996); Taylor et al. (1992); Todarello, Taylor, Parker, and Fanellil (1995); Wise, Mann, and Shay (1992); Yelsma, Hovestadt, Anderson, and Nilsson (2000), and Zeitlin and McNally (1993). However, several additional studies that were not in the original narrative review were identified through PsychInfo and requests posted to APA listserves and added to the meta-analytic sample. These included both published articles and unpublished dissertations.

The final meta-analysis was performed on a total of 42 samples; 33 of these were nonclinical and 9 were clinical. The studies used various measures of alexithymia, including the Toronto Alexithymia Scale (TAS), TAS-20 (a 20-item short form of the TAS), Amsterdam Alexithymia Scale, and Schalling-Sifneos Scale. One study

(Bray, 2003) used the Levels of Emotional Awareness Scale (LEAS), which according to the author of the study, indexes the *absence* of alexithymia. Thus, the direction of the male-female difference from this study was reflected to be consistent with the differences found in the remaining studies.

Each sample was coded for population type (clinical vs. nonclinical), publication type (published article vs. dissertation), and alexithymia measure used. Table 1 lists the publication sources for the samples included in the meta-analysis, as well as reporting the population and measure of alexithymia used for each study.

Table 1
Studies Comparing Males and Females On Measures of Alexithymia

Study	Population	Measure	d	v_d
Bagby, Parker, & Taylor-Study 1 (1994)	Nonclinical	TAS-20	0.19	.004
Bagby, Parker, & Taylor-Study 2 (1994)	Nonclinical	TAS-20	0.00	.010
Bagby, Parker, & Taylor-Study 2 (1994)	Clinical	TAS-20	0.06	.019
Blanchard, Arena, & Pallmeyer (1981)	Nonclinical	SSS	0.25	.017
Berenbaum, Davis, & McGrew (1998)	Nonclinical	TAS-20	0.29	.065
Bermond, Vorst, & Vingerhoets-Study 1 (1999)	Nonclinical	AAS	0.19	.012
Bermond, Vorst, & Vingerhoets-Study 2 (1999)	Nonclinical	AAS	0.23	.011
Bray (2003)	Nonclinical	LEAS	0.56	.008
Bressi, Taylor, Parker, Bressi, Brambilla, Aguglia et al. (1996)	Nonclinical	TAS-20	0.18	.020
Bressi, Taylor, Parker, Bressi, Brambilla, Aguglia et al. (1996)	Clinical	TAS-20	−0.09	.006
Carpenter & Addis (2000)	Nonclinical	TAS-20	0.37	.027
Delaney (2002)	Clinical	TAS-20	0.23	.049
Dennison (2001)	Nonclinical	TAS-20	2.09	.160
Feiguine, Jones, & Kassel (1988)	Clinical	MMPI	0.35	.028
Haviland, Hendryx, Shaw, & Henry (1994)	Clinical	TAS-20	−0.30	.020
Honkalampi, Kintikka, Tanskanen, Lehtonen, & Viinamaki (2000)	Nonclinical	TAS-20	0.37	.010
Eiken (2004)	Nonclinical	TAS-20	0.28	.011
Joukamaa, Solhman, & Lehtinen (1995)	Nonclinical	TAS-20	0.07	.006
Jyvasjarvi, Joukamaa, Vaisanen, Larivaara, Kivela, & Keinanen-Kiukaanniemi (1999)	Clinical	TAS-20	0.34	.023
Kirmayer & Robbins (1993)	Nonclinical	TAS-20	−0.08	.017
Kokkonen, Veijola, Karvonen, Laksy, Jokelainen, Jarvelin, & Joukamaa (2003)	Nonclinical	TAS-20	0.43	.001
Lane, Sechrest, Riedel, Weldon, Kaszniak, & Schwartz (1996)	Nonclinical	TAS-20	0.30	.011
Larsen, vanStrien, Eisinga, & Engels (2006)	Nonclinical	TAS-20	0.16	.017
Levant, Richmond, Majors, Inclan, Rossello, Heesacker, et al. (2003)	Nonclinical	TAS-20	0.16	.003
Loas, Corcos, Stephan, Pellet, Bizouard, Venisse et al. (2001)	Nonclinical	TAS-20	0.08	.005
Loas, Corcos, Stephan, Pellet, Bizouard, Venisse et al. (2001)	Clinical	TAS-20	0.06	.006
Lumley & Sielky (2000)	Nonclinical	TAS-20	0.25	.038
Millard & Kinsler (1992)	Nonclinical	TAS	−0.17	.022
Parker, Bagby, Taylor, Endler, & Schmitz-Sample A (1993)	Nonclinical	TAS-20	0.37	.015
Parker, Bagby, Taylor, Endler, & Schmitz-Sample B (1993)	Nonclinical	TAS-20	0.17	.014
Parker, Bagby, Taylor, Endler, & Schmitz-Sample C (1993)	Nonclinical	TAS-20	0.43	.010
Pandey, Mandal, Taylor, & Parker (1996)	Nonclinical	TAS-20	−0.25	.014
Parker, Taylor, & Bagby (1989)	Nonclinical	TAS-20	−0.13	.040
Parker, Taylor, & Bagby (1993)	Nonclinical	TAS-20	0.30	.020
Parker, Taylor, & Bagby (2001)	Nonclinical	TAS-20	0.30	.001
Parker, Taylor, & Bagby (2003)	Nonclinical	TAS-20	0.28	.011
Taylor, Bagby, Ryan, Parker, Doody, & Keefe (1988)	Clinical	TAS-20	0.75	.094
Taylor, Parker, Bagby, & Bourke (1996)	Nonclinical	TAS-20	0.42	.017
Taylor, Ryan, & Bagby (1985)	Nonclinical	TAS	0.20	.009
Saarijarvi, Salminen, Tamminen, & Aarela (1993)	Clinical	TAS-20	0.60	.018
Salminen, Saarijarvi, Aarela, Toikka, & Kauhanen (1999)	Nonclinical	TAS-20	0.44	.006
Vingerhoets, Heck, Grim, & Bermond-Study 2 (1995)	Nonclinical	AAS	0.71	.026

Note. d = effect size expressed as *Hedges' d*; v_d = variance of d; AAS = Amsterdam Alexithymia Scale; LEAS = The Levels of Emotional Awareness Scale; MMPI = Minnesota Multiphasic Personality Inventory-Alexithymia Scale; SSS = The Schalling-Sifneos Scale; TAS = Toronto Alexithymia Scale; TAS-20 = Toronto Alexithymia Scale-Revised (20 items).

Full citations for these studies are in the reference section, noted by asterisks.

Data Analytic Strategies

A random effects meta-analytic approach was used (e.g., Hedges & Olkin, 1985). The MetaWin v. 2 software (Rosenberg, Adams, & Gurevich, 2000) was used to perform the calculations. Random effects models assume that effect sizes (e.g., the size of the difference between males and females) may vary from study to study due not only to sampling error, but also because of a true random component of variation. This is in contrast to fixed-effects meta-analytic models, which assume that all studies of a given type share a single "true" effect size. Although there is some debate in the literature, the current consensus supports using random effects models when one wishes to generalize the meta-analytic results beyond the specific samples used in the meta-analysis (Hedges & Vevea, 1998). Gender effect sizes in the current study were compared by calculating *Hedges' d* for all samples where values of the mean and standard deviation of alexithymia scores were available for both men and women. In a few of the samples, the means and standard deviations were not available, but a value of *d* could be derived from a reported correlation coefficient or the *t* statistic resulting from a comparison of the gender group means.

As a first step a "no structure" meta-analytic model was tested, treating all samples as representative of a single population. Following this, the potential for categorical moderator effects was explored using a mixed effects model to determine whether effect sizes varied systematically by sample characteristics such as the clinical versus nonclinical nature of the population. The *Q* statistic (evaluated as a chi-square) was used to determine whether a given moderator variable significantly explained variability in gender effect sizes.

Following calculation of the meta-analytic effect size estimate, a fail-safe calculation was performed to address the issue known as the file-drawer problem (e.g., Rosenthal, 1979). Briefly, the file-drawer problem refers to the fact that there is likely a publication bias operating that makes studies with significant results more likely to be published, and thus accessible to the researcher, whereas studies with nonsignificant results are relegated to the file drawer and thus, less likely to be known and available for meta-analysis. We used Rosenberg's method (see Rosenberg et al., 2000) for performing the fail-safe calculation. This calculation indicates the number of additional nonsignificant studies that would need to be combined with the current sample of studies to change the resulting effect size estimate from significant to nonsignificant. If the fail-safe calculation result is large (Rosenthal, 1979, suggests fail-safe values of $5n + 10$ or greater, where n is the number of samples in the meta-analysis), this indicates that there would need to be a large pool of nonsignificant studies that had not been included in the analysis, to call into question the observed significant meta-analytic result.

Results

Table 1 lists the *d* scores and their variances (v_d) for each sample, using the convention that positive values of a *d* score indicate that males score higher than females, and vice versa. Figure 1 displays the *d* effect sizes graphically, plotting each study's sample size along the *x*-axis against its corresponding *d* value along the *y*-axis. The solid dots indicate studies with nonclinical populations, the open dots are studies using clinical populations. The majority of points in Figure 1 cluster together, indicating a pattern of studies with sample sizes ranging from small to moderately large, and having somewhat similar effect sizes. Furthermore, most of the effect sizes are positive in value, indicating that males had higher mean alexithymia scores than did females. Finally, we performed an analysis to determine whether there was any linear relationship between the estimated effect size and the study sample size, and, as desired, this result was nonsignificant (*Kendall's tau* $= -.070$, $z = -.651$, $p = .515$).

However, two studies stand out from the central pattern shown in Figure 1. One of these is the Kokkonen et al. (2003) study, which is an outlier by nature of its comparatively large sample size ($N = 4601$). This study is represented by the dot at the extreme right-hand side of Figure 1. Being an outlier because of large sample size does not pose a potential problem to the meta-analysis; indeed, note that the value of *d* calculated from this study is very consistent with others in the meta-analytic sample. However,

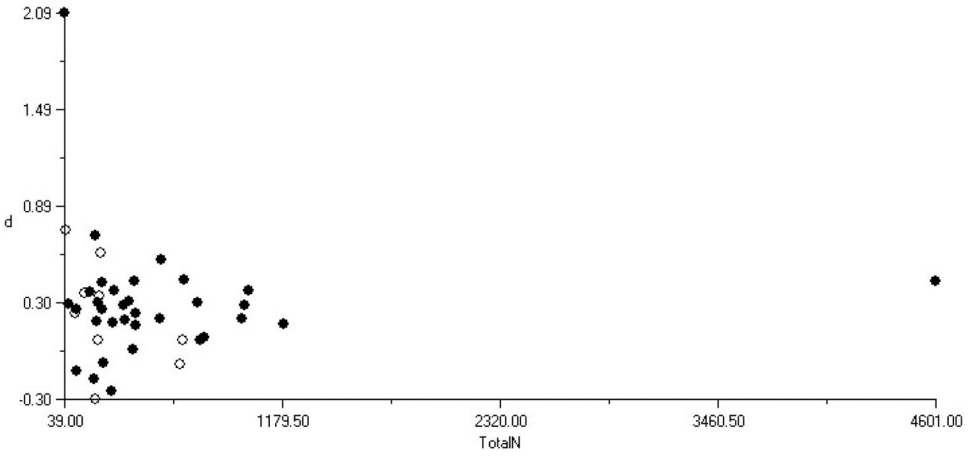

Figure 1. Meta-analytic estimates of *Hedges' d* effect sizes by study sample sizes ("Total *N*") for clinical (○) and nonclinical (●) populations.

the other outlier dot, which can be seen to the far left and toward the top of Figure 1, is more problematic. This study, an unpublished dissertation (Dennison, 2001) based on a relatively small sample size ($N = 39$), had an extremely large observed effect size ($d = 2.090$) in comparison to the remaining samples whose effect sizes ranged from values of −.301 to +.747. Thus, we performed all meta-analytic calculations twice, both including this study and excluding it from consideration.

The overall mean effect size (mean *Hedges'd*) across the complete set of 42 studies was .233 (pooled variance estimate = .037). This value had a bias-corrected bootstrapped 95% confidence interval (CI) of .160 to .304. Because the CI does not include zero, we conclude that the effect is statistically significant. The positive sign of the effect indicates higher average scores on alexithymia for men than for women, thus supporting our expectations. The fail-safe calculation indicated that 687 *more* nonsignificant studies would need to be added to the current sample of studies to change the resulting effect size estimate from significant to nonsignificant. Finally, the Q test for heterogeneity of effect sizes was not statistically significant, $Q = 55.229$, $df = 41$, $p = .068$, suggesting limited potential for moderator effects (see top row of results in Table 2).

To determine whether the Dennison (2001) study substantially biased the value of the overall effect size estimate or of its accompanying confidence interval, we repeated the overall

Table 2
Summary of Meta-Analysis Results

	Moderator analysis		Statistics in sample			95% CI		Homogeneity
	$Q_{between}(df)$	$Q_{within}(df)$	*k*	*n*	*d*	Lower	Upper	*Q*(*df*)
Overall effect	—	—	42	20,819	.233	.160	.304	55.229 (41)
Overall effect minus outlier study	—	—	41	20,780	.221	.149	.278	41.179 (40)
Type of population	.714 (1)	43.044 (39)						
Nonclinical			32	18,321	.234	.169	.298	
Clinical			9	2,459	.163	−.009	.349	
Type of measure	3.405 (1)	38.067 (39)						
TAS			35	18,879	.195	.120	.271	
Other			6	1,901	.374	.239	.528	

Note. *k* = number of samples; *n* = total number of observations, *d* = estimate of *Hedges' d*; TAS = Toronto Alexithymia Scale (all versions). Moderator analyses were performed dropping the outlier study.

meta-analysis excluding this study. The resulting overall effect size estimate based on the remaining 41 samples was still positive in sign and statistically significant, mean $d = .221$, with a pooled variance estimate of .032 and a bias-corrected 95% CI = .149–.278. Rosenthal's fail-safe statistic indicated that 599 additional nonsignificant studies would be needed to negate the significance of the effect. Again, the Q statistic did not indicate significant heterogeneity in effect sizes, $Q = 41.179$, $df = 40$, $p = .419$.

Although the nonsignificant Q statistics suggest that the observed variability in effect sizes was no larger than would be expected by random sampling error, this statistic may have had low statistical power because of the modest number of studies included in the meta-analysis. Because of this, as well as to further address issues raised in the Levant et al. (2006) narrative review, we proceeded with some additional tests of moderator effects. These tests were repeated both with the full set of 42 samples, and with the extreme outlier sample dropped. Conclusions did not differ, so we report only the results based on the 41 nonextreme samples. These results are summarized in Table 2.

The first moderator investigated was whether the clinical versus nonclinical nature of the sample influenced the gender effect size. The estimate of mean effect size for the nine clinical samples (mean $d = .163$) was smaller than that for the 32 nonclinical samples (mean $d = .234$). However, the Q test for this distinction was nonsignificant ($p = .428$), indicating that there was not a significant difference in the mean effect size estimates. Note that the lower 95% CI for the estimate based on clinical samples is negative (although only very slightly so), suggesting that the mean effect size estimate may not be significantly different from zero. This is likely because of the small number of clinical studies ($k = 9$), and is not immediately concerning given that the moderator tests do not support viewing these groups as separate populations.

Although the difference in mean effect size estimates for TAS-based (mean $d = .195$) versus non-TAS-based studies (mean $d = .374$) was not statistically significant ($p = .065$), the conclusion about differences based on measurement method is less firm given the borderline nature of the result. There were only six studies that reported using measures of alexithymia other than the TAS, and three of these studies had values of d that were noticeably higher than the mean d in the entire sample of studies. Specifically, one of the three AAS-based studies had a $d = .71$, the LEAS-based study had a $d = .56$, and the MMPI-based study had a $d = .35$. However, two of the AAS-based studies and the SSS-based study had values close to the mean d of the entire set of studies. All of the non-TAS-based studies had reasonable sample sizes (average $N = 316$, with a range from 144 to 548).

Discussion

General Conclusions About Gender Differences in Alexithymia

The primary purpose of this study was to make a meta-analytic estimate of the direction and extent of gender differences in alexithymia. Our results clearly show men score higher, on average, than women on measures of alexithymia, based on the accumulation of empirical findings across several measures of alexithymia and combining both clinical and nonclinical samples. Importantly, the fail-safe calculations indicate there would need to be a substantial number (600 or over) *additional* unidentified nonsignificant studies for the mean gender effect size to become nonsignificant.

As expected, the estimated effect size was not large, and the distributions of alexithymia scores in males and female have substantial overlap. Indeed, we expected a relatively small effect size given our theoretical rationale that proposed that only a particular subpopulation of men who received strong traditionally masculine gender role socialization as boys would exhibit symptoms of alexithymia. It should, however, be noted that small effect sizes are not to be ignored (Rosenthal, 1991). Cohen, Cohen, West, and Aiken (2003) caution against the strict application of the labels of "small, medium, large" effect sizes and instead urge interpretation to be based on theoretical assumptions and prior findings.

Status of Evidence for Moderators of the Gender Effect

We also did not find compelling support for moderators of the gender effect size. Neither the comparison of clinical versus nonclinical

samples, nor the type of alexithymia measure used was conclusively shown to make a difference. However, these conclusions should be interpreted cautiously because of the relatively small number of samples available to test them. For example, a limitation of the current meta-analysis is that six of the eight studies identified by Levant et al. (2006) that had to be excluded (because of insufficient statistical information for calculation of the effect size) used clinical populations. This meant that the moderator analysis based on the remaining nine clinical studies for which we were able to calculate effect sizes had low statistical power. Although we did not find significant differences in the mean effect sizes for clinical and nonclinical populations, this is a finding that could easily be revised as more studies of clinical populations are conducted. Indeed, we note that a number of the excluded clinical studies verbally described the gender effect as nonsignificant, as also noted in the earlier narrative review by Levant et al. (2006).

We also add that the designation of a population as "clinical" leaves much room for variability in the characteristics of the respondents. Depending upon the individual study, a clinical sample may include inpatients or outpatients, and the diagnoses of the respondents may vary widely. Furthermore, it may be that some clinical subpopulations (such as patients suffering from PTSD, psychosomatic disorders, or substance abuse) would not be expected to show gender differences in alexithymia, because alexithymia is associated with their psychological disturbance (Sifneos, 1967, 1988; Krystal, 1982).

With respect to the moderator analyses of TAS-based versus non-TAS-based measures of alexithymia, we again note that there is room for revision of the current findings. Although not significantly different, the mean effect size estimate for the non-TAS-based measures was substantially larger than for the TAS-based measures. However, because this result was based on only six studies, we reserve judgment about the meaning of this finding. Interestingly, very recent work on the nature of the alexithymia construct by the authors of the TAS supports the idea that self-report measures of alexithymia such as the TAS tap a dimensional and continuous construct that varies in normal populations (Parker, Keefer, Taylor, & Bagby, 2008) rather than a disorder that is categorical in nature (i.e., present or not). This view is consistent with our findings of reliable but relatively small mean gender differences in normal populations.

We thank an anonymous reviewer for noting that there is a related issue that could be further investigated in studies using the TAS to measure alexithymia (currently the most prevalent method). The TAS has three subscales that were derived using factor analysis: (a) difficulty identifying feelings, defined as "the capacity to identify feelings and to distinguish between feelings and the bodily sensations of emotional arousal;" (b) difficulty describing feelings, "the inability to communicate feelings to others;" and (c) externally oriented thinking, a cognitive style that focuses on the external details of everyday life (Bagby, Parker, & Taylor, 1994, p. 27). Limited reporting of gender differences on the subscales in the studies used in this meta-analysis prohibited an exploration of whether the gender difference held uniformly across the three subscales. However, it could be expected that the various subscales might show gender differences to different degrees. In fact, Levant et al.(2006) theorized that the men who suffer from NMA would not show the *pensee operatoire* cognitive style that focuses on the external details of everyday life, and thus would not likely score higher than women on the externally oriented thinking subscale.

Implications of Findings for Theory

Our original theoretical rationale for investigating gender differences in alexithymia was an expectation that they would exist based on the Normative Male Alexithymia hypothesis. The meta-analytic finding of significantly higher mean levels of alexithymia in men is indeed consistent with the NMA hypothesis. However, it is important to note that this evidence, although supporting the NMA hypothesis and contradicting previous claims of no gender difference, does not constitute definitive proof of the theory. Most importantly, the meta-analysis does not tell us anything about the reasons for the observed gender difference. Gender socialization may indeed be a full or partial cause of the observed gender difference, but it may also be completely attributable to other causes.

Additional evidence outside of this study is necessary to demonstrate the potential links between socialization and the development of higher levels of alexithymia in men. For example, the previously mentioned finding that traditional masculinity ideology accounts for unique variance in alexithymia in men (see Levant et al., 2003) does suggest a potential role for socialization. In addition, as alexithymia in men continues to be studied, findings that the gender effect is malleable or reversible, or that there are cross-cultural differences that map onto different socialization practices, might be useful in arguing against a strict biological explanation for the difference. It is also possible that there might be physiologically based gender differences, perhaps tied to some of the neurological mechanisms mentioned in the introduction, which are then further exaggerated by traditional masculinity socialization processes. Regardless, we again remind readers of the interpretational problems associated with making a causal attribution to gender per se, rather than considering that the gender variable is a proxy for a more psychological factor or process (e.g., Quintana et al., 2001).

However, we feel relatively confident that the gender difference in alexithymia has now been established as a phenomenon worthy of further study, regardless of what the sources of this difference are ultimately found to be. In addition, as we address next, a lack of certainty about the theoretical roots of this difference does not mean that we should wait to explore means of addressing it in a therapeutic setting if it is a cause of distress in men's lives.

Implications of Findings for Clinical and Counseling Practice

Although the study of gender differences is fraught with political and clinical ramifications (e.g., Eagly, 1995), ignoring the potentially detrimental effects of higher levels of alexithymia in men, whether because of traditional male gender role socialization or not, would be a disservice to both these men and the society with which they interact. Our results are consistent with a growing body of literature that speaks to the need for clinicians to attend to men's issues in counseling and to sometimes adjust traditional treatment methods and provide interventions to make psychotherapy more amenable to traditionally socialized men (Good, Thompson, & Brathwaite, 2005; Mahalik, Good, & Engler-Carlson, 2004; Schaub & Williams, 2007). A first step in this process is for clinicians to be aware of the increased potential need to address alexithymia issues in their male clients.

Some helpful tools have recently been developed that may prove useful when men with subclinical levels of alexithymia are considering therapy or have entered into it. For example, Rochlen, Blazina, and Raghunathan (2002) demonstrated how the wording of brochures could work to counter social messages about counseling (i.e., framing help seeking as a brave choice vs. admitting a weakness) and making counseling more attractive. One promising route is to use psychoeducational approaches, particularly in early stages of treatment or as a bridge into traditional counseling. Levant (2006) developed a six-session psychoeducational intervention to raise men's awareness about societal messages regarding emotional expression and help men work to become better able to navigate their emotional lives. This intervention, referred to as Alexithymia Reduction Treatment, has recently been manualized in both group and individual therapy formats (Levant, Halter, Hayden, & Williams, 2009). This intervention or a similar approach could be used as a preparatory therapy to help traditionally socialized men feel more comfortable with the counseling process and enhance their commitment to therapy (Schaub & Williams, 2007).

In summary, the results of the current study highlight the importance of continuing the development and study of approaches to dealing with issues of emotional awareness and expression in men. To do so is likely in the long run to benefit the well-being of both men and women, as our understanding of the roots of emotional restriction are increased and therapeutic tools for addressing problems around emotional expression are further developed and evaluated.

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