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Effectiveness of Cognitive Rehabilitation Following Acquired Brain Injury: A Meta-Analytic Re-Examination of Cicerone et al.'s (2000, 2005) Systematic Reviews

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The present study provides a meta-analysis of cognitive rehabilitation literature ($K = 115$, $N = 2{,}014$) that was originally reviewed by K. D. Cicerone et al. (2000, 2005) for the purpose of providing evidence-based practice guidelines for persons with acquired brain injury. The analysis yielded a small treatment effect size ($ES = .30$, d_+ statistic) directly attributable to cognitive rehabilitation. A larger treatment effect ($ES = .71$) was found for single-group pretest to posttest outcomes; however, modest improvement was observed for nontreatment control groups as well ($ES = .41$). Correction for this effect, which was not attributable to cognitive treatments, resulted in the small, but significant, overall estimate. Treatment effects were moderated by cognitive domain treated, time postinjury, type of brain injury, and age. The meta-analysis revealed sufficient evidence for the effectiveness of attention training after traumatic brain injury and of language and visuospatial training for aphasia and neglect syndromes after stroke. Results provide important quantitative documentation of effective treatments, complementing recent systematic reviews. Findings also highlight gaps in the scientific evidence supporting cognitive rehabilitation, thereby indicating future research directions.

Keywords: cognitive rehabilitation, neuropsychological rehabilitation, TBI treatment, stroke treatment, meta-analysis

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Rehabilitation of impaired cognitive processes has come to be a standard component of medical care after traumatic brain injury (TBI) or stroke (Mazmanian, Kreutzer, Devany, & Martin, 1993; McCrea et al., 2008). This increase in clinical application of cognitive rehabilitation has been accompanied by a rapidly expanding literature detailing an ever-increasing set of candidate treatments. Cicerone and colleagues have performed the most exhaustive search of the literature to date, finding 655 articles through 1997 and an additional 315 published from 1998 to 2002 (Cicerone et al., 2000, 2005). With so much recent research activity, it is not surprising that numerous reviews, as well as two recent edited volumes, have been published regarding the history, theoretical foundations, range of techniques, and effectiveness of cognitive rehabilitation (Halligan & Wade, 2005; High, Sander, Struchen, & Hart, 2005). However, only two meta-analytic studies have been published that examined the effectiveness of treatments for specific domains of cognitive function, such as language (e.g., Robey, 1998) and attention (Park & Ingles, 2001). The present study attempts to complement and augment the existing systematic reviews by providing a quantitative assessment of cognitive rehabilitation in general, using broad cognitive domains developed by previous review authors to categorize the literature.

Due to the complex and ambiguous nature of the cognitive rehabilitation literature, the National Institutes of Health (NIH) and several professional organizations, in both Europe and the United States, have become involved in synthesizing findings to provide evidence-based practice guidelines to clinicians. In 1992, the Brain Injury-Interdisciplinary Special Interest Group (BI-ISIG) of the American Congress of Rehabilitation Medicine published practice guidelines for rehabilitation after TBI and stroke (Harley et al., 1992). Although these guidelines provided much needed standards of care, they have been criticized as being based more on expert opinion than on empirically demonstrated effectiveness that might better quantify the degree of effectiveness of different treatments (Cappa et al., 2003). A follow-up NIH study panel examined the cognitive rehabilitation literature from 1988 to 1998. The panel expressed concern that interpretation of the scientific record regarding the effectiveness of cognitive rehabilitation was limited by the heterogeneity of participants, as well as the interventions and outcomes that had been studied (Carney, Chestnut, Maynard, Mann, & Hefland, 1999; NIH Consensus Development Panel on Rehabilitation of Persons with Traumatic Brain Injury, 1999).

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More recently published systematic reviews have come to a different conclusion than did the NIH study panel. For example, Sohlberg et al. (2003) and Lincoln, Majid, and Weyman (2000) found evidence supporting the effectiveness of direct attention training after TBI. Furthermore, Jutai et al. (2003) and Pierce and Buxbaum (2002) found evidence supporting the effectiveness of visual scanning techniques for unilateral neglect as well as other treatments for specific visuospatial deficits after stroke. Basso (2005) found too much conflict in the literature to either support or refute the effectiveness of aphasia treatments but found clear evidence for long-term cognitive therapy as an effective strategy transcending particular treatments. Wilson (2005) argued that, in general, there was little evidence in favor of rehabilitation of memory impairments. However, there was evidence for effective treatment of memory disabilities (i.e., performance on everyday tasks that require remembering). This finding highlights an important distinction in the rehabilitation literature between recovery of cognitive function and learning compensatory strategies for coping with chronic cognitive deficits. It is a critical and controversial issue for many researchers and clinicians in the rehabilitation field.

The distinction between impaired cognitive processing (e.g., a reduction in the spatial extent of the "spotlight" of visual attention that leads to a reduced ability to detect objects in the periphery) as opposed to disabilities in performing complex real-world tasks (e.g., driving a car) illustrates the difference in focus that rehabilitative treatments can take. Cognitive rehabilitation typically involves one of these two basic strategies (Park & Ingles, 2001). The first approach attempts to retrain directly those cognitive processes that have been impaired by injury based on the notion that damaged neural circuits can be retrained if they have been partially or substantially spared after injury (Robertson & Murre, 1999). The second approach seeks to develop new compensatory skills to enhance performance on everyday tasks, such as driving or baking a cake. This second method of rehabilitation is based on the assumption that the individual will learn to compensate for deficits with newly learned strategies using retained cognitive skills and functional reorganization of the brain (Backman & Dixon, 1992; Vanderploeg et al., 2006). We take the position that treatments using either approach are appropriate for the present meta-analytic review, as long as the outcome variables are reasonable measures of traditional cognitive domains of function (e.g., memory, attention, visuospatial skills).

The development of evidence-based treatment guidelines has relied on systematic reviews that focus on the methodological rigor of the studies. In this approach, evidence from stronger research designs, referred to as Class I studies, has greater influence on the final recommendations of the reviewer(s). The European Federation of Neurological Societies Task Force on Cognitive Rehabilitation was created in 1999 to evaluate the existing evidence for the clinical effectiveness of, and treatment guidelines for, cognitive rehabilitation in stroke and TBI (Cappa et al., 2003). This systematic review and a subsequent update (Cappa et al., 2005) found limited high-quality evidence (i.e., Class I) supporting some forms of cognitive rehabilitation; specifically, treatments for visual neglect and apraxia after stroke, impairments of attention after TBI, and memory dysfunction after either TBI or stroke. However, the authors noted the low number of randomized controlled trials (RCTs) that used pre- and posttreatment assessments on experimental and control groups and expressed reservations about having to rely on a relatively large number of single-case studies. Strong concern was also expressed about the wide variety of outcome measures used across studies. The authors stressed a need to better characterize patient samples for both TBI and stroke patients.

The BI-ISIG has updated its practice guidelines with an emphasis on evidence-based treatments published by Cicerone et al. (2000). A follow-up set of practice guidelines was subsequently published by Cicerone et al. (2005). Cicerone et al.'s (2000) initial review concluded that there was strong evidence for the effectiveness of treatments for language and visuospatial perception after stroke and of attention, memory, functional communication, and executive functioning after TBI. Cicerone et al.'s (2005) update, based on 5 additional years of research (i.e., 1998 to 2002), noted continuing evidence supporting the effectiveness of language treatments for aphasia after stroke and new evidence supporting the effectiveness of apraxia treatments after stroke. In agreement with Cappa et al. (2003, 2005), the latest BI-ISIG update (Cicerone et al., 2005) singled out visual neglect treatments after stroke in particular as being strongly supported. They also suggested that memory dysfunction after TBI is amenable to strategy training to remediate the functional disabilities associated with everyday remembering (e.g., use of external reminders). The latest update further reiterated their earlier conclusion that attention impairments could be effectively remediated.

The study groups of Cappa et al. (2003, 2005) and Cicerone et al. (2000, 2005) used systematic review procedures to analyze the literature. These evidence-based reviews can make important contributions to an understanding of how particular treatments might or might not be efficacious across a diverse set of literatures. Systematic reviews can present preliminary findings and conclusions using a wider variety of literature covering many different research designs, including single-case designs and single-group designs. On the other hand, meta-analyses are limited with respect to the literature included, because each included study must present group data for effect size (*ES*) generation, which typically comes from more mature lines of investigation. Thus, a disadvantage of meta-analysis is that a large number of studies using single-case designs cannot be incorporated. Although many of these excluded studies are less rigorous, they may still provide an initial foundation for future more rigorous research to demonstrate the safety and feasibility of alternative treatments.

On the other hand, the meta-analytic approach to research synthesis has become increasingly common in neuropsychology (Demakis, 2006). Meta-analysis is a statistically rigorous set of methods for integrating results across studies that is often viewed as an alternative to the qualitative review process but that has great potential to augment and complement qualitative reviews of a scientific literature (Cooper & Hedges, 1994). Indeed, meta-analysis is particularly useful when conflicting findings from studies of differing methodological rigor are synthesized. Moreover, meta-analysis provides tools for testing hypotheses regarding heterogeneity of effects when there is concern about the variety of individuals, treatments, and outcomes used across a range of studies. In short, meta-analysis can be helpful by providing a summary of a pattern of objective observations.

Unlike systematic reviews, meta-analysis requires that *ES* be generated, which is the basic unit of analysis. In our case, we were interested in estimating the magnitude of the true effect of rehabilitation upon cognitive functioning and comparing and combin-

ing *ES*s across studies. Not only can *ES*s be combined into a single estimate, but patient and study variables can be evaluated to determine if *ES*s are influenced (i.e., moderated) by other variables, an option that is unavailable to systematic reviewers. The present meta-analysis will attempt to identify aspects of study design and patient characteristics that moderate the effect of cognitive treatment. To date, there have been no meta-analytic studies comparable in scope to the broad systematic reviews of Cicerone et al. (2000, 2005). Robey (1998) performed a meta-analytic review of aphasia treatments after stroke, and Park and Ingles (2001) performed a meta-analytic review of attention treatments after TBI.

The main goal of the present study is to provide a meta-analytic review of the literature on cognitive treatments that had previously been reviewed by Cicerone et al. (2000, 2005). Our approach will allow for an evaluation of the statistical support for the effectiveness of cognitive rehabilitation on overall cognitive function, as well as an evaluation of the effectiveness of classes of treatment for more focal impairments within cognitive domains, which may lead to domain-specific improvements in outcome variables. Because meta-analysis provides methods for synthesizing and quantifying results across designs that differ in methodological details, we hope to augment the systematic reviews of Cicerone et al. (2000, 2005) and Cappa et al. (2003, 2005) that were used to develop practice guidelines for cognitive rehabilitation after TBI and stroke.

Method

Sample of Studies

The present study is a meta-analytic review of the same set of studies of cognitive treatment effectiveness that had previously been systematically reviewed by Cicerone and colleagues (Cicerone et al., 2000, 2005). First, studies of cognitive rehabilitation after TBI or stroke indexed by the MEDLINE electronic database through 1997 (Cicerone et al., 2000) and indexed by PubMed and Infotrieve from 1998 to 2002 (Cicerone et al., 2005) were identified. Detailed description of the method of selection and exclusion are reported in the Cicerone et al. articles. Briefly, studies were selected from electronic search engines using the following keywords: attention, awareness, cognition, communication, executive, language, memory, perception, problem solving, reasoning, rehabilitation, remediation, and training. Additional studies were identified by consulting the reference lists of the articles identified and from additional articles known to the committee members involved in the review process. The result was a sample of 967 articles published in or prior to 2002. The authors then applied the following 10 exclusionary criteria: (1) articles not addressing intervention, (2) theoretical articles or descriptions of treatment approaches, (3) review articles, (4) articles without adequate specification of interventions, (5) articles that did not include participants primarily with a diagnosis of TBI or stroke, (6) studies of pediatric patients (7) single-case reports without empirical data, (8) non–peer-reviewed articles and book chapters, (9) articles describing pharmacologic interventions, and (10) articles not written in English. The result was a sample of 258 articles retained for further review that had been systematically reviewed by either Cicerone et al. (2000) or Cicerone et al. (2005).

For the present study, we applied additional exclusionary criteria to the Cicerone et al. (2000, 2005) sample of 258 articles to obtain a reduced sample of studies appropriate for meta-analysis. Note that we use the term *study* to refer to a separately sampled group and that some included articles involved multiple studies. We screened outcome variables measuring motor deficits (i.e., apraxia), emotionality (e.g., depression, anxiety, or irritability), social interactions (e.g., marital status or social skills), or difficult to define measures of real-world function (e.g., employment or measures of self-sufficiency). We also removed single-case studies or multiple-case studies with $N < 3$ (110 studies removed), as meta-analytic methods require estimates of variability that are not available when the study sample size is less than 4. Finally, we removed 41 studies due to insufficient data for the coding of an aggregate *ES*. To be included, a study had to provide sufficient statistical information to estimate an *ES* expressed as a standardized mean difference (Hedges & Olkin, 1985).

This process yielded 101 articles with 119 distinct studies (i.e., presented as separate experiments in the published article, with unique nonoverlapping samples of participants) comprised of 119 treatment samples ($N = 2{,}014$) and 47 distinct control (i.e., nontreatment) samples ($N = 870$). This sample of studies included 72 single-group pre-post (SGPP) designs (i.e., included a single sample of patients who were assessed pretreatment and posttreatment). Nonrandomized studies that reported multiple treatment groups within a single manuscript (e.g., Rattock et al., 1992) were also considered SGPP designs, with each treatment group analyzed separately. There were also 47 independent groups pre-post (IGPP) designs (i.e., patients were randomly assigned to treatment and control groups and assessed pre- and posttreatment), but there were no posttest-only designs. The average number of participants in the typical treatment study was 16.9, and the average number of participants in the typical control sample was 18.5.

An outlier analysis resulted in removal of four studies. Two of these studies used SGPP designs, and the other two used IGPP designs. The *ES*s for the treatment groups in these studies were more than 2.5 *SD* above the overall unweighted mean *ES* in the distribution of unweighted treatment group *ES*s. As a result, they were deemed outliers using criteria developed by Huffcutt and Arthur (1995). The final sample of studies included 97 articles reporting on 115 studies that included 115 distinct treatment (70 SGPP and 45 IGPP) and 45 distinct control samples.

The 45 control samples used the same dependent variables (DVs) as their matching treatment groups within each IGPP study. Each of the 115 sampled treatment groups were evaluated on 1 to 42 DVs, and the set of DVs used varied greatly from study to study. A total of 233 distinct DVs were used 1 to 14 times each across the 115 treatment samples, resulting in 980 nonunique uses of the DVs ($M = 8.5$ DVs per treatment sample). Specifically, 90% of the DVs were used four times or less across the set of 115 treatment samples, with only 10 (of 233) DVs being used for 9 to 14 treatment samples.

Study Coding Reliability

Of the sample of studies coded, 20% were coded by at least two coders for the purposes of calculating interrater reliability. The

entire sample of studies was coded by one of the four coauthors involved in the study, with the lowest coder accounting for the coding of 16% of the studies and the highest coder accounting for 33% of the studies. Interrater reliability was calculated on a variety of moderator and demographic variables. Specifically, the coding of two researchers on each of the following variables were cross-checked: (a) data/no data, (b) etiology or type of lesion (i.e., TBI or stroke), (c) recovery level, (d) chronicity, (e) number of treatment groups, (f) sample size of treatment group, (g) number of control groups, (h) sample size of control group, (i) number of coded DVs, (j) *ES* method of calculation, and (k) mean *ES* per sample. In summary, there were 253 coded variables checked by two coders each, for 506 data points. Seven outliers were excluded from the final analyses. Results were all generated in the form of a correlation coefficient (e.g., Pearson's *r*, phi coefficient, or Cramer's *V*). These coefficients ranged from .77 to 1.00. After each coefficient was converted with the Fisher *z* transformation, the mean coefficient for these 11 critical variables was .96. More specifically, the Pearson *r* for the overall *ES* per study was .98. The corresponding author (M.L.R) ultimately adjudicated all disagreements to adjust *ES* difference among coders. All articles included in the Cicerone et al. (2000, 2005) reviews were checked against our additional meta-analytic exclusionary criteria, the basic design was classified, the sufficiency of reported statistics for determination or estimation of an *ES* was judged, general study information was recorded, and values for potential moderator variables were coded.

Moderators

We focused on a specific set of potential moderator variables suggested by the Cicerone et al. (2000, 2005) and Cappa et al. (2003, 2005) reviews, as well as by our reading of the literature. Table 1 presents the distribution of studies across levels of several qualitative candidate moderator variables, as well as descriptive statistics for quantitative candidate moderator variables. Table 2 presents the cross-tabulation of the remaining qualitative candidate variables. We included study design variables (i.e., study class as used by Cicerone et al. and study design), treatment variables (i.e., cognitive domain, duration of treatment), and participant variables (age, etiology, and time since injury).

Table 1
*Descriptive Statistics for Candidate Moderator Variables for Study Sample (*K = *115)*

Moderator variable	Count	Percent
Study class		
I	30	26.1
II	35	30.4
III	50	44.5
Study design		
Single-group pre-post	70	60.9
Independent groups pre-post	45	39.1
Independent groups posttest only	0	0
	M	*SD*
Age by etiology		
Stroke treatment group	59.4	7.7
Mixed treatment group	37.4	9.2
TBI treatment group	29.1	15.9
Treatment duration (weeks)	13.3	14.2

Note. TBI = traumatic brain injury.

A major focus of the Cicerone et al. (2000, 2005) systematic reviews was to develop evidence-based practice guidelines for cognitive rehabilitation. A fundamental aspect of this approach is the idea that not all scientific evidence is of equal value. Specifically, evidence from higher quality or better controlled research designs should be given greater weight in the synthesis (Woolf, 1992). As is common in evidence-based systematic reviews, Cicerone et al. (2000) defined three classes of studies (i.e., identified as I, II, and III) in descending order of quality. Class I studies "had well designed, prospective, randomized controlled trials." Class II studies "consisted of prospective, nonrandomized cohort studies" or "retrospective, nonrandomized case-control studies." Class III studies included "clinical series without concurrent controls, or studies with results from one or more single cases that used appropriate single-subject methods" (p. 1598). As shown in Table 1, even after a large number of single-case studies were removed, 44% of the remaining studies were coded as Class III studies.

Studies of treatment effectiveness typically included in meta-analytic reviews fall into one of three major research designs, independent groups posttest (IGP) only, SGPP designs, and IGPP designs. Because the present study provides another way to categorize study quality, we included study design in our moderator analysis.

A second focus of the Cicerone et al. (2000, 2005) studies was the idea that cognitive rehabilitation treatments are designed to address deficits from one of several broad domains of cognitive functioning. In their first review, these researchers identified seven treatment domains: attention, visual perception and constructional abilities, language and communication, memory, problem solving and executive functioning, multimodal interventions, and comprehensive-holistic cognitive rehabilitation. For their 2005 review, they combined the comprehensive-holistic and multimodal categories into a single comprehensive category and added apraxia. Because we did not deem apraxia rehabilitation similar enough to other domains of cognition for inclusion, and because the first review by Cicerone et al. (2000) did not include this domain, we chose not to include apraxia studies in the present meta-analysis. We also chose to combine the attention and executive function domains, as we only identified four executive function rehabilitation studies that met our inclusion criteria. This latter decision seemed consistent with what has been suggested in the literature, in that measures of attention are often similar enough to measures of executive function that they may represent a single domain (Bowden et al., 1998; Clark & O'Carroll, 1998; Demakis, 2004). The result was a treatment domain variable that included the following five broad cognitive domains: (a) attention/executive, (b) visuospatial, (c) language, (d) memory, and (e) comprehensive.

Research on cognitive rehabilitation after acquired brain injury has focused primarily on stroke and TBI (Cappa et al., 2003). These two etiologies are likely to lead to different patterns of cognitive impairments that may be differentially receptive to cognitive rehabilitation during various periods of postinjury treatments (Halligan & Wade, 2005; High et al., 2005). As a result, we chose to evaluate etiology (i.e., stroke vs. TBI) and recovery level (i.e., $\leq$1 year vs. $>$1 year) as potential moderators. As can be seen in Tables 1 and 2, this may prove to be difficult considering the

Table 2
Count (Percent) of Studies by Treatment Domain, Etiology, and Recovery Level

		Etiology				Recovery			
Treatment domain	*K*	Stroke	Mixed	TBI	NR	<1 yr	Mixed	>1 yr	NR
Attention/executive	14	1 (7)	2 (14)	11 (79)	0	2 (14)	1 (7)	9 (65)	2 (14)
Visuospatial	29	23 (80)	3 (10)	2 (7)	1 (3)	13 (45)	3 (10)	2 (7)	11 (38)
Language	34	30 (88)	0	4 (12)	0	7 (21)	13 (38)	14 (41)	0
Memory	14	0	6 (43)	8 (57)	0	1 (7)	1 (7)	10 (71)	2 (14)
Comprehensive	24	0	7 (29)	16 (67)	1 (4)	4 (17)	1 (4)	18 (75)	1 (4)
Total	115	54 (47)	18 (16)	41 (36)	2 (2)	27	19	53	16

Note. TBI = traumatic brain injury; NR = not reported in study.

obvious overlap between etiology and these other potential moderator variables, such as age, time since injury, and the cognitive domain for which treatment is the focus.

The age of a patient may also influence the plasticity of the nervous system in terms of recovery and reorganization of function after brain injury (e.g., Disterhoft & Oh, 2006). Therefore, we examined the influence of mean age of patient samples in moderating treatment *ES*s. However, mean age distributions for stroke and TBI studies were completely nonoverlapping. Therefore, identification of unique or independent contributions of these variables to cognitive rehabilitation *ES* was not possible.

Statistical Analysis

Raw ES estimates. The *ES* is the basic unit of analysis in meta-analysis; however, choices must be made early in the process of planning a meta-analysis because there are two basic families of *ES*s, each of which have become associated with a distinct set of statistical procedures. One can quantify an *ES* using correlations, expressing the *ES* as the Pearson product–moment correlation *r* (Rosenthal, 1994), between scores on the DV and treatment and control groups. The other approach quantifies the *ES* as a standardized difference between means, Hedge's *g* (Hedges & Olkin, 1985). Most of our methodology follows from the work of Hedges and colleagues. In particular, we used the variant of this approach that was advanced by Morris and Deshon (2002), which focuses on *ES*s for pre-post designs.

The appropriate *g* variant *ES* for the SGPP design is based on the difference between the pre- and posttest means for the single treatment group, divided by the *SD* of the pretest scores (Morris & Deshon, 2002). Equation 1 presents the basic single-group pre-post *ES* (SGPP-*ES*). It is worth noting that because the denominator of Equation 1 does not include the *SD* of pre-post change scores, this is not a change score *ES* expressed in change score units. Rather, our *ES* uses the *SD* of raw pretest scores and, as such, is a traditional raw score *ES* (Dunlap, Cortina, Vaslow, & Burke, 1996).

$$\text{SGPP-}ES = (\text{Mean}_{\text{Post}} - \text{Mean}_{\text{Pre}})/SD_{\text{Pre}} \quad (1)$$

The appropriate *ES* for an IGPP design (Becker, 1988) is simply the SGPP-*ES* for the treatment group with the SGPP-*ES* for the control group subtracted out. The independent group pre-post *ES* (IGPP-*ES*) is presented in Equation 2.

$$\text{IGPP-}ES = [(\text{Mean}_{\text{Expt,Post}} - \text{Mean}_{\text{Expt,Pre}})/SD_{\text{Expt,Pre}}] - [(\text{Mean}_{\text{Con,Post}} - \text{Mean}_{\text{Con,Pre}})/SD_{\text{Con,Pre}}] \quad (2)$$

The *ES*s presented in Equations 1 and 2 require reporting of the pre- and posttreatment mean (or the equivalent mean change score), as well as the pretreatment *SD* for each group. The majority of studies in the sample met this standard and the raw score metric *ES*s (Equations 1 and 2) were directly computed for each DV. When this information was not reported, but adequate information to compute a change score *ES* was reported (i.e., mean and *SD* change scores, repeated measures *t* or *F*), we computed a change score *ES* and used typical equations (Morris & Deshon, 2002) to convert the change score *ES* into a raw score equivalent (same as Equation 1). In our final sample of 115 studies, *ES*s were computed directly (Equations 1 and 2) for 73% (51 of 70) of the SGPP design studies and 69% (31 of 45) of the IGPP design studies. The remaining studies had *ES*s first computed in change score metric and then converted to the raw score metric of Equation 1.

Statistical model. Standard meta-analytic techniques require a set of statistically independent *ES*s. Specifically, each sample is allowed to contribute only one *ES* to an analysis, regardless of how many DVs have been included in the original study design. Therefore, it is often necessary to combine *ES*s when multiple DVs are included (Rosenthal & Rubin, 1986). We chose to use the simple method of taking the arithmetic mean of *ES*s for all DVs within a study. Because the *ES*s, which are variants of *g*, expressed in Equations 1 and 2 are somewhat biased, we used the transformation to an unbiased *ES* estimator (i.e., *d*) suggested by Hedges and Olkin (1985). Finally, aggregate mean *ES*s were computed using a weighted means procedure to calculate the d_+ statistic (Hedges & Olkin, 1985), in which each *ES* is weighted by the inverse of its sampling variance. This results in *ES*s that are estimated with more certainty (i.e., have a smaller sampling variance), thus they will hold greater weight in determining the final resultant weighted mean *ES*.

The weighted mean *ES* for each study can be seen as estimates of the population mean *ES* for exact replications of that study. The variation in study context and design, including the variation in the unique combination of DVs and the variation in demographics and brain injury characteristics of participants, can be seen as adding random variation to the true *ES* across studies. This situation calls for use of random-effects models to estimate *ES*s (Hedges & Olkin, 1985; Hedges & Vevea, 1998; Raudenbush, 1994). Such models estimate the *ES* for each study as an additive combination

of a fixed parameter (i.e., true *ES*) and a random study parameter that estimates the random fluctuations in the true *ES* from study to study due to design variations.

We used restricted maximum likelihood to estimate random-effects models that yielded several statistics of interest for each subanalysis. A Q statistic, with an approximately chi-square distribution, with $k - 1$ degrees of freedom (k = number of independent *ES*s) is used to test the homogeneity null hypothesis that all cognitive treatments are producing the same true *ES* regardless of variation in study designs. A significant result indicates that the random-effects parameter that estimates random variation due to varying design is significantly different from zero. In other words, there is statistically significant variation in the true *ES*s in the set of studies analyzed that may be accounted for by adding a random study effect parameter to the model. Fixed-effect moderator variables may also account for significant heterogeneity of *ES*s, or a combined mixed model may be necessary to fit fully the data. The random-effects fitting procedure used also yielded a mean *ES*, a standard error (*SE*) of the mean, and a *z* statistic and probability value for a test of the null hypothesis that the population's mean *ES* equals zero. This information was used to create 95% confidence intervals (CIs) for the mean *ES*s. Finally, the random-effects model allowed for an estimate of the random study variance parameter and its *SE*.

Synthesizing ESs across designs. Because the IGPP-*ES* results from an IGPP design, the IGPP-*ES* provides an estimate of the true treatment *ES* with the control groups' spontaneous recovery and practice effects subtracted out. We refer to these as *retest effects*. Specifically, the retest effect is estimated using the controls' SGPP-*ES* calculation, based on the pretest scores (Time 1) and posttest (Time 2) scores. These score differences within control groups cannot logically be related to any specific treatment that was administered to the experimental group. The SGPP-*ES*, being estimated from a treatment group only, is clearly biased in the presence of retest effects (i.e., when there was no control used in the study design). To determine whether it was reasonable to assume a lack of retest effects, we analyzed control groups from the IGPP studies as if they came from their own SGPP study. In particular, we computed SGPP-*ES*s for the control groups and submitted them to random-effects modeling.

As will be discussed in the Results section, our finding of significant retest effects in the control groups resulted in the need to adjust the treatment group *ES*s from the SGPP designs. We used a simple subtractive method (Becker, 1988; Morris & Deshon, 2002). In this method, an SGPP-*ES* (Equation 1) is computed for each control group and for each treatment group, regardless of design. In essence, because of the assumption of random assignment to treatment and control group designs, each group's *ES* can be analyzed as if it was the only group in the design (i.e., statistically independent *ES*s). A random-effects model was then used to estimate the weighted mean SGPP-*ES* for the control groups.

When a moderator variable was included, then the weighted mean SGPP-*ES* for each level of the moderator variable (e.g., treatment domains) was estimated for the control groups. The control groups' mean SGPP-*ES*s were then either subtracted from similarly estimated weighted mean SGPP-*ES*s for the treatment groups (i.e., including all 115 treatment groups regardless of design) or subtracted from each treatment group's SGPP-*ES* prior to model fitting. The latter approach has the advantage of fitting the variability of the adjusted treatment group's *ES*s, because the adjustment is performed prior to model fitting and allows a wider range of inferential tests on the adjusted treatment *ES*s. Subtracting control means after model fitting of unadjusted treatment SGPP-*ES*s allows more limited inferential tests but requires fewer statistical assumptions. Because adjusting SGPP-*ES*s depends on the quality of control group data and assumptions about distortions of *ES* independence, we also analyzed the IGPP-*ES*s from the subset of 45 IGPP studies. Although limited in number, this subset of studies provides the highest quality data for quantitative meta-analysis and provides critical comparisons for the full analyses of the 115 treatment groups' adjusted mean SGPP-*ES*s.

Cognitive Domain of Outcome Measures

We conducted analyses on the mean *ES* of subsets of DVs grouped by cognitive domain. We used a standard neuropsychology assessment handbook (Lezak, Howieson, Loring, Hannay, & Fischer, 2004) as well as the results of a recent factor analytic study of neuropsychological tests (Tulsky & Price, 2003), commonly used to diagnose and document cognitive deficits, to categorize each DV by cognitive domain. Each DV was categorized as belonging to one of the following 12 cognitive functions: attention, auditory memory, executive function, general miscellaneous, language, learning, perceptual organization, processing speed, sensory perceptual, verbal comprehension, visual memory, and working memory. Finally, we mapped each of the 12 cognitive functions onto the following five broad cognitive treatment domains (see *Treatment Group Analysis* subsection): (a) attention/executive, (b) visuospatial, (c) language, (d) memory, and (e) comprehensive.

The cognitive function subcategories of attention and executive function were classified into the attention/executive treatment domain. The cognitive function subcategories of perceptual organization and sensory perceptual were classified into the visuospatial treatment domain. The cognitive function subcategories of verbal comprehension and language/aphasia were classified into the language treatment domain. The cognitive function subcategories of learning, visual memory, auditory memory, and working memory were classified into the memory treatment domain. Finally, the cognitive function subcategories of processing speed, general, and miscellaneous were classified into the comprehensive treatment domain. By creating subsets of DVs based on cognitive domain, we were able to test hypotheses regarding the specificity of treatments from each cognitive domain. A treatment can be said to be specific to the extent that it improves cognitive functions in its own cognitive domain to a greater extent than for other cognitive domains.

Results

Study, Treatment, and Participant Variables

Table 1 presents the distribution of studies across levels of study class (I, II, or III) and study design (IGPP, SGPP, and IGP only). Of interest is the fact that 44% of our sample was comprised of Class III studies, and a full 61% of the studies were SGPP designs that did not include controls. Reliance on SGPP designs assumes that there are no pre-post retest effects (e.g., practice effects or spontaneous improvement of cognitive function from pre- to posttest). To preview, our results (see Table 3) will document wide-

Table 3
Statistics of Interest for Analyses of Single-Group Pre-Post Effect Sizes (SGPP-ESs) by Treatment Domain, Etiology, and Recovery Level[a]

	Control groups			Treatment group statistics (adjusted by control group means)							
							95% CI				
	K	*M ES*	*SE*	*K*	Adj *M ES*	*SE*	Lower	Upper	*z*	Q	σ^2_θ (*SE*)
Treatment domain											
Overall	45	.41	.05	115	.30	.04	.22	.37	7.43**	108.83	.12 (.02)
Attention/executive	9	.39	.11	14	.27	.12	.04	.50	2.34*	12.18	
Visuospatial	13	.23	.08	29	.54	.08	.39	.69	6.99**	28.56	
Language	12	.53	.08	34	.18	.08	.03	.33	2.39*	30.20	
Memory	5	.21	.15	14	.61	.12	.37	.85	4.91**	15.55	
Comprehensive	6	.68	.12	24	.03	.08	−.13	.19	0.36	22.32	
Etiology											
Overall	40	.42	.06	95	.27	.04	.18	.35	6.37**	91.17	.11 (.02)
Stroke	24	.36	.07	54	.40	.05	.29	.50	7.23**	58.61	
TBI	16	.52	.09	41	.09	.06	−.04	.21	1.36	32.57	
Recovery level											
Overall	31	.42	.06	80	.25	.05	.16	.35	5.44**	73.51	.12 (.03)
<1-yr postinjury	18	.39	.08	27	.43	.08	.28	.59	5.63**	31.58	
>1-yr postinjury	13	.46	.10	53	.15	.06	.03	.26	2.55*	41.93	

Note. TBI = traumatic brain injury.
[a] Analysis of treatment group SGPP-*ES*s for all studies, with control group means from independent groups pre-post (IGPP) studies subtracted out.
* $p < .05$. ** $p < .01$.

spread retest effects among control groups that comprise roughly half the *ES* observed in SGPP studies using a treatment group only. This finding raises concern about the potential bias that might be introduced by an overreliance on the SGPP designs in the cognitive rehabilitation literature.

Table 1 also presents the mean age of participants in studies using either pure stroke or TBI groups or a mixed etiology sample. Participants in TBI groups averaged about 29 years of age, whereas participants in stroke groups averaged about 59 years of age. In fact, we examined the distributions of mean age of participants for stroke and TBI studies and found no overlap. For the pure etiology studies, mean age for participants and etiology (stroke vs. TBI) were perfectly correlated or confounded in the literature. The implication for the search for variables that moderate cognitive rehabilitation *ES*s will be that either both or neither of these two variables will be found to be moderators.

Table 2 presents two other confounds involving treatment domain with both etiology and recovery level. Across the sampled literature, attention treatments were tested predominantly on TBI patients, visuospatial and language treatments were tested predominantly on stroke patients, and memory and comprehensive treatments were tested on a balanced sampling of mixed patients or TBI groups. Because of this correspondence, etiology and treatment domain likely account for highly overlapping portions of the variability in *ES*s, and so identification of independent contributions to *ES*s for these two variables will be hampered. A similar problem occurs with the pattern of recovery level and treatment domain. In fact, all three variables are highly confounded, with stroke patients less than 1-year postinjury and TBI patients more than 1-year postinjury being studied most often.

Global Treatment Effects

Our first set of analyses examined the effect of cognitive rehabilitation on global cognitive function. For these analyses, a global *ES* was computed for each study by taking the unweighted means of the *ES*s across all DVs included in that study. We first computed an SGPP-*ES* for each control group (i.e., from IGPP studies), treating them as if they came from a single-group design. This analysis allowed us to see whether retest effects were a likely source of bias for the SGPP-*ES*s for the treatment groups from the SGPP studies. Finding significant *ES*s in control groups would mean SGPP-*ES*s for treatment groups would need to be adjusted by subtracting the control group estimates. This approach also assumes that SGPP-*ES*s from treatment groups from SGPP designs do not differ from SGPP-*ES*s from treatment groups from IGPP designs.

Control group analysis. Significant SGPP-*ES*s in the control groups would indicate testing effects (e.g., learning, and/or spontaneous recovery of function from pre- to posttest). The fixed-effects homogeneity test revealed significant deviation from homogeneity, $Q(44) = 167.47$, $p < .001$, supporting adoption of a random-effects model and/or a fixed-effects model with a moderator variable. As shown in the left portion of Table 3 and in Figure 1, the random-effects mean *ES* ($M = .41$, $SE = .05$) was significantly different from zero, $z = 7.41$, $p < .001$, with a 95% CI of .29 to .51, indicating a small-to-medium *ES* for control groups for the global *ES* across the full range of outcome variables. This significant SGPP-*ES* for control groups greatly complicated our analysis due to the large (61%) number of SGPP treatment studies in our sample. SGPP-*ES*s from treatment groups in these studies must now be assumed to be positively biased.

Our sample of 45 IGPP studies included a range of control groups. Consider, for example, Salazar et al. (2000) who compared an intensive inpatient cognitive treatment regimen to the alternative of sending patients home with instructions to play card games, read magazines, watch TV, and adopt a program of regular physical exercise. The group sent home falls somewhere between a no-treatment control group and a specifically designed sham cognitive control

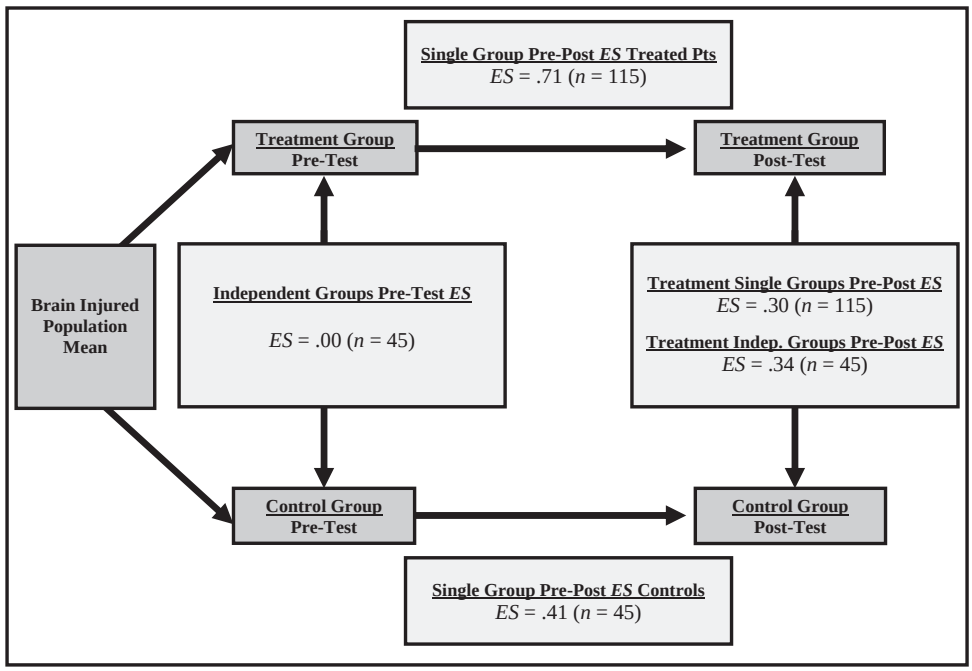

Figure 1. Graphic illustration of study designs for meta-analysis of cognitive rehabilitation.

group and is an example of the heterogeneity of control groups in this literature. We examined the full range of the control groups in our sample of 45 IGPP studies and coded all 45 control groups into one of four broad categories. First were the no-treatment groups ($n = 21$ or 47%), which involved no professional treatment of any type or may have been a wait-list control. Second were the standard treatment groups ($n = 6$ or 13%), which involved traditional medical care by medical staff, nursing, and recreational therapies. Third were the attention/placebo groups ($n = 12$ or 27%), which might involve passive concentration games or support group discussions about cognitive problems. Finally, there were sham cognitive control group ($n = 6$ or 13%), which might involve training in memory or motor skills when the treatment group was receiving attention training in an attempt to improve patients' attentional skills. Analyses of these four types of control groups revealed no significant differences among them on either the unweighted retest effect, $F(3, 41) = .86$, $p = .47$, or the treatment effect, $F(3, 41) = 1.01$, $p = .40$. Across all the cognitive outcome measures, the unweighted mean *ES* for each type of control groups ranged from a low of .16 (standard) to a high of .45 (placebo).

Some may disagree with our assignment of the category of cognitive sham control group in some situations, for example, when the sham treatment was actually another treatment in the same cognitive domain. However, when the motivation for the treatment in question presented in the target article indicated inclusion because it was predicted to be less, or likely not at all, efficacious than the experimental treatment (e.g., Kaschel et al., 2002), the treatment was coded as a sham cognitive control. This choice was difficult, but consensus of the raters was reached in all cases. In cases where the decision went the other way, that is, the cognitive treatment was not deemed as sham, we treated the group as an additional experimental treatment group in a separate SGPP. We could have excluded these studies altogether, but we considered that the least preferred option, and our statistical analysis of the control groups' *ES*s supports this choice.

From our analyses of the control groups' data, we were able to generate a global treatment *ES* for each of the 115 included studies, which is illustrated in Figure 1. The unweighted *ES*s distribution is presented in Figure 2 as a modified stem-and-leaf plot. For studies in which a control group did not exist, we subtracted an estimated retest effect generated from the IGPP studies of .41 from each treatment SGPP-*ES*. Descriptive statistics for unweighted treatment *ES*s are presented in Table 4, which details both measures of central tendency and variability.

We estimated random-effects models for the set of moderator variables of interest listed in the study coding subsection for the control groups. The mean SGPP-*ES*s for control participants varied significantly across cognitive treatment domain, $Q(4) = 13.62$, $p = .009$, for the attention ($M = .39$), visuospatial ($M = .23$), language ($M = .53$), memory ($M = .21$) and comprehensive ($M = .68$) domains (see left portion of Table 3). Because no treatment was actually applied to these control groups, this result must be due to differences in DVs or patients across cognitive domains. The random-effects variance was estimated at $v = .052$ with a standard error of .019, and the test of homogeneity with the random-effects parameter set to zero was significant, $Q(40) = 110.44$, $p < .001$, supporting the random-effects model. Moreover, the homogeneity test for only the fixed-effects (i.e., treatment domain levels) portion of the model was not significant, $Q(40) = 38.18$, $p = .55$, indicating a lack of evidence for continued heterogeneity of *ES* once the fixed treatment domain and random study effects had been taken into account. In addition, a separate regression analysis revealed that mean age of study participant was a significant moderator of the retest effect,

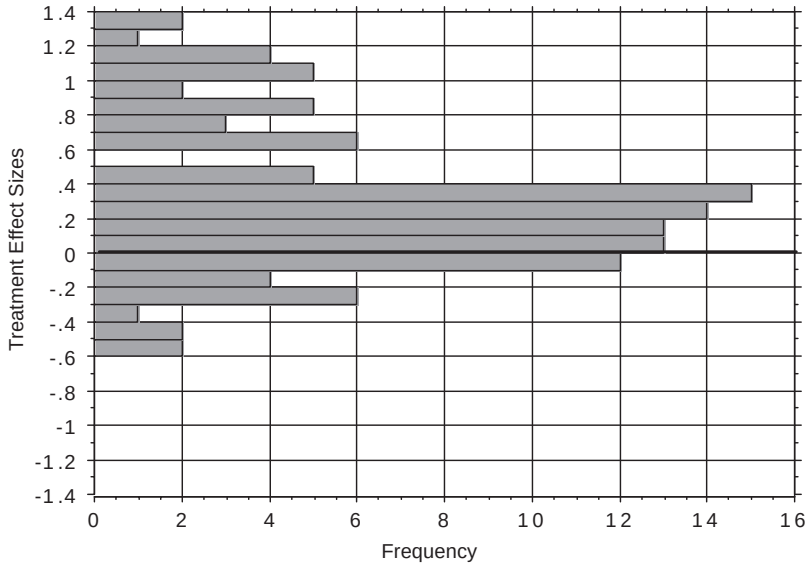

Figure 2. Modified stem-and-leaf plot of estimated treatment effect sizes (*ES*s) for all 115 included cognitive rehabilitation studies.

Q(1) = 4.29, $p = .038$, with older age being correlated with smaller SGPP-*ES*s for control groups.

None of the other variables of interest, study class (I, II, and III), etiology (TBI vs. stroke), treatment duration, and recovery stage ($\leq$1 year vs. $>$1 year) were significant moderators of SGPP-*ES* for control groups (all *p*s $> .26$). Because of the significant mean SGPP-*ES*s for the control groups, and the significant moderation of these effects, the SGPP-*ES*s for the treatment groups must be adjusted to remove the retest effects from the treatment groups' SGPP-*ES*s in the SGPP-*ES* treatment studies (see Figure 1 for a graphic illustration of this relationship). The estimation of a modest overall *ES* in the .50 range strongly suggests that SGPP-*ES*s will be greatly biased upward by testing effects among the treatment groups in these studies.

Treatment group analysis. The SGPP-*ES*s for each treatment group, regardless of study design (SGPP and IGPP), were submitted to a random-effects model with a fixed-candidate moderator variable, after first being adjusted by subtracting the weighted mean SGPP-*ES* for the control groups using the same model. Table 3 and Figure 1 present the mean *ES* and *SE* of *ES* for the control groups, as well as the adjusted mean *ES*, the *SE*, 95% CI, *z* test for difference from zero, and Q test for homogeneity of *ES* within each level of the moderator variable for the treatment groups. Three significant moderator variables were identified with this procedure. As can be seen in the right portion of Table 3, each of the resultant models estimated a significant overall mean SGPP-*ES* in the .25 to .30 range (all *p*s $<.001$) for the overall cognitive treatment effect on global cognitive functioning.

Table 4
Descriptive Statistics for Unweighted Treatment Effect Sizes, Including Measures of Central Tendency and Variability

Measures of central tendency		Measures of variability	
Number of studies (*K*)	115	*SD*	0.43
Mean	0.31	Median absolute deviation	0.22
Median	0.24	Maximum	1.35
Mode	0.35	Minimum	−0.51
10% trimmed mean	0.28	Range	1.86
		Interquartile Range	0.45
		Skewness	0.62
		Kurtosis	−0.23

The moderator variable of treatment domain was significant, Q(4) = 29.09, $p < .001$, and each cognitive treatment domain produced a significant *ES* (*p*s $< .02$), with the exception of the comprehensive domain ($M = .03$, $p = .72$). The visuospatial and memory treatment domains yielded moderate effects, $M = .54$ and .61, respectively, and the attention/executive and language treatment domains yielded small effects, $M = .27$ and .18, respectively. All tests of homogeneity failed to reach significance, indicating that there was not enough evidence to reject the null hypothesis of equal *ES*s within each level of the moderator variable as well as for the model as a whole. Moreover, the homogeneity test for the fully fixed-effects model (i.e., with the random- effect parameter variability set to zero) was significant, Q(110) = 406.93, $p < .001$, supporting the inclusion of the random-effects parameter in the model.

The moderator variable of etiology was evaluated for the 95 pure etiology studies in our sample (i.e., studies with mixed etiology patients were excluded) and was significant, Q(1) = 13.53, $p < .001$. The stroke groups yielded a significant moderate effect ($M = .40$, $SE = .05$, $p < .001$), but the TBI groups did not ($M = .09$, $SE = .06$, $p = .17$). All tests of homogeneity failed to reach significance, indicating that there was not enough evidence to reject the null hypothesis of equal *ES*s within each level of the moderator variable, as well as for the model as a whole. Moreover, the homogeneity test for the fully fixed-effects model (i.e., with the random-effect parameter

variability set to zero) was significant, Q(93) = 323.06, $p < .001$, supporting the inclusion of the random-effects parameter in the model.

The moderator variable of recovery level was evaluated for the 80 pure recovery level studies in our sample (i.e., studies of mixed recovery level patients were excluded) and was significant, Q(1) = 8.65, $p = .003$. The ≤1-year postinjury groups yielded a significant moderate effect ($M = .43$, $SE = .08$, $p < .001$), whereas the >1-year postinjury groups yielded a small effect ($M = .15$, $SE = .06$, $p = .011$). All tests of homogeneity failed to reach significance, indicating that there was not enough evidence to reject the null hypothesis of equal *ES*s within each level of the moderator variable as well as for the model as a whole. Moreover, the homogeneity test for the fully fixed-effects model (i.e., with the random-effect parameter variability set to zero) was significant, Q(78) = 315.51, $p < .001$, supporting the inclusion of the random-effects parameter in the model.

The variables of study design (IGPP vs. SGPP) and study class (I, II, or III) were not significant moderators of *ES*. Because of the limitation of our procedure for subtracting out retest effects from the treatment effects to models with qualitative moderators, we saved the moderator evaluation of age and treatment duration for the analysis of IGPP-*ES*s.

IGPP analysis. We evaluated the set of potential moderator variables presented in the study coding section by fitting random-effects models to the mean IGPP-*ES* for each study. Study class (I, II, or III), study type (SGPP or IGPP), and treatment duration (weeks) failed to reach significance (i.e., *p*s > .05). Three moderator variables were identified using this procedure (see Table 5). As in the analysis of SGPP-*ES*s, treatment domain significantly moderated mean IGPP-*ES*, Q(4) = 18.75, $p < .001$. Comparison of the mean *ES*s in the SGPP-*ES* analysis (see Table 3) with the IGPP-*ES* analysis (see Table 5) reveals a similar pattern of significant *ES*s across the two analyses. Examination of Table 5 reveals that attention and language treatments yielded small effects, $M = .27$ and .32, respectively (*p*s < .026), and visuospatial rehabilitation treatment yielded a moderate effect ($M = .62$, $p < .001$). However, memory and comprehensive rehabilitation did not result in significant treatment effects (*p*s > .30). The discrepancy for estimates of the memory treatment *ES* for global cognitive functioning not withstanding, the analyses of SGPP-*ES*s and IGPP-*ES*s are otherwise in strong agreement and provide a consistent picture regarding cognitive rehabilitation effects on global cognitive functioning.

Etiology significantly moderated mean IGPP-*ES*, Q(1) = 13.14, $p < .001$. Treatment yielded a moderate effect for stroke ($M = .48$, $p < .001$). TBI groups did not produce a significant treatment effect for global cognitive function ($M = .08$, $p = .38$). Recovery time (≤1 year or >1 year) significantly moderated mean IGPP-*ES*, Q(1) = 6.84, $p = .009$. Treatment yielded a moderate effect for ≤1-year postinjury groups ($M = .40$, $p < .001$), but >1-year postinjury groups did not produce a significant treatment effect on global cognitive function ($M = .06$ $p = .54$). Both of these moderator variable analyses of IGPP-*ES*s corresponded closely to the results of similar analyses of SGPP-*ES*s (i.e., compare Tables 3 and 5). Mean age of participants also significantly moderated IGPP-*ES*s, Q(1) = 20.08, $p < .001$. *ES* was significantly positively correlated with mean age at time of cognitive rehabilitation.

As in the analyses of SGPP-*ES*s presented in Table 3, the homogeneity tests for all levels of all moderator models failed to reach significance, indicating a lack of evidence to claim heterogeneous *ES*s. Moreover, the homogeneity test for each significant model presented in Table 5 and the mean age model were all significant for all of the matching fixed-effects-only models (*p*s < .01), supporting inclusion of the random study effect for each model.

Table 5
*Statistics of Interest for Analyses of Independent Groups Pre-Post Effect Sizes (IGPP-*ES*s) by Treatment Domain, Etiology, and Recovery Level*[a]

				95% CI				
	K	Adj. *M ES*	*SE*	Lower	Upper	*z*	Q	σ^2_θ (*SE*)
Treatment domain								
Overall	45	.34	.05	.24	.43	6.67**	38.64	.12 (.02)
Attention/executive	9	.27	.12	.04	.50	2.24*	10.34	
Visuospatial	13	.62	.09	.44	.80	6.79**	18.10	
Language	12	.32	.10	.13	.51	3.32**	7.02	
Memory	5	.18	.18	–.16	.53	1.03	0.55	
Comprehensive	6	–.01	.12	–.25	.23	–0.08	2.63	
Etiology								
Overall	40	.34	.05	.23	.44	6.34**	36.07	.03 (.02)
Stroke	24	.48	.07	.35	.61	7.26**	30.05	
TBI	16	.08	.09	–.10	.25	0.87	6.03	
Recovery level								
Overall	31	.27	.06	.15	.39	4.39**	28.70	.05 (.03)
≤1-yr postinjury	18	.40	.08	.24	.55	5.08**	25.14	
>1-yr postinjury	13	.06	.10	–.13	.26	0.61	3.56	

Note. TBI = traumatic brain injury.
[a] Analysis of IGPP-*ES*s from 45 IGPP studies only.
* $p < .05$. ** $p < .01$.

Specificity of Treatment Effects

The cognitive rehabilitation approach is unique because cognitive psychology theory is brought to bear on a domain to identify candidate processes within a domain to be targeted for a custom-designed treatment (e.g., Park & Ingles, 2001). To see whether cognitive rehabilitation *ES*s are moderated by a specific combination of treatment domains and outcome domains, we assigned each DV into one of the five broad cognitive domains used to group treatments and computed an unweighted mean *ES* for each study within each of the cognitive domains used by that study. For each cognitive treatment domain, we fit a random- effects model for each of the outcome measured cognitive domains with at least three studies in the sample (see Table 6). As with the analyses of global cognitive function *ES*s (see Tables 3 and 5), we fit two versions of each model—one set based on the 115 treatment group SGPP-*ES*s (with control group mean SGPP-*ES*s subtracted out), and the other set based on the 45 IGPP-*ES*s. Unlike the earlier analyses, this time SGPP-*ES*s were adjusted at the aggregate level, that is, weighted mean SGPP-*ES*s were fit by separate random-effects models and the resultant means were subtracted.

The results are presented in Table 6. To the extent that rehabilitation has a specific treatment effect, we expected the *ES*s to be larger when the treatment and outcome domains were matched compared to when they were mismatched. Again, as with the analyses of global *ES*s presented in Tables 3 and 5, the two methods of estimating weighted mean treatment effects for cognitive domain-specific *ES*s corresponded to a high degree. The exceptions were memory treatments and memory outcome measures, for which the mean *ES* from the larger sample of 115 studies was significant but the mean *ES* for the smaller sample of 45 IGPP studies was not.

The treatment domain of attention demonstrated a pattern of domain-specific effects. The results for the SGPP-*ES* and IGPP-*ES* analyses were $M = .35$ and .38, respectively, and these were both statistically significant (ps $< .05$). However, the mean *ES*s for the domain-specific treatments of language, memory, and comprehensive rehabilitation in studies that focused on the treatment of attentional problems were not significantly different from zero for either the SGPP-*ES* or the IGPP-*ES* analyses.

Two other treatment domains yielded ambiguous results that do not rule out domain-specific effects but are inconclusive. The memory treatment domain exhibited weak support for domain-specific treatment effects. The analysis of adjusted SGPP-*ES*s yielded a significant *ES* for the memory outcome measures ($M = .52$, $p < .01$) but not for the attention outcome measures ($M = .12$, $p > .05$). However, this pattern was not found in the analysis of IGPP-*ES*s, in which the *ES* for neither memory nor attention outcome measure were significant (ps $> .05$). A different sort of ambiguity was observed for the language treatments for which both analyses yielded small but significant *ES*s ($M = .22$ and .36) for the SGPP-*ES* and IGPP-*ES* analyses, respectively ($p < .05$ and $p < .01$). However, not enough language rehabilitation studies included outcome measures from domains other than language to provide estimates of treatment effects in other cognitive domains.

Table 6
*Statistics of Interest for Analyses of Effect Sizes (*ES*s) by Cognitive Domain of Treatment and Outcome Measures*

				Comparison of two estimates of cognitive treatment effect					
	Control group SGPP-*ES*s			All 115 tx group SGPP-*ES*s[a]			IGPP-*ES*s from 45 studies		
Outcome domain	*K*	*M ES*[b]	*SE*	*K*	Adj. *M ES*[b]	*SE*	*K*	*M ES*[b]	*SE*
Treatment domain: Attention/executive									
Attention/executive	7	.34**	.11	12	.35*	.14	7	.38*	.15
Language	3	.24	.19	4	.23	.28	3	.28	.18
Memory	3	.17	.13	5	.10	.21	3	.08	.17
Comprehensive	8	.45**	.13	9	.06	.21	8	.08	.16
Treatment domain: Visuospatial									
Visuospatial	10	.20**	.06	18	.44**	.09	10	.47**	.12
Attention/executive	5	.11	.10	13	.51**	.13	5	.46**	.16
Language	8	.16	.11	11	.60**	.16	8	.53**	.10
Comprehensive	12	.32**	.10	25	.56**	.14	12	.78**	.13
Treatment domain: Language									
Language	12	.53**	.09	32	.22*	.11	12	.36**	.10
Treatment domain: Memory									
Memory	4	.34*	.14	14	.52**	.17	4	.20	.17
Attention/executive	3	.18	.19	3	.12	.28	3	.11	.27
Treatment domain: Comprehensive									
Comprehensive	5	.63**	.15	22	.10	.18	5	.16	.19
Attention/executive	4	.52**	.11	12	.07	.14	4	.20	.17
Visuospatial	3	.55**	.10	9	−.05	.15	3	.03	.21
Language	3	.36*	.17	8	−.03	.21	3	.11	.15
Memory	4	.56**	.08	14	.00	.11	4	.01	.10

Note. tx = treatment; SGPP-*ES*s = single-group pre-post effect sizes; IGPP-*ES*s = independent groups pre-post effect sizes; Adj = adjusted.
[a] Analysis of treatment group adjusted SGPP-*ES*s for all studies, with control group means from IGPP studies subtracted out. [b] All mean *ES*s estimated using random effects model using restricted maximum likelihood estimation.
* $p < .05$. ** $p < .01$.

Therefore, there might be domain-specific treatment effects operating in the language treatment domain, but we cannot confirm or disconfirm with the available evidence.

Finally, non-domain-specific treatment effects were found for visuospatial treatments. Significant modest treatment effects were observed in both the SGPP-*ES* and the IGPP-*ES* analyses for the attention, visuospatial, language, and comprehensive outcome measure domains (all ps $< .01$). In fact, the estimated mean *ES*s for visuospatial treatments were lowest for the visuospatial domain outcome measures in our sample. In addition, no significant *ES*s were observed in either analysis for comprehensive treatment studies in any of the five cognitive domains (all ps $> .10$). Therefore, no evidence for domain-specific effects was found for treatments in the domains of visuospatial and comprehensive rehabilitation.

Discussion

The present study provides a meta-analysis of cognitive rehabilitation research that complements the practice reviews of Cicerone et al. (2000, 2005; see also Cappa et al., 2003, 2005). As such, our focus is on the large-scale view, with an orientation toward the following questions: Does cognitive rehabilitation provide an effective class of treatments after neurological injury? Do cognitive treatments differ in effectiveness across cognitive domains? Is there evidence for domain-specific treatment effects? Are there any large-scale moderator effects that influence the treatment effectiveness?

Our analyses of treatment effects on global cognitive function yielded cognitive rehabilitation effects that were relatively modest yet statistically significant, in the .25 to .34 range ($M = .30$; see Tables 3 and 5 and Figure 1). Given this finding, we conclude that there is a scientific base, albeit limited, to support the premise that cognitive rehabilitation is effective for persons with acquired brain injury. This is based on a rather large sample of studies of the cognitive rehabilitation literature. The literature was identified from the even larger sample used by Cicerone et al. (2000, 2005) for their systematic reviews of the cognitive rehabilitation literature. Combined, these reviews were based on an initial 967 articles, but only a reduced set of 258 articles met their inclusion criteria. We took their 258 articles and removed another 155 as being inappropriate for meta-analytic methods, primarily due to inadequate reporting of outcome measures to allow calculation of an *ES* and for use of a single-subject design.

Retest Effects

In addition to the significant treatment *ES* of .30, our analyses revealed a modest mean *ES* of .41 for the control groups in the IGPP studies. This is, in essence, an estimate of a sizable retest effect. Specifically, participants were not exposed to a cognitive treatment yet they demonstrated improved performance on cognitive outcome measures from pretest to posttest. Several possible factors for this effect are changes in motivation from pretest to posttest, placebo effects due to additional individualized attention received by participation in a research study, practice effects on the tests themselves, and spontaneous recovery of cognitive function during the study period.

Moderator Effects on Global Cognitive Rehabilitation

In addition to the main finding of a small significant treatment effect of cognitive rehabilitation for persons with acquired brain injury, our results revealed four significant moderators of the global *ES*: treatment domain, etiology, recovery level, and mean age of participants. Treatment for attention, visuospatial, and language deficits produced significant improvements, whereas memory treatments produced equivocal results and comprehensive treatments failed to produce a significant improvement. Generally, stroke groups experienced significant treatment effects, but TBI groups did not. Moreover, treatment effects were observed for patients less than 1-year postinjury but not for patients more than 1-year postinjury. Older patients tended to improve more, although this effect is confounded with the moderator variable of type of brain injury (i.e., stroke vs. TBI). Moderator variables, although statistically significant, were highly confounded. For pure etiology groups, the correlation between mean age of participants and etiology was perfect, as stroke and TBI groups had nonoverlapping mean age distributions. As demonstrated in Table 2, treatment domain was highly confounded with both etiology and recovery level. Although not presented in a table, recovery level and etiology were also confounded.

In light of these confounds, the effectiveness of cognitive rehabilitation for combinations of these moderators is of interest. For example, there is modest evidence for an effect of attention treatment on global cognitive function in samples of TBI patients, who tend to be younger and less than 1-year postinjury, which is consistent with the findings of Mathias and Wheaton (2007). There is also strong evidence for an effect of visuospatial training and modest evidence for an effect of language training on global cognitive function in samples of individuals with stroke who are older and more than 1-year poststroke. This at first may seem counterintuitive, but it may simply be the result of limited evidence available to partial out the influence of other moderator variables.

Of course, the confounding of treatment, etiology, age, and recovery level is to be expected, as stroke and TBI appear to commonly result in different patterns of loss of cognitive functions and, perhaps, different patients are available for enrollment in studies at different periods postinjury (Halligan & Wade, 2005; High et al., 2005). This leads to far more frequent application of treatments in each cognitive domain to one etiology than to the other one. Etiology and age are related, because TBI tends to be the result of car accidents whereas the risk of stroke increases with age. For example, our sample only contained four studies of language treatment after TBI. We fit these studies to separate random-effects models for the IGPP control, IGPP treatment, and all treatment groups. The single IGPP study had a negative *ES*, that is, the control group produced greater pre-post improvement than did the treatment group. Yet, the mean *ES* for the treatment groups across all four studies was .80 ($SE = .24$), but it does not make sense to use the single IGPP control group to adjust this mean. Instead, if we use all 12 control groups (i.e., of stroke, mixed, TBI, and not recorded etiology) from Table 3 ($M = .53$), we get an estimated adjusted mean language treatment effect in TBI of .27. The most we can conclude is weak evidence in favor of effective language rehabilitation after TBI, given a negative *ES* for the one study in our sample that included a control group.

Treatment Specificity

One issue of importance (e.g., Cappa et al., 2003; Cicerone et al., 2000) has been the extent to which cognitive rehabilitation has its effects specifically on those cognitive functions targeted by the treatment technique. For example, when an intervention is targeted to improve patients' attentional skills, it should have the largest effect on the outcome measures of attention. On the other hand, less of an impact should result from the attentional intervention on patients' visuospatial or language ability. We might then expect that a study that involved an intervention designed to improve attention, which includes measures of attention, visuospatial skills, and language, would have its biggest treatment effect on the attentional measure. Some generalization may occur, such that the visuospatial and language measures may also demonstrate improvement, but improvement in the other domains is expected to be less than that generated in the domain of attention.

Attention rehabilitation provided the only strong evidence for treatment specificity, as the mean *ES* was significant for attention outcome measures but not for language, memory, and comprehensive measures. On the other hand, visuospatial rehabilitation suggested treatment effects across several domains. That is, for patients participating in visuospatial treatment, moderately sized and significant treatment effects were found for memory, language, and comprehensive treatment measures. Additionally, in the visuospatial rehabilitation, the sample visuospatial outcome *ES* mean was the smallest compared to other outcome *ES*s for the adjusted SGPP-*ES* analysis, and it was the second smallest for the IGPP-*ES* analysis (see Table 6). This suggests a modest effect of visuospatial treatments upon global cognitive function. Language and memory treatments produced ambiguous results concerning the issue of treatment specificity. Not enough language studies used outcome variables from other cognitive domains to provide separate *ES* estimates. We are therefore left with a significant language treatment effect for language therapy using language outcome measures, but we have no comparison *ES*s from other cognitive domains. Memory treatments produced a moderately sized and statistically significant mean *ES* for memory measures and a quite small ($M = .12$) attention *ES* in the SGPP-*ES* analysis. Both mean *ES*s, however, were nonsignificant in the IGPP-*ES* analysis. This pattern leaves us with weak evidence for a specific memory treatment effect, matching the weak evidence for a memory treatment effect in the global *ES* analysis. Finally, not only was there no evidence for a treatment-specific effect for comprehensive rehabilitation, there also was no evidence for any significant treatment effects when outcome measures were categorized by cognitive domain.

Comparison With Two Prior Meta-Analyses of Cognitive Rehabilitation

Two meta-analytic reviews targeted to specific cognitive rehabilitation domains have recently been published (Park & Ingles, 2001; Robey, 1998). Although those authors conducted some analyses beyond the scope of this paper, these studies provide important points of comparison. It is worth noting that neither of these studies used random-effects models. They also did not report results from homogeneity tests or have residual heterogeneity that is unaccounted for by the models used. Finally, statistical tests of moderator variables were not reported in either study. Nonetheless, the present results support a small-to-moderate ($M = .35$ and .38, for adjusted SGPP-*ES* and IGPP-*ES* analyses, respectively) treatment-specific effect for attention rehabilitation. This result is only partially consistent with the results of a meta-analysis of attention training conducted by Park and Ingles (2001). They found a large attention training effect ($M = .68$) in their analysis of SGPP-*ES*s for treatment groups but a small nonsignificant effect ($M = .15$) in their analysis of IGPP-*ES*s. These authors concluded that there was little evidence for a significant effect of attention treatment on measures of attention and executive function when appropriate controls are included in the design.

It is important to note that Park and Ingles (2001) did not adjust their SGPP-*ES*s for retest effects observed in control groups. In fact, they did not report SGPP-*ES*s for controls, so their large attention rehabilitation effect for SGPP-*ES*s in treatment groups was most likely biased upward by the presence of retest effects. The present study found a small-to-moderate statistically significant effect ($M = .34$) of attention treatment on attention measures in the IGPP-*ES* control group studies. If this effect is added to the present adjusted mean SGPP-*ES*, the result ($M = .69$) is similar to that which was obtained by Park and Ingles for unadjusted SGPP-*ES*s. Much of the discrepancy in the estimates of the attention training effect in the IGPP-*ES* analyses of Park and Ingles (2001) versus the present meta-analysis (i.e., .15 vs. .38) is attributable to the fact that the previous study separated out five measures of attention skills in applied settings, all with *ES*s in the .45 to 1.15 range (see Park & Ingles, 2001, Table 1), from their set of more focused measures of attention processes (see Park & Ingles, 2001, Table 2). We combined the *ES* estimates for these two groups of outcome measures from the Park and Ingles tables and computed a simple unweighted mean of the reported weighted means and found that the resultant mean *ES* of .31 is comparable to our estimate of .38.

Robey (1998) reported a meta-analytic review of aphasia treatments across all outcome measures in each study ($K = 57$, total individual *ES*s = 75), which corresponds to our global *ES* analysis of language rehabilitation. Robey reported a mean *ES* of .57 for treatment groups that were 3 to 12 months postinjury and of .34 for control groups. Applying our difference of means adjustment to Robey's findings yielded a mean *ES* of .23. This corresponds closely to our findings: We estimated random-effects models for the adjusted IGPP-*ES*s (using the same method as in our main analyses) for language treatment groups in the 3- to 12- month postinjury period and found a mean *ES* of .24 ($p < .05$). Robey (1998) reported the largest effects of aphasia treatment for participants in the acute phase, combined with treatments of at least 2-hr per week. We were unable to substantiate these conclusions because there were no language studies in our sample that reported groups in the less than 3-month postinjury period, and the present meta-analysis did not find a significant effect of treatment duration.

Cicerone et al. Practice Guideline Reviews

Based on their systematic evaluation of the published studies in their sample, Cicerone et al. (2005) concluded, "There is now a substantial body of evidence demonstrating that patients with TBI or stroke benefit from cognitive rehabilitation" (p. 1689). The present meta-analytic review provides modest quantitative support for this statement; specifically, a mean *ES* in the .25 to .34 range

(estimated mean *ES*s; for 95% CIs, see Tables 3 and 5). The estimate of overall *ES* for global cognitive function varies across models due to the fact that inadequacies in study designs did not allow fitting of an overall single model of moderator variables that can reasonably be argued to be the "correct" model.

In their cumulative summary of both of their reviews, Cicerone et al. (2005) argued that there is substantial evidence to support the efficacy of visuospatial and language rehabilitation following strokes leading to aphasia and neglect syndromes. In support of this argument, our meta-analysis suggested a medium-to-large effect of visuospatial training for stroke groups that is in the range of .54 to .62 for global cognitive functions and is of a similar magnitude across all cognitive domains. Our results also yielded a small-to-medium language treatment effect in the range of .18 to .36. Additionally, Cicerone et al. (2005) claimed that there was substantial evidence supporting the efficacy of memory, attention, and language (more specifically, functional communication) rehabilitation for individuals with TBI. Our results are not as supportive of these claims. Although our meta-analysis suggests a small-to-medium effect of attention training for stroke groups in the range of .35 to .38, the results for memory rehabilitation are mixed and weak. We did not observe a significant effect of memory training in our analyses of IGPP-*ES*s for either the global *ES* or the *ES* based only on memory measures. A medium-to-large memory training effect emerged in our analyses of SGPP-*ES*s ($M = .61$ and .52, for the global and memory measures, respectively), but this evidence comes primarily from SGPP designs without controls. The difference between that which was recommended by Cicerone et al. (2005) and the present findings may be related to severity of injury. Cicerone et al. (2005) restricted their recommendations for memory training to those patients who suffered from milder injures, specifically TBI patients. We were not able to partial out the influence of severity of TBI on treatment effectiveness. Our results were collapsed across severity, and benefits that may have been evident for the more mildly injured patients may have been obscured by the ineffective results obtained from more severely injured patients. As discussed earlier, there were only four language treatment studies after TBI in our sample. Of these, only one had a control group, and the treatment *ES* from that study was negative. In summary, our findings did not provide an adequate quantitative basis to support all of the general practice guidelines suggested by the systematic review.

Cicerone et al. (2000, 2005) included more studies in their systematic review than we did; however, we would be surprised if our results differed appreciably with the inclusion of those additional articles. Despite the fact that single-subject design studies constituted 43% of the study sample reviewed by Cicerone and colleagues, the total number of participants included in all 110 single-subject design studies was approximately 275 ($M = 2.5$ per single-subject design study), which would have accounted for 12% of our total sample. The typical single-subject design study uses participants as their own controls, such that these same 275 patients would constitute 24% of our controls and 16% of the entire sample of both patients and controls. Because meta-analysis weights study *ES*s by the inverse of the sample variance, which is highly influenced by sample size, there is limited ability for small studies to influence an outcome. In fact, for the sake of argument, let us assume that the *ES* generated from all single-subject design studies had been as large as 1.0 *SD* units, which is three to four times as large as the *ES* generated from the group studies (*ES* = .25 to .34). If this were then combined with the current reported outcome, the resulting final global *ES* outcome would have increased to only .34 and .42. There is not a large difference between these two ranges, despite the assumption of a very large positive outcome from all the single-subject design studies.

Finally, we concur with Cicerone et al. (2005) that "future research should move beyond the simple question of whether cognitive rehabilitation is effective, and examine the therapy factors and patient characteristics that optimize the clinical outcomes of cognitive rehabilitation" (p. 1681). Recognizing the usefulness of quantified *ES*s, but perhaps underestimating the evidence existing in the literature, Cicerone et al. suggested that truly making progress in identifying factors that optimize cognitive outcomes will require much better reporting of quantified outcome measures documenting clinical efficacy.

Limitations

The most significant limitation of our findings is the strong reliance in the cognitive rehabilitation literature on SGPP designs (61% of our sample). This would not have been such a concern had it not been for the sizable retest effects necessitating analysis of adjusted means. SGPP designs are weak in the face of significant retest effects, as, in and of themselves, they do not allow separate estimation of test and treatment effects. The benefit of our approach is a conservative test of the treatment effect. That is, the focal issue is one of added treatment effect, beyond a broad and rich control experience. In other words, to what extent does cognitive rehabilitation result in a reliable effect in addition to general activities of daily living experienced by patients as they recover in a typical inpatient or outpatient setting.

We argue that to include only control conditions in a restrictive sense would have overestimated the true *ES* for cognitive rehabilitation. More practically, the alternative would have excluded valuable data from our overall analyses. That is, intervention researchers often use alternative activities for the control group due to apparent hesitation to place patients in true no-treatment control groups. These limitations in the literature—the high percentage of SGPP designs and the varied control conditions for IGPP designs—point to future research that is needed to provide a stronger scientific support for cognitive rehabilitation.

An additional factor for consideration is the heterogeneity of the control conditions in the sample of studies. Our criteria for including a group as a control group was that there be random assignment to groups treated differently, that the treatment group experience cognitive rehabilitative therapy not experienced by the control group, and that the control group not receive a cognitive treatment deemed to be efficacious. However, what constitutes a control condition is variable in this literature, varying from true no-treatment groups in some outpatient studies, to placebo comparison groups that experience sessions of unstructured conversation with therapists in others, to inpatient comparison groups given a standard occupational therapy treatment. Moreover, some studies included multiple comparison groups. When selecting the most appropriate control condition, we selected the control group that was most similar to a no-treatment control. Some of these might be considered alternative treatments or

treatment as usual. Despite this, we found no evidence that there were differential effects between control group types in terms of the retest effect.

The moderator variable of treatment duration was not significant statistically, so we were not able to substantiate the influence of treatment duration. This likely was due to several limitations within the rehabilitation literature. Unlike the psychotherapeutic outcome literature, the cognitive treatments we investigated were not "manualized," and thus little detail was available regarding such things as number of sessions per week, length of sessions, and total time of treatment. High variability in reporting relevant information prohibited coherent description and analysis, thus precluding any conclusions regarding treatment duration and effectiveness. These types of variables have been well controlled in the treatment literature for reading disabilities, which could be a model for how such variables might best be measured or manipulated in cognitive rehabilitation in the future.

As we already noted, other limitations in our analyses included the frequent confounding of treatments by etiology, by age, and by recovery level. Without better separation of these variables, it will be impossible for future meta-analysts of this literature to determine the separate or synergistic influences of these variables. Furthermore, without a better incorporation of severity measures and duration of treatment measures, we will continue to have problems determining a dose–response effect for treatment, similar to that which exists in the pharmaceutical industry.

It is true that some functional treatment outcomes described in our sample's studies were not incorporated into this article. Would the results of our current meta-analysis be different if we had included all the measures that were available in the studies we reviewed? We think it unlikely. Not that many measures of real-world functions were excluded, and it is difficult for a few measures to have a significant impact on the resulting *ES*. Furthermore, generalization from a laboratory task to real-world settings typically results in lower performance. In our clinical experience, patients who show improvement "in vivo" often return to baseline when the task is "in vitro." That is not to say that future rehabilitation efforts should not focus on functional outcomes. Improving a patient's ability to perform a laboratory task is obviously far less important than helping a patient actually adapt and cope with everyday problems. Researchers in cognitive rehabilitation can incorporate functional tasks in future, well-designed treatment studies. Nevertheless, operational definitions are needed, and, if used, psychometric tools have to be valid in assessing outcomes from functional treatment activities (e.g., use of external memory aids). It is not uncommon for patients and providers to report improvements in the real-world task of compensating for memory impairment, while the psychometric measures show little or no change.

Future Directions

One goal in support of evidence-based practices in cognitive rehabilitation per an American Psychological Association policy statement (Levant, 2005) is to uncover meaningful patterns in the scientific record through the synthesis of high-quality research. Findings from this study indicate several necessary steps to attain this goal. First, as noted in recent reviews (Cappa et al., 2003, 2005; Cicerone et al., 2000, 2005), a major challenge facing researchers in the development of evidence-based practice guidelines is to find sufficient evidence from high-quality designs to support practice recommendations. Although our results revealed no differences in outcome based on variable study class, we remain convinced that high internal validity in study design leads to better understanding of the relevant factors in a treatment outcome study before external validity can be tested. In general, Class I studies, such as randomized clinical trials (RCTs), have higher internal validity than Class III studies or single-subject designs. As research supports the definition of relevant treatment parameters, researchers need to reduce reliance on single-subject and single-group designs.

We understand that newly developed potentially efficacious treatments must go through a maturational research sequence, which might start with a single-subject design study and, depending upon results, proceed through to a Class I RCT (Gonzalez-Rothi, 2006). However, the speed with which these treatments proceed through the sequence, the need for replication of previously examined treatments, and the need to publish results from each of the study designs in the sequence can lead to significant disputes among researchers. The roots of the cognitive rehabilitation literature go as far back as the 1960s. In fact, our analysis of Cicerone (2000; Cicerone et al., 2005) includes literature that span three decades. Furthermore, the literature we examined included over 250 articles. We believe that this is deep literature, and experts in the field need to accept its maturity. That is not to suggest that the field will not benefit from some level of single-subject designed studies for recently developed treatments. Rather, we believe that the field is at a point in which third-party payers have reduced reimbursement for a variety of treatments due to a lack efficacy data, and it is time for researchers in the field to accept that third-party payers will not wait any longer for data to be generated from substandard methodology before they make decisions regarding reimbursement.

Additionally, research designed to test potential moderators of treatment effectiveness will provide the evidence needed for more detailed quantitative analysis. In fact, it is the perspective afforded by this meta-analysis that placed the confounding of the significant moderators of age, treatment domain, etiology of acquired brain injury, and recovery level into clear view. To appreciate the influence of variables such as age, we recommend that future researchers stratify their sample. For example, treatment groups could differentiate adults younger than 55 years old from adults older than 55 years to investigate how age influences outcome and ultimately to determine how age leads to differing treatments in rehabilitation. Better experimental control (i.e., crossing) of treatment domain, etiology, and recovery level built into future research designs would allow for the assessment of the independent contributions of these factors on cognitive rehabilitation effects. Furthermore, treatment duration as a factor affecting treatment effectiveness requires experimental manipulation. At a minimum, clinical researchers can be more specific regarding the documentation of their participants' time in treatment, supporting successful investigation of this obviously relevant variable by subsequent meta-analyses.

Future studies also need to take into account the influence of litigation and potential malingering. Two unfortunate aspects of the entire cognitive rehabilitation literature involve the failure of researchers to document an influential variable on test performance. First, none of the studies we reviewed indicated whether the participants were involved in litigation or were being compensated for their injuries. Moreover, none of the studies reported measures of effort or symptom validity. Several researchers (e.g., Constantinou, Bauer, Ashendorf, Fisher, & McCaffery, 2005; Green, Rohling, Lees-Haley, & Allen, 2001; Stevens, Friedel, Mehen, & Merten, 2008) have illustrated the major impact such variables can have on the assessment of cognitive functioning. Some treatments may have been more effective than the current data would suggest because some of the participants may have had reason to perform more poorly than they were capable of doing in order to obtain financial benefits. This was a factor in a publication of a dose–response relationship in TBI (Rohling, Meyers, & Millis, 2003). We encourage all researchers not to limit what they might be able to learn about the treatments they are providing by not including measures of effort or response bias in their protocols.

Finally, it is a complex task to balance internal validity with external validity. Clinical researchers often worry that attempts to improve internal validity will come at the cost of external validity. In treatment outcome studies, however, external validity is bounded by internal validity. Thus, we will be unable to substantiate the efficacy or effectiveness of any particular cognitive treatment without consideration of how well we controlled potential confounds within our study designs. For example, this study showed that improved delineation of outcome measures—those that are expected to respond to the treatment and those that could demonstrate generalization versus control—is needed. Some may believe that certain preferred treatments are externally valid (e.g., memory notebook training); but without proper research evidence to this effect, the treatment may never be supported by third- party payers and thus not provided to patients who might otherwise benefit from its administration.

In summary, the field of cognitive rehabilitation has been hampered by several factors: (a) insufficient description of heterogeneous neural damage, (b) single-shot studies of treatments instead of replications under different combinations of potential moderator variables, (c) lack of a broad standard set of cognitive outcome measures that test both cognitive processes and skills, (d) inadequate reporting of outcome measures to allow computation of effect size measures, (e) overreliance on small sample sizes and single-group research designs, and (f) failure to take suboptimal effort or symptom validity into account. Due to these limitations, much of the existing scientific record contains weak and conflicting evidence that precludes a definitive judgment regarding the effectiveness of candidate treatments.

Clinical Implications

A few concrete principles for cognitive rehabilitation are proposed based on these findings. The significant moderator variable regarding time postinjury (e.g., $\leq$1 year vs. $>$1 year) suggests that it is better to start patients in treatment as early as possible rather than waiting for more complete neurological recovery. Second, even older patients (e.g., $\geq$55 years old) can and do benefit from cognitive rehabilitation, particularly if the brain injury is due to stroke. Furthermore, comprehensive nontargeted interventions appear to be less effective and generalization does not happen as well as many may have hoped (see Vanderploeg et al., 2006, for description of the different models of targeting interventions). Thus, clinicians should focus their efforts on direct cognitive skills training (e.g., training for visual spatial neglect) rather than broad generalized interventions with the expectation of subsequent generalization to broader use in the real world (e.g., see Lillie & Mateer, 2006, for review of constraint-induced movement therapy [CIMT] as a model for TBI rehabilitation).

Conclusion

The present analysis provides quantitative evidence for some of the claims made by Cicerone et al. (2000, 2005) in their practice guideline reviews of cognitive rehabilitative treatments for TBI and stroke. Of the five major claims of effectiveness within treatment domain–etiology combinations made by Cicerone et al. (2005), our meta-analysis supported three. However, our results found that two of their five recommendations were premature due to problems with poorly estimated retest effects in uncontrolled designs. We also brought to the fore the problem of retest effects that bias estimates of treatment *ES*s in single-group designs. If these two recommendations are to be substantiated, better designed interventions studies will need to be conducted with proper control of the potential confounding or moderating variables.

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