Systematic review and meta-analyses of word production abilities in dysfunction of the basal ganglia: Stroke, small vessel disease, Parkinson's disease, and Huntington's disease

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Abstract

Clinical populations with basal ganglia pathologies may present with language production impairments, which are however often described in combination with comprehension measures and/or attributed to motor, memory, or processing-speed problems. In this systematic review and meta-analysis, we studied word production in four (vascular and non-vascular) pathologies of the basal ganglia: stroke affecting the basal ganglia, small vessel disease, Parkinson's disease, and Huntington's disease. We compared scores of these clinical populations with those of matched cognitively unimpaired adults on four well established production tasks, namely picture naming, category fluency, letter fluency, and past-tense verb inflection. We conducted a systematic search in PubMed and PsycINFO with terms for basal ganglia structures, basal ganglia disorders and language production tasks. A total of 114 studies were included, containing results for one or more of the tasks of interest. For each pathology and task combination, effect sizes (Hedges' g) were extracted comparing patient versus control groups. For all four populations, performance was consistently worse than that of cognitively unimpaired adults across the four language production tasks (p-values < 0.010). Given that performance in picture naming and verb inflection across all pathologies was quantified in terms of accuracy, our results suggest that production impairments cannot be fully explained by motor or processing-speed deficits. Our review shows that language production difficulties in these clinical populations are not negligible, but more evidence is necessary to determine the exact mechanism that leads to these deficits and whether this mechanism is the same across different pathologies.

Keywords: *diaschisis; hypoperfusion; morphological encoding; speech; verbal fluency; white matter hyperintensity*

1. Introduction

The ability to produce words to express one's thoughts is essential for efficient communication. This ability, referred to as conceptually driven word production, is commonly measured with tasks such as picture naming or word generation. Deficits in word production like anomia, that is, a failure in retrieving words, are common in individuals with damage to perisylvian brain areas in the left hemisphere (Croquelois & Bogousslavsky, 2011). Basal ganglia structures have also been argued to play an important role in lexical-semantic aspects of word production (Copland, 2003; Copland et al., 2000; Crosson, 1985; Wallesch & Papagno, 1988), but this view is not widely adopted. Furthermore, language deficits in people with basal ganglia pathologies, such as a stroke affecting the basal ganglia, small vessel disease (SVD), Parkinson's disease (PD), or Huntington's disease (HD), might be overlooked or mislabeled (e.g., as memory or motor problems), further muddling this issue. Moreover, existing reviews describing language difficulties in these pathologies often bundle together measures of language comprehension and production (e.g., Gagnon et al., 2018; Radanovic & Mansur, 2017). Thus, the effect of basal ganglia dysfunction on conceptually driven word production remains unclear.

To address this question, we performed a systematic review and meta-analyses of

conceptually and lexically driven word production tasks in four vascular and non-vascular diseases that affect the basal ganglia, that is, stroke, small vessel disease (in which the basal ganglia may be affected in addition to periventricular damage), PD, and HD. We examined four different word production tasks: picture naming, category-based fluency, letter-based fluency, and past-tense verb inflection (Figure 1), with the first two tasks being conceptually driven and the last two (at least) lexically driven.

Picture naming, category fluency, letter fluency, and past-tense verb inflection are spoken word production tasks that go beyond the motor components of speech, tapping especially well into the conceptual, lexical, and morpho-phonological levels of production. For example, to name a picture, first a concept needs to be identified, which then drives lexical selection and the encoding of the morphological and phonological form. Similarly, in generating words of a particular semantic category (e.g., animal names) or words whose names begin with a particular letter (e.g., words starting with a *k*) within a time limit, referred to as verbal fluency tasks, conceptual and orthographic/phonological information needs to be retrieved. In a category fluency task, word production is conceptually driven, whereas in a letter fluency task, the target letter drives the retrieval from lexical memory of words with corresponding onset phonemes. In producing the past tense of a verb, lexical memory needs to be accessed to determine whether the verb takes a regular or an irregular past-tense form. Moreover, picture naming and verbal fluency are well established tasks in neuropsychological assessments, and come with normative data, allowing for a standardized age- and education-adjusted comparison to individuals without cognitive impairments. Because all four tasks necessarily require access to words in memory, they cannot be performed solely using sublexical phonological and motor strategies, such as grapheme-to-phoneme conversion, as is often possible in the case of reading aloud. It could be argued that verbal fluency might be affected by motor deficits, as these are typically timed tasks, but accuracy in picture naming and in verb inflection should be largely independent of motor issues. These four tasks have been widely administered in all four clinical

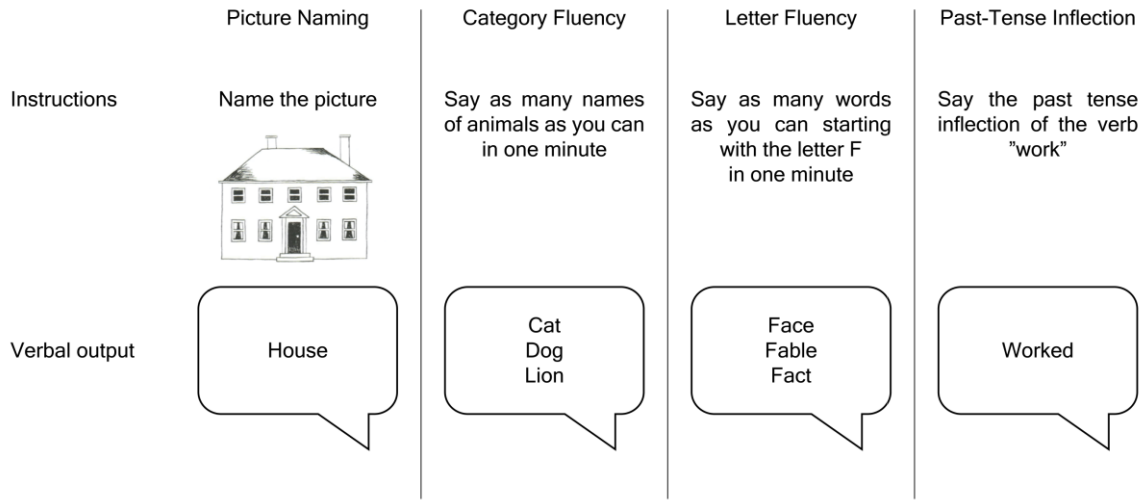

Figure 1. Word production tasks examined in the present study.

populations, making them prime candidates to assess word production deficits related to basal ganglia damage.

In regards to word production, Bohsali and Crosson (2016) distinguishes two loops connecting the basal ganglia to cortical regions, each one composed of three circuits. One loop, consisting of the pre-supplementary motor area (SMA) and the basal ganglia, is assumed to be involved in lexical selection: A direct circuit selects the appropriate lexical item, whereas an indirect circuit inhibits competing alternatives. When the selection has been made, a hyperdirect circuit resets the system so that the process can start over. In a similar manner, another loop consisting of portions of the left inferior frontal gyrus ("Broca's area") and the basal ganglia, is assumed to be responsible for selection of the proper phonological and articulatory representations. According to this view, the function of the basal ganglia, in collaboration with cortical regions, is to make processing more efficient by increasing the signal-to-noise ratio during the different processing stages required for word production. As such, basal ganglia dysfunction may not lead to severe impairment of all language production abilities, as may be the case in Broca's aphasia for instance, but deficits may nevertheless be clinically relevant and should be considered during neuropsychological, behavioral neurological, or linguistic assessments (Bohsali & Crosson, 2016).

1.1. Disorders of the basal ganglia

Stroke is caused by the lack of blood supply to the brain due to the occlusion or bleeding of blood vessels. Aphasia symptoms, such as comprehension, repetition, or naming deficits, are common after middle cerebral artery cortical strokes in the dominant hemisphere, but may also arise after stroke of the basal ganglia (Crosson & Haaland, 2003). According to one recent review of studies on language comprehension and production, one possible mechanism of lexical-semantic deficits following stroke of the basal ganglia is a dysfunction in cortical hemodynamics due to ischemia/hypoperfusion in middle cerebral artery territories (Radanovic & Mansur, 2017). However, previous studies in people with basal ganglia stroke (BG stroke, henceforth) have reported conflicting results or are often based on single cases. Moreover, lexical deficits in individuals with basal ganglia disease not caused by a stroke lesion cannot be readily explained by the presence of hypoperfusion in cortical areas supplied by the middle cerebral artery. Still, given the extensive connectivity with cortex, damage to the basal ganglia or its connections may disrupt neuronal input to the cortex and thereby impair cortical processes (see for discussion Radanovic & Mansur, 2017).

Another disease that may impair basal ganglia function is cerebral small vessel disease (SVD), which is considered a disconnection syndrome due to the presence of vascular lesions (i.e., white matter hyperintensities, lacunes, and microbleeds) at crucial locations between frontal and subcortical areas (Pantoni, 2010). SVD is mainly of the sporadic type, it is widely prevalent in older adults aged 60 years and older (de Leeuw et al., 2001), often associated with vascular risk factors and is the most important vascular cause of cognitive impairment and dementia (Banerjee et al., 2016; Prins & Scheltens, 2015). Although executive dysfunction and worsening of processing speed are among the most commonly documented cognitive consequences of SVD, changes in other cognitive domains may occur as well, yet have only recently received more attention (Ter Telgte et al., 2018). Although

deficits in verbal fluency have been found in people with SVD, the impact of SVD on language remains poorly understood because language is not commonly assessed in this population (Camerino et al., 2021; Vasquez & Zakzanis, 2015).

PD is characterized by cell loss in the substantia nigra compacta (SNc), which leads to less activation through the direct circuit of the basal ganglia (Zarei et al., 2013). This loss also leads to less inhibition through the indirect circuit, and these phenomena combined lead to the characteristic hypokinetic motor symptoms of PD. PD is also associated with cortical pathology at various stages of the disease (e.g., Hu et al., 2000). Non-motor cognitive impairments are also observed, with people with PD scoring significantly lower than cognitively unimpaired adults, particularly on executive functioning and memory tests (Verbaan et al., 2007). Additionally, language production problems have been reported, for example difficulties with morphosyntax, lexical-semantics, and word finding (Auclair-Ouellet et al., 2017; Magee et al., 2019). Ullman et al. (1997) observed that people with PD were worse at producing the past tense of regular compared to irregular verbs (e.g., say "walked" to the cue "to walk" versus say "sought" to the cue "to seek"). In line with a dual-system account (Pinker, 1999; Pinker & Ullman, 2002), Ullman et al. (1997) proposed that regular past-tense verb inflection requires application of a grammatical rule, whereas irregular verbs are generated by associative memory and inhibition of the regular rule. These processes are assumed to be subserved by the direct and indirect circuits of the basal ganglia, respectively. Because verbal fluency has already been thoroughly investigated in PD in recent years (see for reviews Henry & Crawford, 2004; Kudlicka et al., 2011; Muslimović et al., 2007; Vos et al., 2021; Wyman-Chick, 2016), we restricted our systematic review and meta-analysis to picture naming and verb inflection in the present study as these tasks might provide new insights into the deficits of people with PD.

HD is an autosomal dominant neurodegenerative disease caused by a mutation in the huntingtin gene that leads to death of medium spiny neurons (MSN) of the striatum in the indirect circuit, followed by loss of MSNs in the direct circuit (Plotkin & Surmeier, 2015). The early cell loss in the indirect circuit leads to the most characteristic motor HD symptom: chorea, i.e., a movement disorder characterized by abnormal, involuntary, brief, abrupt, unpredictable and irregular movements (Gagnon et al., 2018). Cognitive decline can be observed many years before the onset of motor symptoms, with people with HD showing deficits in executive function, processing speed, and visuomotor integration (Papoutsi et al., 2014). Regarding language production, people with HD show word finding difficulties (e.g., Azambuja et al., 2012), reduction of syntactic complexity in spontaneous speech (e.g., Murray & Lenz, 2001), or intact syntactic complexity but impaired grammaticality (e.g., Jensen et al., 2006). Moreover, and complementary to the PD results, people with HD have been shown to regularize irregular verbs, suggesting a lack of inhibition of the regular rule (Ullman et al., 1997). However, in a recent review, Gagnon et al. (2018) noted that although language impairment seems to be present in HD, the exact nature of this deficit still remains unclear.

1.2. The present study

Previous reviews on populations with basal ganglia dysfunction have often bundled together

comprehension and production tasks, which makes it difficult to disentangle and understand the role of basal ganglia in language-related processes. Moreover, language deficits are usually attributed to hypoperfusion of language-related cortical areas in cases of basal ganglia strokes (Radanovic & Mansur, 2017), and mixed motor, executive, and general cognitive ability impairments in cases of PD (Altmann & Troche, 2011) and HD (Gagnon et al., 2018). Here, we used the same search strategy (see below) and methodological approach to perform a systematic review and meta-analyses of four conceptually or lexically driven word production tasks, comparing the performance of individuals with basal ganglia stroke, SVD, PD, and HD to that of matched cognitively unimpaired adults. By systematically quantifying performance in terms of verbal fluency, accuracy in picture naming, and in verb inflection, we ensured that poor performance in word production could not be easily explained by motor deficits. Moreover, by looking across vascular and non-vascular pathologies of the basal ganglia, we sought evidence for an account of the role of the basal ganglia in language production that would extend beyond the mechanism of cortical hypoperfusion.

2. Methods

The preparation of this systematic review and meta-analyses was carried out following the PRISMA guidelines (Page et al., 2021). This study was pre-registered in the Open Science Framework (OSF, available at https://osf.io/z9k6s, including data and code to reproduce the present results). Our initial goal was to perform a scoping review on language production deficits as a consequence of basal ganglia damage or disorder. As such, the search string used was developed to include all basal ganglia related disorders and diseases and all production tasks. During the study selection process, it became clear that quantification in the form of a meta-analysis was feasible given the substantial number of studies found for some pathologies and some tasks. Our inclusion criteria were then adapted accordingly to only select studies that reported the pathologies and tasks of interest, as reported in the present meta-analyses.

2.1. Search strategy

Two databases, PubMed and PsycINFO, were used. The search strategy was developed with the help of a librarian from Radboud University's library and included MeSH terms and the equivalent PsycINFO controlled vocabulary terms (see Appendix). The search was completed in November 2019 and updated in March 2022. After the search was completed, duplicates were removed and screening was performed using the Covidence tool (*Covidence Systematic Review Software*; https://www.covidence.org/). First, each title and abstract were independently screened by two random reviewers following one restriction: at least one reviewer was one of the authors (I.C., J.F., V.P.), whereas the second reviewer could be a research assistant. After title and abstract screening, the full-text of the included studies was examined by two reviewers, following the same rule. The reviewers evaluated the studies independently and any discrepancy in the decisions were resolved after discussion among the reviewers. Additionally, the reference lists of 22 relevant review articles found with the search string were manually screened to identify potentially relevant articles to be added to the present review.

2.2. Eligibility criteria and study selection

A study was included if: 1) patients were diagnosed with a) ischemic or hemorrhagic stroke in the basal ganglia confirmed by neuroimaging, b) SVD based on the presence of MRI markers of SVD, c) symptomatic PD in individuals not treated with deep brain stimulation, or d) HD confirmed by genetic testing (all but two studies were on manifest HD, with two studies on pre-manifest HD, Mason et al. (2015) and Van den Stock et al. (2015); 2) it reported sufficient quantification of the word production tasks of interest (i.e., mean, SD, and sample size, or standardized effect sizes, or exact *t*- or *F*-values); 3) it provided sufficient information on the cognitive status of the participants to exclude the presence of dementia (note that this criterion was not applicable to the stroke and HD groups[1]); 4) there were no cortical lesions of any kind; 5) it was peer reviewed and published in a scientific journal; 6) written in English; 7) presence of a control group in the same study or normative data available. Given previous reviews and meta-analyses on verbal fluency for PD (Henry & Crawford, 2004; Kudlicka et al., 2011; Muslimović et al., 2007; Vos et al., 2021; Wyman-Chick, 2016), we did not include verbal fluency for PD in our meta-analysis. If two different studies reported results of the same task(s) from the same cohort, the study with the largest sample size was selected to avoid duplicate data. Detailed eligibility criteria for the study selection procedure are shown in the PRISMA flow diagram (Figure 2).

2.3. Data extraction

The determinants of interest were the presence of the diagnosis of a) an ischemic or hemorrhagic stroke in the basal ganglia, b) SVD, c) Parkinson's disease, or d) Huntington's disease. The outcome of interest was word production performance assessed with the following tests: a) category fluency (number of words produced within a time limit), b) letter fluency (number of words produced within a time limit), c) picture naming (accuracy) or d) past-tense inflection (accuracy, described as percentage of correct responses, number of correct responses, or number of errors).

Three researchers (I.C., J.F., and V.P.) extracted the data. For each of the four pathologies investigated, we extracted information about study design, type of word production task, and the respective performance of patients and, when available, of the control group. When a study did not provide language performance of a matched control group, we searched for normative data of the task (in peer-reviewed publications, books, and dissertations, in other languages than English), matching for language and where possible age, sex and education (see Table 1). If the published normative scores were stratified, for example by sex, education, or age, we combined the mean scores (Higgins et al., 2019), so they would best match the patient group. In some cases where the exact sample size for the stratified normative published scores was not available, we used the sample size of the non-stratified sample (which impacted the quality assessment of that study, see below).

[1] In the case of stroke, dementia is not usually assessed unless it is suspected; as such, the vast majority of studies screened and included did not report dementia status for stroke. In the case of HD, as the disease progresses, there is loss of general cognitive abilities, and as such, cognitive screening scores for this group are usually bellow cut-off values.

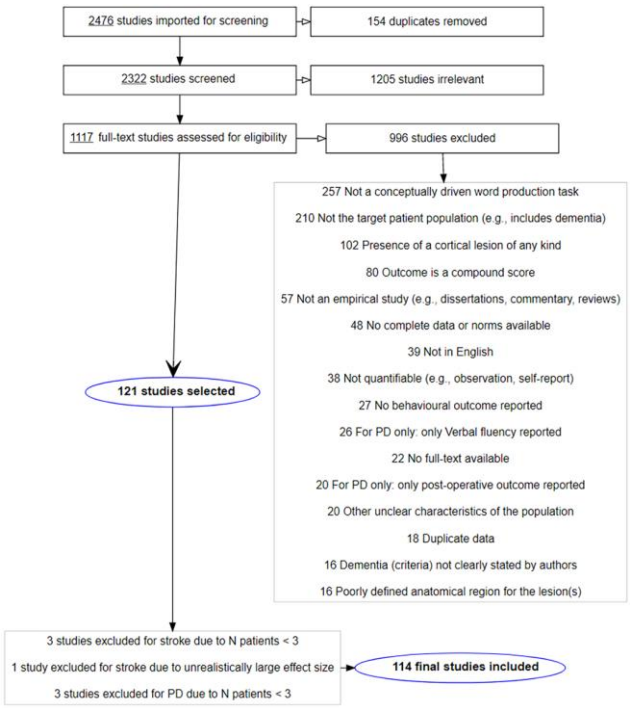

Figure 2. PRISMA flowchart.

For stroke studies, ten studies reported single cases. The scores of single-case studies were first converted into *z* scores relative to a control group or normative data. For each task, the scores were then combined (Higgins et al., 2019) and entered as a single study in the meta-analysis. Note that we also performed additional meta-analyses on studies *not* including the single cases. For papers reporting individual data for which only part of the sample met our criteria, data were extracted from subgroups that matched our criteria (e.g., when a study reported individual-level performance of multiple stroke patients with only a subgroup meeting the criterion of not having a cortical lesion). In studies with more than one participant group of the same population that matched our criteria, scores were combined and entered as a single group in the meta-analysis (Higgins et al., 2019). It is known that people with sporadic SVD differ from people with CADASIL in terms of etiology and age of onset of the disease (Charlton et al., 2006; Joutel et al., 1996). For this reason, SVD studies were categorized as either CADASIL (i.e., cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy) when this genetic variant was present, or sporadic for the remaining cases to be used in a follow-up subgroup analysis. Parkinson's is a degenerative disease characterized by progressive loss of cognitive functions and as such it is informative to have a measure of disease progression. Consequently, for PD studies, disease severity scores (UPDRS motor subscale, Fish, 2011, or Hoehnn and Yahr scale, Bhidayasiri & Tarsy, 2012) were extracted when available to be used as moderators in a follow-up analysis. For PD, in case performance was reported for both "on" and "off" medication, the "on" condition was used. When examining longitudinal studies, we took the baseline/first score for HD and PD because these groups may or are known to develop dementia at later disease stages (Massman et al., 1990; Turner et al., 2002). By contrast, for stroke patients, we took the score at the most chronic stage because cognitive performance at the subacute phase is known to be most severe and likely to improve considerably over time (Middleton et al., 2014). Finally, for all groups, if naming performance was reported for both object and action naming, object naming was used since this is the most common type of naming task.

2.4. Quality assessment and publication bias

Risk of bias for the studies included in the meta-analyses was determined for each disease and task combination by using a modified version of the Newcastle-Ottawa Quality Assessment Scale Cohort Studies (Peterson et al., 2011). This rating

system allowed us to evaluate the quality of each study by assigning a maximum of 9 stars. Our assessment was based on 1) the quality of the participants selection (maximum 3 stars); 2) quality of the outcome (maximum 2 stars); and 3) the comparability of the study groups (maximum 4 stars) in terms of age, sex, educational attainment, and whether the exact sample size was known in the case of normative scores. Additionally, to check for publication bias, we used funnel plots and calculated Egger's *t* statistics for all tasks combined for each pathology. The reason to combine tasks per pathology was that there is, in principle, no reason to assume any publication bias in a task-specific manner (except for past-tense inflection), and by combining across tasks we increase power and improve interpretability of the publication bias analyses. However, the past-test inflection task, for being purely experimental rather than part of standard neuropsychological evaluation, was an exception in this case. Given that experimental studies may tend to suffer more from publication bias (i.e., only published when there are significant results), this task was not combined with the other tasks for the publication bias analyses.

2.5. Effect size calculation

The extracted data from the studies was analyzed in RStudio (R Core Team, 2020), using the package *metafor* (Viechtbauer, 2010). First, all scores were transformed into a standardized mean difference, Hedges' *g* (using 'escalc', vtype = "AV"), which is a corrected effect size statistic for small sample sizes, and its corresponding (estimated) sampling variance using the sample-size-averaged estimator (Lin & Aloe, 2021). For one study (Hochstenbach et al., 1998), the *F* value was converted into Hedges' *g* using the R package *esc* (Lüdecke, 2019). For PD and HD studies with the inflection task, we also calculated for each group the difference between regular and irregular verb inflection: the mean difference in inflection was calculated as irregular minus regular. For SDs we calculated the pooled SD using Equation 1, after Cumming (2012).

$$SD_{po} = \frac{\sqrt{SD_{IRREG}^2 + SD_{REG}^2}}{2} \quad (1)$$

Two post-hoc decisions were made on additional studies to exclude (based on a Reviewer's suggestions): Studies with a patient sample size < 3 and studies with unrealistically large effect sizes. For the latter, the cut-off was set at 5 because this is the most extreme effect size commonly found in the neuropsychological field (Bezeau & Graves, 2001). One exception was made for the inflection task in HD, see Section 3.1 below for details.

2.6. Statistical analysis

Following conventions, an effect size of 0.20 was considered small, 0.50 medium, and 0.80 large (Cohen, 1988). A negative sign of the effect sizes reflected the patient group performing worse than the cognitively unimpaired comparison group.

We used random-effects models to obtain pooled estimates for each task and each pathology combination that had a sufficient number of studies (>4). If only 2-4 studies were found for a specific task and pathology, a fixed-effects model was used instead (which is indicated in the results when applicable). The alpha level was set at 0.05 for all analyses.

For all disease and task combinations that had enough studies (>4), a subgroup moderator analysis was done on the variable "type of comparison group" (i.e., whether the comparison

group originated from *norms* or a matched *control* group of individuals recruited within the same study). Note that for the inflection task, all studies had a control group, so a moderator analysis was not performed. For stroke, this subgroup analysis was conducted without the single case studies, since those were combined into a single score for the main analysis, containing both norms and control studies. For SVD, a subgroup moderator analysis was performed for CADASIL vs sporadic SVD studies. For PD, a moderator analysis was performed for each disease severity rating scale (UPDRS motor subscale and Hoehn and Yahr scale) for PD studies for which this information was available. In the subgroup moderator analysis, we used Cochran's Q-test to test for heterogeneity. The QM statistic refers to a test of differences between subgroups; a significant test result suggests that the moderating variable influences the heterogeneity. The QE statistic indicates the residual heterogeneity after taking the moderator into account. Alpha for the Q-test was set at $p = .10$, as typically done (Pereira et al., 2010). We evaluated heterogeneity by visually checking the overlap of the confidence intervals displayed in the forest plots and the *I*-squared statistic. We interpreted the level of heterogeneity by following the recommendation of the Cochrane Handbook (Higgins et al., 2019). Additionally, we calculated tau-square to estimate the extent of the between-study variance.

3. Results

3.1. Search results and study characteristics

The search returned a total of 2,322 studies after exclusion of duplicates, leading to a final number of 121 studies being selected after screening (Figure 2; details in Table 1). Of the 121 selected studies, 7 studies were excluded due to unrealistically large effects sizes or a patient group sample size < 3, and for da Silva et al. (2011) and Robin and Schienberg (1990) the naming tasks were excluded but the category fluency was kept because of this same criteria. From the final 114 included studies, 22 were on BG stroke, 25 on SVD, 60 on PD and 11 on HD. Four studies (Longworth et al., 2005; Randolph et al., 1993; Tröster et al., 1998; Ullman et al., 1997) included samples of HD and PD patients that met our criteria, and were included in both analyses. For stroke, 15 studies reported naming tasks (3/15 with a control group, 9/15 were single case studies), 13 reported category fluency tasks (2/13 with a control group, 6/13 were single case studies), and 6 reported letter fluency tasks (1/6 with a control group, 4/6 were single case studies). For SVD studies, 16 reported naming tasks (9/16 with a control group, 2/16 belonging to the CADASIL group), 16 reported category fluency tasks (10/16 with a control group, 5/16 belonging to the CADASIL group), and 7 reported letter fluency tasks (3/7 with a control group, 1/7 belonging to the CADASIL group). For the PD studies, 54 reported naming tasks (27/54 with control group, 15/54 with UPDRS motor subscale scores, 11/54 with Hoehn and Yahr scale scores), and 6 reported the inflection task, with scores for both regular and irregular production. Finally, from the HD studies, 8 reported naming tasks (6/8 had a control group), 2 reported category fluency tasks, 3 reported letter fluency tasks, and 2 reported the inflection task, with scores for both regular and irregular production. One of the HD studies had an effect size > 5 for irregular inflection only. However, we still decided to include this study despite the post-hoc exclusion criteria, since for inflection for HD only two studies were selected, and as such it would be informative to include all regularities in our analysis.

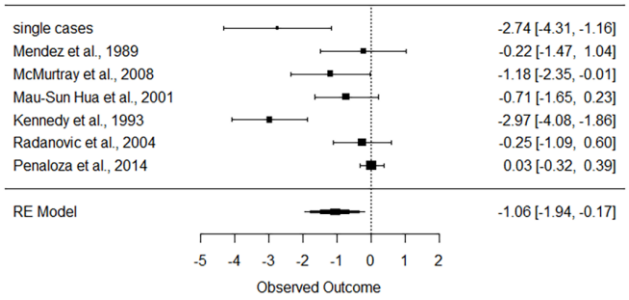

Figure 3. Forest plot of the studies with naming tasks in individuals with basal ganglia stroke versus comparison group. The study labeled "single cases" corresponds to the combined single-case studies.

3.2. Quality assessment

Quality assessment for all task and pathology combinations is described in Supplementary Tables S1, S2, S3 and S4. Studies scored between 4 and 9 stars. On the participant selection criteria, studies lost one star if they had *norms* as type of comparison group (stroke: 12/15 for naming, 11/13 for category fluency, 5/6 for letter fluency; SVD: 7/16 for naming, 6/16 for category fluency, 4/7 for letter fluency; PD: 28/54 for naming; HD 2/8 for naming, 1/3 for letter fluency). Also on the participant selection criteria, all five CADASIL studies lost one star for representativeness due to external validity related to our research question because their SVD is not sporadic SVD (age-related) but rather due to a variation of the NOTCH3 gene. All studies scored the maximum number of stars on the assessment of outcome. On comparability, studies lost stars if patients and the comparison group was not matched on demographic characteristics (stroke: 13/15 for naming, 13/13 for category fluency, 5/6 for letter fluency; SVD: 10/16 for naming, 5/16 for category fluency, 6/7 for letter fluency; PD: 37/54 for naming, 2/6 for inflection; HD: 4/8 for naming, 1/2 for category fluency, 2/3 for letter fluency, 2/2 for inflection). Finally, for comparability, studies lost stars if the exact *n* for the stratified normative published scores was unavailable (stroke: 1/15 for naming, 4/13 for category fluency, 3/6 for letter fluency; SVD: 1/16 for category fluency, 5/7 for letter fluency; PD: 3/54 naming).

3.3. Meta-analyses

3.3.1. Stroke

Overall, people with BG stroke performed worse than cognitively unimpaired adults across all examined tasks (i.e., naming, category fluency, and letter fluency).

All naming task effect sizes for the included studies of BG stroke are shown in Figure 3 (*N* studies = 15). Analysis showed a large effect size with a Hedges' *g* of −1.057 (*SE* = 0.453, 95% *CI* [−1.945, −0.170], τ^2 = 1.142), with a high heterogeneity in terms of between-study differences in variation (I^2 = 85.03%), with people with BG stroke performing worse than cognitively unimpaired adults (*p* = 0.020). Moreover, the subgroup moderator analysis that compared studies with norms vs controls (*N*

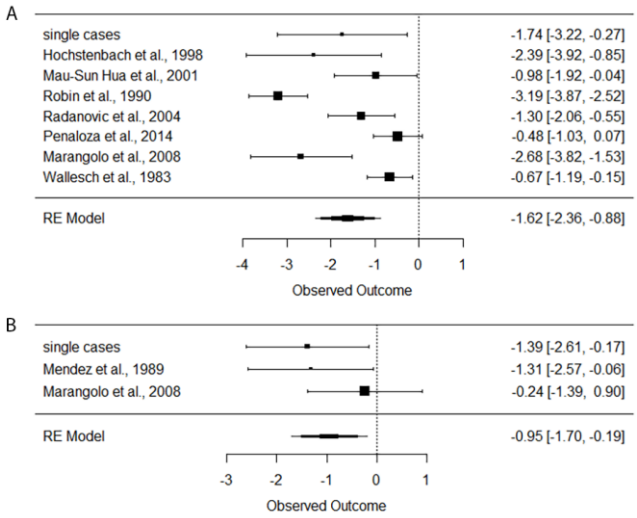

Figure 4. Forest plot of the studies with (A) category fluency tasks and (B) with letter fluency tasks in individuals with basal ganglia stroke versus comparison group. The aggregated single cases are labeled accordingly.

studies = 12 vs 3) indicated that type of comparison group did not explain the heterogeneity (QM = 0.065, df = 1, p = 0.798), with high residual heterogeneity (QE = 24.454, df = 4, p < 0.001).

Category fluency results (N studies = 13) showed a large effect size with a Hedges' g of −1.617 (SE = 0.377, 95% CI [−2.356, −0.878], τ^2 = 0.947), with a high heterogeneity in terms of between-study differences in variation (I^2 = 84.56%), with a worse performance for people with BG stroke compared to cognitively unimpaired adults (p < 0.001), as presented in Figure 4A. Subgroup moderator analysis that compared studies with norms vs controls (N studies = 11 vs 2) indicated that type of comparison group did not explain the heterogeneity (QM = 0.001, df = 1, p = 0.986), with high residual heterogeneity (QE = 52.264, df = 5, p < 0.001).

As shown in Figure 4B (N studies = 6), the effect size for letter fluency was large, with a Hedges' g of -0.948, SE = 0.386, 95% CI [−1.705, −0.190], τ^2 = 0.262), with a low heterogeneity in terms of between-study differences in variation (I^2 = 15.31%), with people with BG stroke performing worse than cognitively unimpaired adults (p = 0.014). Subgroup moderator analysis was not conducted since there were not enough studies left for the comparison.

Since single-case studies were combined and introduced in the analysis as one single study, we additionally performed all of the described above analysis without the single-case studies. This post-hoc analysis yielded similar results to the main analysis (Hedges' g of −0.831 for naming, N studies = 5; −1.610 for category fluency, N studies = 7; and −0.744 for letter fluency, N studies = 2), with people with BG stroke performing worse than cognitively unimpaired adults across all tasks.

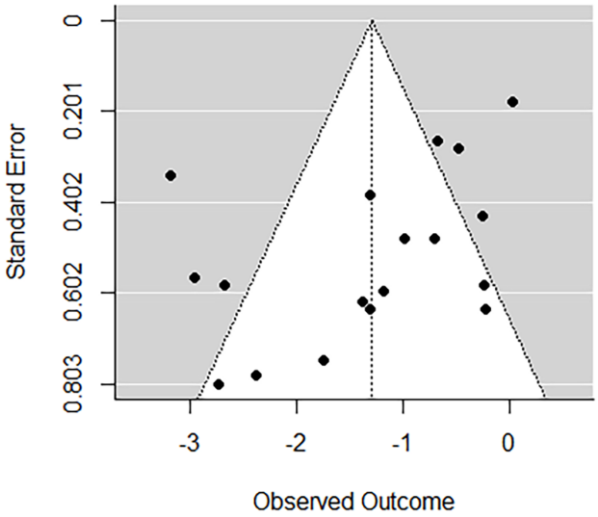

Figure 5. Funnel plot of all studies with naming, category fluency, and letter fluency tasks for the basal ganglia stroke analyses.

The funnel plot for stroke for all tasks was not fully symmetrical as depicted in Figure 5 (N studies = 22), and the rank correlation test for funnel plot asymmetry was statistically significant (Kendall's τ = −0.343, p = 0.048).

3.3.2. Small vessel disease

Overall, people with SVD performed consistently worse than cognitively unimpaired adults on all examined tasks (i.e., naming, category fluency, and letter fluency), regardless of the type of comparison group. Moreover, CADASIL individuals seem to perform slightly worse than the sporadic SVD on naming.

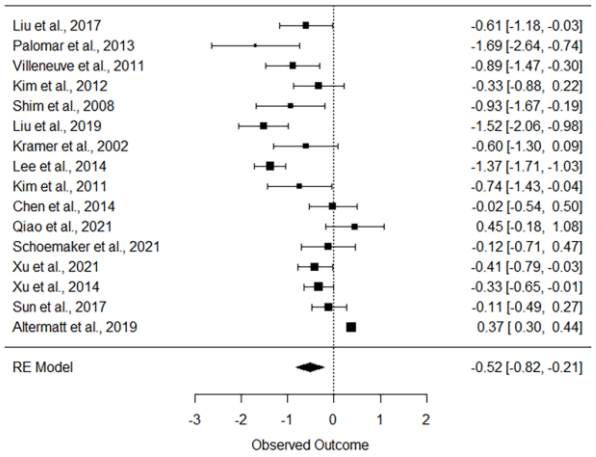

Figure 6. Forest plot of the studies with naming tasks in individuals with small vessel disease versus comparison group.

All naming task effect sizes for the included studies of SVD are shown in Figure 6 (N studies = 16). Results showed a moderate effect size with a Hedges' *g* of −0.518 (*SE* = 0.155, 95% *CI* [−0.821, −0.215], τ^2 = 0.305), with a high heterogeneity in terms of between-study differences in variation (I^2= 89.31%), with people with SVD performing worse than cognitively unimpaired adults (*p* = 0.001). The first moderator analysis that compared studies with norms vs controls (*N* studies = 7 vs 9) indicated that type of comparison group did not explain the heterogeneity (*QM* = 1706, *df* = 1, *p* = 0.192), with high residual heterogeneity (*QE* = 111.079, *df* = 14, *p* < 0.001). The second moderator analysis indicated that the difference between people with SVD and cognitively unimpaired adults was not significant for CADASIL vs sporadic SVD individuals (*N* studies = 2 vs 14, *QM* = 2.857, *df* = 1, *p* < 0.091), although the residual heterogeneity was still high (*QE* = 214.293, *df* = 14, *p* < 0.001).

For the category fluency task depicted in Figure 7A (*N* studies = 16), we found a large effect size with a Hedges' *g* of −0.723 (*SE* = 0.141, 95% *CI* [−0.999, −0.448], τ^2 = 0.260), with a high heterogeneity in terms of between-study differences in variation (I^2 = 94.74%), showing that people with SVD performed worse than cognitively unimpaired adults (*p* < 0.001). The first moderator analysis that compared studies with norms vs controls (*N* studies = 6 vs 10) indicated that type of comparison group did not explain the heterogeneity (*QM* = 0.851, *df* = 1, *p* = 0.356), with high residual heterogeneity (*QE* = 72.405, *df* = 14, p < 0.001). The second moderator analysis indicated that the difference between people with SVD and cognitively unimpaired adults was larger for CADASIL vs sporadic SVD individuals (*N* studies = 5 vs 11, *QM* = 4.942, *df* = 1, *p* = 0.026), although the residual heterogeneity was still high (*QE* = 120.447, *df* = 14, *p* < 0.001).

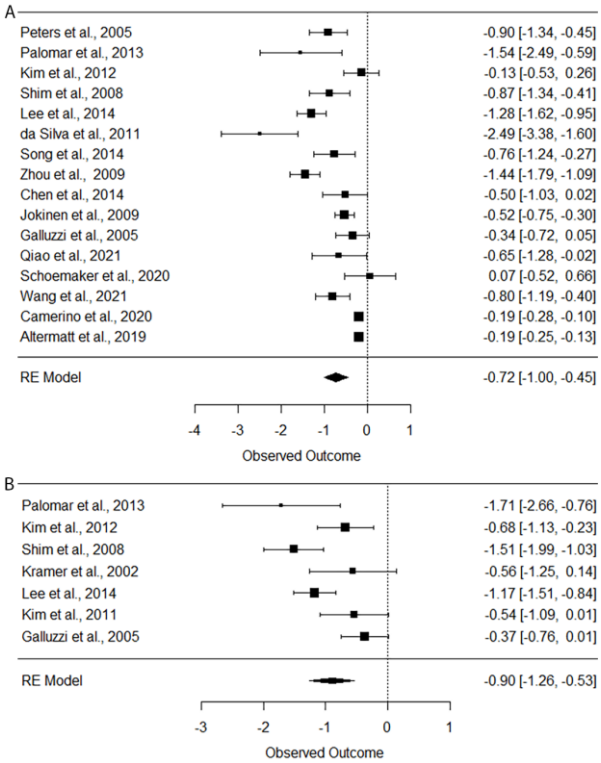

Figure 7. Forest plot of the studies with (A) category fluency tasks and (B) with letter fluency tasks in individuals with small vessel disease versus comparison group.

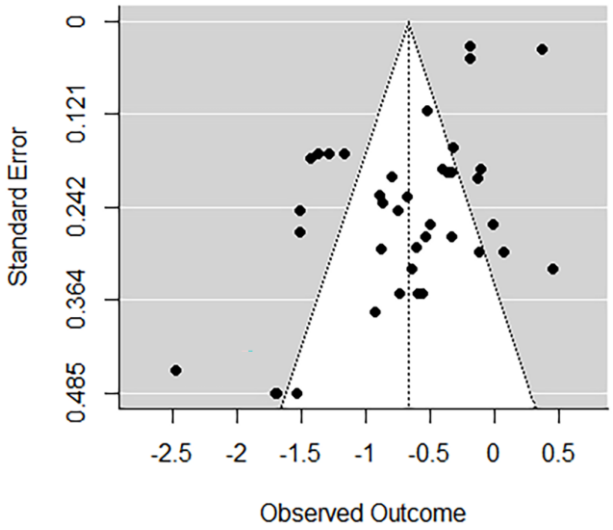

Figure 8. Funnel plot of all studies with naming, category fluency, and letter fluency tasks in individuals with small vessel disease versus comparison group.

As depicted in Figure 7B (*N* studies = 7), the letter fluency analysis showed a large effect size with a Hedges' *g* of −0.896 (*SE* = 0.185, 95% *CI* [−1.259, −0.533], τ^2 = 0.165), with a moderate to high heterogeneity in terms of between-study differences in variation (I^2 = 72.86%), showing that people with SVD performed worse than cognitively unimpaired adults ($p < 0.001$). Unlike naming and category fluency, letter fluency subgroup analysis showed no difference between studies with norms vs controls (*N* studies = 4 vs 3, *QM* = 0.736, *df* = 1, *p* = 0.391; *QE* = 18.044, *df* = 5, *p* = 0.003); nor for CADASIL vs sporadic SVD (*N* studies = 1 vs 6, *QM* = 1.910, *df* = 1, *p* = 0.167; *QE* = 19.171, *df* = 5, *p* = 0.002).

Finally, the funnel plot for all tasks for SVD was not fully symmetrical as depicted in Figure 8 (*N* studies = 25), but the rank correlation test for funnel plot asymmetry was not statistically significant (Kendall's τ = −0.014, *p* = 0.904).

3.3.3. Parkinson's disease

Overall, people with PD performed worse than cognitively unimpaired adults across all examined tasks (i.e., naming and inflection, both regular and irregular), regardless of type of comparison group or disease severity.

All naming task effect sizes for the included studies of PD are shown in Figure 9 (*N* studies = 54). We found a moderate effect size with a Hedges' *g* of −0.457 (*SE* = 0.099, 95% *CI* [−0.651, −0.263], τ^2 = 0.455), with a high heterogeneity in terms of between-study differences in variation (I^2 = 89.77%), with people with PD performing worse than cognitively unimpaired adults ($p < 0.001$). Moderator analysis that compared studies with norms vs controls (*N* studies = 27 vs 27) indicated that type of comparison group did not explain the heterogeneity (*QM* = 0.315, *df* = 1, *p* = 0.575), with high residual heterogeneity (*QE* = 438.651, *df* = 55, $p < 0.001$). Disease severity moderator analysis for the UPDRS motor subscale (*N* studies = 15) showed disease severity not to explain heterogeneity (*QM* = 1.267, *df* = 1, *p* = 0.260), with high residual heterogeneity (*QE* = 193.440, *df* = 13, $p < 0.001$). Moderator analysis for the studies reporting the Hoehn and Yahr scale (*N* studies = 11) showed a similar pattern (*QM* = 4.235, *df* = 1, *p* = 0.040; *QE* = 86.063, *df* = 9, $p < 0.001$). The funnel plot for the naming task for PD was not fully symmetrical (Figure 10). However, the rank correlation test for funnel plot asymmetry was not statistically significant (Kendall's τ = −0.025, *p* = 0.788).

Forest plots for the inflection task are depicted in Figure 11 (*N* studies = 6). In the inflection task for regular verbs (Figure 11A), we found a large effect size with a Hedges' *g* of −1.103 (*SE* = 0.304, 95% *CI* [−1.699, −0.506], τ^2 = 0.430), with a high heterogeneity in terms of between-study differences in variation (I^2 =

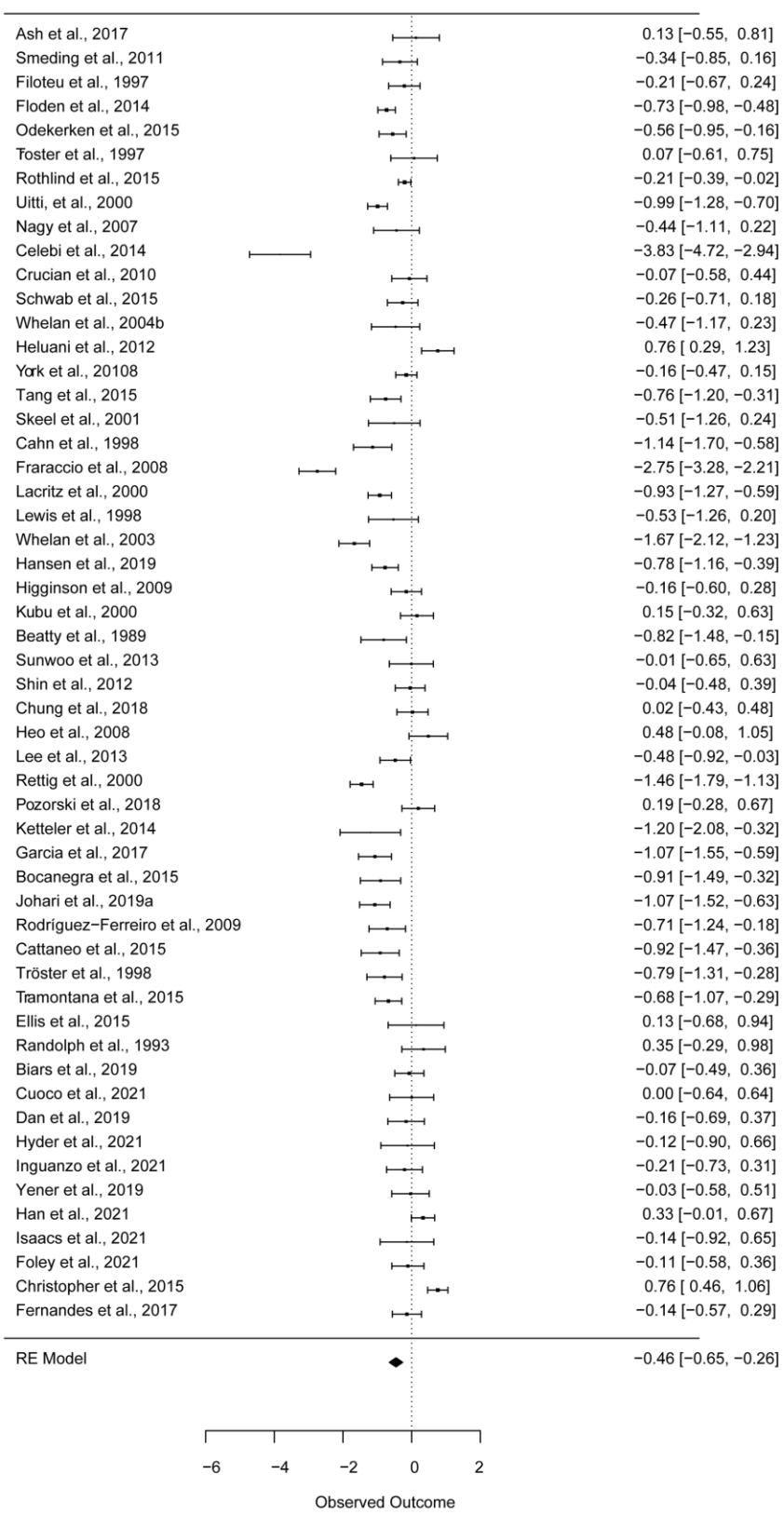

Figure 9. Forest plot of the studies with naming tasks in individuals with Parkinson's disease versus comparison group.

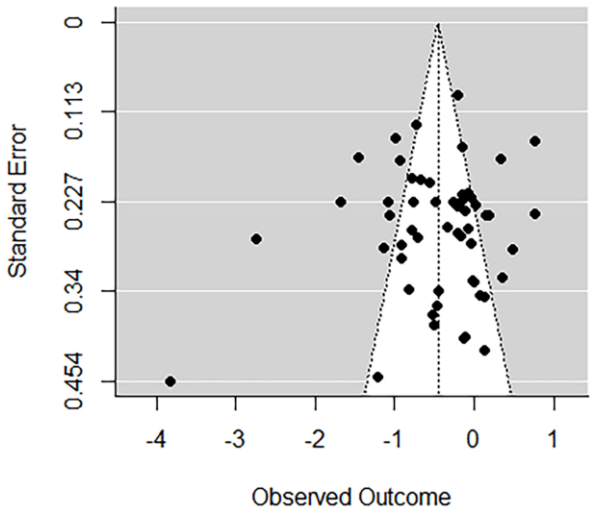

Figure 10. Funnel plot of the studies with naming tasks in individuals with Parkinson's disease versus comparison group.

than cognitively unimpaired adults ($p < 0.001$). A similar pattern was found for the inflection task with irregular verbs (Figure 11B): meta-Hedges' g of −0.786 (SE = 0.283, 95% CI [−1.341, −0.231], $\tau^2 = 0.358$), with high heterogeneity (I^2 = 78.98%), and with people with PD performing worse than cognitively unimpaired adults (p = 0.006). When comparing irregulars with regulars directly, as can be seen in Figure 11C, we did not find any difference between people with PD and cognitively unimpaired adults (p = 0.500): meta-Hedges' g of 0.216 (SE = 0.320, 95% CI [−0.412, 0.844], $\tau^2 = 0.489$), with high heterogeneity (I^2 = 83.71%).

3.3.4. Huntington's disease

Overall, across all examined tasks (i.e., naming, category fluency, letter fluency, and inflection, both regular and irregular), people with HD performed worse than cognitively unimpaired adults.

All naming task effect sizes for the included studies of HD are shown in Figure 12A (N studies = 8). Results showed a large effect size with a Hedges' g of −1.636 (SE = 0.345, 95% CI [−2.312, −0.960], $\tau^2 = 0.784$), with a high heterogeneity in terms of between-study differences in variation (I^2 = 83.55%), with people with HD performing worse than cognitively unimpaired adults ($p < 0.001$). Subgroup moderator analysis that compared studies with norms vs controls (N studies = 2 vs 6) indicated that type of comparison group did not

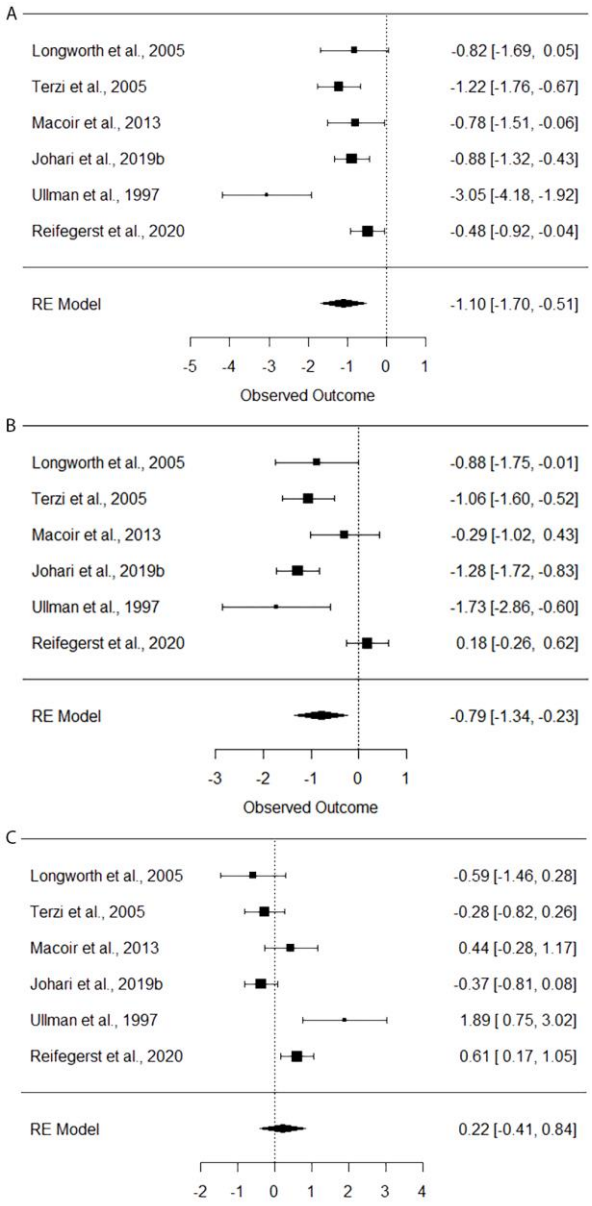

Figure 11. Forest plot of the studies with inflection tasks for (A) regular verbs, (B) irregular verbs and (C) irregular minus regular verbs in individuals with Parkinson's disease versus comparison group.

explain the heterogeneity (QM = 0.188, df = 1, p = 0.665), with high residual heterogeneity (QE = 42.326, df = 6, p < 0.001).

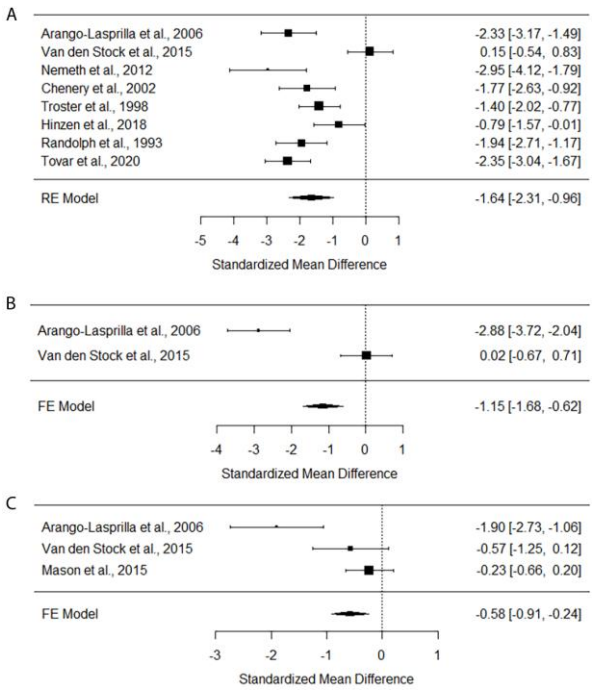

Figure 12. Forest plot of the studies with (A) naming tasks, (B) category fluency tasks, and (C) letter fluency tasks in individuals with Huntington's disease versus comparison group.

As depicted in Figure 12B (N studies = 2), the fixed effects analysis for the category fluency task showed a large effect size, with a Hedges' g = −1.148 (SE = 0.271, 95% CI [−1.679, −0.617]), with a high heterogeneity in terms of between-study differences in variation (I^2 = 96.38%, note that this is based on only two studies), with people with HD performing worse than cognitively unimpaired adults (p < 0.001). The fixed effects analysis for the letter fluency task (Figure 12C, N studies = 3) showed a moderate effect size with a Hedges' g of −0.579 (SE = 0.171, 95% CI [−0.914, −0.243]), with a high heterogeneity in terms of between-study differences in variation (I^2 = 83.40%), with people with HD performing worse than cognitively unimpaired adults (p < 0.001).

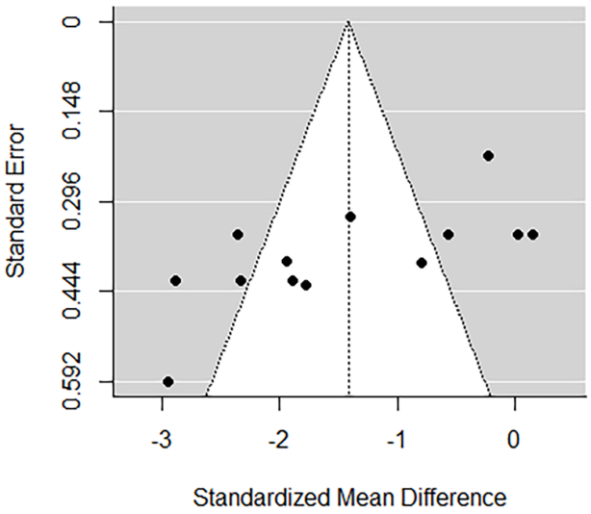

Figure 13. Funnel plot of the studies with naming and fluency tasks in individuals with Huntington's disease versus comparison group.

The funnel plot for naming and fluency tasks for HD (N studies = 13) was not fully symmetrical (Figure 13), and the rank correlation test for funnel plot asymmetry was statistically significant (Kendall's τ = −0.477, p = 0.029).

Forest plots for the inflection task for HD are depicted in Figure 14 (N studies = 2). For regular verbs (n = 2, Figure 14A), a large effect size was observed with a Hedges' g of −2.497 (SE = 0.356, 95% CI [−3.194, −1.800]), with a high heterogeneity in terms of between-study differences in variation (I^2 = 92.93%), with people with HD performing worse than cognitively unimpaired adults (p < 0.001). A similar result was observed for irregular verbs (Figure 14B), with a Hedges' g of −3.704 (SE = 0.356, 95% CI [−4.400, −3.007]), with a high heterogeneity in terms of between-study differences in variation (I^2 = 96.26%), and with people with HD performing worse than cognitively unimpaired adults (p < 0.001). Finally, when comparing irregulars with regulars directly, patients seem to perform worse than cognitively unimpaired adults (p = 0.027), showing particular difficulties with irregulars

(Figure 14C): meta-Hedges' *g* size of −0.786 (*SE* = 0.356, 95% *CI* [−1.483, −0.089]), with low heterogeneity (I^2 = 0.00%). We note that these results are based on two studies, a fixed-effects analysis and including one study with effect size > 5 despite our exclusion criteria. Thus, these results should be taken with extreme caution.

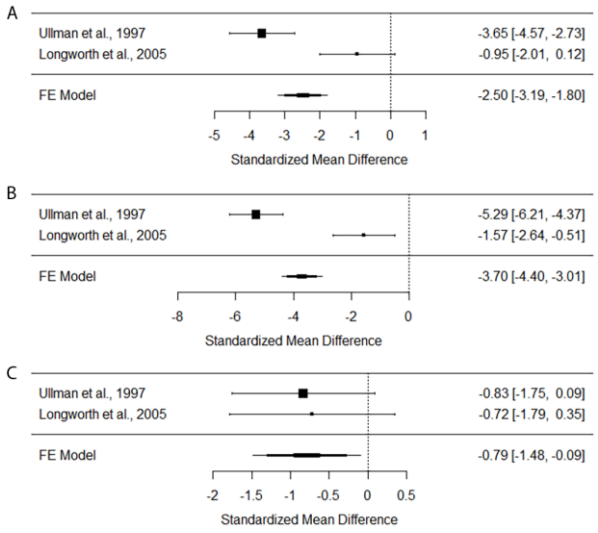

Figure 14. Forest plot of the studies with inflection tasks for (A) regular verbs, (B) irregular verbs and (C) irregular minus regular verbs in individuals with Huntington's disease versus comparison group.

4. Discussion

In the present systematic review and meta-analyses, we demonstrated that individuals with BG strokes, SVD, PD, or HD perform significantly worse than cognitively unimpaired adults on naming, verbal fluency, and verb inflection tasks. These findings are based on a measure of effect sizes that is corrected for small sample sizes and the most accurate effect size estimate in light of unequal variance (Lin & Aloe, 2021; Marfo & Okyere, 2019). Previous reviews have described language impairments in these populations (e.g., Gagnon et al., 2018; Radanovic & Mansur, 2017; Smith & Caplan, 2018; Vasquez & Zakzanis, 2015), conflating comprehension and production abilities, possibly painting a picture of heterogeneity in terms of language deficits. By looking into a set of well-described tasks that tap into the conceptual and lexical levels of spoken word production and by directly comparing patients' scores to those of matched cognitively unimpaired participants, we were able to show a more fine-grained picture of production deficits following basal ganglia pathology.

4.1. Mechanism that leads to the deficits

Given that differences between patients and controls are not limited to timed tasks (i.e., category or letter fluency), our results support the notion that the word production deficits observed in these populations cannot solely be explained by deficits in processing speed or motor speech. This observation suggests that the basal ganglia are involved in conceptually and lexically driven word production. One proposed mechanism for basal ganglia dysfunction leading to language deficits is related to disruption of cortical hemodynamics (Nadeau & Crosson, 1997; Radanovic & Mansur, 2017). As such, the deficits in word production we observed in people with BG stroke could be due to hypoperfusion of the perisylvian cortical areas, in particular the inferior frontal gyrus. However, as an explanation, this mechanism is limited to the cerebrovascular patient group. Although there is evidence for hypoperfusion of cortical areas in people with PD (Borghammer, Cumming, Aanerud, Förster, et al., 2009; Borghammer, Cumming, Aanerud, & Gjedde, 2009; Eckert et al., 2007; Fernández-Seara et al., 2012), there is little support for cortical hypoperfusion in people with HD (Hasselbalch et al., 1992; Sax et al., 1996), for which cortical pathology (e.g., cell loss) may be more likely to impact cortical function (Estrada-Sánchez et al., 2013; Rüb et al., 2016). In PD,

cognitive deficits may also relate to cortical dysfunction, (Lewy body) pathology, and noradrenergic and cholinergic changes, rather than just reduced input from basal ganglia (e.g., Aarsland et al., 2021; Hu et al., 2000). Relatedly, another possibility is that cortical areas, such as the inferior frontal gyrus, are dysfunctional due to a loss of neuronal input from the damaged basal ganglia (i.e., diaschisis). Such physiological dysfunction of cortical areas may hold for all diseases examined (i.e., stroke, small vessel, PD, HD). Another, not mutually exclusive possibility, is that SVD, which is present in virtually every individual over the age of 60, explains our findings across all tasks and populations examined.

It thus remains unclear what the exact mechanism is that leads to word production deficits in basal ganglia dysfunction, and it is an open question whether this mechanism is the same across different pathologies. Moreover, although we selected basal ganglia specific pathologies, we cannot exclude that word production in these patients might be affected by additional (and independent) cortical or periventricular pathology as well, as discussed above. However, our findings clearly indicate that subcortical dysfunction is not negligible and should be taken into consideration in the understanding of word production impairments. Future studies should further investigate functional and structural connections between the basal ganglia and frontal-temporal-parietal cortical regions in relation to language production.

While our results highlight the involvement of the basal ganglia in conceptually and lexically driven word production, it remains unknown how this process is implemented in the cortico-basal loops. The fact that basal ganglia related pathologies do not give rise to marked production deficits such as those observed in cortical aphasia suggests that this circuit facilitates production, but is not essential to it. This circuit could be directly responsible for some processes such as rule application and inhibition in inflection (Pinker, 1999; Pinker & Ullman, 2002). According to this proposal, people with PD or HD disease should show opposite symptoms, with people with PD showing more difficulties with regular verb inflection and people with HD with irregular inflection. However, the present meta-analyses do not fully support this prediction (note that the meta-analysis for HD only included two studies). Alternatively, the basal ganglia may have a more general and regulatory role of increasing the signal-to-noise ratio during the selection of the appropriate lexical or phonological items and their corresponding motor programs (Bohsali & Crosson, 2016). These functions would be of particular importance in situations of uncertainty and conflict, such as overcoming dominant responses, integrating and updating information, or sequencing linguistic elements (Copland et al., 2021). These processes may either take place in the basal ganglia itself or be accomplished in conjunction with cortical areas and the circuits involved may vary depending on the cognitive demands involved. We encourage future studies to disentangle this issue by administering tasks that tap into the different levels of the core processes in word production in these patient populations.

The core processes are made explicit by a prominent, computationally implemented theory of word production that was originally proposed by Levelt et al. (1999) and that has been further developed and extended during the past two decades. Extensions include computer

simulations of word production impairments due to stroke (Roelofs, 2014) or neurodegenerative disease (Roelofs, 2022). Word production is taken to consist of conceptual preparation, lexical selection, and word-form encoding, which is further subdivided into morphological, phonological, and phonetic encoding. Whereas inflection tasks target morphological encoding, windows into other core processes are provided by a number of picture-naming paradigms, including picture-word interference, continuous naming, and blocked-cyclic naming (see de Zubicaray and Piai, 2019, for a review). These paradigms manipulate contextual variables, like semantic or phonological relationships, to elucidate lexical selection and phonological encoding, among other processes, and may be administered in patients with BG strokes, SVD, PD, or HD.

Given the results of our systematic review and meta-analyses, the inclusion of a thorough language production examination in clinical assessment could be important to understand the full clinical picture of people with pathologies of the basal ganglia. For example, in people with SVD, language production is not routinely assessed. In agreement with Telgte and colleagues (2018), we argue that the cognitive profile of SVD is more diverse than previously recognized, and that future neuropsychological evaluations of people with SVD should not only include tests of executive function and processing speed, but also language production. Moreover, neuropsychological evaluation of patients in the four pathologies here examined usually relies on cut-off scores specifically created for aphasia diagnosis. As we demonstrated, word production deficits can be present, albeit in milder forms, even though patients do not perform below the cut-off score for aphasia on production tests. Although these mild symptoms can often go undetected, they should not be neglected as they can significantly affect patients' communication abilities and consequently their quality of life. Since these traditional cut-off scores lack sensitivity for less severe levels of impairment, an updated cut-off range that will allow clinicians to detect these milder deficits could proof useful. We believe a more comprehensive assessment can aid a better diagnosis, and more importantly, assist in providing tailored interventions to improve communication in daily life.

The populations included in this review have different forms of pathology that impact on basal ganglia function either through direct damage, neurotransmitter dysregulation, or disconnection. In terms of stroke, striatocapsular infarcts commonly involve the putamen, the head of the caudate, and the internal capsule (anterior limb) although the globus pallidus, external capsule and periventricular corona radiata can also be damaged (Nadeau & Crosson, 1997). SVD primarily involves subcortical vascular lesions that are heterogenous in location and extent. Subcortical white matter lesions are often observed in periventricular white matter and in the region of the external capsule and corona radiata (Duering et al., 2013) while lacunes are commonly observed in the basal ganglia, thalamus, internal capsule and pons (Pantone et al., 2010). A hallmark feature of PD is dopaminergic loss in the nigrostriatal system leading to altered striatal output, with dopamine depletion progressing from the dorsal to ventral striatum with disease progression and cognitive symptoms varying depending on endogenous and exogenous dopamine levels (Cools, 2006). As described above, the early stages of HD are characterized by striatal degeneration impacting the indirect loop followed by the direct loop.

Overall, while there is variation in which subcortical structures may be impacted in these populations depending on the nature of the vascular insult or disease progression, the disruption of striatal output is commonly observed across the groups, allowing us to draw conclusions regarding the association between basal ganglia dysfunction and language production deficits.

4.2. Limitations

Our study has a number of limitations that may impact our conclusions. Firstly, we excluded studies if the patient group of SVD and PD had a diagnosis of dementia or if this information was not clear from the methods. For people with BG stroke, it may be less common practice to formally assess dementia. Consequently, we cannot rule out that some patients with BG stroke in the studied cohorts would meet the criteria for dementia in the strict sense. However, when clinicians suspect the presence of dementia, people with BG stroke are tested for cognitive and functional decline and a positive diagnosis is then explicitly reported on scientific publications, making it less likely that the study samples included individuals with apparent dementia. Secondly, the results of the present meta-analyses showed a highly heterogeneous in-between study variation for multiple tasks in all four pathologies. One possible factor that could be driving this heterogeneity is the variability in disease stages in the patient groups. Although the moderator analysis for disease severity for people with PD was not statistically significant, this information was only available for a small number of studies. Similarly, for HD, two of the included studies had patients in the pre-manifest stage of the disease. However, it is worth noting that the pattern of results of these studies are in line with the manifest patients. Future studies should further address the effect of disease severity in language abilities after basal ganglia pathology. Despite our attempts, heterogeneity remained high even within subgroup analyses, indicating that the factors we took into account in subgroup analyses did not fully explain the heterogeneity. This issue remains a limiting aspect of our meta-analyses.

As outlined in the methods section, our initial goal was to perform a scoping review looking at basal ganglia pathologies more broadly and all language production tasks. Consequently, we did not include terms such as “Parkinson” or “Huntington” for pathologies or “fluency” or “inflection” for the tasks in our pre-registered search strategy, which might have led to some studies initially being left out. However, we then manually screened the reference lists of existing reviews on these pathologies to address this caveat. We did not fully comply with our pre-registration of a scoping review as it became clear during the review process that specific meta-analyses were possible for certain disease groups and word production tasks. We thus chose to prioritize the meta-analyses over the pre-registration.

A large number of studies lost stars in the quality assessment, because the normative group found was not optimally comparable. For example, not all normative scores were stratified both for age and education. Although this does not affect the quality of the studies we included themselves, it makes the comparison with the normative group less accurate, possibly impacting the results of our meta-analyses. This was taken into account in our quality assessment by means of deducting one star for these studies, but note that it does not mean that the study itself had less quality.

We found evidence of publication bias for HD and stroke. This bias may affect some of our conclusions, as the effect of basal ganglia damage on performance in language production tasks may have been overestimated due to unpublished null-results.

4.3. Conclusions

In the present meta-analyses, we have presented converging evidence from stroke of the basal ganglia, SVD, PD and HD that conceptually and lexically driven word production is affected. Verbal fluency tasks are well-established neuropsychological measures of lexical access and we have shown that patients score lower than matched cognitively unimpaired adults. These deficits may be the result of impaired executive and/or motor functions in most of these patient groups. However, we have also shown the same pattern of results for accuracy in naming, which puts only minimal strain on executive and motor components of language production, in comparison to fluency. Finally, results from an experimental task like past-tense inflection, which also relies on lexical and phonological access in addition to morphology, showed that both people with PD or HD consistently performed worse than cognitively unimpaired adults. With the present evidence, it is not yet possible to draw conclusions on the exact mechanism for basal ganglia involvement in conceptual, lexical, and phonological processes in word production. Moreover, how basal ganglia dysfunction leads to production deficits remains unclear. However, our results provide new converging evidence that basal ganglia and perisylvian language areas might work together in supporting language production. Models of neurobiology of language should consider updating the classical language network to include subcortical areas which might support language production (cf. Roelofs, 2014; Roelofs & Ferreira, 2019). Finally, our results indicate that conceptually and lexically driven word production deficits after basal ganglia pathology are not negligible and should be taken into consideration during diagnosis, which can hopefully contribute to the development of interventions to help patients better cope with language production difficulties.

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Table 1. Details of the studies selected in the present review and meta-analyses

Reference	*n* patients	Etiology	Language assessment	Type of comparison group
Adrover-Roig et al. (2011)	1	Stroke	BNT and category fluency	Norm
Altermatt et al. (2019)	878	SVD	BNT and category Fluency	Norm
Arango-Lasprilla et al. (2006)	11	HD	BNT, animal fluency and letter fluency	Control
Ash et al. (2017)	15	PD	BNT (30)	Control
Beatty & Monson (1989)	25	PD	BNT	Control
Benke et al. (2003)*	2	Stroke	Category fluency	Norm
Biars et al. (2019)	24	PD	BNT	Norm
Bocanegra et al. (2015)	23	PD	Naming (action naming)	Control
Cahn et al. (1998)	13	PD	BNT	Norm
Camerino et al. (2021)	442	SVD	Category Fluency	Norm
Cappa et al. (1997)	1	Stroke	Oral naming, category fluency and letter fluency	Norm
Cattaneo et al. (2015)	28	PD	Naming	Control
Celebi et al. (2014)	10	PD	BNT	Control
Chen et al. (2014)	30	SVD (LACI)	BNT and category fluency	Control
Chenery et al. (2002)	13	HD	BNT	Control
Christopher et al. (2015)	11	PD	BNT	Norm
Chung et al. (2018)	182	PD	BNT	Norm
Crucian et al. (2010)	40	PD	BNT	Control
Cuoco et al. (2021)	17	PD	Naming	Control
da Silva et al. (2011)	6	SVD (CADASIL)	BNT* and category fluency	Norms
Dan et al. (2019)	25	PD	BNT	Control
Ellis et al. (2015)	12	PD	BNT	Control
Fernandes et al. (2017)	18	PD	BNT	Control
Filoteo et al. (1997)	20	PD	BNT	Norm
Floden et al. (2014)	85	PD	BNT	Norm
Foley et al. (2021)	26	PD	Naming	Norm
Fraraccio et al. (2008)	15	PD	BNT	Norm

Galluzzi et al. (2005)	29	SVD (MCI)	Category fluency and letter fluency	Norm
Garcia et al. (2017)	33	PD	Naming (action naming)	Control
Garcia-Caballero et al. (2007)	1	Stroke	Category fluency	Norm
Godefroy et al. (1994)*	2	Stroke	BNT, category fluency and letter fluency	Control
Gurd et al. (1988)	1	Stroke	Category fluency	Norm
Han et al. (2021)	41	PD	BNT	Norm
Hansen et al. (2019)	29	PD	BNT	Norm
Heluani et al. (2012)	20	PD	BNT	Norm
Heo et al. (2008)	46	PD	BNT	Norm
Higginson et al. (2009)	22	PD	BNT	Norm
Hinzen et al. (2018)	19	HD	BNT	Norm
Hochstenbach et al. (1998)	12	Stroke	Category fluency	Control
Hua et al. (2001)	18	Stroke	Naming (60) and category fluency	Control
Hyder et al. (2021)	13	PD	BNT	Control
Inguanzo et al. (2021)	26	PD	BNT	Control
Isaacs et al. (2021)	12	PD	BNT	Control
Johari et al. (2019a)	40	PD	Naming	Control
Johari et al. (2019b)	40	PD	Inflection (regulars and irregulars)	Control
Jokinen et al. (2009)	524	SVD	Category fluency	Control
Kennedy & Murdoch (1993)	4	Stroke	Naming (object)	Norm
Ketteler et al. (2014)	8	PD	BNT	Control
Kim et al. (2011a)	4	Stroke	Naming (KWAB)	Norm
Kim et al. (2011b)*	14	SVD (MCI)	BNT (60) and letter fluency	Norm
Kim et al. (2012)	25	SVD (MCI)	Naming, category fluency and letter fluency	Norm
Kramer et al. (2002)	12	SVD (SIVD)	BNT and letter fluency	Control
Kubu et al. (2000)	18	PD	BNT	Norm
Lacritz et al. (2000)	40	PD	BNT	Norm
Lee et al. (2013)	40	PD	BNT	Control

Lee et al. (2014)	67	SVD (svMCI)	BNT, category fluency and letter fluency	Control
Lewis et al. (1998)	12	PD	BNT	Control
Liu et al. (2017)	25	SVD (VCIND)	BNT	Control
Liu et al. (2019)	29	SVD (SIVD)	BNT	Control
Longworth et al. (2005)	8, 10	PD, HD	Inflection (regulars and irregulars)	Control
Macoir et al. (2013)	15	PD	Inflection (regulars and irregulars)	Control
Marangolo & Piras (2008)	3	Stroke	Category fluency and letter fluency	Norm
Mason et al. (2015)	29	HD (pre-manifest)	Letter fluency (FAS)	Norm
McMurtray et al. (2008)	8	Stroke	BNT	Control
Mendez et al. (1989)	7	Stroke	BNT	Control
Nagaratnam & Gilhotra (1998)	1	Stroke	Naming (WAB)	Norm
Nagy et al. (2007)	16	PD	BNT	Control
Naidoo et al. (2008)	1	Stroke	BNT (15)	Norm
Nemeth et al. (2012)	7	HD	Naming (81)	Control
Odekerken et al. (2015)	114	PD	BNT	Norm
Palomar et al. (2013)	9	SVD (CADASIL)	BNT (60), category fluency and letter fluency	Control
Penaloza et al. (2014)	40	Stroke	BNT and category fluency	Norm
Peters et al. (2005)	65	SVD	Category fluency	Control
Pozorski et al. (2018)	29	PD	BNT	Control
Qiao et al. (2021)	22	SVD	BNT and category fluency	Control
Radanovic et al. (2004)	8	Stroke	BNT and category fluency	Norm
Randolph et al. (1993)	8 (HD) and 10 (PD)	HD and PD	BNT	Norm
Reifegerste et al. (2020)	41	PD	Inflection (regulars and irregulars)	Control
Rettig et al. (2000)	42	PD	BNT	Norm

Robin & Schienberg (1990)	10	Stroke	Naming (responsive)* and category fluency	Norm
Rodríguez-Ferreiro et al. (2009)	28	PD	Naming (objects)	Control
Rothlind et al. (2015)	276	PD	BNT	Norm
Schoemaker et al. (2020)	24	SVD (CADASIL)	Category Fluency	Control
Schoemaker et al. (2021)	24	SVD (CADASIL)	BNT	Control
Schwab et al. (2015)	40	PD	BNT	Control
Sebastian et al. (2020)	1	Stroke	BNT	Norm
Seghier et al. (2014)	1	Stroke	Naming (objects)	Norm
Shim et al. (2008)	19	SVD (vMCI)	BNT (19), category fluency and letter fluency	Norm
Shin et al. (2012)	43	PD	BNT	Control
Skeel et al. (2001)	14	PD	BNT (30)	Control
Smeding et al. (2011)	40	PD	BNT	Norm
Song et al. (2014)	52	SVD (CADASIL)	Category fluency	Control
Sun et al. (2017)	39	SIVD (combined svMCI and SVNCI)	BNT (30)	Norm
Sunwoo et al. (2013)	46	PD	BNT	Norm
Tang et al. (2015)	27	PD	BNT (30)	Norm
Terzi et al. (2005)	27	PD	Inflection (regulars and irregulars)	Control
Tramontana et al. (2015)	30	PD	BNT (15)	Norm
Tröster et al. (1997)	9	PD	BNT	Norm
Tröster et al. (1998)	30, 24	PD, HD	BNT	Control
Tovar et al. (2020)	20	HD	BNT	Control
Troyer et al. (2004)	1	Stroke	BNT (60), category fluency and letter fluency	Norm
Uitti et al. (2000)	57	PD	BNT	Norm
Ullman et al. (1997)	5, 8	PD, HD	Inflection (regulars and irregulars)	Control
Vallar et al. (1988)	1	Stroke	Category fluency and letter fluency	Norm

Van den Stock et al. (2015)	20	HD (pre-manifest)	BNT and category fluency	Control
Van Lancker Sidtis et al. (2006)*	2	Stroke	BNT (60) and letter fluency	Norm
Van Lancker Sidtis et al. (2021)	1	Stroke	BNT	Norm
Villeneuve et al. (2011)	21	SVD (vascular MCI-WML)	BNT	Control
Wallesch et al. (1983)	16	Stroke	Category fluency	Norm
Wang et al. (2021)	74	SVD	Category Fluency	Control
Whelan et al. (2002)*	2	PD	BNT	Control
Whelan et al. (2003)	21	PD	BNT	Norm
Whelan et al. (2004a)*	2	PD	BNT	Norm
Whelan et al. (2004b)	16	PD	BNT	Control
Whelan et al. (2005)*	2	PD	BNT	Norm
Wolfe et al. (1994)	1	Stroke	BNT (15) and letter fluency	Norm
Xu et al. (2014)	74	SVD (Combined VaMCI and SIVD)	BNT (30)	Norm
Xu et al., 2021	101	SVD	BNT	Norm
Yener et al. (2019)	25	PD	BNT	Control
York et al. (2008)	51	PD	BNT	Norm
Zhou & Jia (2009)	56	SVD (MCI-SVD)	Category fluency	Control

Notes. BNT = Boston naming test; HD = Huntington's disease; LACI = lacunar infarcts; MCI = mild cognitive impairment; PD = Parkinson's disease; SIVD = subcortical ischemic vascular disease; SVD = small vessel disease; svMCI = subcortical vascular mild cognitive impairment; VaMCI = vascular mild cognitive impairment; SVNCI = subcortical vascular no cognitive impairment; VCIND = vascular cognitive impairment no dementia; WML = white matter lesions; (K)WAB = (Korean) western aphasia battery; CADASIL = cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy; studies indicated with and * were excluded post-hoc as indicated in section 3.1.

Appendix

Mesh terms used in PubMed: (("Stroke" [Mesh] OR "Infarction" [Mesh] OR "Basal Ganglia Diseases" [Mesh] OR Brain Infarction [Mesh] OR "Brain Diseases"[Mesh] OR "Cerebrovascular Disorders"[Mesh] OR "Lacunar"[Mesh] OR "Basal ganglia Hemorrhage"[Mesh] OR "Leukoaraiosis"[Mesh] OR "Basal Ganglia Cerebrovascular Disease"[Mesh] OR "Brain Damage [Mesh], Chronic"[Mesh] OR "Cerebral Small Vessel Diseases"[Mesh] OR "Brain Small Vessel Disease with Hemorrhage"[Mesh] OR "Intracranial Hemorrhages"[Mesh]) NOT "Cerebellum"[Mesh]) AND (("Basal Ganglia"[Mesh] OR "Subcortical"[Mesh] OR "Putamen"[Mesh] OR "Caudate Nucleus"[Mesh] OR "Ventral striatum"[Mesh] OR "Striatal"[Mesh] OR "Globus pallidus"[Mesh] OR "Corpus striatum"[Mesh] OR "Neostriatum"[Mesh] OR "Substantia Nigra" [Mesh]) NOT "Cerebellum"[Mesh]) AND ("Language"[Mesh] OR "Language Test"[Mesh] OR "Linguistics"[Mesh] OR "Language Disorder"[Mesh] OR "Semantics"[Mesh] OR "Speech disorders"[Mesh] OR "Speech"[Mesh] OR "Speech Production Measurement"[Mesh] OR "Speech Articulation Tests"[Mesh] OR "Language Tests"[Mesh] OR "Communication"[Mesh] OR "Linguistics"[Mesh] OR Anomia[Mesh]). These Mesh terms were adapted and used to search in PsycINFO (add the list as supplementary material).

Terms used in PsycINFO: basal-ganglia.mp. or exp Basal Ganglia/ OR striatum.mp. or exp Striatum/ OR neostriatum.mp. or exp Striatum/ OR subcortical.mp. OR exp Subthalamic Nucleus/ or subthalamic.mp. OR subcortex.mp. NOT cerebellum.mp. or exp Cerebellum/ AND language.mp. or exp Language/ OR
speech.mp. or exp Oral Communication/ OR naming.mp. or exp Naming/ OR procedural.mp. OR oral communication.mp. or exp Oral Communication/ OR aphasia.mp. or exp Aphasia/ OR AND damag*.mp. OR lesion*.mp. or exp Lesions/ OR disease*.mp. OR stroke*.mp OR exp Cerebrovascular Accidents/ or cerebrovascular accident*.mp. OR vascular lesion*.mp. OR basal ganglia disease*.mp.

Supplement

Table S1. Quality assessment of the studies for the naming task.

Reference	Group	Selection criteria			Comparability of study groups criteria				Outcome criteria		Total stars	Notes on norms
		Representative-ness of the patient cohort	Selection of the control cohort	Ascertainment of exposure	Age	Sex	Ed.	*N* of controls known	Outcome	Same method		
Adrover-Roig et al. (2011)	stroke	*		*	*			*	*	*	6	Allegri et al. (1997)
Cappa et al. (1997)	stroke	*		*	*				*	*	5	Novelli et al. (1986); Perani et al. (1987)
Godefroy et al. (1994)*	stroke	*	*	*	*		*	*	*	*	8	
Kennedy & Murdoch (1993)	stroke	*		*				*	*	*	5	Milman et al. (2014)
Kim et al. (2011b)*	stroke	*		*				*	*	*	5	H. Kim & Na (2004)
Mau-Sun Hua et al. (2001)	stroke	*	*	*	*		*	*	*	*	8	
McMurtray et al. (2008)	stroke	*	*	*	*	*	*	*	*	*	9	
Mendez et al. (1989)	stroke	*	*	*	*	*	*	*	*	*	9	
Nagaratnam & Gilhotra (1998)	stroke	*		*	*		*	*	*	*	7	Zec et al. (2007)
Naidoo et al. (2008)	stroke	*		*			*	*	*	*	6	Kent & Luszcz (2002)
Penaloza et al. (2014)	stroke	*		*				*	*	*	5	Alegret et al. (2012)
Radanovic et al. (2004)	stroke	*		*	*		*	*	*	*	7	Leite et al. (2017)
Robin & Schienberg (1990)*	stroke	*		*				*	*	*	5	Borod et al. (1980)
Sebastian et al. (2020)	stroke	*		*	*		*	*	*	*	7	Kent & Luszcz (2002)
Seghier et al. (2014)	stroke	*		*	*	*		*	*	*	7	From the manual, thanks to David Howard
Troyer et al. (2004)	stroke	*		*	*		*	*	*	*	7	Na & King (2019)
Van Lancker et al. (2006)*	stroke	*		*	*		*	*	*	*	7	Zec et al. (2007)
Van Lancker et al. (2021)	stroke	*		*	*		*	*	*	*	7	Zec et al. (2007)

Wolfe et al. (1994)	stroke	*			*	*		*	*	*	*	7	Kent & Luszcz (2002)
Altermatt et al. (2019)	SVD	*			*		*	*	*	*	*	8	Berres et al., (2000)
Chen et al. (2014)	SVD	*	*		*	*	*	*	*	*	*	9	
da Silva et al. (2011)*	SVD (CADASIL)				*	*		*	*	*	*	6	Da Silva (2017)
Ji et al. (2014)	SVD	*	*		*	*	*	*	*	*	*	9	
Kim et al. (2011a)	SVD	*			*	*		*	*	*	*	7	Kim & Na (1999)
Kim et al. (2012)	SVD	*			*	*		*	*	*	*	7	Kim & Na (1999)
Kramer et al. (2002)	SVD	*	*		*	*		*	*	*	*	8	
Liu et al. (2017)	SVD	*	*		*	*		*	*	*	*	8	
Liu et al. (2019)	SVD	*	*		*	*	*	*	*	*	*	9	
Palomar et al. (2013)	SVD (CADASIL)		*		*	*	*	*	*	*	*	8	
Qiao et al. (2021)	SVD	*	*		*	*	*	*	*	*	*	9	
Schoemaker et al. (2020)	SVD (CADASIL)		*		*	*	*	*	*	*	*	8	
Shim et al. (2008)	SVD	*			*	*		*	*	*	*	7	Kim & Na (1999)
Sun et al. (2017)	SVD	*			*	*		*	*	*	*	7	Lee et al. (2012)
Villeneuve et al. (2011)	SVD	*	*		*	*		*	*	*	*	8	
Xu et al. (2014)	SVD	*			*	*		*	*	*	*	7	Lee et al. (2012)
Xu et al. (2021)	SVD	*			*	*		*	*	*	*	7	
Ash et al. (2017)	PD	*	*		*	*		*	*	*	*	8	
Beatty & Monson (1989)	PD	*	*		*	*		*	*	*	*	8	
Biars et al. (2019)	PD	*			*	*		*	*	*	*	7	Zec et al. (2007)
Bocanegra et al. (2015)	PD	*	*		*	*	*	*	*	*	*	9	
Cahn et al. (1998)	PD	*			*			*	*	*	*	6	Zec et al. (2007)
Cattaneo et al. (2015)	PD	*	*		*	*		*	*	*	*	8	
Celebi et al. (2014)	PD	*	*		*		*	*	*	*	*	8	
Christopher et al. (2015)	PD	*			*	*	*	*	*	*	*	8	
Chung et al. (2018)	PD	*			*	*		*	*	*	*	7	Kim & Na (1999)
Crucian et al. (2010)	PD	*	*		*	*	*	*	*	*	*	9	
Cuoco et al. (2021)	PD	*	*		*	*		*	*	*	*	8	
Dan et al. (2019)	PD	*	*		*	*	*	*	*	*	*	9	
Ellis et al. (2015)	PD	*	*		*	*		*	*	*	*	8	
Fernandes et al. (2017)	PD	*	*		*	*	*	*	*	*	*	9	
Filoteu et al. (1997)	PD	*			*			*	*	*	*	6	Zec et al. (2007)
Floden et al. (2014)	PD	*			*			*	*	*	*	6	Zec et al. (2007)
Foley et al. (2021)	PD	*			*	*			*	*	*	6	
Fraraccio et al. (2008)	PD	*			*			*	*	*	*	6	Zec et al. (2007)
Garcia et al. (2017)	PD	*	*		*	*	*	*	*	*	*	9	

Han et al. (2021)	PD	*		*				*	*	4	Lee et al. (2012)
Hansen et al. (2019)	PD	*		*			*	*	*	5	Zec et al. (2007)
Heluani et al. (2012)	PD	*		*	*			*	*	5	Mansur et al. (2006)
Heo et al. (2008)	PD	*		*	*		*	*	*	6	Kim & Na (1999)
Higginson et al. (2009)	PD	*		*		*	*	*	*	6	Zec et al. (2007)
Hyder et al. (2021)	PD	*	*	*	*	*	*	*	*	9	
Inguanzo et al. (2021)	PD	*	*	*		*	*	*	*	7	
Isaacs et al. (2021)	PD	*	*	*		*	*	*	*	7	
Johari et al. (2019a)	PD	*	*	*	*	*	*	*	*	9	
Ketteler et al. (2014)	PD	*	*	*	*	*	*	*	*	9	
Kubu et al. (2000)	PD	*		*			*	*	*	5	Zec et al. (2007)
Lacritz et al. (2000)	PD	*		*		*	*	*	*	6	Zec et al. (2007)
Lee et al. (2013)	PD	*	*	*	*	*	*	*	*	9	
Lewis et al. (1998)	PD	*	*	*	*	*	*	*	*	9	
Nagy et al. (2007)	PD	*	*	*	*	*	*	*	*	9	
Odekerken et al. (2015)	PD	*		*			*	*	*	5	Marien et al. (1998)
Pozorski et al. (2018)	PD	*	*	*	*	*	*	*	*	9	
Randolph et al. (1993)	PD	*		*	*	*	*		*	7	Zec et al. (2007)
Rettig et al. (2000)	PD	*		*		*	*	*	*	6	Zec et al. (2007)
Rodriguez-Ferreiro et al. (2009)	PD	*	*	*	*		*	*	*	*	8	
Rothlind et al. (2015)	PD	*		*				*	*	4	Zec et al. (2007)
Schwab et al. (2015)	PD	*	*	*	*	*	*	*	*	9	
Shin et al. (2012)	PD	*	*	*	*	*	*	*	*	9	
Skeel et al. (2001)	PD	*	*	*	*	*	*	*	*	9	
Smeding et al. (2011)	PD	*		*	*	*	*	*	*	8	Marien et al. (1998)
Sunwoo et al. (2013)	PD	*		*	*		*	*	*	*	7	Kim & Na (1999)
Tang et al. (2015)	PD	*		*	*			*	*	*	6	Lee et al. (2012)
Tramontana et al. (2015)	PD	*		*	*		*	*	*	*	7	Lansing et al. (1999)
Tröster et al. (1997)	PD	*		*			*	*	*	*	6	Zec et al. (2007)
Tröster et al. (1998)	PD	*	*	*	*		*	*	*	*	8	
Uitti, et al. (2000)	PD	*		*			*	*	*	*	6	Zec et al. (2007)
Whelan et al. (2002)*	PD	*	*	*			*	*	*	*	7	
Whelan et al. (2003)	PD	*		*			*	*	*	*	6	Zec et al. (2007)
Whelan et al. (2004b)	PD	*		*			*	*	*	*	6	
Whelan et al. (2004a)*	PD	*	*	*				*	*	*	6	Worrall et al. (1995)
Whelan et al. (2005)*	PD	*		*	*		*	*	*	*	7	Zec et al. (2007)

Yener et al. (2019)	PD	*	*	*	*	*		*	*	*	8	
York et al. (2008)	PD	*		*			*	*	*	*	6	Zec et al. (2007)
Arango-Lasprilla et al. (2006)	HD	*	*	*	*	*	*	*	*	*	9	
Chenerey et al. (2002)	HD	*	*	*	*	*	*	*	*	*	9	
Hinzen et al. (2018)	HD	*		*	*		*	*	*	*	7	Allegri et al. (1997)
Nemeth et al. (2012)	HD	*	*	*	*	*	*	*	*	*	9	
Randolph et al. (1993)	HD	*		*	*	*				*	5	Tombaugh & Hubiey (1997)
Tröster et al. (1998)	HD	*	*	*	*		*	*	*	*	8	
Trovar et al. (2020)	HD	*	*	*	*	*	*	*	*	*	9	
Van den Stock et al. (2015)	HD	*	*	*	*			*	*	*	7	

Notes: studies indicated with * were excluded post-hoc as indicated in section 3.1 of the main text.

Table S2. Quality assessment of the studies for the category fluency task.

Reference	Group	Selection criteria			Comparability of study groups criteria				Outcome criteria		Total stars	Notes on norms
		Representativeness of the patient cohort	Selection of the control cohort	Ascertainment of exposure	Age	Sex	Ed.	N of controls known	Outcome	Same method		
Adrover-Roig et al. (2011)	stroke	*		*	*			*	*	*	6	Benito-Cuadrado et al. (2002)
Benke et al. (2003)*	stroke	*		*	*			*	*	*	6	Luck et al. (2018)
Cappa et al. (1997)	stroke	*		*	*				*	*	5	Novelli et al. (1986)
Garcia-Caballero et al. (2007)	stroke	*		*			*	*	*	*	6	Benito-Cuadrado et al. (2002)
Godefroy et al. (1994)*	stroke	*	*	*	*		*	*	*	*	8	
Gurd et al. (1988)	stroke	*		*		*		*	*	*	6	Acevedo et al. (2000)
Hochstenbach et al. (1998)	stroke	*	*	*	*		*	*	*	*	8	
Marangolo & Piras (2008)	stroke	*		*	*				*	*	5	Novelli et al. (1986)
Mau-Sun Hua et al. (2001)	stroke	*	*	*	*		*	*	*	*	8	
Penaloza et al. (2014)	stroke	*		*			*	*	*	*	6	Benito-Cuadrado et al. (2002)
Radanovic et al. (2004)	stroke	*		*	*		*		*	*	6	Radanovic et al. (2004)
Robin & Schienberg (1990)	stroke	*		*				*	*	*	5	Borod et al. (1980)
Troyer et al. (2004)	stroke	*		*	*		*	*	*	*	7	Tombaugh et al. (1999)
Vallar et al. (1989)	stroke	*		*	*				*	*	5	Novelli et al. (1986)
Wallesch et al. (1983)	stroke	*		*	*			*	*	*	6	Luck et al. (2018)
Altermatt et al. (2019)	SVD	*		*	*	*	*	*	*	*	8	Berres et al., (2000)
Camerino et al. (2021)	SVD	*		*	*	*	*	*	*	*	8	de Vent et al., (2016)
Chen et al. (2014)	SVD	*	*	*	*	*	*	*	*	*	9	
da Silva et al. (2011)	SVD (CADASIL)			*	*		*	*	*	*	6	da Silva (2017)
Galluzzi et al. (2005)	SVD	*		*	*				*	*	5	
Lee et al. (2014)	SVD	*	*	*	*	*	*	*	*	*	9	
Jokinen et al. (2009)	SVD	*	*	*	*	*		*	*	*	8	
Kim et al. (2012)	SVD	*		*	*		*	*	*	*	7	Ryu et al. (2012)
Palomar et al. (2013)	SVD		*	*	*	*	*	*	*	*	8	
Peters et al. (2005)	SVD (CADASIL)		*	*	*	*	*	*	*	*	8	

Qiao et al. (2021)	SVD	*	*	*	*	*	*	*	*	*	9	
Schoemaker et al. (2020)	SVD (CADASIL)		*	*	*	*	*	*	*	*	8	
Shim et al. (2008)	SVD	*		*	*		*	*	*	*	7	Ryu et al. (2012)
Song et al. (2014)	SVD (CADASIL)		*	*	*	*	*	*	*	*	8	
Wang et al. (2021)	SVD	*	*	*	*	*	*	*	*	*	9	
Zhou & Jia (2009)	SVD	*	*	*	*	*	*	*	*	*	9	
Arango-Lasprilla et al. (2006)	HD	*	*	*	*	*	*	*	*	*	9	
Van den Stock et al. (2015)	HD	*	*	*	*			*	*	*	7	

Notes: studies indicated with * were excluded post-hoc as indicated in section 3.1 of the main text.

Table S3. Quality assessment of the studies for the letter fluency task.

Reference	Group	Selection criteria			Comparability of study groups criteria				Outcome criteria		Total stars	Notes on norms
		Representati-veness of the patient cohort	Selection of the control cohort	Ascertainment of exposure	Age	Sex	Ed.	N of controls known	Outcome	Same method		
Cappa et al. (1997)	stroke	*		*	*				*	*	5	Novelli et al. (1986)
Godefroy et al. (1994)*	stroke	*	*	*	*		*	*	*	*	8	
Marangolo & Piras (2008)	stroke	*		*	*				*	*	5	Novelli et al. (1986)
Mendez et al. (1989)	stroke	*	*	*	*	*	*	*	*	*	9	
Troyer et al. (2004)	stroke	*		*	*		*	*	*	*	7	Tombaugh et al. (1999)
Vallar et al. (1989)	stroke	*		*	*				*	*	5	Novelli et al. (1986)
Van Lancker et al. (2006)*	stroke	*		*			*	*	*	*	6	Tombaugh et al. (1999)
Wolfe et al. (1994)	stroke	*		*	*		*	*	*	*	7	Tombaugh et al. (1999)
Palomar et al. (2013)	SVD (CADASIL)		*	*	*	*	*	*	*	*	8	
Galluzzi et al. (2005)	SVD	*		*	*				*	*	5	
Lee et al. (2014)	SVD	*	*	*					*	*	5	
Kramer et al. (2002)	SVD	*	*	*	*		*	*	*	*	8	
Kim et al. (2012)	SVD	*		*	*		*		*	*	6	Yi et al. (2020)
Kim et al. (2011a)	SVD	*		*					*	*	4	Yi et al. (2020)
Shim et al. (2008)	SVD	*		*	*		*		*	*	6	Yi et al. (2020)
Arango-Lasprilla et al. (2006)	HD	*	*	*	*	*	*	*	*	*	9	
Mason et al. (2015)	HD	*		*	*			*	*	*	6	Tombaugh et al. (1999)
Van den Stock et al. (2015)	HD	*	*	*	*			*	*	*	7	

Notes: studies indicated with * were excluded post-hoc as indicated in section 3.1 of the main text.

Table S4. Quality assessment of the studies for the verb inflection task.

Reference	Group	Selection criteria			Comparability of study groups criteria				Outcome criteria		Total stars
		Representativeness of the patient cohort	Selection of the control cohort	Ascertainment of exposure	Age	Sex	Ed.	N of controls known	Outcome	Same method	
Johari et al. (2019b)	PD	*	*	*	*	*	*	*	*	*	9
Longworth et al. (2005)	PD	*	*	*	*	*		*	*	*	8
Macoir et al. (2013)	PD	*	*	*	*	*	*	*	*	*	9
Reifegerst et al. (2020)	PD	*	*	*	*	*	*	*	*	*	9
Terzi et al. (2005)	PD	*	*	*	*	*	*	*	*	*	9
Ullman et al. (1997)	PD	*	*	*	*		*	*	*	*	8
Longworth et al. (2005)	HD	*	*	*	*	*	*	*		*	8
Ullman et al. (1997)	HD	*	*	*	*	*	*		*	*	8

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For references of the selected papers for meta-analyses, see main text

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