Review Article

Pseudo-dementia: A neuropsychological review

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Abstract

Ever since Kiloh (1961)[2] coined the term pseudo-dementia, it has been used a little loosely for describing the cognitive deficits in depression, especially, which is found in old age. However, several diagnostic dilemmas persist regarding the nosological status of this condition. Teasing out these individual diagnostic problems is important not only for administering appropriate therapy, but also for preventing them from the unnecessary diagnostic assessments towards the other diagnoses. Thus, it is important to have a detailed knowledge of the cognitive or neuropsychological deficits in this condition. In this review, we start by addressing the important issue of diagnostic confusion between dementia and pseudo-dementia. Subsequently, we proceed by reviewing the present scientific literature on the cognitive deficits found in this clinical condition. For the sake of convenience, we will divide the cognitive deficits into:

1. Memory deficits
2. Executive function deficits and
3. Deficits in speech and language domains.

Finally, we will look at the progression of this condition to see the components of this condition, which can be actually called "Pseudo".

Key Words

Cognition, executive functions, memory, pseudodementia

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Introduction

The term pseudo-dementia (PDEM) was coined by Kiloh (1961)[2] to describe the cases, which closely mimicked the picture of dementia. Since then, the term has been used to describe the cognitive profile of various psychiatric disorders, especially depression in old age, which present with cognitive deterioration in dementia. After the term came into the academic use, there have been several arguments against its usage[3,4] as well as in favor of it.[5] Inspite of these arguments, PDEM remains an important descriptive denotation for describing cognitive deficits in psychiatric disorders, especially depression. Clinically, PDEM has become synonymous with the cognitive deficits seen in patients with major depressive disorder. As the term signifies, it is the clinical condition, which presents with the picture of a full-blown dementia but actually is a different entity. This means that actually this condition has two components, which is also reflected in its name:

1. "The dementia component" which is the combination of various cognitive deficits found in these psychiatric disorders and
2. "The pseudo component" which denotes the actual lack of the neurodegenerative dementia.

Both these components are important and will be dealt with separately in this review. The relationship between depression and dementia is complex and has been the topic of many debates recently. This complexity arises from the two facts that cognitive impairments can be found in depression[50] and that dementia can manifest with depressive symptoms as well. This issue of the diagnostic dilemma will be addressed in the subsequent sections.

There are multiple reasons why there has to be a clearer understanding of this condition. Most importantly, it needs to be properly understood because of the role it plays in the functional impairment of depressive disorders. In the recent review by McIntyre *et al*.,[51] it was found that cognitive deficits accounted for the largest percentage of variance with respect

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to the link between psychosocial dysfunction and major depressive disorder (MDD). Thus, cognitive deficits are clearly important functional predictors of MDD. In this review, we will try to bring forth a better and clearer picture of cognitive deficits in depression. First, we will focus on the issue of diagnostic dilemmas between dementia and pseudo-dementia. Subsequently, we will focus on the individual cognitive deficits and the progression of this condition.

Pseudodementia vs. Dementia — Understanding the Diagnostic Dilemma

The importance of distinguishing primary dementing processes from functional disorders has been highlighted time and again since Kiloh coined this term in 1961. In his own words, such patients *"may be in danger of therapeutic neglect and perhaps of unnecessary neurosurgical investigations"*. However, the author also mentioned that this term does not have any nosological significance and only describes a condition. Now, we know that this condition is far more important to understand for establishing a diagnosis of either dementia or depression. The timely recognition and treatment of depression in the elderly is thus important not only to prevent the patient from the consequences of progressing depression but also to prevent them from unnecessary investigative evaluations for dementia. The difficulty in diagnostic assessments of PDEM and pervasive developmental disorder (PDD) is particularly evident in elderly patients as compared to young adults because of the additional confusion created by age-related cognitive deficits. No wonder that there have been reports of high rates of both false-positive and false-negative errors in the diagnosis of dementia.[52-55] This points the necessity of improved clinical diagnostic techniques. Along with this normal age-related cognitive decline, multiple health problems, and the common use of several different medications are often the additional factors obscuring appropriate diagnosis of depression in the aged people.

One of the earliest and still most cited descriptions of cognitive deficits of PDEM was provided by Small *et al*.[56] In their classical descriptions, depressive PDEM patients had equal loss for recent and remote events, were especially characterized by patchy or specific memory loss, their attention and concentration were intact and gave frequent "Don't know" answers. More specifically, their performance on similarly difficult neuro-psychological tasks were much variable.

A clearer picture of this diagnostic dilemma was provided by Kaszniak[58] who delineated four clinically important reasons that make the differentiation between these two clinical states difficult:

1. Cognitive changes in the elderly blur the distinction between normal aging and early signs of PDD
2. Cognitive impairment frequently accompanies depression and can be severe enough to cause confusion between dementia and depression
3. Signs of some neurologic diseases associated with progressive decline (e.g., Alzheimer's disease [AD] and Parkinson's disease [PD]) have symptoms that overlap with depression
4. Dementia and depression can co-exist.

Cognitive Deficits in Depression: Detailing the "Dementia" Component

One of the earliest descriptions of overall cognitive deficits in depressive disorders was given by Caine[59] who characterized patients with such "neuropsychiatric disorders" as having deficits in the following areas:

1. Arousal, attention, and concentration;
2. Mood and affect;
3. Perception (both ideational and physical, internal and external);
4. Specific intellectual functions (e.g., memory, language); and
5. Personality.

This classification summarizes the cognitive deficits in depressive disorders. However, it lacked specificity for the cognitive deficits. Further, along this review, we will focus more on specific cognitive domains, and will do it as a comparison with the cognitive deficits in dementing disorders. We will limit present discussion to memory, executive functions and speech and language deficits among the other cognitive deficits.

Memory function deficits

Memory has been the most assessed function for evaluation of cognitive differences between depression and dementia. It is now commonly accepted that depression presents with a number of deficits in the domains of episodic memory and learning.[23] Considerable body of evidence shows that there are multiple cognitive impairments in depression as compared to normal subjects. This finding has been consistent across most studies and appears to involve both explicit verbal and visual memory functions and is similarly affected in patients suffering from both melancholic (endogenous) and non-melancholic (non-endogenous) depression types.[7] However, these patients have intact performances on implicit memory tasks, and some other memory functions appear to be spared.[24-27] Although the neurobiological underpinnings of these memory deficits are not clear, they can be traced to the temporal lobe related abnormalities. It is well known that temporal lobe lesions result in impairments in episodic memory. This fact along with the finding that reductions in hippocampal volume are demonstrated in patients with major depression[28] may point towards temporal lobe deficit as the culprit for these deficits.

Hart and Kwentus[35] examined memory scanning and incidental memory performance in depressed elderly (n = 15) and normal control patients (n = 16). They employed the Stemberg task[60] and the digit symbol (DS) subtest of the WAIS-R, Wechsler[82] both of which are measures of basic psychomotor speed, as well as incidental memory (DS) and information processing efficiency (ST). They found depressed patients' reaction time to be significantly slowed down. However, incidental memory for symbols (on DS) and information processing capacity (slope function of the ST) were not affected by the depressive status. Authors raised the possibility that the psychomotor slowing in depressed patients was more due to "motivational" factors that are mediated by catecholaminergic systems.

Till now, we have focused on memory deficits in depression patients in comparison to normal individuals. However, there have been many other studies, which show that memory

deficits in depression are considerably mild when seen in comparison to dementia.

Early work by Whitehead addressed the issue of verbal learning and memory deficits in depression by comparing elderly depressed and patients with mild dementia. They posited that variation in response strategies was the core reason for the memory-related differences observed in these groups of patients.[29] Authors had observed that depressed patients tended to take a conservative approach in making responses, thereby showing less random variation in their responses, whereas patients with mild dementia committed more random and false positive errors.[29] However, their findings were based on relatively small samples of patients (n = 12) with weakly defined clinical diagnoses. Miller & Lewis[44] took a step ahead for evaluating this finding and verified depressed patient's conservative response strategies with well-matched and more clearly defined samples. They found that the accuracy of depressed patients on a recognition memory task was significantly better than that in dementia of Parkinson's disease patients. La Rue[36] compared depressed patients (n = 41), AD patients (n = 19), and patients with other organic impairments (e.g., PD, multi-infarct dementia; n = 20) on the Object Memory Evaluation (OME). A general finding was that depressed patients performed better than AD patients on "all initial learning and recall as well as delayed recall measures." Dannenbaum *et al*[38] used the Brown-Peterson task in discriminating between these delayed recall performances of normal, elderly depressed and Alzheimers dementia patients. They found superior delayed recall performance of depressed elderly relative to patients with DAT.

In addition to AD, other subcortical neurodegenerative disorders also have been reported with impairments in cognitive processing including Friedreichs Ataxia[39] and Parkinson's disease[41]. Niederehe[41] conducted a series of studies aimed at distinguishing between depressed, PDD patients, and normal individuals on measures of "episodic", "semantic",[42] and constructive memory tests. His studies were based on small sample size (n = 24) and consisted of well-matched groups. A consistent outcome of his studies was a qualitatively worse performance of PDD patients as compared to depressed patients.

Hart *et al*.[43] with their study results suggested that depressed and AD patients might be distinguished on the basis of the rapidity with which they forget initially encoded information. In their study, the depressed patients, AD patients, and normal subjects were differentially exposed to the to-be-remembered information. Patients were presented line drawings for 2, 4, and 8 s (for normal, depressed, and Alzheimer's patients, respectively), in view of the fact that the levels of learning impairments groups were suffering was different. Their results indicated that AD patients showed a much more rapid rate of forgetting as compared to depressed and normal individuals who forgot information at the same rate. However, there was relatively equivalent general intellect, verbal fluency, and concentration ability.

Some early studies have assessed recall-memory functions using several different standardized neuropsychological tools. In a study using the incidental memory manipulation with the DS subtest of the WAX-R (mentioned above), Hart *et al*.[40] found that depressed (n = 15) and mildly demented patients (n = 15) were similar in performance with respect to psychomotor speed, but the recall of symbols demonstrated by depressed patients was significantly more than mild AD patients. Another study examining memory-recall performance using the selective reminding procedure Buschke[81] produced similar results showing that depressed patients (n = 14) were impaired on total recall and proportion of items retained from trial to trial relative to normals (n = 16), but were superior to mild AD patients (n = 15) across all memory indices.[40]

Work by La Rue and collaborators have examined elderly depressed patients' performance on a variety of clinical memory tasks.[36,37] Normal elderly (n = 10), depressed patients (n = 10), and patients with PDD (n = 10) were compared with respect to their performance on the Benton Visual Retention Test (BVRT), Inglis Paired Associate Learning Test (IPA), and Fuld Object Memory Evaluation (OME).[37] Depressed patients performed at or below the level of normal controls on most BVRT and IPA indices, and were generally superior to dementia patients. However, the OME was far better at distinguishing between groups.

In view of these findings of memory impairments in depression, the question of etiopathogenesis becomes all the more important. Till now, there is no 'unified theory' for these cognitive deficits but most theories point towards an encoding problem in such patients. This encoding deficit can be seen in the context of different information processing stages of memory.[30] From the perspective of a "levels of processing" approach of depression,[31] such patients might not be able to encode information to the same "depth" as normal subjects. In a small sample study, Weingartner *et al*.[4] examined normal and depressed subjects' ability to employ elaborative encoding strategies when learning new information. Their findings suggested that depressed patients failed to engage in encoding strategies that would maximize the likelihood of subsequent recall which however, improved in performance when material was presented in a predecided organized fashion. Weingartner[32] on the other hand looked at depressed patients' encoding abilities as a function of the relative amount of effort expended in initial learning.[33] He found that depressed patients were less successful than normals when engaging in "effort demanding" encoding exercises. This led, Weingartner[32] to propose that, "biological systems associated with motivation, effort, and arousal appear to be linked to the performance of effort demanding cognitive operations in depressed patients."

To summarize, depression presents with several memory-function deficits most common being those related to tasks of recall or remembering. However, the sample sizes of these studies have been relatively small (generally l0-30 subjects per group), and the methodologies vary widely, which have been the main limitations of these studies. Additionally, these memory deficits inspite of being significantly worse than normal individuals, are comparably better than those associated with dementia Regarding the basic neurobiology of these deficits, most researchers have suggest that such differences represent general cognitive inefficiency and attention problems rather than a fundamental lack of ability due to structural deficits.

This can be seen in the differences between depression and dementia patients as well as in the temporariness of these deficits in the clinical picture of depression, which will be dealt in next sections.

Executive function deficits

Executive function deficits are prevalent in depressive disorders and recently they have been found to produce clinically significant effects, which may in fact be a significant mediator of the functional impairments found in such patients. Task switching or the set-shifting abilities have been the most commonly found impairments among executive functions in depressed patients. In a recently well-reviewed article by McIntyre,[51] authors found that executive function deficits occur at a rate of 20-30% of patients suffering from MDD. Studies in past decade examining impairment in executive tasks have produced somewhat consistent findings of impairments in set shifting tasks in depressed patients.[6-9,57] In general, significant impairments have been observed in subjects with more severe depression.[10-12] For example, Beats *et al*.[8] found a severely depressed elderly sample to be most prominently impaired on verbal fluency and attentional set-shifting. Same finding has been reflected even in younger patients suffering from moderate depression by Purcell *et al*.[9] so that no impairment on working memory was found but impairments on measures of motor speed and attentional set-shifting were observed, with half of the depression group even failing to complete all stages of this task. Same study also revealed that these 'impaired' subjects had higher rates of admissions for treatment of depression, suggesting that those with overall greater illness severity were more impaired on set-shifting tasks. Similar to these findings, the studies by Channon[61] and Channon & Green[62] showed that even patients suffering from mild to moderate depression (mean Beck Depression Inventory[63] scores of 17-21) suffered from impairment in executive functions of matching, task completion and task switching based on their performance in Wisconsin Card Sorting Test even though they belonged to younger age group (mean 20-40 years). Similar findings of set shifting ability deficits have been observed in several other studies.[6,7,57] An interesting comparison of executive function of depressed patients with manic patients was conducted by Murphy *et al*.[57] In a study comparing the performance of subjects with depression and mania on a novel affective set-shifting task, they found that subjects with depression were impaired in their ability to shift the focus of attention (corresponding to the set-shifting component of the WCST), while patients with mania were impaired in their ability to inhibit behavioural responses. This impairment corresponds to the inability to prevent the corresponding to the interference effect of the Stroop test as described by Golden[18]. There have also been attempts to evaluate the executive functions on various subgroups of depression. Some particularly interesting studies were conducted by Austin *et al*..[6,7] They divided depression patients into endogenous and non-endogenous subsets using standardized definitions of endogenous depression by the Newcastle system.[19] Further, they subdivided the samples into melancholic and non-melancholic according to the CORE instrument.[17] In the Austin *et al*.)[7] study, subjects with endogenous/melancholic depression were impaired (as in the Austin *et al*. 1992a study)[6] on working memory (digits backwards) as well as on tasks heavily reliant on set-shifting (Trails B, and digit symbol substitution) as well as an increased perseverative response on the Wisconsin Card Sorting Task.[34]

Other than these set-shifting problems, several other executive function deficits have been observed. Bomstein, *et al*.,[64] studied a sample of elderly depressed patients ($n = 62$) for the presence of the "Fuld profile"[65] on the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler)[82] which was thought to be relatively specific (though not necessarily sensitive) to disorders in which there is prominent temporal lobe pathology, most notably AD. Results from this study indicated that the Fuld profile was present although less frequently in depressed elderly patients (16%) than in the AD samples (44%) as reported in 1984 by Fuld, but similar to that observed in a normal subject group (12.8%).[66] These findings suggest that the elderly patients with depression do present with executive function deficits but less than AD patients. Recently, the trend of such assessment studies has been to employ comprehensive batteries and/or intelligence tests for examining neuropsychological function in depressed patients. Gray, *et al*.,[67] looked at the performances of depressed ($n = 30$) PDD ($n = 26$) and general neurological patients ($n = 24$) on subtests of the Halstead-Reitan Neuropsychological Battery (HRB). They found that depressed patients were generally less impaired, as measured by the Halstead impairment index, than PDD and neurological groups. In fact, depressed individuals were superior to the other groups on all HRB measures except, Tactual Performance Test location, Speech Sounds Perception Test, and Trail-Making Test A.[67] In a similar vein, McCue, Goldstein, and Shelly[68] compared the performances of depressed ($n = 45$) and PDD patients ($n = 34$) on a short form of the Luria Nebraska Neuropsychological Battery (LNNB). They were able to differentiate between the groups on all the 10 clinical scales of the form employed. Patients with depression who were misclassified as having PDD tended to have more extreme degrees of depressive symptomatology and lower educational levels.[68] These studies, with larger sample sizes and a broader range of cognitive domains assessments, further strengthen the general finding of substantial differences between depressed and demented individuals. In a most recent study, Egerhazi *et al*.[14] used Cambridge Automated Neuropsychological Battery in patients suffering from MDD during their acute stage. They found that during the acute episode, delayed matching to sample, paired associate learning, spatial recognition memory, rapid visual processing and visuospatial planning were impaired. In remission, improvements in the domains of visual learning ability, spatial recognition memory, psychomotor speed, and executive function were observed. The authors concluded that MDD is associated with neurocognitive dysfunctions in different domains, the most prominent deficit being found in the paired associate learning test, which requires both the elaboration of "frontal strategies" and the "mnemonic processes".

Processing speed impairment is another important executive function deficit observed in depression. In a recent study by Brown *et al*.,[13] it was found that impairments in processing speed partially mediated the effect of depression on functioning in patients suffering from minimal cognitive deficits. A more clinical outcome of this processing speed impairment is psychomotor slowing, which is almost universally found in the depressed patients, irrespective of the number of episodes. However, there also have been exceptions to these near-universal findings. Elliott *et al*.[16] conducted a study on middle-aged subjects with moderate, predominantly chronic depression, and demonstrated impaired abilities of these

patients on the tasks of Tower of London, verbal fluency and spatial working memory but intact performance on a modified and easier version of the Cambridge Neuropsychological Battery (CANTAB) set-shifting task.[15] Works by Bieliauskas and colleagues (Bieliauskas *et al*.; Bieliauskas & Lamberty; Bieliauskas, *et al*.)[20,21,22] have repeatedly presented the findings showing the lack of significant differences between normal and depressed patients on EF and other cognitive measures. In a series of studies using screening measures such as the MMSE and the Neurobehavioral Cognitive Status Exam[58] no differences were found between depressed (n = 15) and non-depressed patients (n = 33) across a wide array of cognitive and motor tasks (with the exception of the NCSE Attention subtest). On the basis of these findings, they put forth the notion that differences between normal and depressed patients are sufficiently subtle to not significantly influence scores on standard cognitive screening instruments.

Speech and language function deficits

Small number of studies has specifically examined speech-language function in elderly depressed patients. While most of the tasks used to measure cognitive abilities in above-mentioned studies have obvious receptive and expressive language components, it is important to highlight some studies which have looked at specific language skills. For instance, in the study by Emery and Breslau[67] it was observed that depressed patients performed better than AD patients on measures of naming, repetition, general reading skill, syntax, and auditory verbal comprehension. At the same time, normal individuals tended to show better language performance than depressed patients overall, though these differences were considerably less obvious than those noted between depressed and AD patients.[67] It is to be noted that "verbal fluency" tasks are recognized as sensitive indicators of general cognitive impairment; so this finding has to be seen as a measure of general impairment rather than specific language related impairment.[48] However, Hart *et al*.[46] found no statistically significant difference between depressed and AD patients on the familiar controlled oral word association test (a.k.a., F-A-S test; Benton & Hamsher)[70] between depressed and AD patients so that both of them showed reduced verbal fluency, However, depressed patients outperformed AD patients on a categorical fluency task (i.e., "animals"). Thus, finally a reduction in psychomotor speed was considered as the main reason to explain depressed patients' weakness on this task rather than true deficits in the domain of language and speech.[46]

Another commonly used measure of language function has been the Boston Naming Test.[45] Using a 30-item version of the BNT, Speedie *et al*.[49] reported that depressed patients were impaired to the same extent as patients with irreversible dementias whereas prominent language deficits (including confrontation naming) are not thought to be common in depression.[59] Most of the findings of language related impairments have only been incidental while examining for other cognitive domains.

Progression of Pseudo-Dementia: Perspectives for the "Pseudo" Component

As we highlighted in the previous sections, it is difficult to tease out true cases of pseudo-dementia given the overlaps between the clinical findings in dementia and depression with regards to both depressive symptoms and cognitive impairments. Saez-fonseca[71] found in their 5-7 year follow up study that 71.4% of those suffering from PDEM had converted into dementia at follow-up compared to only 18.2% of the conversion in the cognitively intact group. Kral & Emery[1] conducted a similar study of progression of pseudo-dementia. Forty-four elderly patients of both sexes (mean age 76.5 years) suffering from depressive PDEM were intensively treated for the depression. When the depression subsided, cognitive function also reverted to premorbid level. Patients were regularly interviewed and retested at six months intervals for four to 18 years (average 8). Some patients experienced, during the follow-up period, a recurrence of the depression for which they were again successfully treated. For testing the progression of these cognitive deficits with time, several different study designs have been used. Some studies have used the more direct method of comparing cross-sectionally the performance of subjects who have recovered from depression with that of matched controls. Paradiso *et al*.[72] found significant cognitive impairments on the set-shifting tasks in subjects who had recovered from unipolar depression as compared to normal controls. Additionally, these cognitive deficits were not related to medication status which suggested the independence of these deficits from treatment related variables. In the same line, Marcos *et al*.[73] found persistent deficits in both immediate memory and delayed recall of visual and verbal material, and block design in patients of melancholia after 3 months of their recovery.

Although these studies provide a cross-sectional perspective regarding poor neuropsychological performance of depressed patients in comparison to normal controls, definitive findings can only be provided by testing the cognitive status before and after recovery so that any baseline cognitive deficits are eliminated. Abas *et al*.[74] tested elderly patients with endogenous depression on several memory measures and reported that nearly half of those performing poorly at baseline were poor performers inspite of absence of clinically evident dementia or minimal cognitive deficits. In a similar sample of elderly patients, Beats *et al*.[8] also found that many, but not all deficits had remitted upon recovery: Specifically, measures of simple and choice reaction times, perseveration on the set shifting task and verbal fluency did not fully recover.

There have also been studies showing that cognitive impairments improve with treatment. In one of the earliest studies, Sternberg & Jarvik[75] reported that in endogenous depression subjects responding to a tricyclic antidepressant treatment, although performance on learning and short-term memory tasks remained impaired after treatment, there was improvement in immediate memory and this was related to degree of depressive recovery. Similar findings were reported by Calev *et al*.[76] and Bazin *et al*.[27] neither of which found residual impairments in either explicit (verbal and visual) or implicit memory tasks upon recovery. Similarly, Trichard *et al*.[78] in a controlled study of executive task performance in middle-aged subjects with severe depression, reported improved performance on the verbal fluency task but not the Stroop test upon recovery. A very significant finding was reported by Peselow *et al*.[79] who in a study of patients with unipolar depression treated with imipramine for 4 weeks, found

significant improvement in all mnemonic measures only those responding to treatment. They concluded that in memory tasks performance, recovery of mood was associated with significant cognitive improvement. These findings have been reminded in a recent study by Egerhazi *et al*.,[14] where cognitive impairment was found to improve partly in remission, suggesting that an individual's current mood interacts with the ability to perform a cognitive task.

To summarize, results suggest that several cognitive domains especially those related to memory functions are improved with treatment of depressive disorders; however, several of them do persist. Thus a residual deficit in mnemonic and executive function appears to remain in some patients with a history of depression and specifically need to be investigate further because reversible cognitive impairment in late-life moderate to severe depression appears to be a strong predictor of dementia. More studies are needed to exactly understand the relationship of cognitive deficits in depression to crucial epidemiological variables such as age, treatment, duration and chronicity of illness and number of episodes.[80] Thus, it is recommended to have a full dementia screening for patients suspected to have PDEM.

Conclusion

Present review suggests that over past few decades, enough study results point to the fact that depressive states adversely affect cognitive functions, especially in old-age or geriatric depression. In spite of the methodological and sampling problems encountered when working with these complex populations, the differentiation between depression and early stages of dementia seems to be plausible. Although, earlier researchers have pointed out the inabilities of neuropsychological tests in the context of making these differentiations[2,59] most of the recent data support this practice and should be able to differentiate between true cases of dementia, depression and the ill-defined intermediate stage of pseudo-dementia. Subsequent endeavors in this area with more well-defined populations and properly designed studies are needed to generalize these conclusions.

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