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APOE Genotype Knowledge and Its Impact on Cognitive Beliefs and Performance

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Abstract

As genetic testing for Alzheimer's disease (AD) risk becomes increasingly accessible, it is important to understand how individuals respond to knowledge of their genetic risk. In this study, we examined whether awareness of being an APOE ε4 carrier, a genetic risk factor for late-onset AD dementia, adversely affects subjective and objective cognition. Participants were 195 cognitively unimpaired older adults (aged 63-79, *M*age= 71.18), recruited from the Alzheimer's Prevention Initiative's GeneMatch program. All had undergone APOE testing, with 94 ε4 carriers and 32 non-carriers aware of their genotype, and 41 ε4 carriers and 28 non-carriers unaware. Subjective cognition was assessed using measures of memory control, attention control, and memory anxiety. Objective cognition was assessed with short-term memory, working memory, and episodic memory tasks. Results showed that ε4 carriers aware of their genotype were less confident that they could influence their cognitive functioning through effort and less confident that they could control their attention. Additionally, non-carriers aware of their genotype were less concerned they were currently developing AD, suggesting that disclosure may provide reassurance when genetic risk is absent. Awareness of genotype did not reliably affect objective cognition; however, exploratory analyses found that among ε4 carriers, awareness was associated with poorer working memory performance when it was assessed early in the test battery but better performance when assessed later. Together, these findings suggest that being aware of a heightened genetic risk for AD can undermine older adults' perceived cognitive control and, under certain conditions, produce transient adverse effects on working memory performance.

Keywords: APOE genotype, genetic testing, subjective cognition, memory control beliefs, stereotype threat

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As genetic testing becomes more accessible, more people are learning about their risk for diseases like Alzheimer's disease (AD), whether through clinical disclosure or direct-to-consumer services. This growing accessibility makes it increasingly important to understand how learning about genetic risk information influences individuals' thoughts, feelings, and behavior. In the current study, we examined whether knowing that one is an APOE ε4 carrier, a genetic risk factor for AD, influences subjective perceptions of cognitive ability and objective cognitive performance.

The APOE gene, located on chromosome 19, is a well-established genetic risk factor for late-onset AD and other neurodegenerative diseases (Chapman et al., 2001). This gene has three common allelic variants -- ε2, ε3, and ε4 -- with individuals inheriting one allele from each parent. In terms of AD risk, the ε3 allele is considered neutral, ε2 is protective, and ε4 is associated with increased risk (Genin et al., 2011; Koutsodendris et al., 2022). The risk associated with the ε4 allele is dose-dependent: Individuals with one copy (ε4 heterozygotes) are approximately three times more likely to develop mild cognitive impairment or dementia, while those with two copies (ε4 homozygotes) are about six times more likely (Qian et al., 2017). Moreover, ε4 homozygosity is linked to an earlier onset of AD symptoms, lowering the average age of onset from 84 to 68 years (Corder et al., 1993).

Clinicians are often reticent to disclose APOE genotyping results to asymptomatic individuals because APOE genotype alone has limited predictive value for AD and, in the absence of symptoms APOE genotype does not yet guide treatment or prevention decisions. However, genetic testing is increasing in popularity, both in clinical settings and through commercialized direct-to-consumer genetic testing services. Survey research suggests that 79%

of people are willing and interested in taking a genetic test for AD (Neumann, et al., 2001; see also Kopits et al., 2011; Waterink et al., 2023) and 15.5% of primary care doctors who treat AD have been asked to order APOE genotype testing by an asymptomatic patient (Chase et al., 2002).

Although disclosure to asymptomatic individuals is not yet common, once patients begin to exhibit signs of cognitive impairment, clinicians now routinely disclose APOE genotype because this information directly informs decisions about monoclonal antibody (mAb) therapies. Currently there are two FDA-approved mAb therapies that have demonstrated efficacy in reducing amyloid deposits in the brain and slowing cognitive decline: lecanemab (*Leqembi;* McDade et al., 2022; Swanson et al., 2021; van Dyck et al., 2023) and donanemab (*Kisunla;* Sims et al., 2023). However, the risks of treatment-related adverse events for mAb therapies vary by APOE genotype. Lecanemab produces higher amyloid-related imaging abnormalities (ARIA) due to edema/effusion (ARIA-e) and hemorrhage (ARIA-H) in APOE ε4 carriers compared to non-carriers (Cummings et al., 2023; van Dyck et al., 2023). Likewise, donanemab produces higher ARIA-e rates in APOE ε4 carriers compared to non-carriers (Sims et al., 2023). Because of these increased risks, APOE genotyping is recommended for all candidates prior to initiating either lecanemab (Cummings et al., 2023) or donanemab treatment (Rabinovici et al., 2025).

Prior research suggests that APOE disclosure does not negatively impact psychological well-being (for reviews, see Bemelmans et al., 2016; Marshe et al., 2019; Rahman et al., 2012). For instance, the Risk Evaluation and Education for Alzheimer's Disease (REVEAL) study examined the safety and feasibility of sharing APOE results with individuals, particularly first-degree relatives of AD patients. The results suggested that, when supported by genetic counseling, APOE disclosure does not lead to serious psychological distress, although some

individuals may experience transient test-related distress (Green et al., 2009; see also Alber et al., 2021; Langbaum et al., in press). Overall, 80% of participants in the REVEAL study who learned their APOE genotype reported that the information had a positive impact, 17% perceived it as neutral, and only 3% (n = 1) perceived it as negative (LaRusse et al., 2005).

However, some evidence suggests that learning one's APOE genotype can affect subjective cognition. In a qualitative study, 46% of older individuals who learned they were APOE ε4 carriers reported a negative impact of this knowledge on their subjective cognition. These individuals became more "sensitive" or "alert" to their memory, noticing "senior moments" more frequently and associating forgetful moments to concerns about AD. In contrast, 45% of non-carriers reported a positive impact, feeling less worried about occasional forgetfulness after learning their APOE genotype (Largent et al., 2021).

Knowledge of APOE genotype can also influence older adults' ratings of their own memory abilities. In a study by Lineweaver, Bondi, Glasko, and Salmon (2014), APOE ε4 carriers aware of their genotype rated their memory capacity *lower* than ε4 carriers without such knowledge. In contrast, ε4 non-carriers aware of their genotype rated their memory capacity *higher* than ε4 non-carriers without such knowledge. Among APOE ε4 non-carriers, those aware of their genotype also gave more favorable ratings of their retrospective memory functioning and reported fewer problems remembering what they had read compared to ε4 non-carriers who were unaware of their genotype.

In the current study, we tested whether awareness of being an APOE ε4 carrier similarly alters other aspects of subjective cognition among older adult ε4 carriers. Specifically, we focused on control beliefs (related to both memory and attention), and on memory-related concerns and anxiety. These outcomes were chosen because they represent important ways in

which subjective cognition can influence everyday functioning and well-being. Control beliefs serve as a core self-regulatory resource, guiding motivation, effort, and strategy use, and higher levels of control have been linked to better health, well-being, and cognitive functioning (for a review, see Lachman et al., 2011). In contrast, anxiety about memory is associated with poorer memory performance (Davidson & Hultsch, 1991), particularly in older adults (Andreoletti et al., 2007). We reasoned that learning about a heightened genetic risk of AD could undermine older adults' sense of control, potentially diminishing their confidence in their ability to manage their own memory and attention. We also anticipated that learning about a heightened genetic risk of AD might lead some older adults to feel concern or anxiety about their cognitive functioning, which could potentially interfere with their cognitive performance.

We also examined whether awareness of being an APOE ε4 carrier impacts objective cognitive performance. To our knowledge, only one prior study has directly examined this question, finding modest negative effects on a subset of memory measures for older adults. Specifically, Lineweaver et al. (2014) reported that APOE ε4 carriers who were aware of their genotype performed significantly worse on both the immediate and delayed recall tests of the Logical Memory subtest from the *Weschler Memory Scale – Revised*. However, no such effect was observed on the Rey-Osterrieth Complex Figure Test, which involves copying a complex abstract line drawing and later reproducing it from memory, either immediately or after a delay. Given these mixed findings, the current study tested whether awareness of being an APOE ε4 carrier would impact older adults' performance on a variety of additional memory tests, including those measuring short-term memory (Digit Span Forward), working memory (Digit Span Backward), and episodic memory. Within our assessment of episodic memory, we separately evaluated memory for individual items and memory for associations between items,

given the well-established associative memory deficits that occur in normal aging (Old & Naveh-Benjamin, 2008).

Current Study

The goal of this study was to examine whether awareness of being an APOE ε4 carrier influences both subjective and objective cognition in older adults. We focused on control beliefs and memory-related anxiety as these outcomes are central to everyday functioning and well-being, and we assessed objective performance across multiple cognitive domains, including short-term memory, working memory, and episodic memory. To maximize the likelihood of detecting adverse effects of APOE ε4 awareness, we explicitly framed the study as an investigation of how APOE genotype influences cognition. This framing was chosen because such instructions have the potential to evoke stereotype threat, a process in which individuals worry that their behavior may confirm a negative stereotype about a group to which they belong. Previous research has shown that such concerns can reduce self-efficacy, heighten anxiety, and impair performance (Steele & Aronson, 1995; Spencer et al., 2016), including in older adults (Barber, 2020). We reasoned that describing the current study as an investigation of how APOE genotype influences cognition might cause ε4 carriers aware of their genotype to worry that their performance would reflect their heightened genetic risk for AD. By explicitly emphasizing the link between genotype and cognition as part of our study instructions, this study was structured to create conditions under which any detrimental effects of APOE knowledge, if they exist, would be most likely to emerge.

Method

Transparency and Openness

All procedures were approved by the Institutional Review Board at Georgia State University (protocol H21155 titled "APOE Genotype and Cognitive Performance"), but study procedures and analyses were not pre-registered. The data reported here has not been previously published and is available from the corresponding author.

Recruitment

Participants were recruited from the Alzheimer's Prevention Initiative's GeneMatch program, which is a registry designed to connect individuals with AD-focused research opportunities (Langbaum et al., 2019). Upon enrolling in GeneMatch, participants submitted a cheek swab for APOE genotyping, which was conducted by a Clinical Laboratory Improvement Amendments certified laboratory (clinicaltrials.gov NCT02564692). All participants provided consent for their APOE results to be shared with the research team.

The current study did not disclose APOE results to participants. However, as part of our recruitment efforts, we oversampled individuals from GeneMatch who had previously participated in the Generation Program clinical AD trials (clinicaltrials.gov NCT02565511 and NCT03131453). These clinical trials disclosed APOE genotype to participants as part of eligibility screening (Langlois et al., 2019; Lopez et al., 2019). This disclosure did not result in clinically significant psychological symptoms (Langbaum et al., in press).

Invitations to the current study were sent through participants' GeneMatch accounts. The invitations described the study as an online investigation of how genes influence mental abilities and emphasized that individuals with a range of different genetic profiles were being invited to participate. Invitees had up to four weeks to accept the study invitation before it expired.

Participants and Power Analysis

The goal of this study was to evaluate how subjective and objective cognition are impacted by APOE genotype and by participant's awareness of their APOE genotype. A total of 214 participants completed this study, but we excluded one individual whose score of 10 on the Geriatric Depression Scale (GDS-15; Sheikh & Yesavage, 1986) was suggestive of moderate to severe depression. Because the goal of this study was to evaluate how subjective and objective cognition are influenced by awareness of APOE genotype, we also excluded participants if they reported having learned their APOE genotype but had forgotten or were unsure whether they carried an ε4 allele (n = 14). We also excluded participants who reported knowing their *APOE* genotype but who were mistaken about whether they were ε4 carriers (n =2; both individuals were ε4 heterozygotes who mistakenly believed they were non-carriers).

Our final sample consisted of 195 older adults, aged 63 to 79 years (M = 71.18). Of these, 69 individuals reported never having learned their APOE genotype. Among these genotype-unaware individuals, 42 were ε4 carriers and 28 were non-carriers. The remaining 126 participants reported having learned their genotype and could accurately recall whether they were ε4 carriers or not. Among these genotype-aware individuals, 94 were ε4 carriers and 32 were non-carriers. Among individuals aware of their genotype, 89.7% had learned their APOE genotype at least three years prior, and 84.9% reported that a health professional was present to explain their results when they first received them (see Table 1). Participants were predominately White (96.9%), well-educated (M = 16.80 years of education), and mostly women (60.5%). Demographic characteristics by APOE ε4 carrier group, and by participants' awareness of their APOE genotype, are further detailed in Table 1.

A sensitivity analysis was conducted using G*Power 3.1.9.7 (Faul et al., 2007) to determine the effect size that this study was powered to observe. We specified an ANOVA with

an alpha level of .05, power of 0.80, a sample size of 195 participants, four groups, and a numerator *df* of 1. This analysis indicated that the study was powered to observe main effects and interactions having an effect size of $f = 0.20$.

Materials

Perceived Risk of Developing AD

As a manipulation check, we included a question assessing participants' belief that they would develop AD in the future. Specifically, participants responded to the question, *"How would you rate your risk of developing Alzheimer's disease in the future?"* using the following options: *Unlikely*, *Possible*, *Probable*, or *Almost Certain*. We included this measure to ensure that participants who were aware of their APOE genotype understood what this meant in terms of AD risk. We expected ε4 carriers who knew their genotype to report higher perceived risk, and non-carriers who knew their genotype to report lower perceived risk, relative to unaware participants.

Subjective Cognition Measures

Memory Controllability Inventory. The Memory Controllability Inventory (Lachman et al., 1995) assesses perceived memory control, and perceptions of current memory abilities. Within this 12-item scale there are 4 subscales, each consisting of 3-items. For the current study, participants responded to each item using a 1 to 6 scale (*strongly disagree* to *strongly agree*). Subscale scores were computed by averaging the response given to all items within a given subscale. Higher scores reflect higher endorsement of the factor being assessed.

Present Ability Subscale. The Present Ability subscale focuses specifically on appraisals of current memory ability (e.g., *'I can remember the things I need to'; α* = .77).

Potential Improvement Subscale. The Potential Improvement subscale measures participants' confidence in their ability to find strategies that could enhance memory performance (e.g., *'I can find ways to improve my memory'; α* = .72).

Inevitable Decrement Subscale. The Inevitable Decrement subscale measures participants' beliefs about the inevitability of decline in memory function with increasing age (e.g., *'When it comes to memory, there is no way I can make up the for the losses that come with age'; α* = .67).

Effort Utility Subscale. The Effort Utility subscale assesses beliefs about the effectiveness of effort in maintaining or improving memory, including the potential to delay future decline (e.g., '*If I work at it, I can improve my memory'; α* = .78).

Attention Control Scale. Two items designed to assess attentional control were selected from the Attention Control Scale (Derryberry & Reed, 2002). These items were: (1) "*When trying to focus my attention on something I have difficulty blocking out distracting thoughts"* (reverse-scored), and (2) *"When a distracting thought comes to mind, it is easy for me to shift my attention away from it"*. In the current study, participants responded to these items using a 1 to 6 scale (*strongly disagree* to *strongly agree*), and responses to the two items were associated with one another (r = .525, p < .001; α = .68). A composite score was created by averaging participants' responses to the two questions.

Metamemory in Adulthood Questionnaire – Anxiety subscale. The Anxiety subscale of the Metamemory in Adulthood questionnaire (Dixon & Hultsch, 1983) is a 14-item questionnaire designed to assess feelings of stress related to memory performance, and the ability to use memory when in different emotional states.[1] Due to time constraints in the current study, a

[1] Lineweaver et al. (2014) also examined how awareness of being an APOE ε4 carrier influenced responses to items on the Metamemory in Adulthood questionnaire. However, none of the items included the Lineweaver et al. study

shortened version of the questionnaire was administered, consisting of the following five items: (1) "*I get anxious when I am asked to remember something",* (2) "*I feel uneasy when I attempt a problem that requires me to use my memory",* (3) "*I would feel on edge right now if I had to take a memory test or something similar",* (4) "*When someone I don't know very well asks me to remember something I get nervous",* and (5) "*I get tense and anxious when I feel my memory is not as good as other peoples'".* For the current study, participants responded to each item on a 1 to 6 scale (*strongly disagree* to *strongly agree*). The internal consistency of this shortened questionnaire was high (α = .87). Scores were computed as the average mean response for the five items, with higher scores reflecting higher levels of memory anxiety.

Aging Concerns Scale (Lachman et al., 1995). The Aging Concerns Scale was included to assess memory-related concerns that are specific to aging. In the current study, responses were provided on a 1 to 6 scale (*strongly disagree* to *strongly agree*). Subscale scores were calculated as the average response across their respective items, with higher scores indicating stronger endorsement of the assessed factor.

Independence Subscale. The 3-item Independence subscale focuses on beliefs that one can independently manage their own memory as they get older (e.g., '*As I get older, I won't have to rely on others to remember things for me'; α* = .73).

Modified Alzheimer's Likelihood Subscale. The Alzheimer's Likelihood subscale is designed to assess beliefs about the inevitability of AD and the tendency to interpret memory lapses as a sign of developing AD. Although this measure is typically scored as a single composite, its items reflect a mix of related but distinct constructs. Two of the items assess beliefs that one is already developing AD (i.e., *'When I forget something, I am apt to think I have*

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came from the Anxiety subscale. Instead, Lineweaver et al. used items from the Capacity and Change subscales, which focus on perceptions of current memory abilities and perceived changes in memory abilities over time.

Alzheimer's disease' and '*I sometimes think I am developing Alzheimer's disease'*), one item assesses perceived future risk ('*I think there's a good chance I will get Alzheimer's disease')* and one item reflects broader beliefs about AD prevalence (*'Alzheimer's disease is a common problem among the elderly'*). Because the focus of our study was on how APOE genotype awareness might influence current memory-related concerns, we created a targeted subscale using only the two items that specifically assess belief that one is already developing AD. Responses to the two items were associated with one another ($r = .629$, $p < .001$; $\alpha = .77$).

Objective Cognition Measures

Forward Digit Span. This task was a modified version of the standard digit span task (Wechsler, 2008) adapted for online administration. During this task, participants were visually presented with a series of digits and were asked to recall them in the same order. Each trial began with a 2-second fixation cross, followed by the digit sequence, with each digit displayed for 1-second. Within each sequence, a given digit only appeared once, and there were no sequential ascending or descending digits. After the sequence was completed, participants typed their responses into a text box, with no time limit imposed.

The forward digit span task began with three trials of 3-digit sequences. If participants correctly recalled at least one of these sequences, the task advanced to three trials of 4-digit sequences. This progression continued, with task difficulty increasing by one digit at each level, up to a maximum sequence length of 9 digits. Performance was scored as the total number of trials in which participants correctly recalled the entire sequence (without errors or intrusions).

Backward Digit Span. This task followed the same procedure as the Forward Digit Span task, except participants were instructed to recall the digits in reverse order (Wechsler, 2008). The task began with three trials of 3-digit sequences and progressed in the same manner as the

Forward Digit Span task, up to a maximum sequence length of 9 digits. There was only one other procedural difference from the Forward Digit Span task: If on the very first trial a participant entered the digits in forward order, then corrective feedback was provided that reminded them to recall the digits in reverse order. Performance was scored as the total number of trials in which participants correctly recalled the entire sequence in reverse order (without errors or intrusions).

Item and Associative Recognition Test. Stimuli for this task consisted of images of faces from the FACES database (Ebner et al., 2018) and images of places from the Places dataset (Zhou et al., 2017). [2] During the encoding phase of this task, participants were presented with a sequence of 24 critical face-place pairs, each displayed for four seconds. To account for potential primacy and recency effects in episodic memory, two non-critical face-place pairs were added to the beginning of the sequence and an additional two non-critical face-place pairs were added to the end of the sequence. Participants were instructed to study the faces, places, and pairings between the faces and the places for upcoming memory tests. Following encoding, participants completed an unrelated filler task lasting 2-3 minutes. They then completed three episodic memory tests: (1) an item recognition test for the faces, (2) and item recognition test for the places, and (3) an associative recognition test.

In the item recognition test for the faces, participants were shown a series of 12 faces (6 previously seen and 6 new). For each face, they were given 4-seconds to make a yes/ no recognition judgment, indicating whether the face had been presented earlier. The item recognition test for places followed the same structure, but here participants were shown 12

[2] There were 48 target face images in total, evenly divided by gender (24 female, 24 male) and age group (24 middle-aged adults, 24 older adults). All faces displayed neutral expressions. There were also 48 target place images, with approximately half depicting indoor scenes and half depicting outdoor scenes. None of the place images included people or animals. Lures for the item recognition tests were selected from the same databases as the critical stimuli and were chosen to have similar characteristics as the target items. We did not counterbalance which items served as targets versus lures. However, because all participants saw the same sets of targets and lures, fixed item difficulty would not be able to account for any between-group differences in performance.

places (6 previously seen, 6 new). Finally, for the associative recognition test, participants were informed they would encounter two types of pairs: (1) *intact pairs,* where the face and place were originally presented together during encoding, and (2) *rearranged pairs,* where the face and place were both previously seen during encoding, but were not originally paired together. Participants were then shown 12 face-place pairs (6 intact pairs and 6 rearranged pairs), and they were asked to decide whether the face and place had been presented together during encoding. Participants were required to make their yes/no response within 4 seconds.

This entire procedure (encoding, filler task, three memory tests) was then repeated in a second round, with a second set of 24 face-place pairs.[3] Thus, across the two rounds of this test, participants' memory was tested for 48 faces and 48 places. Corrected recognition was calculated as the proportion of hits minus false alarms for each test.

Procedure

Study procedures were conducted online using Qualtrics. After providing informed consent, participants completed a demographics questionnaire reporting their age, gender, ethnicity and race, marital status, educational attainment, employment status, and region of residence. They also answered questions assessing other participant characteristics, including subjective social status, subjective health, and family history of AD. As a measure of depressive symptoms, participants then completed the Geriatric Depression Scale (GDS-15; Sheikh & Yesavage, 1986).

[3] The critical face and place images were randomly paired together and divided into two sets. One set contained all the female face-place pairings (n = 24) and the other set contained the male face-place pairings (n = 24). Within each set, the 24 face-place pairings were distributed across the memory tests as follows: (1) Six used during the face item-memory test, (2) Six used during the place item-memory test, (3) Six used as 'intact' pairs during the associative memory test, and (4) Six used as rearranged pairs during the associative memory test. To minimize potential stimulus effects, we counterbalanced across participants whether a given face-place pair was used in the face item-memory test, the place item-memory test, as an intact pair during the associative memory test, or as a rearranged pair during the associative memory test.

Next, we assessed participants' knowledge of their APOE genotype. First, participants indicated whether they had ever learned their APOE genotype (*yes/ no*). Those who responded "yes" were then reminded that the APOE ε4 allele is associated with an increased risk for late-onset AD and were asked whether they carried this allele. Response options included reporting zero, one, or two copies of the ε4 allele. An additional response option allowed participants to indicate knowledge that they carried at least one ε4 allele while being uncertain whether they had one or two copies. The final response option allowed participants to indicate that they were unsure whether they were an ε4 carrier altogether. Participants aware of their genotype were also asked to indicate when they first learned this information and whether a genetic counselor or doctor was present to explain their results when they learned this information. Immediately following this, participants were asked to rate their own risk of developing AD, which served as a manipulation check.

Participants next completed a series of questionnaires and tasks, including our measures of cognition. The objective memory tests were always administered first, with order of the tasks counterbalanced. As part of this, participants were randomly assigned to one of two task orders. Some completed the Forward and Backward Digit Span tasks first, followed by the item and associative recognition tests. Others began with the item and associative recognition tests, followed by the Forward and Backward Digit Span tasks.[4] At the beginning of each objective memory test, participants were explicitly told that the purpose of the test was to determine how cognitive performance is affected by APOE genotype. Following completion of the objective cognition tests, we assessed subjective cognition. At the outset of this portion of the study,

[4] Within the item and associative recognition test additional counterbalancing was applied. Specifically, we varied whether the female face-place image set appeared in the first or second round of the task. To minimize order effects, across participants we also counterbalanced the order of the three recognition tests (i.e., the item recognition test for faces, item recognition test for places, and associative recognition test of face-place pairings).

participants were told the questions were included *"in order to evaluate whether APOE genotype affects how people perceive their own memory and cognitive abilities*". This was followed by the Memory Controllability Inventory, as well as selected items from the Aging Concerns Scale, the Anxiety subscale of the Metamemory in Adulthood questionnaire, and the Attention Control Scale (see Materials), presented in an intermixed order. The study took approximately one hour to complete. Upon completion, participants were offered a $20 electronic Amazon gift card.

Results

Knowledge of Genotype

As part of this study, participants indicated whether they had ever learned their APOE genotype. Those who had not were classified as "unaware" for the analyses. Among participants who reported learning their genotype, only those who accurately identified whether they were an ε4 carrier were included in the "aware" group.

To be included in analyses, we further required participants in the "aware" group to demonstrate knowledge of whether they had an increased genetic risk for AD based on their APOE genotype. However, they were not required to recall their exact genotype. For example, an individual with an ε3/ ε4 genotype was excluded from analyses if they reported having no ε4 alleles or if they stated they had forgotten whether they carried ε4 alleles. In contrast, an individual with an ε3/ ε4 genotype was included in the "aware" group if they reported any of the following: (1) carrying one copy of the ε4 allele, (2) carrying two copies of the ε4 allele, or (3) acknowledging that they carried the ε4 allele but were unsure whether they had one or two copies.

Perceived Risk of Developing AD

As a manipulation check, we first assessed whether APOE genotype knowledge affected perceived risk of developing AD, based on responses to the question "*How would you rate your risk of developing Alzheimer's disease in the future?*". Responses were analyzed using a 2 (APOE genotype: ε4 carrier vs. non-carrier) X 2 (APOE knowledge: aware vs. unaware) ANOVA. This analysis revealed a significant main effect of APOE genotype, F (1, 194) = 11.87, $p < .001$, and a significant interaction between APOE genotype and APOE knowledge, F (1, 194) = 20.56, $p < .001$. Follow-up pairwise comparisons showed that among ε4 carriers, those aware of their genotype rated their risk of developing AD to be significantly higher (M = 2.20, SD = 0.56) than those who were unaware (M = 1.98, SD = 0.65), t (133) = 2.06, p = .042, d = 0.385. In contrast, among APOE ε4 non-carriers, those aware of their genotype rated their risk as significantly lower (M = 1.50, SD = 0.51) than those who were unaware (M = 2.07, SD = 0.38), t (58) = -4.88, $p < .001$, d = -1.264.

Subjective Cognition

Responses to the subjective cognition questionnaires as a function of APOE genotype and knowledge of APOE genotype are presented in Table 2.

To evaluate the effects of APOE genotype and knowledge of that genotype on subjective cognition we conducted a series of 2 (APOE genotype: ε4 carrier vs. non-carrier) X 2 (APOE knowledge: aware vs. unaware) ANOVAs for each subjective cognition questionnaires. As shown in Table 3, significant interactions between APOE genotype and APOE knowledge emerged for the Effort Utility subscale of the Memory Controllability Inventory (p = .014) and for the Attentional Control subscale (p = .046), which were both measures of control beliefs. A significant interaction between APOE genotype and APOE knowledge also emerged on our modified Alzheimer's Likelihood Subscale of the Memory Concerns Scale (p = .045), which

focused on beliefs that one is currently developing AD. To further explore these interactions, for each of these measures we next conducted pairwise comparisons examining the effect of APOE knowledge separately for e4 carriers and non-carriers.[5]

Memory Controllability Inventory - Effort Utility Subscale.

Among e4 carriers, those aware of their APOE genotype reported significantly lower beliefs that memory could be improved through effort (M = 4.17) compared to those unaware (M = 4.41), t (133) = 2.05, p = .043, d = -0.383. In contrast, for e4 non-carriers, awareness of APOE genotype did not significantly impact Effort Utility scores, t (58) = -1.60, p = .116, d = 0.413. However, scores were numerically higher among non-carriers aware of their genotype (4.50) compared to those unaware (4.24).

Attentional Control Scale.

A similar pattern emerged for the Attentional Control Scale. Among e4 carriers, those aware of their *APOE* genotype reported feeling less able to control their attention through effort (M = 3.85) compared to those unaware (M = 4.20), t (133) = -2.06, p = .041, d = -0.386. In contrast, for e4 non-carriers, attentional control scores did not significantly differ as a function of genotype awareness, t (58) = 0.96, p = .339, d = 0.249, although scores were numerically higher for non-carriers aware of their genotype (M = 3.88) compared to those unaware (M = 3.63).

Aging Concerns Scale – Modified Alzheimer Likelihood Subscale.

Among ε4 non-carriers, those who were aware of their genotype were less likely to think they were currently developing AD (M = 2.17, SD = 1.17) compared to those who were unaware

[5] The interactions between APOE genotype and genotype awareness also approached significance for two additional measures: the Inevitable Decrement subscale of the Memory Controllability Inventory (p = .087) and the Independence subscale of the Aging Concerns Scale (p = .046). However, follow-up pairwise comparisons conducted within each genotype group revealed no significant pairwise differences.

(M = 2.80, SD = 1.05), t (58) = -2.19, p = .032, d = -0.567. No significant differences were observed among ε4 carriers, t (133) = 0.03, p = .980, d = 0.005.

Objective Cognition

Performance on the objective cognitive tests as a function of APOE ε4 carrier group and knowledge of APOE genotype are presented in Table 2.

To evaluate the effects of APOE genotype and knowledge of that genotype on objective cognition we conducted a series of 2 (APOE genotype: ε4 carrier vs. non-carrier) X 2 (APOE knowledge: aware vs. unaware) ANOVAs for each objective cognition measure (i.e., Forward Digit Span scores, Backward Digit Span scores, and the corrected recognition scores for the item memory test on faces, item memory test on places, and associative memory test on face-place pairings). As shown in Table 4, within these analyses there were no significant effects. There was no evidence in any of these analyses that objective cognitive performance was impacted by APOE genotype or by participants' awareness of their genotype.

None of these conclusions changed when task order was included as a covariate. However, when task order was instead treated as a factor in the ANOVA, a significant three-way interaction emerged between APOE genotype, APOE knowledge, and task order for performance on the Backward Digit Span, F (1, 187) = 5.51, p = .020. Follow-up 2 (Genotype awareness) X 2 (Task order) ANOVAs on Backward Digit Span scores indicated that the interaction between these factors was significant for the ε4 carriers, F (1, 131) = 11.13, p = .001, but not for the ε4 non-carriers, F (1, 56) = 0.36, p = .552. Among ε4 carriers, awareness of genotype was associated with poorer Backward Digit Span performance when the digit span tasks were completed first in the test battery (Aware: M = 8.69, Unaware: M = 10.96), t (65) = -2.25, p = .028, d = -0.569. This pattern reversed when the digit span tasks were completed last in the test

battery. Here, the ε4 carriers aware of their genotype had better Backward Digit Span performance than those unaware of their genotype (Aware: $M = 10.35$, Unaware: $M = 7.56$), $t(66) = 2.46$, $p = .016$, $d = .704$.

Discussion

Does awareness of an increased genetic risk for AD adversely affect objective or subjective cognition in older adults? Results from the current study suggest that it might, although effects were generally limited to subjective outcomes.

APOE Awareness and Subjective Cognition

Awareness of being an APOE ε4 carrier was associated with reduced perceptions of cognitive control. Specifically, ε4 carriers who were aware of their genotype reported lower scores on both the Effort Utility subscale of the Memory Controllability Inventory and on items from the Attentional Control Scale, indicating diminished beliefs in their ability to influence or manage their own cognitive functioning. These effects were not present among ε4 non-carriers.

These findings extend prior research demonstrating links between awareness of APOE genotype and other aspects of subjective cognition (Lineweaver et al., 2014; Largent et al., 2021). Whereas previous research focused on how awareness of APOE genotype impacts overall perceptions of current memory capacity and how it compares to past memory abilities, the current study shows that APOE awareness may also influence broader cognitive control beliefs, including the perceived effectiveness of exerting cognitive effort. This is particularly important given that control beliefs predict a range of adaptive outcomes (Lachman et al., 2011). For instance, individuals with stronger control beliefs tend to report better health, greater life satisfaction, and lower depressive symptoms (Lachman & Weaver, 1998). In the cognitive domain, stronger control beliefs are associated with greater use of effective cognitive strategies

(Hertzog et al., 2010; Lachman & Andreoletti, 2006), better memory performance (Raldiris et al., 2020), and greater responsiveness to cognitive training (Rebok et al., 1995). Importantly, both daily diary and longitudinal studies suggest that the direction of this relationship flows from control beliefs to cognitive outcomes, rather than the reverse (Neupert & Allaire, 2012; Windsor & Anstey, 2008). Believing that one can influence one's own cognition may lead to greater engagement and effort, which in turn promotes better outcomes over time (Bandura, 1977). Thus, even small reductions in perceived control, such as those observed in this study, could have meaningful implications over time.

At the same time, it is important to note that not all aspects of perceived control were influenced by APOE genotype awareness in this study. Of the four subscales of the Memory Controllability Inventory, only scores on the Effort Utility subscale (i.e., the belief that effort can help maintain or improve memory) were significantly lower among ε4 carriers who were aware of their genotype. There were no significant effects of APOE genotype awareness on responses to the Present Ability subscale (i.e., perceptions of memory ability / memory self-efficacy), Potential Improvement subscale (i.e., confidence in finding ways to improve memory), or Inevitable Decrement subscale (i.e., belief that memory decline is unavoidable). This pattern suggests that the psychological impact of APOE genotype awareness may be selective. Beliefs that depend on a sense of active agency (such as the perceived usefulness of trying harder) may be particularly vulnerable to disruption when individuals learn they are at an increased genetic risk for AD.

While the current study found no effects of APOE genotype awareness on perceptions of overall memory abilities, this differs from the results reported by Lineweaver et al. (2014). One possible explanation for this discrepancy is study-level differences in the specificity of the

measures. In this study, perceptions of memory ability were assessed using the Present Ability subscale of the Memory Controllability Inventory, which captures a broad, global sense of ability (e.g., "*I can remember the things I need to*"). In contrast, Lineweaver et al. used the Capacity subscale of the Metamemory in Adulthood questionnaire, which assesses perceptions of ability in specific domains (e.g., "*I am good at remembering names*"). Prior research on self-efficacy suggests that global appraisals tend to be more stable, whereas domain-specific assessments are more malleable and more strongly tied to actual task performance (e.g., Beaudoin & Desrichard, 2011; Miyoshi, 2011). Following from this, it is possible that perceptions of memory ability within specific domains may be more susceptible to the influence of APOE awareness as compared to global perceptions of memory abilities. Future research is needed to test this possibility by including both global and domain-specific measures of perceived memory ability within the same study.

In the current study, APOE genotype awareness also influenced participants' concern that they might currently be developing AD. To assess this, we used a modified version of the Alzheimer's Likelihood subscale from the Aging Concerns Scale, including only items that tapped into present-focused concerns (i.e., '*When I forget something, I am apt to think I have Alzheimer's disease'* and '*I sometimes think I am developing Alzheimer's disease'*). Among ε4 non-carriers, those aware of their genotype scored lower on this modified subscale than non-carriers who were unaware of their genotype. This finding aligns with prior research suggesting that genotype disclosure can provide reassurance to individuals who learn they are not at elevated genetic risk (Largent et al., 2021). In contrast, among ε4 carriers, awareness of genotype did not significantly increase concern that they were *currently* developing AD, even though these same individuals rated their future risk as higher. This dissociation suggests that beliefs about

current cognitive status and beliefs about future disease risk are psychologically distinct and may be differentially shaped by genotype knowledge.

APOE Awareness and Objective Cognition

In contrast to the observed effects for subjective cognition, knowledge of APOE genotype did not reliably impact objective cognitive performance. Tests assessing short-term memory (Digit Span Forward), working memory (Digit Span Backward), and episodic memory (item and associative recognition) showed no performance differences based on APOE genotype or genotype awareness. These null effects stand in partial contrast to those reported by Lineweaver et al. (2014), who found that ε4 carriers aware of their genotype performed worse on immediate and delayed recall in the Logical Memory subtest of the WMS-R. However, even in that study, the effects of APOE genotype awareness on objective cognitive performance were not observed across all tasks, as performance on the Rey-Osterrieth Complex Figure Test was unaffected by genotype awareness. Together, these findings suggest that if APOE genotype awareness affects objective memory performance, such effects are likely task-specific and relatively subtle.

Our exploratory analyses of task order effects suggest that any detrimental effects of APOE genotype awareness on objective cognition may also be relatively short-lived. Specifically, we found that among ε4 carriers, awareness of APOE genotype was linked to poorer working memory performance when the digit span tasks were administered first in the test battery, but with better performance when they were administered last. No such effects were observed for ε4 non-carriers. Although this crossover interaction should be interpreted with caution as it comes from an exploratory analysis, this finding aligns with prior research on age-based stereotype threat. In designing the current study, we maximized the likelihood of eliciting

threat by instructing participants that the purpose of the study was to examine how cognition is affected by APOE genotype. For ε4 carriers aware of their genotype, such instructions were expected to heighten concerns about their ability to perform well and increase self-monitoring, potentially impairing performance. Our task order results suggest that these feelings of threat may have been strongest early in the session and may have selectively undermined working memory performance. This would be consistent with other research showing that age-based stereotype threat effects are often largest when the outcome is measured immediately after the threatening cue (Lamont et al., 2015) and that working memory is especially vulnerable to threat (Armstrong et al., 2017). Over time these threat effects may have diminished, perhaps due to habituation or compensatory strategies. The reversal observed when digit span was administered last even raises the possibility of contrast effects, whereby ε4 carriers aware of their APOE genotype ultimately overcorrected to disconfirm negative expectations implied by the instructions. Future research should examine these possibilities more directly, but the current pattern suggests that any detrimental effects of APOE awareness on objective cognition may be transient, domain-specific, and contingent on situational context.

Situational Influences on Subjective and Objective Cognition

The framing of our study is also important for interpreting the findings. By explicitly telling participants that the study was examining how APOE genotype affects cognitive performance, we intentionally created conditions under which threat-related concerns were most likely to arise for the ε4 carriers aware of their genotype. This approach mirrors methods commonly used in the age-based stereotype threat literature. In this line of research, older adults are typically exposed to situational cues that emphasize the negative link between age and memory. For example, older adults in the stereotype threat condition may be told that the

purpose of the study is to understand why memory declines with age. Such instructions often reduce older adults' memory self-efficacy and heighten concerns about cognitive decline (Bouazzaoui et al., 2016; Wong & Gallo, 2019). Although age-based stereotype threat can also impair objective performance (Barber & Mather, 2013; Hess et al., 2003; Mazerolle et al., 2012; Barber, 2020), there is some evidence that age-based stereotype threat exerts a stronger effect on subjective measures. For example, Caughie et al. (2023) found that making age stereotypes salient increased older adults' memory concerns without altering their actual performance. This divergence between subjective and objective outcomes mirrors the pattern we observed in the current study. Awareness of APOE genotype more reliably affected subjective beliefs than it did objective performance.

At first glance, it may seem counterintuitive to describe the effects of APOE genotype awareness in terms of "stereotype" processes, given that the ε4 allele is a well-established genetic risk factor for AD (Corder et al., 1993; Genin et al., 2011; Qian et al., 2017) and can be associated with cognitive decline even in asymptomatic older adults (O'Donoghue et al., 2018; Wisdom et al., 2011). However, the current findings are like those observed in studies of diagnosis threat, which is a conceptually similar phenomenon to stereotype threat. In diagnosis threat studies, simply reminding individuals of a medical condition they have (e.g., a history of head injury) can lead to poorer performance compared to when the condition is not made salient (Suhr & Gunstad, 2002). Although diagnosis threat effects are typically small (Niesten et al., 2023), they nevertheless demonstrate how contextual reminders of health-related risks can exacerbate concerns and shape both subjective and objective cognition.

Looking ahead, a key question is whether the effects of APOE knowledge emerge only under conditions that explicitly highlight the link between genotype and cognition, as in the

present study, or whether they also arise more naturally in clinical and research contexts. Some medical consultations and treatment discussions may reference associations between APOE genotype and AD risk, which may inadvertently trigger threat-related concerns for ε4 carriers aware of their genotype (Regner & Huguet, 2025). However, many other situations that require reliance on memory do not contain explicit reference to APOE genotype. Determining the contexts in which APOE knowledge most strongly undermines cognition will be essential for understanding its real-world consequences and for developing strategies to minimize harm.

Clinical Implications

The current findings also have important clinical implications for how APOE genotype information is disclosed. Although genetic testing for AD risk is becoming increasingly available, this study suggests that such knowledge may reduce perceptions of cognitive control for ε4 carriers. Best practices for disclosure should include pre-test education that clarifies what the test does and does not reveal, assessment of an individual's readiness to receive genetic information, and post-test counseling to contextualize the results and address emotional reactions (Roberts & Green, 2021; Stites et al., 2022). Clinical conversations should also address the possibility that APOE genotype knowledge may alter how individuals appraise their own cognitive functioning. Acknowledging this risk up front may help mitigate its effects and ensure that individuals are making fully informed decisions about whether to learn their APOE results. By preparing individuals for how this information might influence their perceptions, clinicians can help minimize adverse effects and support informed, psychologically safe decision-making. These considerations will become even more critical once preventative treatments for AD become available. Once this happens clinicians may routinely offer APOE testing to

asymptomatic individuals so that they can identify and prioritize ε4 carriers (who carry the highest near-term risk for developing AD) for early intervention.

Limitations

Several limitations of the current study should be noted. First, although the overall sample size was adequate for detecting moderate effects, the study was underpowered to detect genotype awareness effects among ε4 non-carriers. APOE ε4 is less common in the general population than APOE ε3 (Corbo & Scacchi, 1999; Singh et al., 2006), but the current study intentionally oversampled ε4 carriers to ensure sufficient power to detect effects within that group. As a result, our final analytic sample included 195 participants, with 135 ε4 carriers (41 unaware, 94 aware) and only 60 non-carriers (28 unaware, 32 aware). The relatively small subgroup sizes among non-carriers limited our ability to detect potential effects of genotype awareness in this group and may have contributed to null findings. Nonetheless, the current study provides some evidence that knowing one is *not* at elevated genetic risk for AD may be reassuring. Among ε4 non-carriers, those aware of their genotype were significantly *less* likely to endorse present-focused concerns about developing AD on the modified Alzheimer's Likelihood Scale. They also showed numerically higher control beliefs than unaware non-carriers on both the Effort Utility subscale of the Memory Controllability Inventory and the Attentional Control Scale. Although these latter differences were not statistically significant and the study lacked power to evaluate these differences fully, the convergence of effects across multiple measures highlights a potentially important direction for future research.

A second limitation of the current study is that the sample consisted of self-selected volunteers who had agreed to be recontacted after previous APOE testing, potentially limiting generalizability to broader populations who may differ in health status, education, or willingness

to learn genetic information. In addition, the sample was predominantly non-Hispanic White, which further limits the applicability of findings to more diverse racial and ethnic groups, particularly given known group differences in APOE prevalence and racial disparities in AD risk (Gleason et al., 2021).

Third, although we included multiple measures of subjective and objective cognition, our task battery may not have captured more subtle or domain-specific effects of genotype awareness.

Fourth, most participants who were aware of their APOE genotype in this study had learned their result at least three years prior, and most reported that a health professional was present to explain their results. We cannot account for how time since disclosure, or differences in the disclosure method, might have influenced participants' responses.

Finally, because the study design was correlational, causal claims cannot be made. Experimental studies that manipulate genotype disclosure are needed to more clearly determine how learning one's APOE genotype drives changes in subjective cognition. Although we interpret our findings to suggest that awareness of being an ε4 carrier leads to reductions in perceived cognitive control, it is also possible that individuals with lower pre-existing control beliefs may be more inclined to seek out opportunities to learn their APOE genotype. Future experimental research is needed to disentangle the direction of effects and clarify the mechanisms linking APOE knowledge to cognitive outcomes.

Conclusion

In conclusion, the findings from the current study suggest that awareness of being an APOE ε4 carrier can negatively influence older adults' perceptions of their cognitive control. When the testing context makes the association between APOE genotype and cognitive

performance salient, this knowledge may also have short-lasting adverse effects on working memory performance. As genetic testing for AD risk becomes increasingly available, it is critical to consider not only the clinical utility of this information but also its potential psychological impact. Future work should continue to investigate the situational and individual factors that moderate responses to genotype disclosure and identify strategies to support adaptive interpretations of genetic risk information.

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Table 1
Sample Characteristics as a Function of APOE ε4 Carrier Group and APOE Knowledge

	APOE ε4 carriers (individuals with at least one ε4 allele)		*APOE ε4* non-carriers (individuals without the ε4 allele)	
	Aware of genotype	Unaware of genotype	Aware of genotype	Unaware of genotype
Sample size (n)	94	41	32	28
Genotype (n)				
ε3/ ε3	0	0	32	28
ε3/ ε4	66	33	0	0
ε4/ ε4	28	8	0	0
Age (years)	70.55 (4.24)	71.24 (3.45)	71.56 (3.86)	72.75 (3.28)
Gender (%)				
Male	42.6	34.1	40.6	35.7
Female	57.4	65.9	59.4	64.3
Ethnicity and Race (%)				
White	97.9	95.1	100.0	92.9
Asian	0.0	2.4	0.0	0.0
Black / African American	0.0	0.0	0.0	3.6
Hispanic, Latino/Latina	1.1	2.4	0.0	0.0
Biracial	1.1	0.0	0.0	0.0
Education (years)	17.05 (2.01)	16.56 (2.56)	16.78 (2.31)	16.32 (2.58)
Subjective Social Status	7.28 (1.38)	6.66 (1.22)	7.19 (1.09)	7.43 (1.26)
Subjective Health	7.64 (1.14)	7.41 (1.12)	7.41 (1.07)	7.11 (1.26)
Geriatric Depression Scale (GDS-15) score	1.64 (1.78)	1.54 (1.68)	1.44 (2.11)	2.07 (2.02)
Employment Status (%)				
Employed full-time	10.6	9.8	3.1	7.1
Employed part-time	9.6	19.5	12.5	10.7
Retired	77.7	68.3	84.4	82.1
Unemployed	2.2	2.4	0.0	0.0
Relationship Status				
Married	73.4	58.5	71.9	71.4
Divorced	12.8	24.4	15.6	7.1
Never married	2.1	2.4	3.1	10.7
Separated	1.1	4.9	6.3	0.0
Widowed	10.6	9.8	3.1	10.7
Family History of AD (%)				
Yes	88.3	75.6	68.8	67.9
No	10.6	14.6	28.1	21.4
Unsure	1.1	9.8	3.1	10.7

Time Aware APOE genotype (%)		
Less than 6 months	1.1	0.0
6-12 months	0.0	0.0
1-2 years	9.6	9.4
3-5 years	72.3	84.4
More than 5 years	17.0	6.3
Professional Support at Disclosure (%)		
No	12.8	9.4
Yes	84.0	87.5
Unsure	3.2	3.1

Table 2

Means and Standard Deviations for Responses to Subjective and Objective Cognition Measures as a Function of APOE ε4 Carrier Group and APOE Knowledge

	APOE ε4 carriers (individuals with at least one ε4 allele)		APOE ε4 non-carriers (individuals without the ε4 allele)	
	Aware of genotype	Unaware of genotype	Aware of genotype	Unaware of genotype
Subjective Cognition				
Memory Controllability Inventory				
Present Ability subscale	4.25 (0.87)	4.51 (0.89)	4.46 (0.82)	4.36 (0.85)
Potential Improvement subscale	4.45 (0.65)	4.50 (0.80)	4.56 (0.60)	4.32 (0.71)
Inevitable Decrement subscale	3.12 (0.75)	2.99 (0.84)	2.93 (0.88)	3.23 (0.70)
Effort Utility subscale	4.17 (0.65)	4.41 (0.61)	4.50 (0.63)	4.24 (0.63)
Attention Control Scale	3.85 (0.92)	4.20 (0.81)	3.88 (0.99)	3.63 (1.01)
Metamemory in Adulthood				
Anxiety subscale	3.24 (0.89)	2.99 (0.93)	3.04 (1.02)	3.21 (1.00)
Aging Concerns Scale				
Independence subscale	3.87 (0.83)	4.10 (0.79)	4.16 (0.82)	3.86 (0.94)
Modified Alzheimer's Likelihood subscale	2.53 (0.84)	2.17 (1.17)	2.55 (1.08)	2.76 (1.04)
Objective Cognition				
Forward Digit Span	12.96 (3.20)	13.24 (3.34)	12.97 (3.32)	12.86 (3.83)
Backward Digit Span	9.61 (3.73)	9.63 (4.87)	10.50 (5.12)	10.61 (4.95)
Episodic Memory				
Item Corrected Recognition Faces	0.60 (0.22)	0.58 (0.22)	0.58 (0.19)	0.54 (0.17)
Item Corrected Recognition of Places	0.66 (0.22)	0.69 (0.18)	0.65 (0.19)	0.62 (0.23)
Associative Corrected Recognition	0.34 (0.24)	0.32 (0.22)	0.32 (0.22)	0.28 (0.22)

Note: All subjective cognition measures were answered on a 1 (strongly disagree) to 6 (strongly agree) scale.

Table 3

ANOVA Results: Main Effects and Interaction Effects of APOE ε4 Carrier Group and APOE Knowledge on Subjective Cognition

Dependent Variable	***APOE* Genotype** (ε4 carrier vs. non-carrier)	***APOE* Knowledge** (aware vs. unaware)	***APOE* Genotype X *APOE* Knowledge Interaction**
Memory Controllability Inventory			
Present Ability subscale	$F = 0.04$, $p = .842$	$F = 0.35$, $p = .556$	$F = 1.75$, $p = .187$
Inevitable Decrement subscale	$F = 0.02$, $p = .882$	$F = 0.44$, $p = .506$	$F = 2.97$, $p = .087$
Potential Improvement subscale	$F = 0.11$, $p = .742$	$F = 0.67$, $p = .415$	$F = 1.80$, $p = .182$
Effort Utility subscale	$F = 0.57$, $p = .452$	$F < 0.01$, $p = .932$	**$F = 6.20$, $p = .014$**
Aging Concerns Scale			
Independence subscale	$F = 0.03$, $p = .860$	$F = 0.07$, $p = .793$	$F = 3.90$, $p = .050$
Modified Alzheimer's Disease (AD) Likelihood subscale	$F = 0.16$, $p = .688$	**$F = 3.97$, $p = .048$**	**$F = 4.08$, $p = .045$**
Metamemory in Adulthood Anxiety subscale	$F < 0.01$, $p = .933$	$F = 0.09$, $p = .760$	$F = 1.95$, $p = .164$
Attentional Control	$F = 3.40$, $p = .067$	$F = 0.10$, $p = .751$	**$F = 4.03$, $p = .046$**

Note: Degrees of freedom for all F values are (1, 191)

Table 4

ANOVA Results: Main Effects and Interaction Effects of APOE ε4 Carrier Group and APOE Knowledge on Objective Cognition

Dependent Variable	***APOE* Genotype** (ε4 carrier vs. non-carrier)	***APOE* Knowledge** (aware vs. unaware)	***APOE* Genotype X *APOE* Knowledge Interaction**
Forward Digit Span	$F = 0.12$, $p = .725$	$F = 0.03$, $p = .870$	$F = 0.14$, $p = .709$
Backward Digit Span	$F = 1.76$, $p = .187$	$F < 0.01$, $p = .924$	$F < 0.01$, $p = .955$
Item Memory			
Corrected recognition faces	$F = 0.80$, $p = .372$	$F = 0.74$, $p = .390$	$F = 0.06$, $p = .816$
Corrected recognition places	$F = 1.72$, $p = .191$	$F < 0.01$, $p = .933$	$F = 0.80$, $p = .372$
Associative Memory			
Corrected recognition pairings	$F = 0.85$, $p = .357$	$F = 0.81$, $p = .369$	$F = 0.08$, $p = .779$

Note: Degrees of freedom for all F values are (1, 191)

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Data Availability Statement

The data reported here has not been previously published and is available from the corresponding author.

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