

Modelling Decision-Making in Alzheimer's Patients and Healthy Controls



Lecturer
Andreas Højlund

Aarhus University
Jens Chr. Skous Vej
8200 Aarhus N



Abstract

This study investigated the difference in the cognitive processes underlying decision-making in Alzheimer's Disease (AD) patients and healthy elderly controls (HC) during the Iowa Gambling Task (IGT). AD, a leading cause of dementia, is known to impact cognitive functions crucial for decision-making. Several studies have showed decision-making deficits in AD patients based on behavioural performance metrics of the IGT. However, previous analyses of IGT data from AD patients do not suffice in directly discerning the underlying processes that account for these deficits (Haines et al., 2018). Utilising the Outcome-Representation Learning (ORL) model, this project investigates the latent psychological processes driving IGT performance through cognitive modelling. IGT data from two studies involving AD patients and healthy controls, comprising a total of 80 participants, was analysed using hierarchical Bayesian modelling approach. Posterior distributions were estimated for each of the free parameters of the ORL model using two hierarchical models one estimating a group mean for each group, and the other modelling the difference between the groups directly. Based on the first model, the 95% credible interval (CI) of the posterior of the estimated difference in learning rates for reward and punishment excluded 0, providing evidence of differences between AD and HC. This was supported by the second model, as the 95% CI of group mean posteriors did not overlap. In sum, the results suggest that AD patients exhibited poorer performance when it came to recalling prior responses or acquiring new associations between stimuli and rewards linked to specific decks. These findings align both with the parietal atrophy and related cognitive impairment reported in the AD pathology.

Keywords: Alzheimer's Disease, Iowa Gambling Task, Decision-Making

Code availability: All code used for the project can be found on 



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1 Introduction

1.1 Alzheimer's Disease

1.1.1 Neuropathology of Alzheimer's Disease

Ageing is an inevitable natural biological process affecting the structure and function of the brain, leading to increased risk of developing dementia (Sahathevan, 2015). Dementia is a clinical syndrome caused by neurodegeneration which adversely impacts cognitive function, including memory, thinking, and behaviour (Viswanathan et al., 2009). Alzheimer's disease (AD) is the main cause of dementia (Scheltens et al., 2021) and most widespread neurodegenerative pathology globally (Gaubert & Chainay, 2021). Afflicting millions of individuals worldwide (World Health Organization, 2017), AD is a fatal progressive neurodegenerative disorder that poses a significant burden on affected individuals, their relatives, and society as a whole. Neurodegeneration, also known as cerebral atrophy, refers to the destruction of neurons and their connections (Pini et al., 2016). The progression is marked by the buildup of amyloid- β peptide and tau proteins, forming amyloid plaques and neurofibrillary tangles, respectively (Masters et al., 2015; Selkoe, 2001), causing a progressive deterioration in cognitive functioning (Davis et al., 2017; Gleichgerrcht et al., 2010). In the early stages, degeneration typically occurs in the frontal and temporal lobes of the brain, particularly in memory-related areas such as the hippocampus (Masters et al., 2015). Notably, the entorhinal cortex, a cortical region in the medial temporal lobe providing input to the hippocampus, is typically one of the brain regions in which alterations caused by AD are first detected (Bear et al., 2016; Igarashi, 2023; Maruszak & Thuret, 2014). As the disease progresses, additional brain regions become increasingly involved (Braak & Braak, 1991; Gleichgerrcht et al., 2010; Naggara et al., 2006).

1.1.2 Cognitive Impairments in Alzheimer's Disease

The cognitive impairments present in patients with Alzheimer's Disease encompass a broad range of functions, prominently including a deterioration in memory, especially of the episodic and declarative type (Förstl & Kurz, 1999). These memory deficits frequently occur alongside impairments in other cognitive domains, including language processing, visuospatial skills, and executive functioning (EF) (Ramirez-Gomez et al., 2017). As highlighted by Schepers et al., 2023, both episodic memory and EF are essential to learning new information, monitoring performance, and flexibly adapting one's behaviour based on environmental changes. Conse-

quently, AD-related memory and EF impairments hinder patients' ability to effectively store, recall, and update information pertinent to decision-making, thereby interfering their ability to adapt efficiently (El Haj et al., 2020; Schepers et al., 2023). Adaptability is crucial to make decisions that lead to the most advantageous outcomes in uncertain and changing environments (Collins & Shenhav, 2021). In fact, AD is well-established as having a detrimental impact on patients' ability to make advantageous decisions, a fundamental skill crucial for preserving autonomy (Gaubert et al., 2022; Jacus et al., 2018; Kim et al., 2002). Numerous studies consistently report that AD patients underperform compared to healthy controls (HC) in laboratory decision-making tasks, often resorting to random choices and exhibiting reduced strategic stability due to difficulties in learning from feedback (Delazer et al., 2007; Jacus et al., 2018; Sinz et al., 2008; Torralva et al., 2000; Zamarian et al., 2010). This illustrates how learning, memory, and decision-making are closely related to observed behaviour, and how AD patients may lack the cognitive flexibility needed to adjust behaviour based on positive and negative outcomes of decisions.

Research indicates that the neuroanatomical changes in AD can affect the emotional network (Silva et al., 2021; Warren, 2022; Weiss et al., 2008). This has led researchers to link the observed decreased ability to learn and adapt in response to positive and negative outcomes to AD-related deficits in emotional processing (Bucks & Radford, 2004; Chaudhary et al., 2022; Chu, 1997), more particularly apathy, i.e., the most common neuropsychiatric symptom in AD (Dolphin et al., 2023; Nobis & Husain, 2018). Apathy is characterised by a lack of motivation, reduced emotional responsiveness, diminished initiative, and a general disinterest in activities and social interactions (Marin, 1991). Several studies have found evidence that apathy affects cognitive functioning, particularly impacting decision-making abilities (Bayard et al., 2014; Daumas et al., 2023).

In a paper published in 2021, Gaubert and Chainay conducted a systematic review on decision-making deficits in AD patients and its influence on their everyday life. Based on decades of research in this field, they propose that since decision-making appears to be based on cognitive and emotional processes impaired in AD patients, it follows that decision-making abilities of AD patients will likewise be impaired (Gaubert & Chainay, 2021).

While the idea that AD is associated with impaired decision-making is well-established, the specific mechanisms influencing these decision-making processes can vary significantly. Such variations depend on factors such as the individual's characteristics, the nature of the task, and the type of decision to be made. Advancing the knowledge of how cognitive functioning and decision-making processes differ between AD patients and healthy individuals is crucial. This need is underscored by

the rapid growth of the global ageing population, which presents new challenges and significant financial costs to societies worldwide (Livingston et al., 2017; Prince et al., 2013).

One way to advance the scientific understanding of decision-making deficits in AD patients is by developing and applying methods to assess and understand these decision-making deficits and how they translate into specific decision-making challenges faced by AD patients. In response to this need, the Iowa Gambling Task has been applied in several studies on AD, drawing from its proven usefulness in discerning decision-making impairment in other clinical populations.

1.2 The Iowa Gambling Task

■ The Iowa Gambling Task (IGT) is an experienced-based paradigm commonly used in decision-making research within the fields of psychology and cognitive science. In fact, it is the most commonly used behavioural task in literature on decision-making under ambiguity and uncertainty (Moreno-Padilla et al., 2023). It was originally developed by Bechara et al., 1994 to investigate decision-making deficits of patients with lesions to the ventromedial prefrontal cortex. Its design aims to realistically simulate real-life decision-making, incorporating elements of reward, punishment, learning, and decisions made under conditions of risk and uncertainty. To succeed in the task, participants are required to integrate information pertaining to their wins and losses, as well as to weigh the risks and benefits associated with each possible choice throughout the game. As such, the decision-making processes employed in the IGT relies on both learning and memory, as participants must not only process information about past outcomes but also use that information to guide their future decisions effectively.

In the task, participants are presented with four decks of cards (labelled A, B, C, and D) and instructed to successively choose a card from one of the decks. Each choice returns immediate feedback in the form of either a monetary reward or loss. The task comprises 100 trials and the goal is for the participant to maximise their net outcome over the trials. The decks differ in their payoff schemes. Deck A and B are disadvantageous as they are associated with high immediate rewards but even higher unpredictable occasional losses, resulting in negative long-term outcomes. Deck C and D are advantageous decks as they are associated with lower immediate rewards but also smaller occasional losses, resulting in a positive long-term net outcome. Additionally, the IGT varies the frequency of losses among the decks; Decks A and C have frequent losses, while Decks B and D have infrequent losses. The IGT aims to determine whether, across the trials, participants learn to prefer the advantageous decks over the bad, risky decks, since this is the only choice pattern resulting in a

maximisation of long-term net outcomes.

1.3 The Iowa Gambling Task in Alzheimer's Research

Across several studies using the IGT, AD patients have been found to exhibit poorer performance than their age-matched healthy counterparts (Bayard et al., 2015; Bertoux et al., 2012; Gaubert et al., 2022; Hot et al., 2013; Jacus et al., 2018; Moreno & Alameda, 2011; Sinz et al., 2008; Sun et al., 2020; Torralva et al., 2000). These studies typically evaluate performance by examining the obtained net outcome and deck shifting behaviour to assess whether participants succeed in adapting to an advantageous strategy over the course of multiple trials. More specifically, the findings from these studies suggest that AD patients choose disadvantageous decks more often than HC and fail to adapt their strategy based on feedback, resulting in obtaining lower net gains. Furthermore, findings suggest that AD patients tend to shift more frequently among decks, tend to make decisions randomly, and fail to establish an advantageous strategy over the trials (Sinz et al., 2008).

Researchers have employed various approaches in their efforts to explain the causes of AD patients performing more poorly than HC in the IGT. The most prominent approach is to correlate behavioural IGT data with other behavioural measures of cognitive functioning. For instance, Torralva et al. (2000) tested for correlation between deficits in decision-making and deficits in other cognitive functions in AD patients. They found statistically significant correlations between the number of disadvantageous choices in the IGT and impairments in both verbal and visual anterograde memory in AD patients. Optimal task performance in the IGT requires the participant to remember the feedback, that is, the consequence of choosing the different decks, and inability to memorise this information would imply inability to identify or distinguish between the advantageous and disadvantageous decks (Gaubert & Chainay, 2021). Likewise, Alameda-Bailén et al. (2017) used a shortened version of the IGT and found that the propensity of AD patients to make unfavourable choices was attributable to deficits in declarative memory. This also aligns with findings from (Sinz et al., 2008), who reported that AD patients displayed challenges in learning from feedback and subsequently sustaining a consistent strategy over the course of the IGT. In a study conducted by Bayard et al., 2015, participants' explicit understanding of the IGT contingencies was evaluated post-task and it was found that only 10% of the AD participants reported correct full explicit knowledge of the task as compared to 35% of the HC. Ability to acquire full explicit knowledge was found to be a good indicator of making advantageous decisions in the IGT, and all AD patients who made advantageous decisions also displayed correct full explicit knowledge of the IGT contingencies. Behavioural IGT

data has also been correlated with measures of emotional processing and emotional apathy (Bayard et al., 2014; Hot et al., 2013).

Building upon research on emotional and cognitive dysfunctions in AD, Hot et al. (2013) contrasted the decision-making performances on IGT of AD patients with HC under standard and happiness-induced conditions. They found that the happiness condition significantly increased AD patient performance level in the IGT compared with that of the control subgroups, interpreting their findings as support for the idea of affective states modifying performance in the IGT.

In a study from 2014, Bayard et al. examined the impact of apathy on IGT performance. Both MCI and AD patients had higher apathy than HC, and scoring high on apathy was strongly associated to the disadvantageous decision-making profile observed in AD patients (Bayard et al., 2014). The authors interpreted their findings in light of the somatic markers hypothesis (SMH), highlighting that performance on the IGT has previously been attributed to dysregulation of somatic markers (Bechara & Damasio, 2002). The SMH posits that emotional and physiological responses play a crucial role in decision-making (Damasio, 1996). In the context of this study, the authors proposed that the absence of physiological and emotional responses resulting from apathy in aMCI and AD patients deprive them of important internal cues needed for making advantageous decisions in the IGT.

Lastly, attempts have also been made to correlate behavioural IGT data with neuroimaging data. In a study from 2013, behavioural IGT data of AD patients was covaried against T1 MRI scans. They found correlations between impaired IGT performance and atrophy in the parietal and temporal cortex of AD patients (Kloeters et al., 2013).

While these studies provide support for the existence of decision-making deficits in AD patients, they do not enable us to directly discern the underlying psychological processes that account for these deficits. The analyses of these studies typically involve computing individual net-scores for each subject by subtracting the number of times the participant chose a disadvantageous decks from the number of times the participant chose an advantageous deck across a selected interval of trials (e.g. across all 100 trials or per 10-trial or 20-trial blocks). This is often followed by comparisons of mean scores across participant groups at different intervals, which can then be correlated with other measures. Such analyses have often proven useful to discern clinical populations from healthy populations based on behavioural performance data. However, the IGT involves complex decision-making processes, including aspects of learning, risk assessment, and reward anticipation - and investigating such processes requires different methodological approaches that attempt to account for the latent processes that underlie these observed behaviours (Haines

et al., 2018).

1.4 Cognitive Modelling of Iowa Gambling Task Data

❖ Cognitive modelling can be used to evaluate how well theoretical assumptions regarding latent psychological processes driving decision-making fit behavioural data. By simulating and predicting participant choice behaviour at trial level, cognitive modelling can help describe the latent psychological processes driving decision-making behaviour beyond directly observable behavioural data. Several computational cognitive models have been developed to conceptualise and assess latent psychological processes believed to drive performance on the IGT. Put simply, what is common to these models is their assumption that in each trial, participants choice of a deck is influenced by some expectation pertaining the value associated with choosing that deck, which is in turn influenced by aspects of human cognition formalised by a number of mathematical equations. However, the particular aspects of cognition as well as how these are mathematically defined differ depending on the model.

Some earlier computational models proposed for the IGT are the Expectancy-Valence Learning (EVL) model (Stout, 2002), the Prospect Valence Learning Model (PVL; Ahn et al., 2008), the PVL model with Delta rule (PVL-Delta; Ahn et al., 2008; Fridberg et al., 2010; Steingrover et al., 2013), and the Valence-Plus Perseverance Model (VPP; Worthy et al., 2013). All of these models have been used to identify group level differences in cognitive mechanisms between HC and various clinical populations. Nonetheless, Haines et al., 2018 found that neither of these models displayed optimal performance for both short- and long-term prediction accuracy and parameter recovery, three metrics used for evaluating how well such models are capable of capturing the latent cognitive processes driving decision making. This lead them to propose a novel reinforcement-learning model, the Outcome-Representation Learning (ORL) model, which they claim provides the best compromise between the previously-mentioned competing models. Relative to other computational models used in the IGT, the ORL has exhibited enhanced or equivalent results in several critical metrics, including post hoc model fit, simulation performance, and parameter recovery, as reported by Haines and colleagues Haines et al., 2018.

1.4.1 Outcome-Representation Learning Model

❖ The Outcome-Representation Learning (ORL) model is a computational reinforcement learning model assuming that four distinct cognitive strategies drive



decision-making in IGT: maximising long-term expected value, maximising win-frequency, choice perseveration, and reversal learning (Haines et al., 2018).

The ORL model assumes that the decision-maker’s choice is influenced by five free parameters that all capture distinct latent psychological processes driving IGT behaviour. These parameters are: a learning rate for reward (A_{rew}), a learning rate for punishment (A_{pun}), a weight representing the win frequency sensitivity (ω_F) indicating if participant display perseverance with decks associated with higher win frequencies, a memory decay parameter (K) with low values indicating slow forgetting of past deck choices, and lastly, a perseveration weight indicating the tendency to stay or switch decks (ω_P). As such, the ORL model allows for the estimation of these five free parameters. The relations between all the parameters in the ORL model are illustrated in the plate notation in figure 1. We describe each parameter in detail below, based on the model specification presented by Haines et al. (2018).

The ORL model assumes that for each trial, deck selection is guided by a valence model that linearly integrates value, frequency, and perseverance signals:

$$V_t^d = EV_t^d + EF_t^d \cdot \omega_F + PS_t^d \cdot \omega_P \quad (1)$$

This valence model is informed by several parameters: V_t^d denotes the value signal for each deck d on trial t . EV_t^d denotes the expected value of a given deck on a given trial. As EV_t^d is not weighted, ORL assumes it has a weight of 1, thus serving as a reference point which frequency and perseverance effects are evaluated against. EF_t^d denotes the expected outcome frequency of a given deck on a given trial. The win frequency sensitivity parameter ω_F ($-\infty < \omega_F < \infty$) is a weight reflecting the influence of win/loss frequency on the expected value of each deck relative to the outcome magnitude. A value of $\omega_F < 0$ indicates that the decision maker prefers decks with low win frequency. Oppositely, a value of $\omega_F > 0$ indicates a preference for decks with high win frequency. PS_t^d refers to the perseverance of deck d on trial t . The perseverance weight (ω_P) reflects the effect of perseverance on valence relative to outcome magnitude ($-\infty < \omega_P < \infty$). Values of $\omega_P < 0$ indicate a preference for switching decks more frequently, whereas values of $\omega_P > 0$ indicate a preference to stay with recently chosen decks.

The updating of the expected value EV_t^d of the chosen deck d uses separate learning rates for positive and negative outcomes and is formalised by the expected value learning rule:

$$EV_t^d = \begin{cases} EV_{t-1}^d + A_{rew} \cdot (x_{t-1} - EV_{t-1}^d), & \text{if } x_{t-1} \geq 0 \\ EV_{t-1}^d + A_{pun} \cdot (x_{t-1} - EV_{t-1}^d), & \text{otherwise} \end{cases} \quad (2)$$

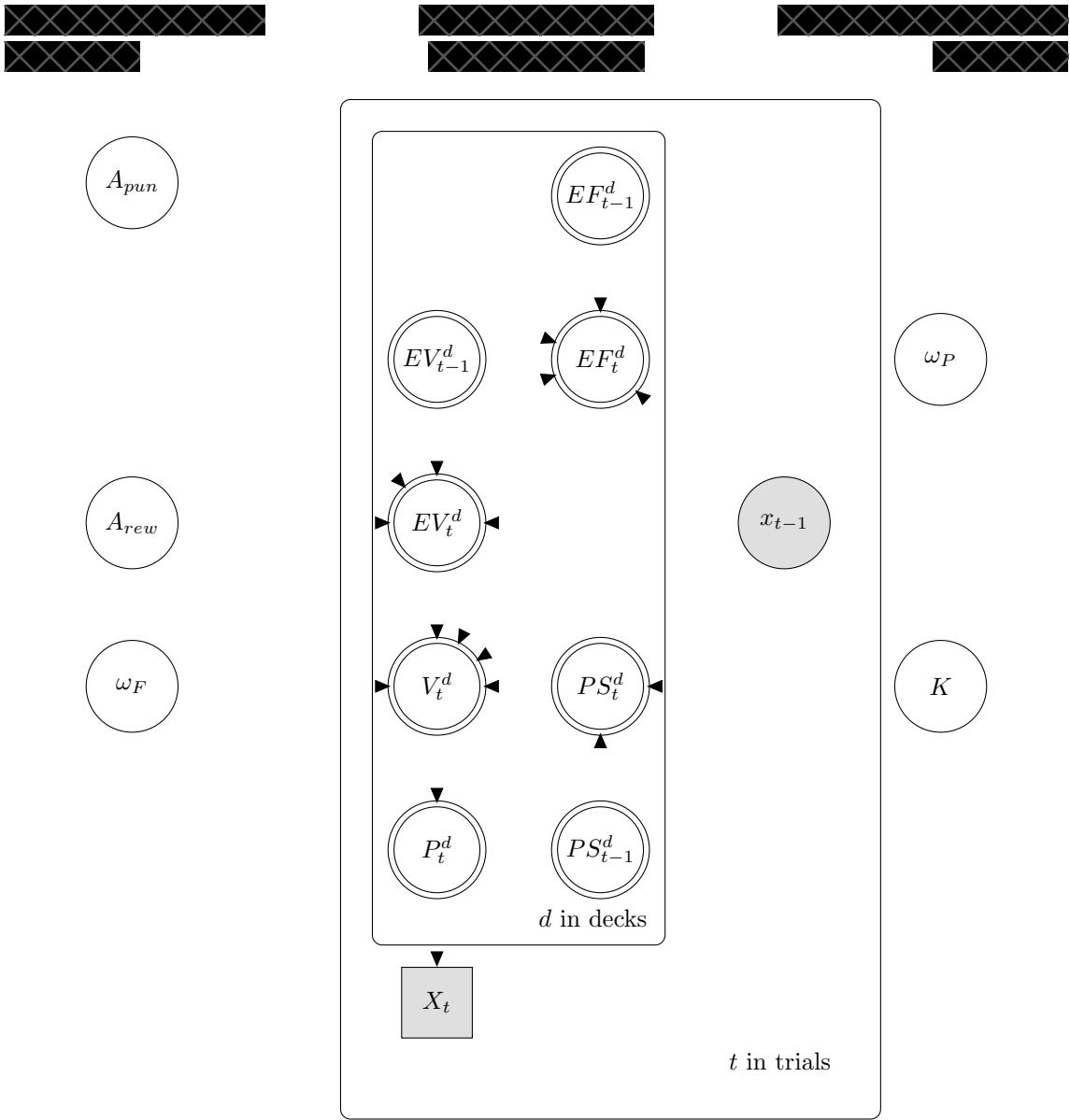


Figure 1: Subject-level plate notation for the ORL model. Circle nodes represent continuous variables, while the square node indicates that X_t is a discrete variable. Single-lined nodes denote stochastic variables, while double-lined nodes signify deterministic ones. Unshaded nodes represent latent variables, whereas shaded nodes denote observed variables.

As depicted in equation 2, the objective outcome on the previous trial used to update expected values is denoted x_{t-1} . The learning rate used to update expectations based on reward ($x_{t-1} \geq 0$) is denoted A_{rew} ($0 < A_{rew} < 1$), while the learning rate used to update expectations based on punishment ($x_{t-1} < 0$) is denoted A_{pun} ($0 < A_{pun} < 1$). By using these two separate learning rates for positive and negative outcomes, the ORL model is able to account for over- and under-sensitivity to losses and gains. The difference between the two learning rates indicate how the decision maker’s learning is dominated by either positive or negative outcomes. A large difference between these two learning rates indicates that learning is more dominated by either positive or negative outcomes. However, literature has shown that individuals, irrespective of the long-term expected value, tend to prefer decks



that are associated with a high win frequency (Chiu and Lin, 2007, as cited in Haines et al., 2018). This means that if two decks have the same long-term value, people would tend to pick cards from the deck associated with the most frequent smaller wins rather than one with less frequent but larger wins. To account for this win frequency effect, the expected outcome frequency, EF_t^d of deck d on trial t is modelled as:

$$EF_t^d = \begin{cases} EF_d^t + A_{\text{rew}} \cdot (sgn(x_{t-1}) - EF_{t-1}^d), & \text{if } x_{t-1} \geq 0 \\ EF_d^t + A_{\text{pun}} \cdot (sgn(x_{t-1}) - EF_{t-1}^d), & \text{otherwise} \end{cases} \quad (3)$$

In equation 3 the learning rates A_{pun} and A_{rew} are the same as described in equation 2. The sign function $sgn(x_{t-1})$ returns 1 if the objective outcome on the previous trial $x_{t-1} > 0$, returns 0 if $x_{t-1} = 0$ and -1 if $x_{t-1} < 0$. Furthermore, the model incorporates a reversal learning component for EF_t^d which models $EF_t^{d'}$, the expected outcome frequency of all unchosen decks, d' , on trial t as follows:

$$EF_t^{d'} = \begin{cases} EF_{t-1}^{d'} + A_{\text{pun}} \cdot \left(\frac{-\text{sgn}(x_{t-1})}{c} - EF_{t-1}^{d'} \right), & \text{if } x_{t-1} \geq 0 \\ EF_{t-1}^{d'} + A_{\text{rew}} \cdot \left(\frac{-\text{sgn}(x_{t-1})}{c} - EF_{t-1}^{d'} \right), & \text{otherwise} \end{cases} \quad (4)$$

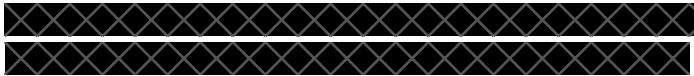
In equation 4, c represents the number of possible alternative choices for the chosen deck. In the standard version of the IGT with four possible cards to choose from, $c = 3$. The learning rates are again shared from the expected value learning rule in equation 2. The implication of equation 4 is that if the outcome of a chosen deck d is negative, the expected value of all unchosen decks d' will be updated using the reward learning rate, A_{rew} . Reversely, all unchosen decks will be updated using the punishment learning rate, A_{pun} in response to a positive outcome.

Additionally, a choice perseverance model is incorporated in the ORL to capture tendencies to stay or switch decks irrespective of the outcome:

$$PS_t^d = \begin{cases} \frac{1}{1+K}, & \text{if } X_t = d \\ PS_{t-1}^d, & \text{otherwise} \end{cases} \quad (5)$$

In equation 5, PS_{t-1}^d refers to the perseverance of deck d on the previous trial. X_t denotes the chosen deck on the given trial. The memory decay parameter K represents how quickly decision makers forget their past deck choices. High values of K indicate that decision makers have short memory of their own deck choices, whereas low values of K indicate that decision makers have long memory of their own deck choices. The perseverance of the chosen deck d on a given trial t is set to $\frac{1}{1+K}$, whereas the perseverance of unchosen decks decay exponentially.

Choice probabilities (P_t^d), i.e., the probability of choosing a particular deck d in



a given trial t , is derived by applying a softmax function:

$$P_t^d = \frac{e^{\theta \cdot V_{t-1}^d}}{\sum_{d=1}^4 e^{\theta \cdot V_{t-1}^d}} \quad (6)$$

The softmax function described in Haines et al. (2018) takes the value signal for each deck d on the previous trial $t - 1$ and includes a choice consistency parameter θ . However, θ is fixed at 1 due to parameter identifiability problems between θ , ω_P and ω_F .

In sum, applying the ORL model to IGT data allows us to estimate the following parameters, each representing distinct latent psychological processes: learning rate for reward (A_{rew}), learning rate for punishment (A_{pun}), a memory decay parameter (K), win frequency sensitivity weight (ω_F) and a perseverance weight (ω_P).

1.5 Research Question and Hypotheses

XXXXXX The primary aim of this study is to investigate the cognitive processes underlying decision-making in AD patients as compared to HC during the performance of the IGT. Previous research using frequentist statistics have found decision-making deficits in AD patients as measured by poorer performance on the IGT. Although attempts have been made to explain these observed deficits by correlating IGT data with other cognitive measures, correlating observable performance measures does not suffice in explaining latent psychological processes driving decision-making behaviour. However, cognitive modelling of IGT data allows theoretical assumptions about such cognitive processes to be tested explicitly through models such as the ORL. Therefore, we see a potential in supplementing these findings with cognitive modelling of IGT data from AD patients using the ORL model.

We hypothesise that these deficits in AD patients arise from a reduced capacity to adjust their behaviour in response to both positive and negative outcomes, indicating slower adaptation in their decision-making strategies during the IGT. This slower adaptation is believed to be a consequence of the memory impairment and apathy characteristic of Alzheimer's disease, which hinders AD patients' ability to effectively recall their previous responses and establish and retain new associations between stimuli and rewards or punishments for specific decks. Consequently, we hypothesise observing lower learning rates for both punishment and reward in the AD group compared to HC.

Furthermore, it is hypothesised that the memory decay parameter K will be notably higher in AD patients than that of the HC group. This higher K value would suggest that AD patients have shorter memories of their previous choices, further contributing to their difficulties in adaptive decision-making during the IGT.

In sum, we present the following hypotheses:

H1: The learning rate for reward (A_{rew}) is lower for AD patients as compared to healthy controls.

H2: The learning rate for punishment (A_{pun}) is lower for AD patients as compared to healthy controls.

H3: The memory decay parameter K is higher for AD patients as compared to healthy controls.

2 Methods

All code used for this project can be found on [\[REDACTED\]](#).

2.1 Data Sources

[REDACTED] This project relies on IGT data from two independent studies (Gaubert et al., 2022; Jacus et al., 2018). In the first study by Jacus et al. (2018), 60 participants were grouped based on clinical status: 20 AD patients (10 females, 10 males, mean age = 80.5, age range = 71 – 90), 20 amnestic mild cognitive impairment patients (11 females, 9 males, mean age = 78.5, age range = 61 – 83) and 20 HC (13 females, 7 males; mean age = 71, age range = 64 – 85 years). The current study uses the data from the HC and AD patient groups. AD patients were professionally diagnosed and recruited from the memory clinic at Centre Hospitalier du Val d’Ariège.

The second study by Gaubert et al. (2022) comprised 40 participants, consisting of 20 AD patients (12 females and 8 males, mean age = 72.7), and 20 age-matched HC (12 females, 8 males, mean age = 72.4). The AD patients were diagnosed based on clinical, neuropsychological, and biological assessments and recruited from three university hospitals.

In both studies, participants were native French speakers and screened for both gambling addictions and other major neurological conditions. Participants were instructed that their objective in the IGT was to maximise their winnings in fictitious money over a series of trials and informed that certain decks might be more advantageous or disadvantageous. However, the specific details of which decks were advantageous or disadvantageous were not disclosed. Each study consisted of 100 trials. Access to the data was obtained through agreements with authors of the original studies. For the first study (Jacus et al., 2018), J. Pierre Jacus, Doctor of Psychology at the University of Toulouse-Jean, provided the data. For the second study (Gaubert et al., 2022), Hanna Chainay, professor at Université Lumière Lyon 2, facilitated access. In both cases, the data was shared upon agreement that it

would be exclusively used for this project and not distributed further.

Jacus et al. (2018) calculated an overall net score by subtracting disadvantageous card selections from advantageous ones for the last part of the IGT (trial 41-100) as a measure of decision-making performance, and found poorer performance in the AD group. Gaubert and Chainay (2021) analysed both net scores at various trial intervals and the number of switches between advantageous and disadvantageous choices. While they found that AD patients switched more frequently between advantageous and disadvantageous deck, suggesting inability maintain an advantageous strategy, they found no effect of participant group on net score. However, it is important to note that the group comparisons of net scores in the two respective studies were tested using different statistical methods and were based on different trial intervals. For full details of the analyses, refer to the original studies.

We combined the two dataset, resulting in a total of 80 participants (40 AD and 40 HC). For each trial for each participant in the combined dataset we calculated the outcome (wins subtracted by the losses) and scaled it by dividing by 100.

2.2 Hierarchical Bayesian Analysis

The five free parameters of the ORL model (A_{rew} , A_{pun} , K , ω_P and ω_F) were estimated using a hierarchical Bayesian analysis (HBA) approach (Gelman et al., 2020; Lee & Wagenmakers, 2014; McElreath, 2020). HBA holds the advantage of allowing for group-level hyper parameters to be estimated simultaneously with the estimation of individual-level parameters rather than estimating parameters for each participant separately. Moreover, HBA estimates probability distributions over the full range of possible parameter values providing more information than just a point estimate. The analysis was conducted using PyStan (Riddell et al., 2021, v.3.8), a Python interface to Stan (Stan Development Team, 2023, v.2.33.0). Stan relies on Hamiltonian Monte Carlo (HMC), a type of Markov Chain Monte Carlo (MCMC), to efficiently sample from probabilistic models. All models are sampled with MCMC for 4000 iterations, using the first 1000 iterations as warm-up on 4 sampling chains resulting in 8000 posterior samples for all parameters.

2.3 Model Specification

The ORL model used in this study was described in detail in Section 1.4.1. The analyses conducted in this study rely on three different model specifications of the ORL: a non-hierarchical model (M1) to estimate the parameters for individuals one at a time, a hierarchical model (M2) that estimates a group mean for each parameter and a hierarchical model with comparison (M3) that estimates group



level parameter differences.

Due to the scope of the current project, the non-hierarchical model and the hierarchical model without group comparison will not be elaborated on, but model files can be found in the GitHub repository. However, the following section elaborates on M3, as the main focus of the paper is to investigate differences between AD and HC.

The hierarchical comparison model (M3) models the free parameters using the overall mean across the two groups, the difference between the groups, and a group-level standard deviation (See Figure 2 for plate notation).

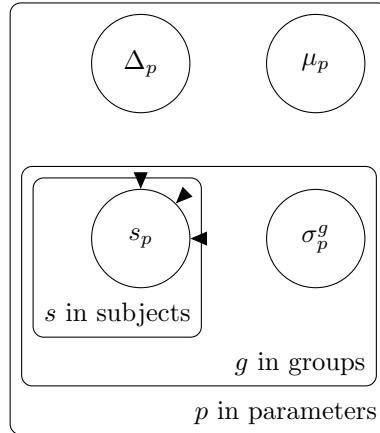


Figure 2: Estimating the five ORL subject-level parameters from an overall parameter mean μ_p , the parameter difference between the two groups Δ_p and a group-level parameter standard deviation σ_p^g .

To demonstrate the modelling the free parameters, we show how A_{rew} is modelled as an example:

$$\mu A_{rew} \sim \mathcal{N}(0, 1) \quad (7)$$

$$\Delta A_{rew} \sim \mathcal{N}(0, 1) \quad (8)$$

$$\sigma A_{rew}^g \sim \mathcal{N}(0, 0.2) \quad (9)$$

The prior of both μA_{rew} and ΔA_{rew} , i.e., the overall mean across the two groups and the difference between the groups is set to a normal distribution with a mean of 0 and standard deviation of 1. The prior of the scale of the group-level distribution (σA_{rew}^g) is set to a normal distribution with a mean of 0 and standard deviation of 0.2.



$$\mu A_{rew}^g = \begin{cases} \mu A_{rew}^g = \mu A_{rew} + \frac{\Delta A_{rew}}{2}, & \text{if } g = \text{HC} \\ \mu A_{rew}^g = \mu A_{rew} - \frac{\Delta A_{rew}}{2}, & \text{if } g = \text{AD} \end{cases} \quad (10)$$

The group-level mean μA_{rew}^g is determined from the overall mean across the two groups and the difference (See equation 10).

$$A_{rew}^{s'} \sim \mathcal{N}(0, 1) \quad (11)$$

$$A_{rew}^s = \text{inv probit}(\mu_{A_{rew}}^g + \sigma_{A_{rew}}^g \cdot A_{rew}^{s'}) \quad (12)$$

The individual-level reward learning rate (A_{rew}^s) is determined by the group-level mean, group-level scale and the individual-level parameter on the unconstrained space ($A_{rew}^{s'}$). The constrained parameters (A_{rew}^s , A_{pun}^s and K^s) are modelled using probit-transformation. The probit function is the inverse cumulative distribution function of the standard normal distribution. We use the inverse probit function which takes a real number between $-\infty$ and ∞ and maps it to a value between 0 and 1. After being inverse probit-transformed, K is further multiplied by 5 following the procedure by Haines et al. (2018), allowing it to range from 0 to 5. Unbounded parameters are modelled in a similar fashion, except that no transformation is applied and the group-level σ prior is set to a half-cauchy(0, 1).

2.4 Parameter Recovery

In order to make valid inference with model parameters (Haines et al., 2018), one must first validate that the model's parameters are actually identifiable (Wilson & Collins, 2019). This can be achieved through a parameter recovery process. Parameter recovery involves simulating data with known parameters, and then applying the cognitive model to this data to assess the model's ability to accurately recover parameters. To match the condition under which the experimental data used on this study was collected, we simulate IGT data using the same payoff structure as employed by Jacus et al. (2018). The simulated outcomes were scaled by dividing by 100, to match scaling of the experimental data mentioned in section 2.1.

2.4.1 Parameter Recovery Procedure

For each of the free parameters, the mean of the posterior estimates were compared to the true sampled parameters. The ability of the model to recover parameters was tested for each of the three models. *M1*: To test how well M1 recovers individual-level parameters, data was generated by simulating responses from 100 participants, drawing parameter values from uniform distributions with



specified minimum and maximum values. We sampled A_{rew} and A_{pun} in the range $[0, 1]$, K in the range $[0, 5]$, and the weighting parameters ω_P and ω_F in the range $[-2, 2]$. Using the same approach as Haines et al. (2018), we set the choice consistency θ to a fixed value of 1.

M2: To test recovery of group-level parameters, we simulate data for 100 groups independently of each other. Group means for each parameter for each group is drawn by sampling from uniform distributions in the ranges previously specified. From a normal distribution with the group-level mean and a standard deviation of 0.05, individual-level parameters are drawn. As A_{rew} ($0 < A_{rew} < 1$) and A_{pun} ($0 < A_{pun} < 1$) and K ($0 < K < 5$), we draw another value if it falls outside of the bounds. We simulate 40 participants in each group, matching the number of participants after pooling the two datasets (40 HC and 40 patients with AD). Before plotting, the estimated μA_{rew} , μA_{pun} and μK are inverse probit-transformed to the constrained space, and μK is further multiplied by 5.

M3: To test how well the M3 recovers the Δ parameters, i.e., the differences in parameters between two groups, we simulate 20 groups using the same procedure as for M2. We recover each Δ parameter for all pairs of simulated groups. As the group-level parameters are informed by the individual-level parameters estimated using inverse probit-transformation, the recovered Δ s are not in an immediately interpretable range. In order to assess the model's capability to recover group-level differences of the bounded parameters (A_{rew} , A_{pun} and K), we normalised each of the estimated bounded parameters to the range of the true parameters using the following formula:

$$x_{normalised} = (b - a) \frac{x - min(x)}{max(x) - min(x)} + a \quad (13)$$

Where b is the maximum of the true parameter, a is the minimum of the true parameter and x is the estimated parameter



2.4.2 Parameter Recovery Results

The parameter recovery results for each of the three models are combined in figure 3.

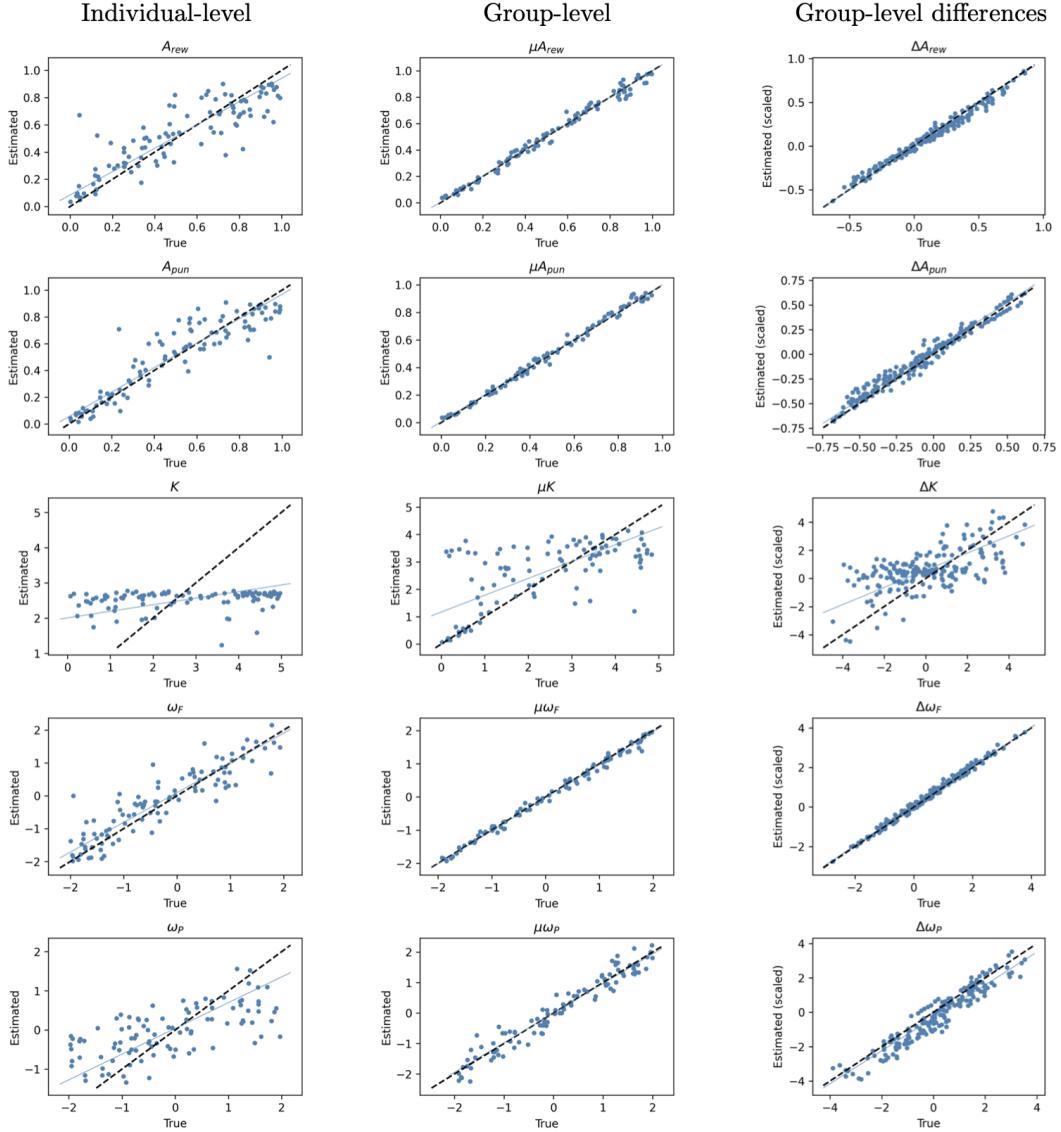


Figure 3: Parameter recovery of individual-level, group-level and group-level differences in column 1, 2 and 3 respectively. The x-axis shows the true parameter value and the y-axis shows the mean of the posterior distribution of the parameter. The black dashed line indicates perfect recovery and the blue line shows a regression line fitted to the points

M1: M1 displayed moderate recovery for the parameters for A_{rew} , A_{pun} , and ω_F within their specified ranges. While recovery of these parameters are scattered away from the dashed line indicating perfect recovery, the estimation error show no signs of systematic over- or underestimation of the true values. This is evident by the blue regression line fitted to the points being close to the dashed line. Recovery of ω_P shows some bias, as the model seems to overestimate smaller values and



underestimate larger values. The parameter K is particularly poorly recovered, not allowing for interpretation of estimated values of this parameter using the non-hierarchical model.

M2: As evident in Figure 3, the group mean of all the free parameters of the ORL model are well-recovered by the hierarchical model, except for K . For K , M2 seems to be highly biased in its recovered estimates.

M3: Patterns similar to the M2 recovery emerge. For each of the parameters ΔA_{rew} , ΔA_{pun} , $\Delta \omega_F$, and $\Delta \omega_P$, the data points are densely clustered around the line of perfect recovery. This suggests that the model is highly accurate in estimating the mean differences for these parameters between the two groups. Again, the parameter ΔK displays poor recovery, particularly with a tendency for overestimation of lower values of ΔK . Thus, estimated values of ΔK should be interpreted with care.

2.5 Parameter Estimation

Having established that the two hierarchical models (M2 and M3) can satisfactorily recover known parameters based on simulated data for all parameters except K , the next step is estimating how decision making processes differ between AD patients and HC from real data. To do so, the hierarchical models are fitted to the IGT data from the two groups collected by Jacus and colleagues (Jacus et al., 2018).

Before making inferences from the posteriors it is crucial to first ascertain the models' descriptive adequacy and convergence diagnostics. The following sections describe this process in detail for M3, the model used for addressing our hypotheses. Descriptive adequacy metrics and convergence diagnostics for M2 can be found in the Appendix C & D.

2.5.1 Posterior Predictive Checking

Assessing the descriptive adequacy of a model involves evaluating how well it captures the observed data. We accomplish this through posterior predictive checking, a method in which data is simulated under the fitted model based on the posterior and then compared to the observed data (Gelman et al., 2020). Specifically, we compare the deck choice as predicted by the model with the deck choice actually made by each participant in each trial. For each trial, the mode of the model's predictions (i.e., the deck the model predicts the most over all samples) is determined. We calculate the accuracy of the model for each participant. We calculate the chance-level at $\alpha = 0.05$ taking the number of decks and trials into account, using the binomial cumulative distribution (Combrisson and Jerbi, 2015).

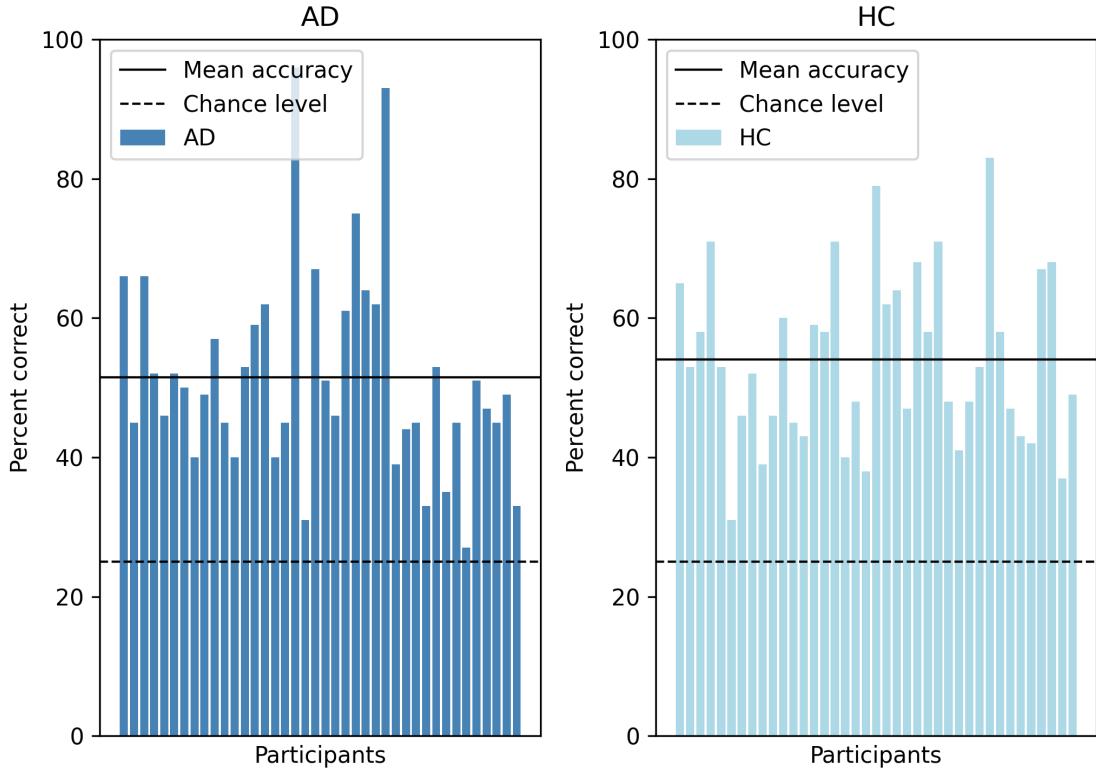


Figure 4: Accuracy of M3 predictions of chosen deck for each participant. The dotted line signifies chance level, with any measurements above this line suggesting that the model has predictive value. The solid line represents the mean accuracy across all participants.

Figure 4 shows the accuracy of M3 in predicting each participant’s chosen deck for each of the two participant groups, providing insight into the overall performance of the model. For some participants, the accuracy of the model’s predictions is considerably above the chance level, while for a few participants, it is closer to the chance threshold. The model performs above chance-level for all participants in both groups, with no indications of systematic differences with respect to the accuracy between the two groups. Overall, the model displayed satisfactory descriptive adequacy.

2.5.2 Convergence Diagnostics

❖ As mentioned in section 2.2, the estimation of posterior probability distributions using Stan relies on Hamiltonian Monte Carlo (HMC). It is important to asses MCMC diagnostics to ensure chain convergence. Convergence refers to the fact that each individual chain explores the right distribution and every chain explores the same high probability region (McElreath, 2020). A useful graphical convergence diagnostic is the trace rank plot. A trace rank plot ranks all parameter samples (lowest rank as 1 and highest equal to the sample count across all chains) (Vehtari



et al., 2021). For each parameter, histograms for the ranks of each chain are built and overlaid. Largely overlapping histograms within the same probability region of the posterior indicates efficient exploration and signifies chain convergence. Figure 5 shows the trace rank plots for the Markov chains of M3.

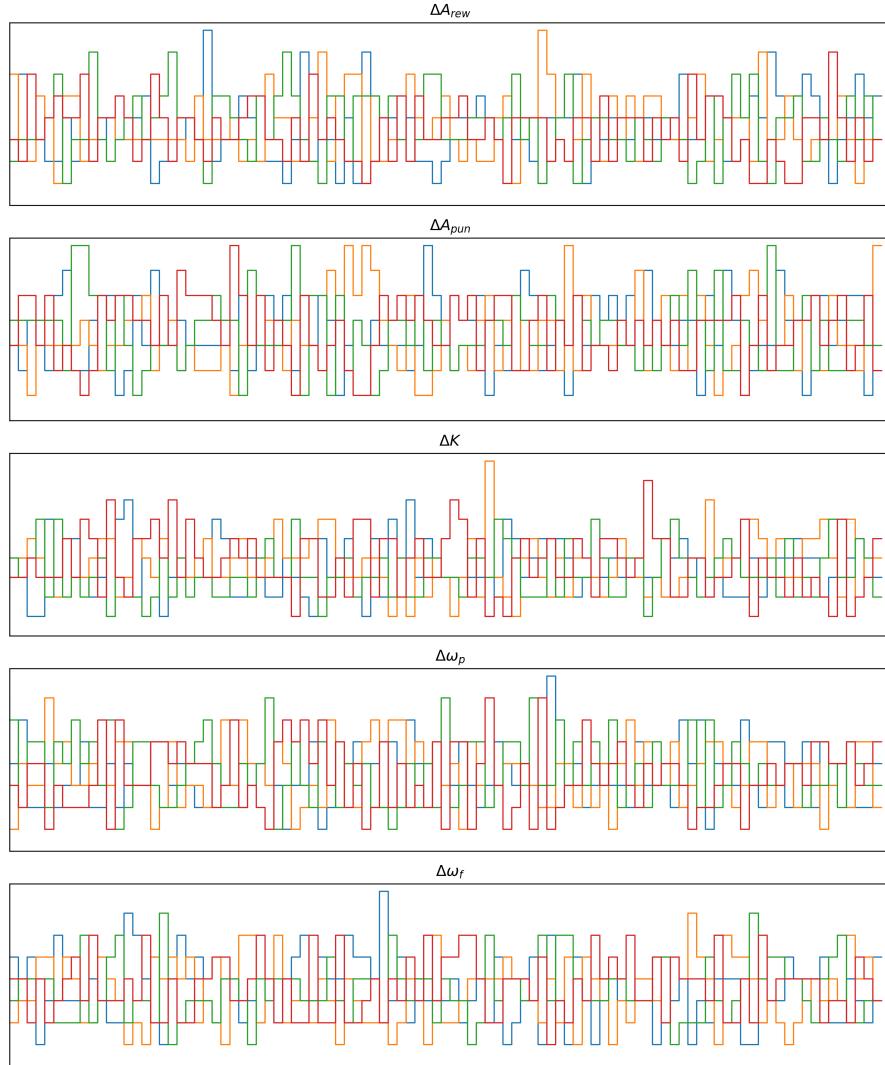


Figure 5: Trace rank plot of group difference parameters Δ

As evident in Figure 5, the Markov chain for each parameter is well mixing and overlapping within the same probability region. Another useful diagnostic for assessing convergence is the Gelman and Rubin potential scale reduction statistic, \hat{R} (Gelman & Rubin, 1992). The \hat{R} statistic quantifies the ratio between the between-chain variability and within-chain variability. An \hat{R} value of, or very close to, 1.0 suggest that the variance within each chain is about the same as the variance across chains, indicating that the chains have converged to a common distribution (Steingroever et al., 2013). Although previous literature suggest that an \hat{R} value less than 1.1 indicate satisfactory convergence of the Markov chains (Gelman & Rubin, 1992;

Gelman et al., 2013), more recent literature advises a more stringent cutoff of 1.01 (Vehtari et al., 2021). In other words, a desirable value of \hat{R} is as close to 1.0 as possible and $= < 1.01$. For all five estimated parameter differences (ΔA_{rew} , ΔA_{pun} , ΔK , $\Delta \omega_F$, and $\Delta \omega_P$) the \hat{R} value was 1.0. Moreover, the number of effective samples were > 1000 for all parameters except for K (see Appendix A).

2.5.3 From Model Evaluation to Inference

Both M2 and M3 displayed satisfactory performance in parameter recovery at both individual and group levels for all parameters except for K . Their ability to accurately capture observed data is further validated by the models descriptive adequacy. Additionally, convergence diagnostics —confirmed by trace rank plots and Gelman and Rubin’s \hat{R} statistic— indicated convergence of the Markov chains. Having established the reliability of the model based on descriptive adequacy and convergence metrics, the next stage is to draw inferences from the models’ application to the experimental data.

We plot the posterior distributions of the delta parameters as well as the distributions of each parameter fitted on the two groups individually. While our main analysis pertains to M3, we plot the estimated group-means from M2 to aid interpretation of numeric values of the bounded parameters.

For each Δ posterior from M3, we assess whether there are differences between AD and HC based on the extracted 95% credible interval (CI). In Bayesian statistics, a 95% CI is interpreted as the interval within which the true value of the parameter has a 95% and probability of falling (Hespanhol et al., 2019). We consider there to be strong evidence of a difference between the groups if the 95% CI of the posterior distribution of the difference parameter (Δ) excludes 0. If the 95% CI of the posterior distribution for a difference parameter (Δ) excludes 0, we consider it substantial evidence for a true difference between the groups.



3 Results

Figure 6 shows the posterior distributions for the Δ parameters, i.e., the differences between AD and HC for each free parameter of the ORL model, as estimated by M3.

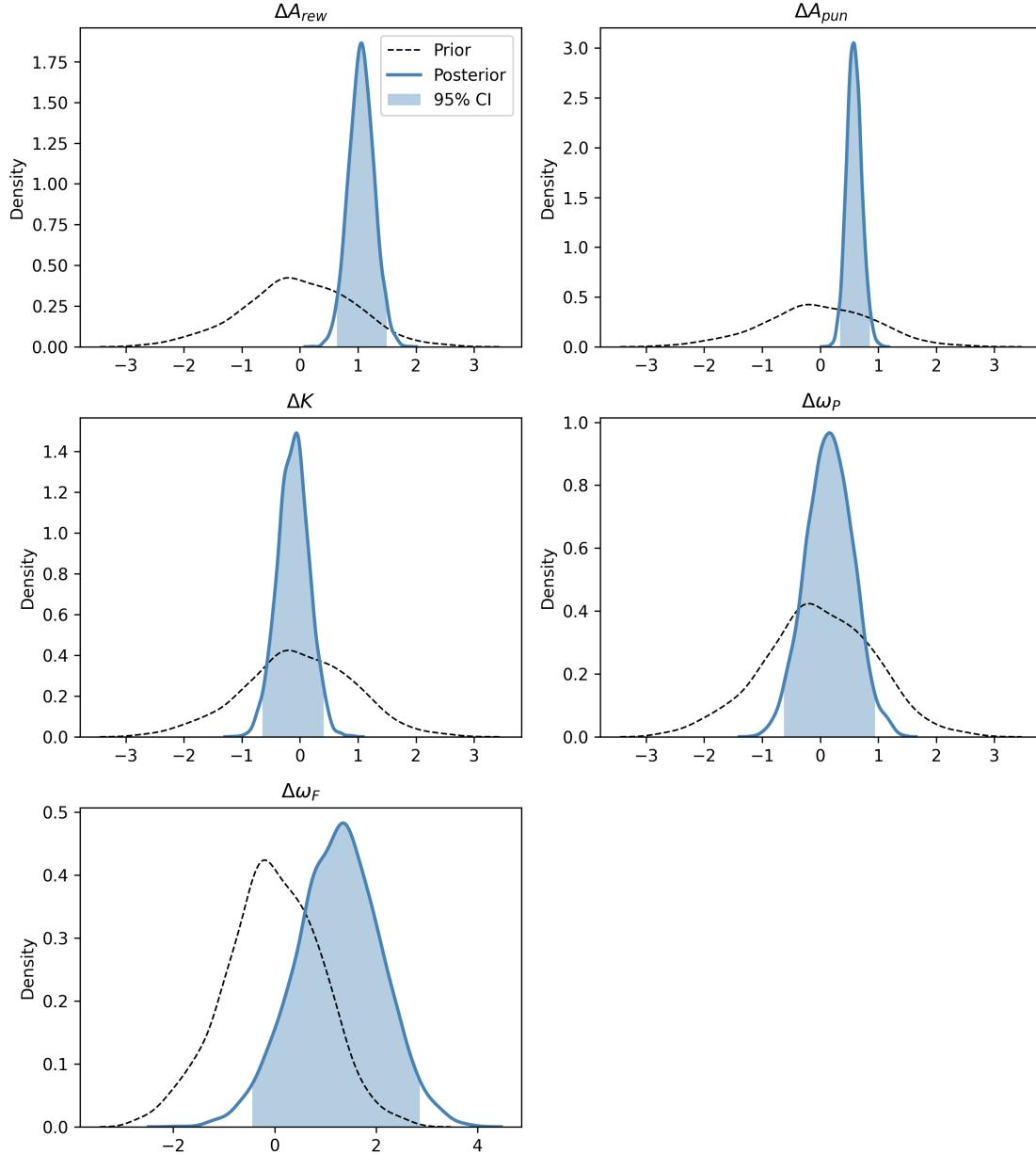
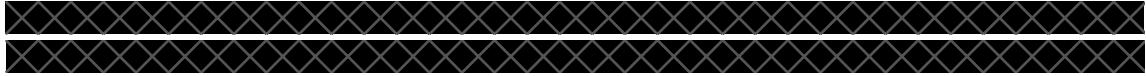


Figure 6: M3 Posterior density distributions of all five parameter differences between AD and HC. The dashed line represents the prior distribution, i.e., the belief about the parameter difference before considering the new data. The solid line represents the posterior distribution, i.e., the updated belief about the parameter difference after the model has seen the data. The shaded blue area under the curve is the 95% CI.

The results of the M3 parameter estimation indicate that the only parameters



for which there is a credible difference between the two groups are ΔA_{rew} ($MLE = 1.19, CI = [0.63, 1.49]$) and ΔA_{pun} ($MLE = 0.59, CI = [0.34, 0.85]$) as the 95% CI for each of these parameters exclude 0. For both parameters, the CI falls within the positive range, signifying that the AD group means for both learning rates are lower than the corresponding group means for HC (refer to Equation for definition of the group mean for each group 10).

\blacksquare The results of the M2 parameter estimation support these findings. This is evident in Figure 7, which shows that the 95% CIs do not overlap for μA_{rew} (HC [0.37, 0.58], AD [0.07, 0.16]) and μA_{pun} (HC [0.07, 0.13], AD [0.02, 0.04]). The M2 parameter estimations show no indications of group differences for the parameters μK , $\mu \omega_P$ and $\mu \omega_F$.

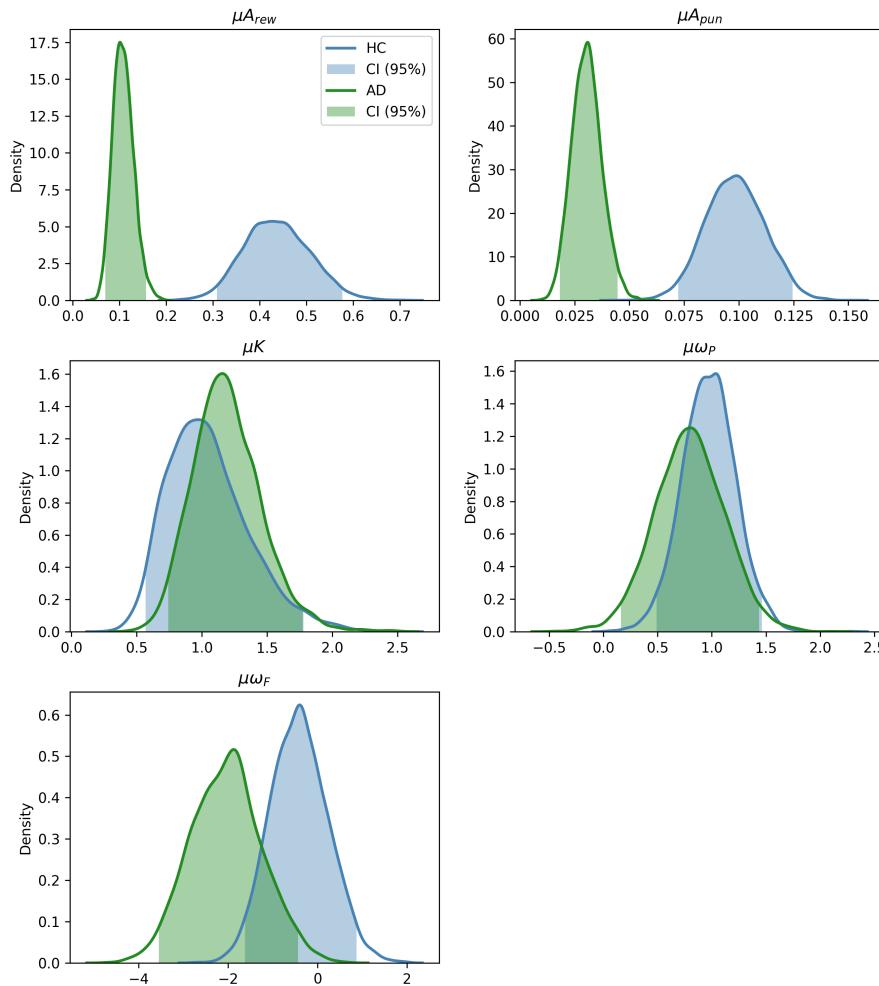


Figure 7: M2 posterior densities of parameters. μA_{rew} , μA_{pun} , and μK have been inverse probit-transformed before plotting. K has additionally been multiplied by 5.

4 Discussion

In this study, we investigated the differences in decision-making between patients with AD and HC by means of cognitive modelling of behavioural IGT data collected from two separate studies.

Compared to HC, AD patients displayed lower learning rates for both punishment and reward, supporting $H1$ and $H2$. This is evident from the posteriors of ΔA_{rew} and ΔA_{pun} from M3 and μA_{rew} and μA_{pun} from M2. These findings suggest that AD patients performed worse in terms of remembering previous responses or learning new stimulus-reward associations for particular decks. These findings align both with the parietal atrophy and related cognitive impairment reported in the AD pathology as mentioned in Section 1.1. The lower learning rates could provide explanation for the decision-making deficits identified in the original studies from which the data for this paper originated.

It was further hypothesised that K would differ between the groups, however given the lacking ability to recover K we fail to support $H3$. Additionally, we note that the 95% CI of the M3 posterior for the difference (ΔK) contains 0 and using M2 to model the groups separately shows great overlap between the μK posteriors.

(EO) Since no a priori hypotheses were established for the remaining parameters, we refrain from drawing specific conclusions based on their posteriors. Nevertheless, we present the posteriors for these parameters to provide a complete overview of our findings. No evidence of difference was found in the perseverance weight parameter ω_P between the groups, as evident both by the 95% CI of $\Delta \omega_P$ containing 0 and overlapping M2 posteriors of the individually fitted groups ($\mu \omega_P$). However, the 95% CI of the $\mu \omega_P$ posteriors estimated by M2 are positive (and exclude 0) for both AD patients and HC, indicating a preference to stay with recently chosen decks.

No strong evidence of a difference in the win frequency sensitivity parameter ω_F between the groups was found, as evident by the 95% CI of $\Delta \omega_F$ containing 0 and overlapping posteriors of the individually fitted groups ($\mu \omega_F$). We see that the 95% CI of the $\mu \omega_P$ posterior for AD patients is negative, suggesting that AD patients prefer decks with low win frequency (See Figure 7).

While cognitive modelling revealed distinctions in decision-making processes between HC and AD patients, the upcoming sections address and discuss certain limitations in our study.

4.1 Model Selection Considerations

☒ The ORL model was proposed based on its ability to detect distinct patterns of decision-making in substance-using populations (Haines et al., 2018). Given that the application of cognitive models on IGT data from AD patients is an unexplored research domain, it remains uncertain if the underlying psychological processes governing decision making in other clinical populations apply to AD patients. This paper takes the first step to explore this area of research by applying the ORL model to IGT data from AD patients and HC. However, as mentioned in section 1.4, various cognitive models can be used to examine latent psychological variables driving decision-making in the IGT task. We based our analysis on the ORL model without testing if any competing models fit our particular dataset equally well or better. A more exhaustive method could involve fitting the data to multiple competing models such as the PVL-Delta or VPP models and subsequently do model comparison to ascertain the best fitting model. This could allow for explicit testing of potential competing hypotheses pertaining the psychological processes driving IGT performance in AD patients. However, conducting such model comparisons falls beyond the scope of the current project. As mentioned in section 1.4 the authors of the ORL model compared it to the PVL-Delta and VPP models and found superior performance of the ORL model in terms of parameter recovery and predictive accuracy. Although we did not do model comparison, we find that the ORL model (as implemented in M2 and M3) showed good parameter recovery, robust descriptive adequacy and effective Markov chain convergence for our dataset. By applying the ORL model to our dataset, we were able to detect distinct decision-making patterns between AD and HC in terms of learning rates, aligning with research indicating impaired performance in AD patients based on their memory impairments and related decreased ability to learn new stimulus-reward relationships.

4.2 Modelling Choices

☒ In this study, we pooled the data from two different studies into a single dataset, despite minor discrepancies in the frequency and magnitudes of gains and losses in their respective payoff schemes. Our parameter recovery was based on simulated data reflecting the payoff scheme from the study by Jacus et al. (2018). This approach did not account for slight variations in payoff schemes for individual subjects in the study by Gaubert et al. (2022), which were generated by a random seed. While combining these datasets likely introduces some level of noise, it also doubles the sample size as compared to having used only one dataset. We assume that this trade-off between the risk of data inhomogeneity and the benefit of a larger

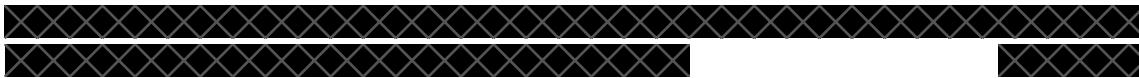
sample size will not negatively influence the certainty of the posterior estimates. To validate this assumption, we further applied the ORL model to each dataset independently using M3 and then compared the standard deviations of the posterior distributions for each parameter in both the individual and combined datasets. This comparison revealed that the uncertainty in the posterior estimates of the pooled data set was either equal to or lower than that in the individual datasets for all parameters (See Appendix B.1: Table 4 and 5).

■ To our knowledge, no published research has modelled Δs directly when comparing groups using the ORL model. Rather, they compare parameter posteriors based on a hierarchical model fit to each group separately using model specifications similar to M2 (e.g., Moreno-Padilla et al., 2023, Haines et al., 2018, Zhang et al., 2022). As we are not only interested in the free parameters of the ORL model but also how they differ across groups, we further include an explicit modelling of the group differences in M3. There are several benefits to this approach, most importantly that we are estimating the variable of interest directly. Using this approach, a prior is set for the difference. This allows for providing the model with prior beliefs about the difference and if relevant calculating the Bayes Factor.

Despite the advantages of modelling the difference directly, the parameterisation of the bounded parameters (A_{rew} , A_{pun} and K) does not allow for direct numeric interpretation of the differences between the groups. This is a result of the inverse probit-transformation of these parameters at individual-level. While this parameterisation allows us to infer both whether groups differ and if so, which group shows a higher mean, determining the credible interval in bounded space is not trivial. Inverse probit-transforming the estimated Δs confines them to a range of 0 to 1. However, ΔA_{rew} and ΔA_{pun} in reality range from -1 to 1. As the mapping from the space in which the deltas are estimated to the inverse probit space is not linear, it is not possible to merely recenter by subtracting -0.5 from the transformed value and multiplying with 2 to allow for the values to range from -1 to 1.

This issue does not pertain to parameters estimated by the hierarchical model without comparison (M2) fitted to the data of each group separately. Here, the variable of interest is the mean, and inverse probit-transforming the estimated μ to get them to the constrained space is not a problem as the boundaries of the inverse probit-function matches the boundaries of the estimated parameter.

Future work should specify a hierarchical comparison model in a way that allows for interpretation of the numeric values of the parameter differences for the bounded parameters. A potential approach, which falls beyond the current project to test, could include substituting the inverse probit-function for another link function or modelling the group level means from an absolute difference such that inverse probit-



transforming the estimated Δ s confines them to the right range.

Another aspect of the model that warrants improvement is the parameterisation of the decay parameter, K , which yielded poor parameter recovery. Haines et al. (2018) conducted further parameterisation of K that was not included in the current study. More specifically, they determined K by $3^{K'} - 1$ as this parameterisation yielded the best performance in estimating K . In the current study, it holds that K was difficult to recover, both at the individual-level and group-level. To interpret posterior estimates of the memory decay parameter, additional optimisation of modelling the parameter should be done, i.e., by testing the parameterisation described by Haines et al. (2018).

Future work should extend from build upon the current research project by comparing competing computational models to ensure that the best fitting model is used and address these aforementioned methodological limitations.

5 Conclusion

With the aim of investigating differences in the latent cognitive decision-making processes between patients with Alzheimer's disease and age-matched healthy controls, the differences between the two groups in the five free parameters of the outcome representation learning model were estimated using hierarchical Bayesian analysis. Strong evidence of a difference in both reward and punishment learning rates was found, with lower learning rates in the Alzheimer's group. Despite methodological limitations, the present study demonstrates that cognitive modelling can 1) provide insights into the latent psychological processes driving IGT behaviour in AD patients and 2) serve as a useful tool for advancing the understanding of decision-making deficits in AD patients.

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References

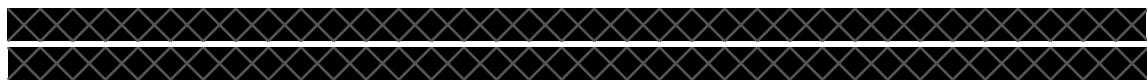
- Ahn, W.-Y., Busemeyer, J. R., Wagenmakers, E.-J., & Stout, J. C. (2008). Comparison of decision learning models using the generalization criterion method.



- Cognitive Science*, 32(8), 1376–1402. <https://doi.org/https://doi.org/10.1080/03640210802352992>
- Alameda-Bailén, J. R., Salguero-Alcañiz, M. P., Merchán-Clavellino, A., & Paíno-Quesada, S. (2017). Cognitive mechanisms in decision-making in patients with mild alzheimer disease. *Current Alzheimer Research*, 14. <https://doi.org/10.2174/1567205014666170417113834>
- Bayard, S., Jacus, J.-P., Raffard, S., & Gely-Nargeot, M.-C. (2014). Apathy and emotion-based decision-making in amnesic mild cognitive impairment and alzheimer's disease. *Behavioural Neurology*, 2014, 1–7. <https://doi.org/10.1155/2014/231469>
- Bayard, S., Jacus, J.-P., Raffard, S., & Gély-Nargeot, M.-C. (2015). Conscious knowledge and decision making under ambiguity in mild cognitive impairment and alzheimer disease. *Alzheimer Disease and Associated Disorders*, 29(4), 357–359. <https://doi.org/10.1097/wad.0000000000000061>
- Bear, M. F., Connors, B. W., & Paradiso, M. A. (2016). Neuroscience: Exploring the brain (Enhanced Fourth Edition). *Jones & Bartlett Learning*, 838–839.
- Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. (1994). Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, 50(1), 7–15. [https://doi.org/10.1016/0010-0277\(94\)90018-3](https://doi.org/10.1016/0010-0277(94)90018-3)
- Bechara, A., & Damasio, H. (2002). Decision-making and addiction (part i): Impaired activation of somatic states in substance dependent individuals when pondering decisions with negative future consequences. *Neuropsychologia*, 40(10), 1675–1689. [https://doi.org/10.1016/s0028-3932\(02\)00015-5](https://doi.org/10.1016/s0028-3932(02)00015-5)
- Bertoux, M., Funkiewiez, A., O'Callaghan, C., Dubois, B., & Hornberger, M. (2012). Sensitivity and specificity of ventromedial prefrontal cortex tests in behavioral variant frontotemporal dementia. *Alzheimer's and Dementia*, 9(5S). <https://doi.org/10.1016/j.jalz.2012.09.010>
- Braak, H., & Braak, E. (1991). Neuropathological stageing of alzheimer-related changes. *Acta Neuropathologica*, 82(4), 239–259. <https://doi.org/10.1007/bf00308809>
- Bucks, R. S., & Radford, S. A. (2004). Emotion processing in alzheimer's disease. *Aging and Mental Health*, 8(3), 222–232. <https://doi.org/10.1080/13607860410001669750>
- Chaudhary, S., Zhornitsky, S., Chao, H. H., van Dyck, C. H., & Li, C.-S. R. (2022). Emotion processing dysfunction in alzheimer's disease: An overview of behavioral findings, systems neural correlates, and underlying neural biology. *American Journal of Alzheimer's Disease and Other Dementias®*, 37, 153331752210828. <https://doi.org/10.1177/15333175221082834>



- Chiu, Y.-C., & Lin, C.-H. (2007). Is deck c an advantageous deck in the iowa gambling task? *Behavioral and Brain Functions*, 3(1), 37. <https://doi.org/10.1186/1744-9081-3-37>
- Chu, C. (1997). The autonomic-related cortex: Pathology in alzheimer's disease. *Cerebral Cortex*, 7(1), 86–95. <https://doi.org/10.1093/cercor/7.1.86>
- Collins, A. G. E., & Shenhav, A. (2021). Advances in modeling learning and decision-making in neuroscience. *Neuropsychopharmacology*, 47(1), 104–118. <https://doi.org/10.1038/s41386-021-01126-y>
- Combrisson, E., & Jerbi, K. (2015). Exceeding chance level by chance: The caveat of theoretical chance levels in brain signal classification and statistical assessment of decoding accuracy. *Journal of Neuroscience Methods*, 250, 126–136. <https://doi.org/10.1016/j.jneumeth.2015.01.010>
- Damasio, A. R. (1996). The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 351(1346), 1413–1420. <https://doi.org/10.1098/rstb.1996.0125>
- Daumas, L., Zory, R., Junquera-Badilla, I., Ferrandez, M., Ettore, E., Robert, P., Sacco, G., Manera, V., & Ramanoël, S. (2023). How does apathy impact exploration-exploitation decision-making in older patients with neurocognitive disorders? *npj Aging*, 9(1). <https://doi.org/10.1038/s41514-023-00121-5>
- Davis, R., Ziolkowski, M. K., & Veltkamp, A. (2017). Everyday Decision Making in Individuals with Early-Stage Alzheimer's Disease: An Integrative Review of the Literature. *Research in Gerontological Nursing*, 10(5), 240–247. <https://doi.org/10.3928/19404921-20170831-05>
- Delazer, M., Sinz, H., Zamarian, L., & Benke, T. (2007). Decision-making with explicit and stable rules in mild alzheimer's disease. *Neuropsychologia*, 45(8), 1632–1641. <https://doi.org/10.1016/j.neuropsychologia.2007.01.006>
- Dolphin, H., Dyer, A. H., McHale, C., O'Dowd, S., & Kennelly, S. P. (2023). An update on apathy in alzheimer's disease. *Geriiatrics*, 8(4), 75. <https://doi.org/10.3390/geriatrics8040075>
- El Haj, M., Boutoleau-Bretonnière, C., & Allain, P. (2020). Memory of decisions: Relationship between decline of autobiographical memory and temporal discounting in alzheimer's disease. *Journal of Clinical and Experimental Neuropsychology*, 42(4), 415–424. <https://doi.org/10.1080/13803395.2020.1744527>
- Förstl, H., & Kurz, A. (1999). Clinical features of alzheimer's disease. *European Archives of Psychiatry and Clinical Neuroscience*, 249(6), 288–290. <https://doi.org/10.1007/s004060050101>



- Fridberg, D. J., Queller, S., Ahn, W.-Y., Kim, W., Bishara, A. J., Busemeyer, J. R., Porrino, L., & Stout, J. C. (2010). Cognitive mechanisms underlying risky decision-making in chronic cannabis users. *Journal of Mathematical Psychology*, 54(1), 28–38. <https://doi.org/10.1016/j.jmp.2009.10.002>
- Gaubert, F., Borg, C., & Chainay, H. (2022). Decision-Making in Alzheimer's Disease: The Role of Working Memory and Executive Functions in the Iowa Gambling Task and in Tasks Inspired by Everyday Situations [Publisher: IOS Press]. *Journal of Alzheimer's Disease*, 90(4), 1793–1815. <https://doi.org/10.3233/JAD-220581>
- Gaubert, F., & Chainay, H. (2021). Decision-Making Competence in Patients with Alzheimer's Disease: A Review of the Literature. *Neuropsychology Review*, 31(2), 267–287. <https://doi.org/10.1007/s11065-020-09472-2>
- Gelman, A., Carlin, J. B., Stern, H. S., Dunson, D. B., Vehtari, A., & Rubin, D. B. (2013, November). *Bayesian data analysis*. Chapman; Hall/CRC. <https://doi.org/10.1201/b16018>
- Gelman, A., Hill, J., & Vehtari, A. (2020, July). *Regression and other stories*. Cambridge University Press.
- Gelman, A., & Rubin, D. B. (1992). Inference from iterative simulation using multiple sequences. *Statistical Science*, 7(4). <https://doi.org/10.1214/ss/1177011136>
- Gleichgerrcht, E., Ibáñez, A., Roca, M., Torralva, T., & Manes, F. (2010). Decision-making cognition in neurodegenerative diseases. *Nature Reviews Neurology*, 6(11), 611–623. <https://doi.org/10.1038/nrneurol.2010.148>
- Haines, N., Vassileva, J., & Ahn, W.-Y. (2018). The Outcome-Representation Learning Model: A Novel Reinforcement Learning Model of the Iowa Gambling Task. *Cognitive Science*, 42(8), 2534–2561. <https://doi.org/10.1111/cogs.12688>
- Hespanhol, L., Vallio, C. S., Costa, L. M., & Saragiotto, B. T. (2019). Understanding and interpreting confidence and credible intervals around effect estimates. *Brazilian Journal of Physical Therapy*, 23(4), 290–301. <https://doi.org/10.1016/j.bjpt.2018.12.006>
- Hot, P., Ramdeen, K. T., Borg, C., Bollon, T., & Couturier, P. (2013). Impaired decision making in alzheimer's disease: A deficit of cognitive strategy selection? *Clinical Psychological Science*, 2(3), 328–335. <https://doi.org/10.1177/2167702613504094>
- Igarashi, K. M. (2023). Entorhinal cortex dysfunction in alzheimer's disease. *Trends in Neurosciences*, 46(2), 124–136. <https://doi.org/10.1016/j.tins.2022.11.006>



- Jacus, J.-P., Gély-Nargeot, M.-C., & Bayard, S. (2018). Ecological relevance of the Iowa gambling task in patients with Alzheimer's disease and mild cognitive impairment. *Revue Neurologique*, 174(5), 327–336. <https://doi.org/10.1016/j.neurol.2017.08.003>
- Kim, S. Y., Karlawish, J. H., & Caine, E. D. (2002). Current state of research on decision-making competence of cognitively impaired elderly persons. *The American Journal of Geriatric Psychiatry*, 10(2), 151–165. <https://doi.org/10.1097/00019442-200203000-00006>
- Kloeters, S., Bertoux, M., O'Callaghan, C., Hodges, J. R., & Hornberger, M. (2013). Money for nothing - Atrophy correlates of gambling decision making in behavioural variant frontotemporal dementia and Alzheimer's disease. *NeuroImage. Clinical*, 2, 263–272. <https://doi.org/10.1016/j.nicl.2013.01.011>
- Lee, M. D., & Wagenmakers, E.-J. (2014, April). *Bayesian cognitive modeling: A practical course*. Cambridge University Press. <https://doi.org/10.1017/cbo9781139087759>
- Livingston, G., Sommerlad, A., Orgeta, V., Costafreda, S. G., Huntley, J., Ames, D., Ballard, C., Banerjee, S., Burns, A., Cohen-Mansfield, J., Cooper, C., Fox, N., Gitlin, L. N., Howard, R., Kales, H. C., Larson, E. B., Ritchie, K., Rockwood, K., Sampson, E. L., ... Mukadam, N. (2017). Dementia prevention, intervention, and care. *Lancet (London, England)*, 390(10113), 2673–2734. [https://doi.org/10.1016/S0140-6736\(17\)31363-6](https://doi.org/10.1016/S0140-6736(17)31363-6)
- Marin, R. S. (1991). Apathy: A neuropsychiatric syndrome. *The Journal of Neuropsychiatry and Clinical Neurosciences*, 3(3), 243–254. <https://doi.org/10.1176/jnp.3.3.243>
- Maruszak, A., & Thuret, S. (2014). Why looking at the whole hippocampus is not enough—“a critical role for anteroposterior axis, subfield and activation analyses to enhance predictive value of hippocampal changes for Alzheimer’s disease diagnosis. *Frontiers in Cellular Neuroscience*, 8. <https://doi.org/10.3389/fncel.2014.00095>
- Masters, C. L., Bateman, R., Blennow, K., Rowe, C. C., Sperling, R. A., & Cummings, J. L. (2015). Alzheimer's disease. *Nature Reviews Disease Primers*, 1(1), 15056. <https://doi.org/10.1038/nrdp.2015.56>
- McElreath, R. (2020, March). *Statistical rethinking* (2nd ed.). Chapman & Hall/CRC.
- Moreno, A., & Alameda, J. R. (2011). Alzheimer's dementia, cognitive impairment and decision making. *European Journal of Investigation in Health, Psychology and Education*, 1(1), 17–29. <https://doi.org/10.3390/ejihpe1010002>
- Moreno-Padilla, M., Alacreu-Crespo, A., Guillaume, S., & Reyes Del Paso, G. A. (2023). The outcome-representation learning model: Impairments in decision-



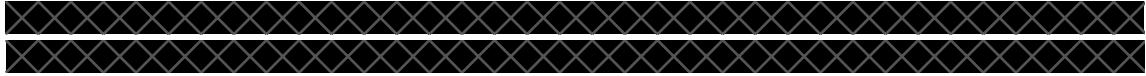
- making in adolescents with excess weight. *Current Psychology*, 42(26), 22404–22414. <https://doi.org/10.1007/s12144-022-03299-1>
- Naggara, O., Oppenheim, C., Rieu, D., Raoux, N., Rodrigo, S., Dalla Barba, G., & Meder, J.-F. (2006). Diffusion tensor imaging in early alzheimer's disease. *Psychiatry Research: Neuroimaging*, 146(3), 243–249. <https://doi.org/10.1016/j.pscychresns.2006.01.005>
- Nobis, L., & Husain, M. (2018). Apathy in alzheimer's disease. *Current Opinion in Behavioral Sciences*, 22, 7–13. <https://doi.org/10.1016/j.cobeha.2017.12.007>
- Pini, L., Pievani, M., Bocchetta, M., Altomare, D., Bosco, P., Cavedo, E., Galluzzi, S., Marizzoni, M., & Frisoni, G. B. (2016). Brain atrophy in alzheimer's disease and aging. *Ageing Research Reviews*, 30, 25–48. <https://doi.org/10.1016/j.arr.2016.01.002>
- Prince, M., Bryce, R., Albanese, E., Wimo, A., Ribeiro, W., & Ferri, C. P. (2013). The global prevalence of dementia: A systematic review and metaanalysis. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 9(1), 63–75.e2. <https://doi.org/10.1016/j.jalz.2012.11.007>
- Ramirez-Gomez, L., Zheng, L., Reed, B., Kramer, J., Mungas, D., Zarow, C., Vinters, H., Ringman, J. M., & Chui, H. (2017). Neuropsychological Profiles Differentiate Alzheimer Disease from Subcortical Ischemic Vascular Dementia in an Autopsy-Defined Cohort. *Dementia and Geriatric Cognitive Disorders*, 44(1-2), 1–11. <https://doi.org/10.1159/000477344>
- Riddell, A., Hartikainen, A., & Carter, M. (2021, March). Pystan (3.0.0).
- Sahathevan, R. (2015). Dementia. In *Diet and nutrition in dementia and cognitive decline* (pp. 187–198). Elsevier. <https://doi.org/10.1016/b978-0-12-407824-6.00018-5>
- Scheltens, P., De Strooper, B., Kivipelto, M., Holstege, H., Chételat, G., Teunissen, C. E., Cummings, J., & van der Flier, W. M. (2021). Alzheimer's disease. *Lancet (London, England)*, 397(10284), 1577–1590. [https://doi.org/10.1016/S0140-6736\(20\)32205-4](https://doi.org/10.1016/S0140-6736(20)32205-4)
- Scheper, I., Brazil, I. A., Claassen, J. A. H. R., Bertens, D., Geurts, S., & Kessels, R. P. C. (2023). Learning capacity in early-stage alzheimer's disease: The role of feedback during learning on memory performance. *Journal of Neuropsychology*. <https://doi.org/10.1111/jnp.12330>
- Selkoe, D. J. (2001). Alzheimer's disease: Genes, proteins, and therapy. *Physiological Reviews*, 81(2), 741–766. <https://doi.org/10.1152/physrev.2001.81.2.741>
- Silva, R. C. R. d., Carvalho, R. L. S. d., & Dourado, M. C. N. (2021). Deficits in emotion processing in alzheimer's disease: A systematic review. *Dementia*



- amp; *Neuropsychologia*, 15(3), 314–330. <https://doi.org/10.1590/1980-57642021dn15-030003>
- Sinz, H., Zamarian, L., Benke, T., Wenning, G. K., & Delazer, M. (2008). Impact of ambiguity and risk on decision making in mild Alzheimer's disease. *Neuropsychologia*, 46(7), 2043–2055. <https://doi.org/10.1016/j.neuropsychologia.2008.02.002>
- Stan Development Team. (2023). *Stan modeling language users guide and reference manual*. Version 2.33.0. <https://mc-stan.org>
- Steingroever, H., Wetzels, R., & Wagenmakers, E.-J. (2013). Validating the PVL-Delta model for the Iowa gambling task. *Frontiers in Psychology*, 4. Retrieved November 24, 2023, from <https://www.frontiersin.org/articles/10.3389/fpsyg.2013.00898>
- Stout, J. (2002). A contribution of cognitive decision models to clinical assessment: Decomposing performance on the Bechara gambling task. *Psychological assessment*, 14, 253–62. <https://doi.org/10.1037/1040-3590.14.3.253>
- Sun, T., Xie, T., Wang, J., Zhang, L., Tian, Y., Wang, K., Yu, X., & Wang, H. (2020). Decision-Making Under Ambiguity or Risk in Individuals With Alzheimer's Disease and Mild Cognitive Impairment. *Frontiers in Psychiatry*, 11, 218. <https://doi.org/10.3389/fpsy.2020.00218>
- Torralva, T., Dorrego, F., Sabe, L., Chemerinski, E., & Starkstein, S. E. (2000). Impairments of social cognition and decision making in alzheimer's disease. *International Psychogeriatrics*, 12(3), 359–368. <https://doi.org/10.1017/s1041610200006463>
- Vehtari, A., Gelman, A., Simpson, D., Carpenter, B., & Bürkner, P.-C. (2021). Rank-normalization, folding, and localization: An improved r^* for assessing convergence of mcmc (with discussion). *Bayesian Analysis*, 16(2). <https://doi.org/10.1214/20-ba1221>
- Viswanathan, A., Rocca, W. A., & Tzourio, C. (2009). Vascular risk factors and dementia. *Neurology*, 72(4), 368–374. <https://doi.org/10.1212/01.wnl.0000341271.90478.8e>
- Warren, A. (2022). Heightened emotion processing as a compensatory mechanism in persons with Alzheimer's disease: Psychological insights from the tri-network model. *Frontiers in Dementia*, 1, 983331. <https://doi.org/10.3389/frdem.2022.983331>
- Weiss, E. M., Kohler, C. G., Vonbank, J., Stadelmann, E., Kemmler, G., Hinterhuber, H., & Marksteiner, J. (2008). Impairment in emotion recognition abilities in patients with mild cognitive impairment, early and moderate alzheimer disease compared with healthy comparison subjects. *The American Journal*



- of Geriatric Psychiatry*, 16(12), 974–980. <https://doi.org/10.1097/jgp.0b013e318186bd53>
- Wilson, R. C., & Collins, A. G. (2019). Ten simple rules for the computational modeling of behavioral data (T. E. Behrens, Ed.). *eLife*, 8, e49547. <https://doi.org/10.7554/eLife.49547>
- World Health Organization. (2017). *Global action plan on the public health response to dementia 2017–2025*. Retrieved December 8, 2023, from <https://iris.who.int/handle/10665/259615>
- Worthy, D., Pang, B., & Byrne, K. (2013). Decomposing the roles of perseveration and expected value representation in models of the Iowa gambling task. *Frontiers in Psychology*, 4. Retrieved January 6, 2024, from <https://www.frontiersin.org/articles/10.3389/fpsyg.2013.00640>
- Zamarian, L., Weiss, E. M., & Delazer, M. (2010). The impact of mild cognitive impairment on decision making in two gambling tasks. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 66B(1), 23–31. <https://doi.org/10.1093/geronb/gbq067>
- Zhang, L., Vashisht, H., Nethra, A., Slattery, B., & Ward, T. (2022). Differences in Learning and Persistency Characterizing Behavior in Chronic Pain for the Iowa Gambling Task: Web-Based Laboratory-in-the-Field Study. *Journal of Medical Internet Research*, 24(4), e26307. <https://doi.org/10.2196/26307>



7 Appendix

A Model Summary Statistics

Table 1: Summary Statistics - Hierarchical Model Without Comparison (M2) (AD)

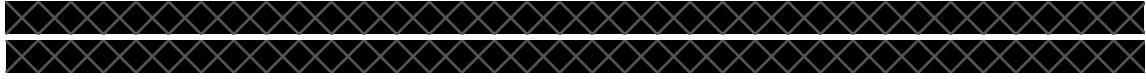
Parameter	Mean	SD	HDI 3%	HDI 97%	MCSE Mean	MCSE SD	ESS	\hat{R}
μA_{rew}	-1.246	0.121	-1.476	-1.024	0.002	0.002	2946	1.00
μA_{pun}	-1.881	0.101	-2.077	-1.703	0.002	0.001	2711	1.00
μK	-0.721	0.17	-1.04	-0.402	0.005	0.003	1312	1.00
$\mu \omega_P$	0.797	0.323	0.158	1.364	0.008	0.006	1639	1.00
$\mu \omega_F$	-2.038	0.796	-3.49	-0.528	0.024	0.017	1054	1.00

Table 2: Summary Statistics - Hierarchical Model Without Comparison (M2) (HC)

Parameter	Mean	SD	HDI 3%	HDI 97%	MCSE Mean	MCSE SD	ESS	\hat{R}
μA_{rew}	-0.161	0.18	-0.478	0.183	0.005	0.004	1096	1.00
μA_{pun}	-1.296	0.079	-1.443	-1.152	0.002	0.001	1743	1.00
μK	-0.824	0.216	-1.214	-0.417	0.007	0.005	1041	1.00
$\mu \omega_P$	0.973	0.246	0.487	1.418	0.006	0.004	1584	1.00
$\mu \omega_F$	-0.41	0.649	-1.629	0.77	0.019	0.013	1157	1.01

Table 3: Summary Statistics - Hierarchical Comparison Model (M3)

Parameter	Mean	SD	HDI 3%	HDI 97%	MCSE Mean	MCSE SD	ESS	\hat{R}
ΔA_{rew}	1.059	0.218	0.665	1.488	0.005	0.004	1746	1.00
ΔA_{pun}	0.586	0.13	0.359	0.848	0.003	0.002	2229	1.00
ΔK	-0.113	0.268	-0.602	0.403	0.009	0.007	846	1.00
$\Delta \omega_P$	0.157	0.4	-0.631	0.864	0.01	0.007	1510	1.01
$\Delta \omega_F$	1.251	0.838	-0.323	2.825	0.023	0.016	1306	1.00



B Posterior Distributions by Data Source

B.1 Dataset 1

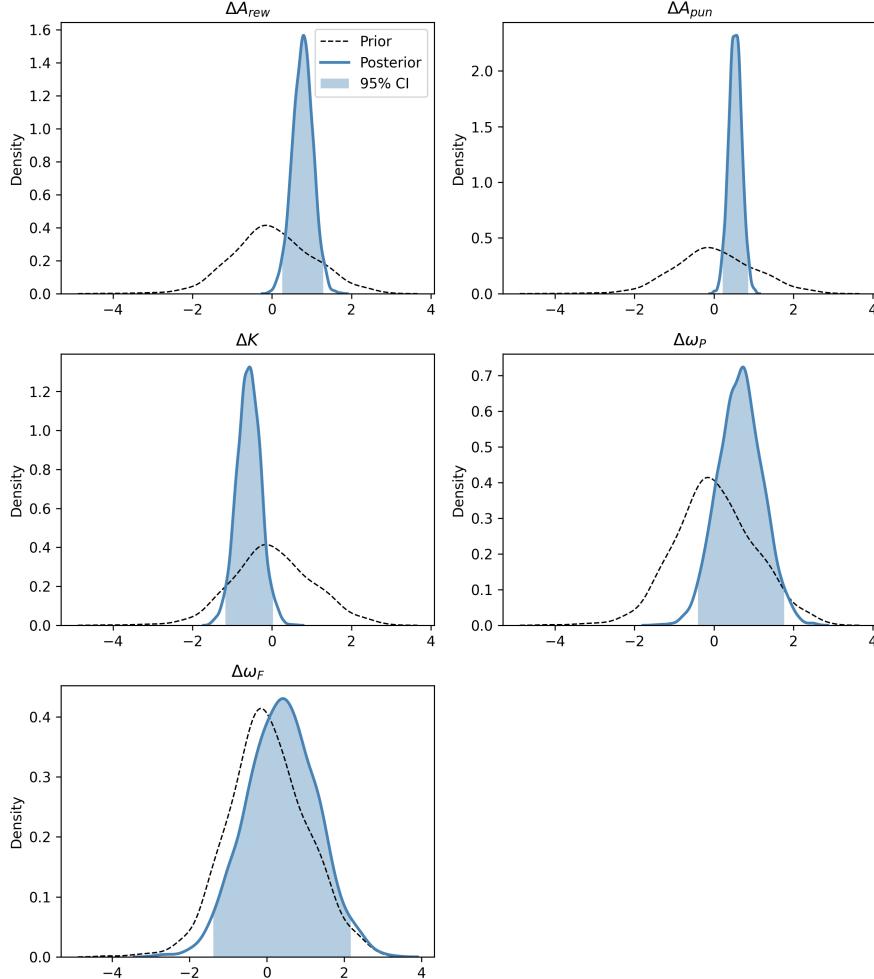
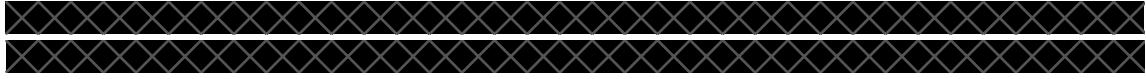


Figure 8: Posterior density distributions from dataset 1 of parameter differences between AD and HC. The blue area under the curve represents the 95% credible interval (M3).

Table 4: Summary Statistics - Hierarchical Comparison Model Dataset 1 (M3)

Parameter	Mean	SD	HDI 3%	HDI 97%	MCSE Mean	MCSE SD	ESS	\hat{R}
ΔA_{rew}	0.776	0.259	0.274	1.259	0.005	0.004	2349	1.0
ΔA_{rew}	0.537	0.162	0.237	0.852	0.003	0.002	2601	1.0
ΔK	-0.582	0.298	-1.143	-0.01	0.006	0.004	2249	1.0
$\Delta \omega_P$	0.661	0.557	-0.391	1.687	0.012	0.008	2309	1.0
$\Delta \omega_F$	0.378	0.91	-1.413	1.99	0.018	0.013	2463	1.0



B.2 Dataset 2

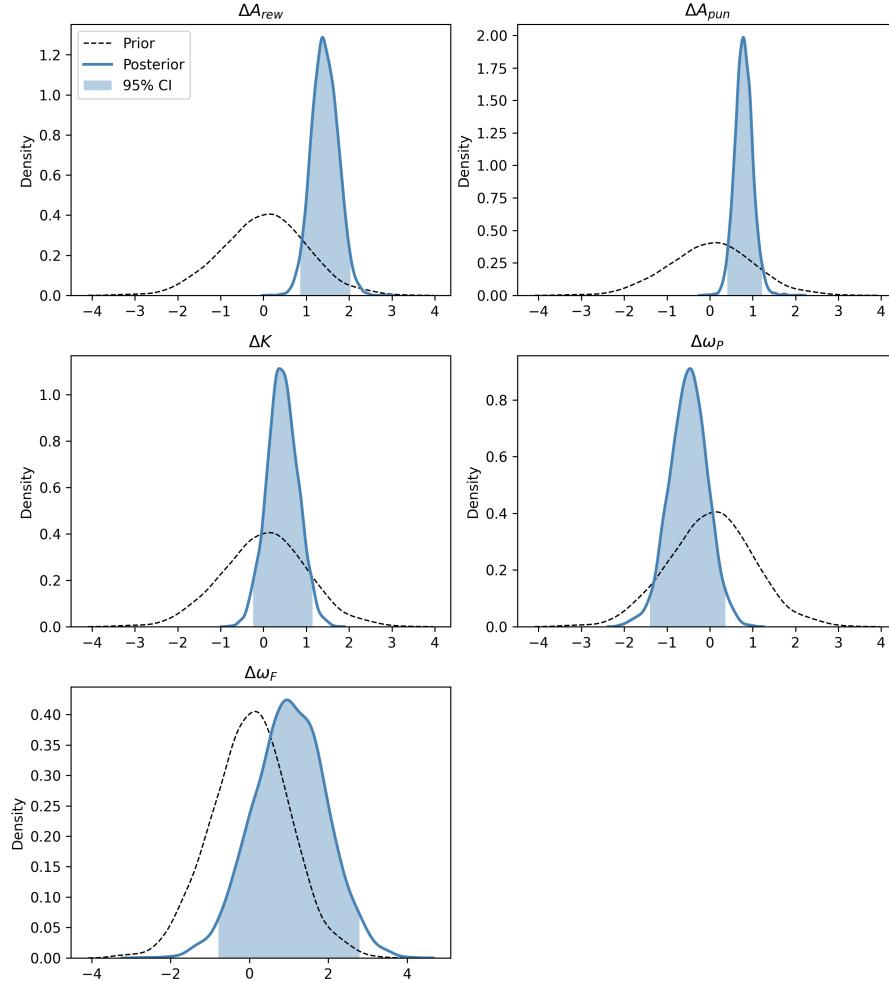


Figure 9: Posterior density distributions from dataset 2 of parameter differences between AD and HC. The blue area under the curve represents the 95% credible interval (M3).

Table 5: Summary Statistics - Hierarchical Comparison Model Dataset 2 (M3)

Parameter	Mean	SD	HDI 3%	HDI 97%	MCSE Mean	MCSE SD	ESS	\hat{R}
$\delta[0]$	1.435	0.304	0.894	2.016	0.007	0.005	2128	1.0
$\delta[1]$	0.794	0.208	0.415	1.191	0.004	0.003	2332	1.0
$\delta[2]$	0.446	0.356	-0.204	1.137	0.011	0.008	1014	1.0
$\delta[3]$	-0.503	0.444	-1.311	0.359	0.009	0.007	2201	1.0
$\delta[4]$	1.017	0.919	-0.614	2.824	0.019	0.013	2390	1.0



C Convergence Diagnostics (M2)

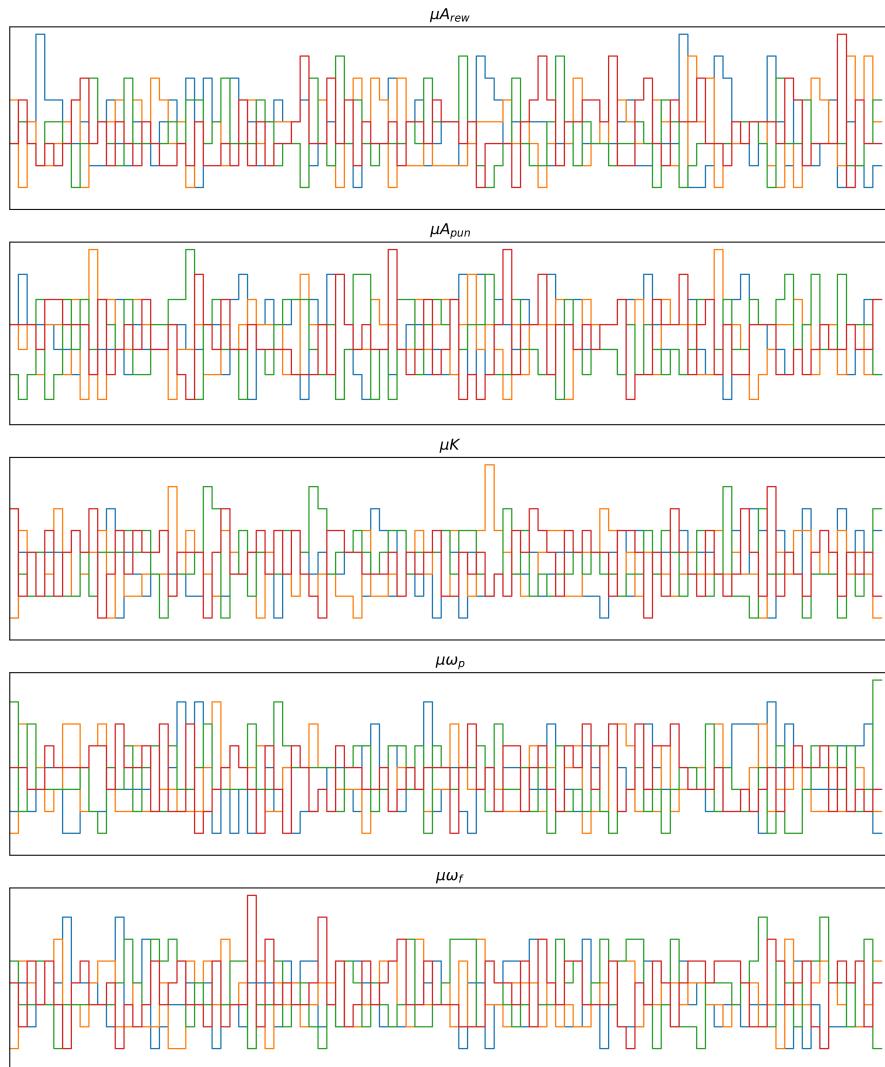


Figure 10: Trace rank plot for AD.

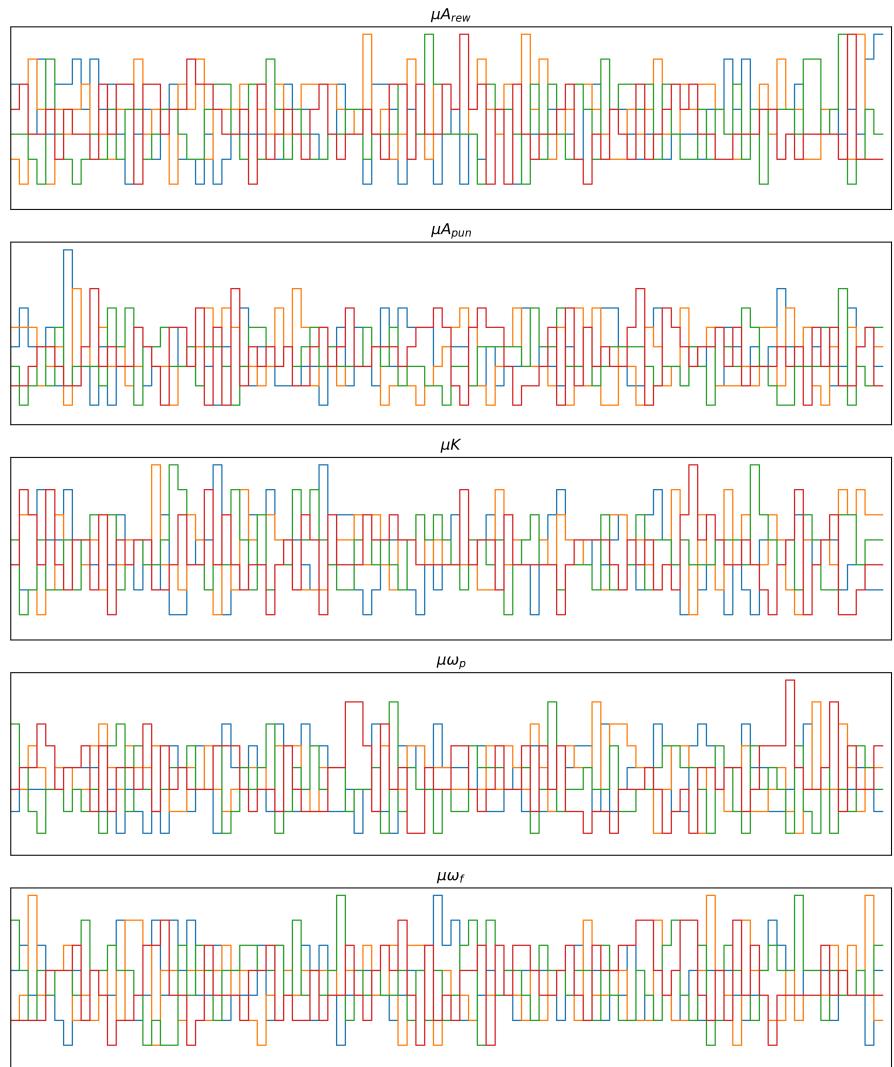


Figure 11: Trace rank plot for HC.



D Descriptive Adequacy (M2)

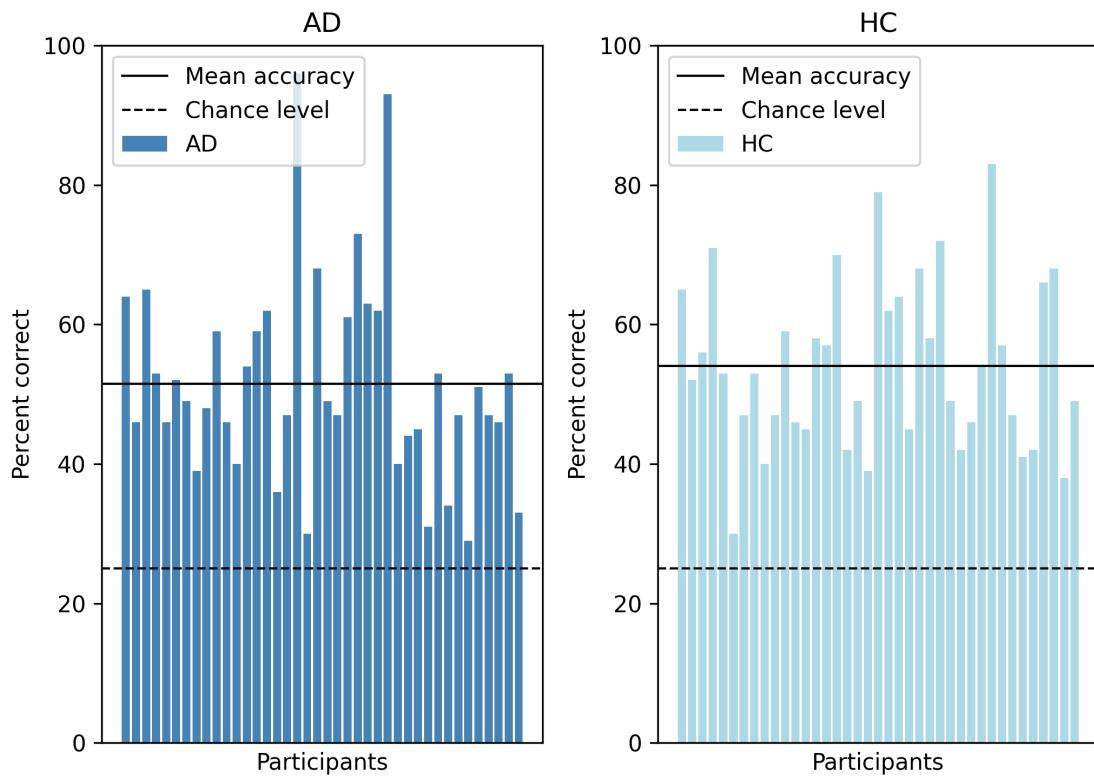


Figure 12: Descriptive adequacy of HD and AD fitted separately using M2