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Research Article

Ambiguous Decision-Making in Adults with Epilepsy

Sally Ann Wakeford[1]*, Neal Hinvest[2], Howard Ring[3], and Mark Brosnan[2]

[1]Institute of Child Health, University College London, UK
[2]Department of Psychology, University of Bath, UK
[3]Department of Psychiatry, University of Cambridge, UK

*Corresponding author
Sally Ann Wakeford, Institute of Child Health, University College London, 71 Hillside Drive, Christchurch Dorset BH23 2RT, United Kingdom; Tel: 44-7961053633; Email: s.wakeford@bath.edu

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Keywords

- Epilepsy
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Abstract

Introduction: There is a high prevalence of autism spectrum disorders (ASD) in epilepsy. ASDs are characterised by a deficit of social interaction, social communication, and restricted, repetitive behaviours. Previous research by Wakeford and colleagues reported higher autistic characteristics in adults with epilepsy who had no diagnosis of an ASD. A subsequent study found that while sameness behaviours were unimpaired, adults with epilepsy reported poor reciprocal social interaction, revealing difficulties in social interactions, a characteristic of autism. The Somatic Marker Hypothesis proposes that neural systems supporting decision-making overlap with components of a neural circuitry which guide social behaviour. Impaired decision-making abilities under ambiguity may indicate compromised somatic marker formation, crucial for social cognition. The present paper aims to investigate ambiguous decision making, and whether the Somatic Marker Hypothesis is a valid explanatory model for these cognitive features of epilepsy.

Method: Our experiment investigated ambiguous decision-making ability measured by the IOWA Gambling Task in adults with epilepsy.

Results: Adults with epilepsy demonstrated impaired decision-making abilities compared to adults without epilepsy, likely to result from compromised somatic marker formation.

Conclusion: The somatic marker hypothesis contributes a neurobiological plausible account of the underlying impairment of decision-making in epilepsy. Given that intact somatic marker formation is important for social cognitive function, this model provides a mechanism for linking somatic function to decision-making and social behaviours in epilepsy, suggesting that disrupted neurobiological factors may be implicated in both.

INTRODUCTION

There is a high prevalence of autism spectrum disorders (ASD) in epilepsy [1,2]. Recent evidence suggests that epileptogenesis during early development may contribute to ASD through disrupted synaptic plasticity, especially in the prefrontal cortex which is implicated in decision making [2,3]. Adults with epilepsy have been found by the Authors of this paper to score higher for autistic trait characteristics than adults without epilepsy [4]. A subsequent study of ours concluded that adults with epilepsy have more social but not non-social characteristics of autism such as sameness behaviours, evidenced by higher scores on a test of reciprocal social interaction [5]. An impairment of social cognition is a core defining characteristic of autism spectrum disorders. Psychosocial maladjustment is defined as an extreme difficulty in dealing appropriately with other people, and is a serious problem in many individuals with chronic epilepsy [6]. Historically, epilepsy is associated with social stigma which has been implicated in causing social isolation, however, it is still unclear to what extent these difficulties are primarily due to dysfunctional social cognition or due to living with a stigmatising condition.

Evidence has shown that epilepsy during early brain maturation is associated with disruption to social cognitive functioning, and early onset is a consistent factor for impaired advanced social cognition [7,8]. Seizure activity has been shown to impair social cognition, and severity of seizures, frequency of seizures and chronic epilepsy have been identified through research to increase the risk of social dysfunction [6,9-11]. Consistent with this, some improvements with increased social relationships and interactions after epilepsy surgery have been reported in both children and adults [12]. Such evidence argues for neurobiological factors related to the genesis of epilepsy as a primary cause of social interaction difficulties. Several researchers argue that investigations into social cognition are

largely neglected in those with epilepsy. At present, there is genuine uncertainty of whether psychosocial maladjustment can be attributed to the social cognitive consequences of having epilepsy in adults with epilepsy.

Given that both higher autistic traits and specific social difficulties have been identified in epilepsy, this raises the question of whether a social cognitive deficit underlies these social difficulties, and if so, what explanatory theory can be identified and tested to address the findings of these initial studies in adults with epilepsy. The Somatic Marker Hypothesis (SMH) offers a neural explanation for how emotions contribute to decision making under ambiguity which precedes explicit insight through somatic states, which guide social behaviour [13].

The word somatic refers to the emotion-based biasing signals which express themselves with positive or negative affect. These signals elicit a somatic state, which influence the processes of response to stimuli. It has been hypothesized that somatic states influence the inhibition of a response which has been previously learned or can introduce a bias in an otherwise deliberate evaluation of dangerous or advantageous outcomes. The SMH is a model of the somatic processes underlying decision-making. This model proposes that somatic marker formation occurs when somatic signals are integrated in the ventral medial prefrontal cortex (vmPFC), and it is well established that prefrontal cortex (PFC) damage results in a severe impairment of decision making and disrupts social behaviour profoundly [14]. This purports that emotions play a central role in social cognition and decision making, and proposes that the formation of somatic signals must be intact to guide social interaction appropriately.

Prefrontal cortex network abnormalities have been identified in absence epilepsy [15], idiopathic generalized epilepsy [16], juvenile Myoclonic Epilepsy (JME) [17], frontal lobe epilepsy (FLE) and other focal epilepsies [18]. While prefrontal cortex network abnormalities have been identified in numerous epilepsies, frequent seizures in this region have been proposed to lead to impaired social behaviour only in FLE, while neural activation in this regions in JME have been proposed to explain difficulties in social adjustment [17,18].

There is some evidence of disadvantageous decision making under ambiguity in patients with JME and mesial temporal lobe epilepsy (mTLE) but not neocortical TLE. In JME, poor performance has been associated with an increased activation in the dorso lateral prefrontal cortex [17,19,20].On the Iowa Gambling Task (IGT) which measures decision making under ambiguity, Wandschneider and colleagues found that a greater proportion of JME patients with seizures than seizure-free JME patients demonstrated poorer decision making abilities. Other research has shown that patients with mTLE but not neocortical TLE showed difficulties in making decisions under ambiguity possibly due to interictal dysfunction within the dorsolateral PFC and medial PFC structures [21,22]. Most patients with mTLE and unilateral hippocampal damage demonstrated disadvantageous decision making in an experiment by Labudda and colleagues, but while poor decision making did not occur for every mTLE patient, those patients who selected disadvantageous alternatives were found to have had earlier seizure onset Chronic epilepsy in mTLE patients was related to worse scores for decision making [23]. This is consistent with the possibility that epileptic seizures may have negative consequences for decision making abilities, as chronic epilepsy is associated with both amygdala damage and progressive hippocampal damage in intractable TLE, regardless of pathology [24,25].

Generally though, there is a lack of research of decision making abilities in epilepsy, especially in heterogeneous groups. From the few studies that have been conducted, the relative contributions of structures that underpin somatic marker formation have not yet been well established. The neural explanation for how emotions regulate decision making under ambiguity through somatic states offered by the SMH which are purported to guide social behaviour, provides a framework for this investigation. Therefore, given the previous finding of self-reported social difficulties reported by a heterogeneous epilepsy adult group, the aim of this investigation is to explore whether there is a deficit in decision making ability under ambiguity related to compromised somatic marker formation. We hypothesized that adults with epilepsy would perform more poorly on a task of ambiguous decision making than adults without epilepsy.

METHODS

Study design

Two groups of adults were recruited for these short assessments: a heterogeneous group with epilepsy, and a control group without epilepsy. This experiment employed the IOWA Gambling Task.

Participants:

Method of recruitment: This study mainly recruited from our previous studies: existing participants (n=10) [4]; additional participants were recruited from adverts on epilepsy charity websites and through University psychology departments (n=2), 6 of 16 control participants were psychology students. The research was conducted at the University of Bath requiring the participant to travel to the research laboratory. Therefore, due to travel and length of experiment time, selection may bias towards more high-functioning adults. However, the sampling method was considered the most appropriate available. The control sample consisted of 87.5 % of students; the epilepsy group consisted of 41.7% students. No participant had vagus nerve stimulation designed to prevent seizures, as research has demonstrated that the vagus nerve is a conduit for afferent somatic signals that can influence decision making [26].

Exclusion criteria: Participants were excluded if they had a diagnosis of an ASD. Only adults (≥ 18 years) participated. No participant had an autism-epilepsy syndrome, e.g., Dravet's Syndrome. Participants self-reported their epilepsy type.

Participant samples: Following the exclusion of one participant (see 4.1.1.2), the participant sample comprised n=27: Control Group n=16 (Female n=12, Male n=4), Epilepsy n=11 (Female n=9; Male n=2). For anti-epileptic drug (AED) use, (Table 3).

Antiepileptic drugs: Participants were asked to self-rate the effectiveness of AEDs for controlling their seizures using a 5-point Likert scale: 1=totally uncontrolled, 2=poorly controlled,

3=partially controlled, 4 = reasonably well controlled, and 5 = well controlled.

Data exclusion: Raw data was checked for evidence of outliers and atypical patterns, such as single choice repetitions and responses faster than one second. One epilepsy participant responded exceptionally fast in the last 2 blocks of the task: less than half a second (0:00-0:35 seconds), revealing that the response key was pressed almost immediately, showing that the participant did not have time to think about the contingencies of each choice. Consequently, this led to the exclusion of this participant (Table 4) for mean RT by group.

Risk Assessment: The studies were conducted in the University of Bath Psychology Laboratory, risk assessed for participants with epilepsy employing the Epilepsy Safety Check Assessment with additional information incorporated from the Epilepsy Risk Assessment [28,29]. Missing from the epilepsy risk assessments were psychological factors known to increase risk of seizures. These include environmental stress triggers such as environmental sensory stimuli and stressors, therefore the researcher conducted an additional assessment for these psychological stressors which may increase risk of seizures. A log book was maintained to record unplanned participant illness and factors affecting comfort during the research

Assessment of ambiguous decision-making

This task employed the IOWA Gambling task by Psychology Experiment Building Language (PEBL) version 0.11 [30]. This program was displayed on standard 36cm x 28cm monitors.

IOWA Gambling Task: The IGT is a neuropsychological behavioural measure of decision making. It was originally developed to assess individuals with vmPFC damage [31]. It comprises of 4 decks of cards presented face down, each card representing a hypothetical monetary gain across 100 trials. Two decks are disadvantageous with high rewards and high losses over time; two decks are advantageous with low rewards and low losses overtime. The decksare constructed so that selection from advantageous decks will lead to an overall gain and vice-versa if selecting from the disadvantageous decks. The risk associated with each selection is unknown and ambiguous. Over the duration of the task, healthy controls without vmPFC damage learn the contingencies of each deck selection and switch selection to advantageous decks (C and D) as the task progresses [31,32]. The computerised version of the original paper version is commonly used, without difference between the versions [33]. Validity for the IGT is good, and measures of IGT performance in healthy controls have shown anticipatory electro dermal responses of anticipated risk prior to a risky card selection, which support the SMH [34]. The IGT is designed to simulate playing a game in which no 'real money' is used, but in which participants are instructed to win as much fictitious money as possible.

Design: The study was conducted as a between-groups design. The independent variable was group: epilepsy and control group. The dependent variable was the participant's total winnings on the IGT, the number of advantageous cards selected, and response time (RT) for each trial in milliseconds.

Procedure

Participants were presented with a computerised version of the IGT showing four decks of cards face down, and $2000 capital gain of 'play' money. Participants were provided with standard instructions, and were asked to select cards with the aim of making as much money as possible, or to minimise losses. Participants were informed that decks can be either advantageous or disadvantageous. The researcher pointed to the display screen features while instructions were read out. Participants were explicitly instructed to play the game as though they were actually accumulating 'real' money, to ensure good validity. No participant was informed that their response time was measured as part of the experimental design. The task comprised of 100 trials lasting 5-10 minutes.

Statistical methods: Following testing for normally distributed data, with correction where appropriate, comparisons were undertaken using SPSS version 18 for Windows, and significance level was set a conventional level of $p < .05$.

Ethical considerations: The research was approved by the University of Bath, Department of Psychology Ethics Committee.

RESULTS

Age but not education was found to be significantly different between group, (Table 1). Kolmogorov-Smirnov test was non-significant revealing normal distribution ($p > .05$), and Levene's test confirmed homogeneity of variance ($p > .05$). There was a significant difference between group ($t(26) = -3.067$, $p = 0.005$), (Table 2). The following analyses were conducted with an independent t-test, and an ANCOVA with age as a covariate to control for group age differences.

Score

An independent t-test explored group differences for score. Kolmogorov-Smirnov test revealed normal distribution ($p>.05$), and Levene's test confirmed homogeneity of variance ($p>.05$). There was no significant difference between group ($t_{(25)} = -0.66$, $p = 0.52$, n.s; $F_{(1,24)}=0.94$, MSE = 30.8, $p = 0.34$, n.s), (Table 4).

Response Time

An independent t-test explored group differences for average time (time 2 to time 100). Response time for the first selection (time 1)was used to explain the display screen features to participants, and removing thistime did not affect measures since there is no consideration to be made based on previous selection. Kolmogorov-Smirnov test revealed normal distribution ($p>.05$), and Levene's test confirmed homogeneity of variance ($p>.05$). Time was approaching significance, however when age was controlled for, there was no significant difference ($t_{(25)} = -1.96$, df=25, p=0.062, n.s.; $F_{(1,24)}=0.80$, MSE=25.76, p=0.38, n.s), (Table 5).

Deck Selection

Score: A mixed design ANCOVA (Group, 2 x Block, 5) with block as a repeated measure and age as a covariate explored score differences between groups. Maunchly's test for sphericity reveals sphericity is assumed (p=0.146). ANCOVA showed a significant main effect for Block ($F_{(4,96)}=5.22$, MSE=483810,

Table 1: Demographic characteristics of epilepsy and control groups.

Characteristic	Controls (n=16)	Epilepsy (n=12)	Sig.
	Mean (SD) Range	Mean (SD) Range	
Gender [Male/Female]	M=4, F=12	M=3, F=9	
Mean Age	28.0 (5.92) 20-45	36.0 (8.47) 23-50	p=0.015, sig.
WAIS-R FSIQ-2	117.19 (8.83) 102-130	120.75 (11.02) 93-136	p=0.35, n.s.
Education [years]	7.10 (2.84) 2-12	7.10 (1.86) 4-9	p=0.87, n.s*
Age at onset of epilepsy [years]		18.6 9.90	(0.3-33)
Duration of epilepsy [years]		18.0 12.10 (4.0-43)	

* Mann-Whitney U test

Table 2: Classification by primary and secondary type, and known pathology.

Epilepsy Type, primary and secondary (reported pathology where known)
1. Temporal Lobe Epilepsy, Both Hemispheres (secondary Tonic Clonic seizures) Dispersed brain damage, lack of oxygen
2. Right frontal lobe epilepsy, left temporal discharge (secondary generalized seizures**) Right frontal cortical dysplasia
3. Complex Partial Epilepsy Scar tissue from brain stem tumour removal
4. Juvenile Myoclonic Epilepsy (secondary Tonic Clonic seizures)
5. Familial Epilepsy Genetic condition
6. Temporal Lobe Epilepsy, Right (secondary Tonic Clonic seizures)
7. Frontal Lobe Epilepsy (secondary Right Mesial Temporal, treated with VNS)
8. Idiopathic Generalized Epilepsy
9. Tonic Clonic Epilepsy(secondary Absence seizures)
10. Temporal Lobe Epilepsy, Right *Scarring on right temporal lobe*
11. Tonic Clonic Epilepsy *Hereditary*
12. Frontal Lobe Epilepsy, Left *Surgery to remove arteriovenous malformation of the left temporal lobe;* *Todd's paresis: full recovery*
All classification of epilepsy type was self-reported by participants ** ILAE (2009) revision of terminology has replaced the term secondarily generalized seizure to the specific seizure components [27]

Table 3: Classification of epilepsy type.

Classification of Epilepsy	N	%
Primary Type:		
Temporal Lobe Epilepsy	4	33.3
Frontal Lobe Epilepsy	2	16.7
Tonic Clonic Epilepsy	2	16.7
Other Focal Epilepsy	1	8.3
Juvenile Myoclonic Epilepsy	1	8.3
Idiopathic Generalised Epilepsy	1	8.3
Familial Epilepsy	1	8.3
Total	**12**	**100**

Table 4: Other Clinical Characteristics.

Clinical Characteristics	N	%
Hemisphere:		
Left	1	8.3
Right	4	33.3
Both	1	8.3
Unknown	6	50

Surgery:		
Brainstem Tumour removal	1	8.3
LTLE Venus malformation removal	1	8.3
No Previous Surgery	10	83.3
Seizure Frequency:		
Daily	4	33.3
Weekly	2	16.7
Monthly	2	16.7
Yearly	3	25
Unknown	1	8.3
Onset of Epilepsy:		
Childhood-onset <18yrs	3	25
Adulthood-onset >18yrs	8	66.7
Unknown	1	8.3
Temporal Lobe Epilepsy:		
TLE	4	33.3
No-TLE	8	66.7
Anti-epilepsy drug seizure control*		
Poorly controlled	3	25
Partially controlled	2	16.7
Reasonably well controlled	2	16.7
Well controlled	1	8.3
Unknown	4	33.3

*Participants were not required to rate whether they were receiving Monotherapy or Polytherapy, as this was not the aim of this research.

Table 5: IOWA Task, Total Mean Score and Mean RT by group.

	Total Score $			**Response Time per choice** (minutes: seconds)		
	Mean	**(SD)**	**Range**	**Mean**	**(SD)**	**Range**
Controls n=16	1746.9	-499	950-2625	0.4472222	(0:491)	0:727 – 2:810
Epilepsy n=11	1881.8	-564	850-2525	2:042	(0:728)	1:101 – 3:371

p=0.001), a significant main effect for Group ($F_{(1,24)}$=7.83, MSE=15304, p=0.010), and a trend for the interaction of block and group ($F_{(4,96)}$=2.21, MSE=204990, p=0.073).

Post-hoc ANCOVA analysis for each block with age as a covariate revealed that deck choice was not significantly affected by Group for Block 1 ($F_{(1,24)}$=2.24, MSE=41828, p=0.133); significant for Block 2 ($F_{(1,24)}$=4.46, MSE=1004570, p=0.045); significant for Block 3 ($F_{(1,24)}$=5.89, MSE=1242397, p=0.023); significant for Block 4 ($F_{(1,24)}$=7.90, MSE=1437960, p=0.010); and non-significant for Block 5 ($F_{(1,24)}$=1.21, MSE=152463, p=0.283), (Figure 1)

Time: A mixed design ANCOVA (Group, 2 x Block,5) with block as a repeated measure and age as a covariate explored time differences between groups. Maunchly's test for sphericity reveals sphericity is violated (*p*=.001), therefore values reported are corrected by a conservative correction factor applied to the degrees of freedom used, Greenhouse- Geisser correction. ANCOVA showed no significant main effects for Block ($F_{(1,24)}$=1.35, MSE = 3.03, p=0.268), there were no significant main effects for Group ($F_{(1,24)}$=0.79, MSE = 1.03, p=0.380), and the interaction of block and group was not significant ($F_{(1,24)}$=1.66, MSE = 3.70, p=0.20).

These results indicate that while there was no difference between group performances for RT, there was a significant effect for block between the groups.

Advantageous and Disadvantageous Deck Selection: Analysis was conducted to explore group differences in advantageous and disadvantageous deck selection, to reveal the extent to which participants learn to select advantageous decks at a higher rate than disadvantageous decks as the task progresses. Decks A and B are disadvantageous with large gains and large punishments resulting in a net $ loss; decks C and D are advantageous with small gains and small punishments resulting in a net $ gain.

The mean advantageous/ disadvantageous (A/D) score was calculated by subtracting the number of disadvantageous deck selections (decks A and B) from the number of advantageous deck selections (decks C and D). A higher score indicated that deck selections were more advantageous. A mixed design ANCOVA (Group, 2 x Block, 5) with block as a repeated measure and age as a covariate explored time differences between groups. Maunchly's test for sphericity reveals sphericity is violated (p=.003), therefore values reported are corrected by a conservative correction factor applied to the degrees of freedom used, Greenhouse - Geisser correction. ANCOVA showed no significant main effects for Block ($F_{(4,96)}$=0.92, MSE = 26.6, p=0.431), there was a significant main effect for Group ($F_{(1,24)}$=4.45, MSE = 42.3, p=0.046), and the

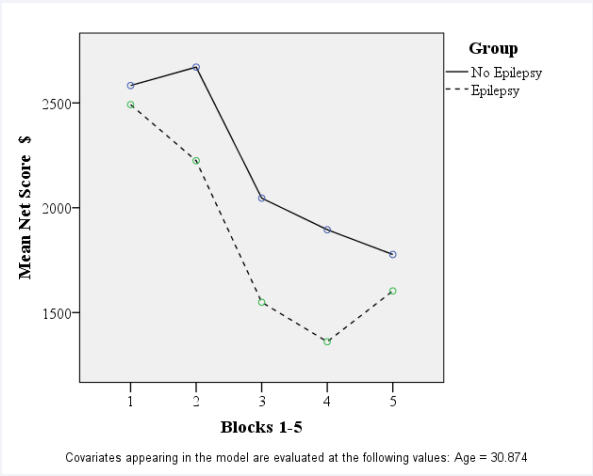

Figure 1 Group Performance on the IOWA gambling task, measured by mean net scores selected for each of 5 blocks, of 20 cards.
*Note: the initial endowment for each participant is $2000

interaction of block and group was not significant ($F_{(4,96)}$ = 0.864, MSE = 24.9, p=0.46).

Post-hoc ANCOVA analysis for each block with age as a covariate revealed that deck choice was not significantly different for Group in Block 1 ($F_{(1,24)}$=0.44, MSE=13.4, p=0.512); significant for Block 2 ($F_{(1,24)}$=6.34, MSE=131, p=0.019); approaching significance for Block 3 ($F_{(1,24)}$=3.54, MSE=23.0, p=0.072); approaching significance for Block 4 ($F_{(1,24)}$=3.29, MSE=110, p=0.082); and not significant for Block 5 ($F_{(1,24)}$=0.113, MSE=4.36, p=0.74), (Figure 2).

Overall, the groups performed significantly different, and differences throughout the task can be seen in (Figure 3) and (Figure 4), which show the pattern of deck selection for each group.

DISCUSSION

The aim of this experiment was to investigate ambiguous decision making in adults with and without epilepsy, and to examine whether the Somatic Marker Hypothesis would be a valid explanatory model. The results showed that adults with epilepsy demonstrated poorer decision making abilities compared to adults without epilepsy. They selected fewer advantageous cards throughout the task as evidenced by the significant group differences in advantageous minus disadvantageous deck selection, and also demonstrated significant differences in selection between the four decks. However, there were no significant group differences for time taken to complete the task, revealing that the epilepsy group did not demonstrate impulsive decision making, but took equivalent time to deliberate their choice.

The task comprised of 5 blocks, and post-hoc analysis revealed a significant difference for choosing fewer advantageous than disadvantageous cards in block 2. This was followed by a non-significant trend in blocks 3 and 4 towards less advantageous decision making. Interestingly though, while adults with epilepsy selected fewer advantageous than disadvantageous cards in blocks 2 to 4, block 5 revealed a sudden increase in advantageous card selection. This overall pattern can be compared to deck selection of the control group. While the control group did not avoid selecting from deck B to the same extent as deck A, they showed a clear preference for advantageous decks C and D than decks A and B by block 2, whereas the epilepsy group demonstrated this pattern much later at block 5, (Figures 3,4). This pattern suggests that the control group initially selected from disadvantageous decks A & B, but early on at block 2 they had learned to choose the advantageous decks C and D, and avoid deck A which is one of the disadvantageous decks, therefore demonstrating good learning. Consequently, even when accounting for deck B in their selection, the control group still showed learning. Indeed, control group increased their selection of advantageous choices selecting deck D as their main choice, (Figure 3). Deck D has high frequency gain and low frequency loss, and is the best choice for a winning strategy.

By comparison to the control group, the epilepsy group did not show they had learned to avoid the bad decks towards the end of block 4. By block 5, the epilepsy group show a delayed

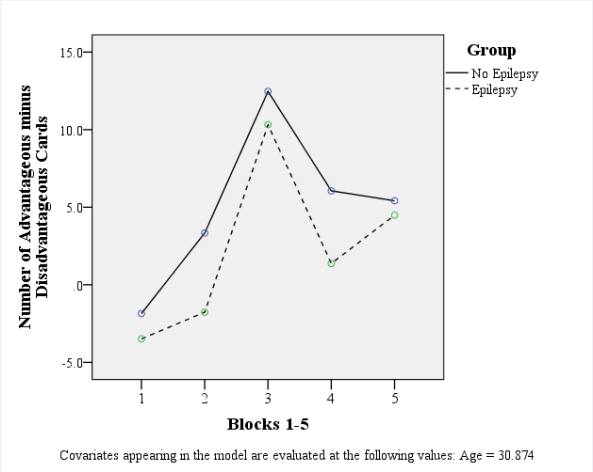

Figure 2 Mean number of cards selected from the advantageous minus disadvantageous decks for each of 5 blocks of 20 cards between adults with and without epilepsy.

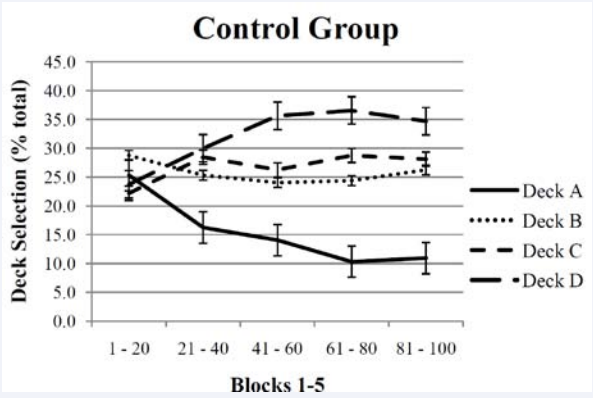

Figure 3 Control group deck selection from each of 4 decks (A,B,C& D) as a percentage of total selection, for each of 5 blocks of 20 cards.

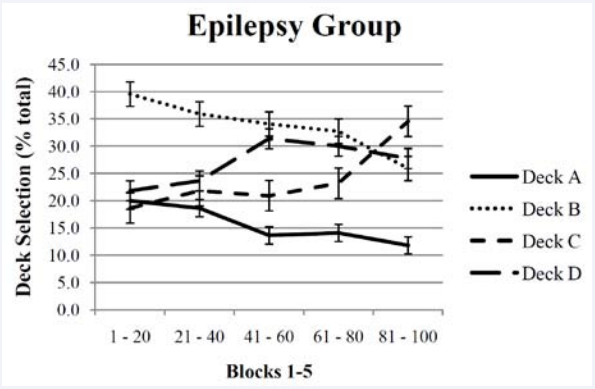

Figure 4 Epilepsy group deck selection from each of 4 decks (A,B,C& D) as a percentage of total selection, for each of 5 blocks of 20 cards.

shift to avoiding the bad decks. A decrease in selection from disadvantageous deck A occurs in later blocks than for the control group. In addition, the epilepsy group appear to sample more from the whole range of decks throughout the task compared to control group who seem to select mostly from decks C and D, and from deck B selections as discussed. For a review on deck B selection in typical subjects see Lin and colleagues [35]. This pattern of shifting reveals a slower learning effect with a fairly consistent selection trend from deck B in association with increased selection from decks C and D.

Impaired decision making ability measured by the IGT implicates degraded vmPFC functioning [36]. Prefrontal cortex network abnormalities have been identified in absence epilepsy, idiopathic generalized epilepsy, FLE, focal epilepsies, and purported to explain difficulties in social adjustment in [15-18]. Our findings of disadvantageous decision making under ambiguity support previous evidence of poor decision making measured by the IGT in epilepsy patients found in JME and mTLE [17,19-21]. Notably, findings by Wandschneider and colleagues found that a greater proportion of JME patients with seizures than seizure-free patients had difficulties in advantageous decision making. Our research not only found poorer decision making but may explain their difficulties in social adjustment, especially considering 10 of 11 participants with epilepsy were from our previous study which found higher autistic behavioural characteristics in a heterogeneous group of adults with epilepsy [4].

Chronic epilepsy in mTLE patients has been related to worse scores for decision making which is consistent with the likelihood that epileptic seizures may have negative consequences for decision making abilities, as chronic epilepsy is associated with both amygdala damage and progressive hippocampal damage in intractable TLE, regardless of pathology [23-25].

Recent evidence has identified that early-life seizures in animal studies can result in lasting alterations in the structure and functioning of the PFC [3]. The researchers' state that these early seizures result in thicker prelimbic PFC which is consistent with the abnormal and increased brain growth associated with ASD (ibid.). Importantly, this recent evidence of abnormal functioning in the PFC would be consistent with impaired decision making for adults with childhood-onset of epilepsy. Generally, the evidence above suggests that there may be a deficit of formation of somatic markers in adults with epilepsy which results in significant differences in decision making which may be consistent with either functional differences or functional connectivity to the medial and ventromedial brain regions.

This paper highlights three possible accounts which, whilst not mutually exclusive, may explain this pattern of performance. The first possibility is an initial delay in formation of associations for somatic markers due to differences in encoding information. This explanation would lead to a pattern of learning which may be identical to the control group, but due to an initial delay, the learning occurs at a later time point. The pattern of learning by the epilepsy group initially differed in block 2, but by block 5 at the end of the task there is no difference. In this experiment, there are fewer previous experiences to draw upon at the beginning of the task in block 1. The data suggests that the epilepsy group are delayed from block 1 at the early stage but not the later stage of the task (Figure 2) plots the advantageous minus disadvantageous deck selection, revealing that the epilepsy group demonstrate fewer advantageous selections throughout the task, but this is worse in block 2 which would be consistent with a delay in the beginning. In support of a delay in learning, the RT data reveals a behavioural change towards advantageous deck selection in the control group was reached at the end of block 1 at mean time of 25.4 seconds, whereas this change was reached by the epilepsy group at the end of block 4 at a mean time of 1 minutes and 26.6 seconds. Therefore, while an initial delay in formation of associations for somatic markers was demonstrated, these differences did not continue through to the end of the task, and this explanation is rejected.

The second possibility is that probabilistic contingencies are not being fully encoded, the resulting upstream effect may be revealed through a weakened formation of somatic markers. Formation of weaker associations may be related to a weakened connection between the amygdala and hippocampus. The strength of the connection between the amygdala and hippocampus is predictive for adaptive learning [37]. However, if formation of somatic markers were weakened, this would not provide an explanation for the sudden learning shown in block 5 by epilepsy group which shows an improvement in performance that is not consistent with this explanation, therefore this explanation is rejected.

The third possibility is that participants with epilepsy encode all information but fail to link associations into meaningful reward/punishment categories immediately, however they store all information correctly until they form the associations, subsequently profiting from this later in the task. This possibility proposes that information is encoded correctly, but formation of associations for somatic markers is hindered, possibly due to functional differences in the vmPFC, resulting in a loss of linking the physiological response to the stimulus. This could explain the pattern of initial poor performance, but after the initial delay the associations are linked leading to a sudden observable change of improved performance. This is evidenced by fast learning in block 5 in the epilepsy group, in contrast to block 4. Once linked, these associations are strong enough to drive advantageous

decision making quickly, providing an outcome comparable to the control group.

The above evaluation suggests that the mostly likely explanation of the three accounts above may be that the adults with epilepsy demonstrated an initial delay at the beginning, possibly due to failure to link associations after correctly encoding information, evidenced by their improved performance towards the end of the task.

LIMITATIONS

There are several limitations of this experiment. The experiment was limited by sample size and sample bias. It is unknown whether performance was influenced by depressive symptoms which are common in TLE [38]. Further, AEDs are related to a decline in performance IQ, concentration and mental speed, and one AED type, valproic acid, can impair complex decision making [39].It is unknown whether participants in the 'AEDs unknown, n=4' group were treated with this AED type. It is also unknown whether lateralisation influenced performance as suggested previously, however recent research has suggested it may not be a factor for decision making [40]. Another limitation is that performance on the IGT cannot be directly contrasted with performance on tasks measuring social cognitive function. However, given the findings in this study, further research of social cognitive function in adults with epilepsy is warranted.

CONCLUSION

The aim of the experiment was to explore ambiguous decision making in a heterogeneous group of adults with epilepsy. The SMH was a potential valid explanatory model for this study, as somatic marker formation is crucial for social cognition, and any deficits in linking associations into meaningful reward/punishment categories demonstrated by the participants with epilepsy could underlie self-reported social difficulties that have been described in previous studies.

The results of this experiment measuring performance on decision making under ambiguity revealed that adults with epilepsy performed significantly worse than adults without epilepsy. This indicated that the epilepsy group demonstrated difficulties with formation of associations for somatic markers, which would typically guide their decision making performance on the Iowa Gambling Task. These findings suggest that somatic marker formation was compromised in the epilepsy group. As somatic markers are crucial for guiding social behaviour, this may provide an explanation for the poor social adjustment and social difficulties identified as autistic-like characteristics found in this population.

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