Reduced autonomic responses to faces in Capgras delusion

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SUMMARY

People experiencing the Capgras delusion claim that others, usually those quite close emotionally, have been replaced by near-identical impostors. Ellis & Young suggested in 1990 that the Capgras delusion results from damage to a neurological system involved in orienting responses to seen faces based on their personal significance. This hypothesis predicts that people suffering the Capgras delusion will be hyporesponsive to familiar faces. We tested this prediction in five people with Capgras delusion. Comparison data were obtained from five middle-aged members of the general public, and a psychiatric control group of five patients taking similar anti-psychotic medication. Capgras delusion patients did not reveal autonomic discrimination between familiar and unfamiliar faces, but orienting responses to auditory tones were normal in magnitude and rate of initial habituation, showing that the hyporesponsiveness is circumscribed.

1. INTRODUCTION

The Capgras delusion (Capgras & Reboul-Lachaux 1923; Ellis *et al.* 1994) is characterized by the patient insisting that others, usually those quite close emotionally, have been replaced by doubles, impostors or robots. This bizarre belief can arise as a monosymptomatic delusion held with conviction despite insight into its irrationality and unbelievability (Alexander *et al.* 1979). It has been noted to follow various types of brain injury (Fleminger & Burns 1993; Förstl *et al.* 1991), has been reported for patients from many different cultures (Christodoulou 1977), and presents a significant risk of violence against the alleged impostors (de Pauw & Szulecka 1988; Förstl *et al.* 1991; Silva *et al.* 1995).

The Capgras delusion has long fascinated psychiatrists (Enoch & Trethowan 1991), and is beginning to attract the attention of philosophers interested in the nature of human consciousness, beliefs and rationality (Dennett 1996). Although usually considered very rare (Enoch & Trethowan 1991), there are grounds for thinking it may have been under-reported (Förstl *et al.* 1991).

Psychodynamic accounts of the Capgras delusion are still invoked by some authors, and this possibility was explored by Capgras himself (Capgras & Carrette 1924). A widely adopted hypothesis has been that conflicting feelings of love and hate toward a close relative are resolved by the delusion, since the double can be hated without guilt (Enoch & Trethowan 1991). Closely related to this is Berson's (1983) idea that the precipitating event is a change in crucial interpersonal relationships, producing feelings of strangeness and eliciting previously unconscious negative feelings. These, claims Berson, lead the patient to conclude that the person who elicits such different feelings is an impostor (Berson 1983, pp. 975–976).

A major problem for psychodynamic accounts, however, is that advances in brain imaging have resulted in many of the case reports of Capgras delusion and related problems of misidentification published in the last 20 years showing clear neurological damage (de Pauw 1994; Förstl *et al.* 1991); these imply that, at the very least, psychodynamic accounts do not offer a full explanation.

Other attempts to explain the Capgras delusion include the cerebral hemisphere disconnection hypothesis (Joseph 1986), which proposes that each cerebral hemisphere independently processes visual information from the face, and that the Capgras delusion arises when the two processes fail to integrate; the categorization failure hypothesis (Cutting 1991), which suggests that the delusion reflects a disturbance in the judgement of identity or uniqueness, owing to a breakdown of the normal structure of semantic categories; and the memory deficit hypothesis (Staton *et al.* 1982), which maintains that there is a failure in the updating of the patient's mental representations of familiar faces, and that Capgras delu-

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sion results from the consequent mismatch between what is seen and its ultimately outdated representation.

There are thus a number of theoretical accounts of possible functional bases for the Capgras delusion in the research literature, but these are usually generated *ad hoc* in discussions of individual cases, and are seldom tested against systematically collected empirical data.

A particular focus of recent interest has been in the hypothesis that diminished affective responses to familiar visual stimuli may play a causal role. Ellis & Young (1990) suggested that the Capgras delusion results from damage to a neurological system involved in orienting responses to seen faces based on their personal significance. On this hypothesis, the basis of the Capgras and other reduplicative delusions lies in damage to neuro-anatomical pathways responsible for appropriate emotional reactions to familiar visual stimuli, and represents the patient's attempt to make sense of the fact that these visual stimuli no longer have appropriate affective significance. This conception can be traced back to Brochado's (1936) description of a thesis by Derombies (1935), and has recently been emphasized by several authors (Anderson 1988; Ellis & Young 1990; Lewis 1987; Weinstein & Burnham 1991). A prediction made by this hypothesis (and by no other existing account) is that people suffering the Capgras delusion will be hyporesponsive to familiar faces. Consistent with this prediction, we show here that Capgras delusion patients do not reveal autonomic discrimination between familiar (famous) and unfamiliar faces. However, orienting responses to auditory tones were normal in magnitude and rate of initial habituation for Capgras cases, showing that the hyporesponsiveness is circumscribed.

Ellis & Young's (1990) prediction has its origins in the findings of covert recognition in prosopagnosic patients by Bauer (1984) and Tranel & Damasio (1985). Prosopagnosia involves an inability to recognize previously familiar faces after brain injury in the occipito-temporal region. Although overt recognition of even the most familiar faces can be lost, some prosopagnosics have been shown to reveal autonomic activity (skin conductance response—SCR) to faces of known individuals; this happens even though, consciously, the prosopagnosic patient is unable to identify the person (Bauer 1984; Tranel & Damasio 1985, 1988).

Covert recognition indexed by autonomic activity in prosopagnosia, it has been argued, results from the continued operation of a system responsive to the signal value arising from the personal emotional significance of the face (Bauer 1984; Tranel & Damasio 1985; Tranel & Damasio 1988; Tranel *et al.* 1985). Hence Bauer (1984) claimed that overt recognition of the face's identity (severely impaired in prosopagnosia) and covert recognition in the form of autonomic responses (relatively preserved in prosopagnosia) involve different neurological pathways. Further evidence of a dissociation between overt and covert face recognition comes from the observation by Tranel *et al.* (1995) of four patients with bilateral ventromedial frontal damage who could recognize faces normally yet did not reveal discriminatory SCRs to these familiar faces. The accounts given by Bauer (1984) and by Tranel *et al.* (1995) differ with regard to the exact neurology of pathways mediating overt and covert recognition, but concur in regarding them as neurologically dissociable.

Ellis & Young (1990) proposed that Capgras delusion is the neurological mirror image of prosopagnosia, resulting from relatively preserved overt recognition (the patient knows that a face is his wife's, or whoever), coupled with a loss of affective responses. The delusion is then a misattribution by the patient of a change in his or her internal world (reduced responsiveness to stimuli with personal relevance) to a change in the external world (replacement by replicas or impostors) (Wright *et al.* 1993; Young *et al.* 1993). Such misattributions to external influences are likely in states of intense suspiciousness (Kaney & Bentall 1989), known to be a common correlate of Capgras delusion (Fleminger & Burns 1993).

On this account, Capgras delusion reflects an unfortunate interaction of different contributory causes, among which we have singled out suspiciousness and loss of appropriate affective responses (Young 1994). The advantage of this hypothesis is that it generates testable falsifiable predictions that do not follow from other existing accounts.

We predicted, then, that if tested with procedures used to investigate covert autonomic recognition in prosopagnosia (Bauer 1984; Tranel & Damasio 1985, 1988), people suffering the Capgras delusion will be hyporesponsive to familiar faces and will not show differential SCRs to familiar faces even though these may be overtly recognized. We tested this prediction in five people with Capgras delusion. Comparison data were obtained from five middle-aged members of the general public, and a psychiatric control group of five patients taking similar anti-psychotic medication was used to rule out the possibility that hyporesponsiveness might be due either to medication or to delusions *per se*.

2. METHOD

(*a*) *Participants*

Five people with Capgras delusion participated in the study. All were psychiatric patients. Table 1 summarizes clinical information for these five individuals. In each case they had expressed the idea that others had been substituted and firmly believed that they were impostors. Comparison data were obtained from a psychiatric control group of five patients taking similar anti-psychotic medication, and a second control group of five middle-aged members of the general public.

(*b*) *Facial identity processing*

Facial identity processing was tested for the five participants with Capgras delusion. Recognition of familiar faces, matching photographs of unfamiliar faces (in the

Table 1. *Background information on participants with Capgras delusion and psychiatric controls*

Capgras patients				
patient	age	sex	symptoms	medication
HS	37	F	Capgras delusion for family, ward staff and patients. Persecutory delusions.	Trifluoperazine
VD	44	F	Capgras for virtually the entire town of residence. Reduplicative paramnesia for hospital. Persecutory delusions. Auditory and visual hallucinations. Conflicting diagnoses: schizophrenia/mania.	Lithium Loxapine
IN	32	M	Capgras for parents. Persecutory delusions. Conflicting diagnoses: paranoid schizophrenia, paranoid psychosis, psychogenic psychosis.	Chlorpromazine Trifluoperazine
VL	34	F	Capgras for a number of people, including family members. Persecutory delusions. Conflicting diagnoses: acute schizophrenic episode/paranoid psychosis/generalized anxiety.	Temazepam Chlorpromazine Trifluoperazine Flupenthixol decanoate
SS	63	F	Capgras for neighbours. Persecutory delusions.	Trifluoperazine
psychiatric controls				
patient	age	sex	symptoms	medication
BR	63	F	Vivid audio-visual hallucinations, persecutory delusions. Diagnosis: organic disorder.	Trifluoperazine
AN	27	M	Delusions of reference, grandeur and persecution. Conflicting diagnoses: schizophrenia/ schizoaffective disorder/recurrent manic episodes.	Lithium Flupenthixol decanoate
MN	42	F	Depressed mood, paranoia, suicidal ideation. Diagnosis: depression.	Chlorpromazine Diazepam, Lofepramine
AY	31	F	Delusions of persecution, guilt and ill-health. Conflicting diagnoses: psychotic depression/ schizophreniform illness/paranoid schizophrenia/ schizoaffective disorder.	Zopiclone Lofepramine Droperidol Lorazepam
NN	21	M	Delusions of reference, grandeur and persecution. Auditory hallucinations. Conflicting diagnoses: schizophrenia/manic depression.	Zopiclone Diazepam Chlorpromazine

Benton test or a task using disguised faces), and recognition memory for faces were assessed. Due to the demands of clinical testing, participants were not able to complete all of these tasks, but everyone was tested on at least two of them. For these tasks, the performance of people with Capgras delusion was compared with that of 40 controls (20 male, 20 female), aged 20–59.

Identification of familiar faces was assessed with 30 highly familiar faces (famous people) and 10 unfamiliar faces, presented in pseudo-random order (Young *et al.* 1995). For each face the subject was asked whether or not it was a familiar person and, if so, his or her occupation and name. An additional four trials with familiar faces and two trials with unfamiliar faces were given first, as practice. The measures of performance involved the number of highly familiar faces recognized as familiar, the number given correct occupation, the number named correctly, and the number of unfamiliar faces correctly recognized as unfamiliar (correct rejections).

In the 'Benton test of facial recognition' (Benton *et al.* 1983), subjects have to choose which of six photographs of unfamiliar faces are pictures of the same person as a simultaneously presented target face photograph. The test includes items involving choice of identical photographs, as well as transformations of orientation or lighting, which are pooled to give an overall total. The unfamiliar face matching test used with patient SS examined ability to match disguised faces (Young *et al.* 1990). Two separate test sheets were used, each showing a 4×4 matrix of faces in which each of the four faces in the top row appeared three times in disguised or undisguised forms elsewhere on the sheet.

The 'Warrington recognition memory test' (RMT) (Warrington 1984) assesses recognition memory separately for faces and words. In the faces part of the RMT, 50 faces are shown at the rate of one every three seconds for a 'pleasant or unpleasant' decision, and recognition memory is then tested immediately by presenting each of the faces paired with a distractor, with the subject having to choose which has been seen before. A similar

procedure is used with words, and was included here to check for general cognitive impairment. Ellis *et al.* (1992) found a substantial impairment on the faces part of this test in a small group of Capgras patients.

The NART-R (Nelson 1991) reading test was also used as an additional background measure providing an estimate of intelligence.

(*c*) *SCRs to faces*

The five Capgras delusion patients, five psychiatric controls and five normal controls were tested with a procedure modelled on that adopted by Tranel and his colleagues for their studies of covert recognition in prosopagnosia (Tranel & Damasio 1985, 1988; Tranel *et al.* 1985). The skin conductance responses (SCRs) were recorded to a series consisting predominantly of unfamiliar faces, with occasional familiar (famous) faces interspersed among them. The difference in mean SCR to familiar and unfamiliar faces was used as an index of the autonomic orienting response to faces with high signal value. Famous faces were used (rather than personally familiar faces) to allow a common set of materials for all subjects, and because the constraints of clinical testing of the patient groups would have prevented assembling sets of family photographs in sufficient time.

The 29 face stimuli were chosen from a set of 328 faces. They were scanned into a Macintosh LC computer as grey-scale PICT images using Desk Scan II, so that each filled a 7.5 cm × 10 cm area at the centre of the monitor. All 328 faces were rated for familiarity by 11 independent judges using a seven-point Likert scale. For the present experiment 20 faces rated '1' (i.e. unknown) by all judges were used for the unfamiliar set of 20, and five with an average rating higher than six (min. 6.33, max. 6.86) were selected as the familiar set. Four unfamiliar faces acted as buffer items, two occurring before and two after the experimental set.

During testing each subject was seated in an adjustable chair so that his or her eyes were approximately 100 cm from the centre of the display computer monitor. The subject was told that a series of faces would be presented, each for 2 s followed by a 20 s interval, and asked to attend to the stimuli while remaining relaxed throughout. There followed a 5 min period during which electrodes were placed in position and baseline SCR levels recorded. Then two buffer items, 25 experimental stimuli, and two further buffers were presented in sequence. During this presentation, the five familiar faces occurred at irregular intervals in the sequence of predominantly unfamiliar faces. After the experiment, the subject was shown all stimuli again to check that he or she correctly knew each to be familiar or unfamiliar. This they all did without error for the five familiar faces and so no adjustment to the familiar face data was necessary. If an unfamiliar face was misclassified as familiar, data for the corresponding trial were omitted; only 2.5% of unfamiliar face trials were dropped for this reason.

The SCRs were recorded on a MacLab/8 device using MacLab software. Recordings were made via 1 cm^2 Ag–AgCl electrodes fixed to the thenar and hypothenar eminences of the non-preferred hand with adhesive collars that allowed a 0.5 cm contact area with the electrolyte. The electrolyte was made up of 0.05 molar NaCl in a unibase cream medium. Subjects' hands had first been thoroughly cleaned with a liquid soap before electrodes were attached. During testing the relaxed arm was supported by a chair rest.

Taking the advice of Venables & Christie (1980) a latency window of 1–3 s from stimulus onset was used to determine an amplitude measure. All responses that did not have their initial deflection within this window were ignored. The amplitude of the largest SCR within each latency window was measured by someone who was blind as to which stimulus (familiar or unfamiliar face) gave rise to each. The largest amplitude of the response to each relevant stimulus was taken as the SCR measure. These values were used to create two averages; one relating to the five latency windows for familiar stimuli, and the other to the 20 latency windows for unfamiliar stimuli. Only after blind scoring were the calculated SCR amplitudes related to corresponding stimulus familiarity.

(*d*) *SCRs to repeated tone*

A further SCR test examined habituation of the orienting response to a repeated tone. Our intention was to create a task in which SCR could be measured to an auditory instead of a visual stimulus, and in which the rate of change in response to a repeated signal could be determined. This additional test also served to ensure that, for each subject, good contact existed between hand and electrodes.

Four of the Capgras delusion patients and five normal control subjects participated as subjects. The tones were 0.5 s bursts of 90 dB white noise, presented via headphones at irregular intervals which were on average 20 s apart. SCRs were measured in the manner described above, and the maximum amplitude calculated for the defined 1–3 s window following each tone. Habituation occurred rapidly, with some SCRs in both groups showing zero response to the tones after five trials.

3. RESULTS

The mean SCR amplitudes to familiar and unfamiliar faces for each of the three groups of participants are shown in figure 1*a*. Because the prediction made was for an interaction between face familiarity and subject group a two-factor ANOVA was used. It revealed a main effect for group ($F = 4.99$, d.f. $2, 12$, $p < 0.05$), and a main effect for familiarity ($F = 20.70$, d.f. $1, 12$, $p < 0.001$). The most important result, however, was a highly significant group × familiarity interaction ($F = 7.37$, d.f. $2, 12$, $p < 0.01$). Simple effects analyses showed that the three groups differed significantly in their SCRs to familiar faces ($F = 6.75$, mean s.e. $= 0.019$, $p < 0.05$), but not to unfamiliar faces ($F = 2.06$, mean s.e. $= 0.009$, $p = 0.17$). The normal controls and the psychiatric controls each revealed higher mean SCRs to familiar compared with unfamiliar faces; normal controls ($F = 29.67$, mean s.e. $= 0.003$, $p < 0.001$), psychiatric controls ($F = 5.78$, mean s.e. $= 0.003$, $p < 0.05$). The Capgras delusion group, however, revealed no differential SCRs to familiar and unfamiliar faces ($F < 1$).

Although ANOVA was our preferred method of analysis, because of the predicted interaction, a possible concern is that SCRs are not always normally distributed. While ANOVA is reasonably robust when data distributions are either not normal or are skewed (Stevens 1990), it was thought prudent to carry out a supplementary analysis involving

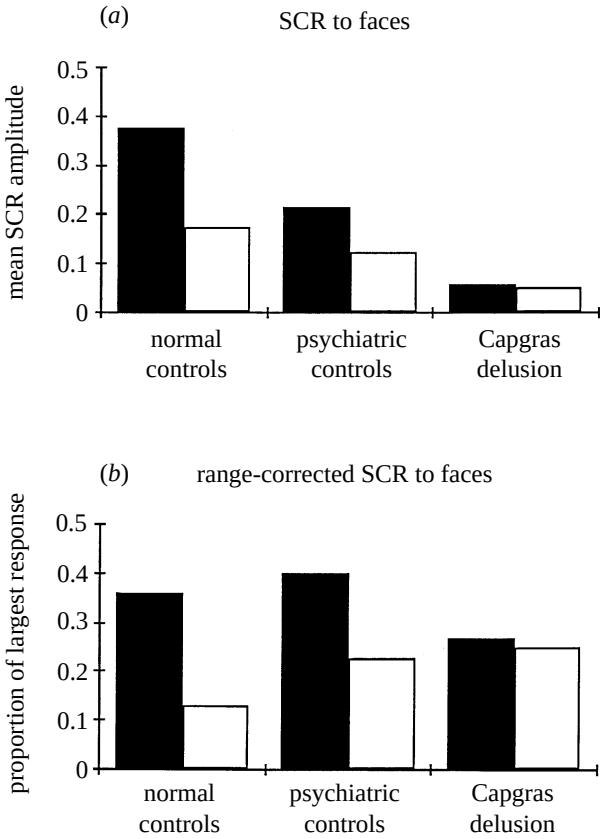

Figure 1. Mean SCR amplitude to familiar (filled column) and unfamiliar (open column) faces for normal controls, psychiatric controls and people with Capgras delusion. Figure 1*a* gives amplitudes in μS; figure 1*b* shows range-corrected responses.

a non-parametric procedure. Binomial tests comparing each group's mean responses to familiar and unfamiliar faces revealed significant differences for the normal group ($p = 0.03$) and the psychiatric control group ($p = 0.03$). There was no such effect for the Capgras group ($p > 0.2$).

Although these data are consistent with the predicted lack of an SCR to familiar faces in people with Capgras delusion, they also show a strong hint of more generalized hyporesponsiveness. Even though the SCRs to unfamiliar faces did not differ significantly across groups, there seemed to be a trend toward lower responses from the people with Capgras delusion. Potentially, therefore, the interpretation might be contaminated by a floor effect arising from the reduced responses of the Capgras delusion group overall. Consequently, we carried out a supplementary analysis using range-corrected scores to eliminate the main effect of subject group (Boucsein 1992). The particular transform we used was suggested by Lykken & Venables (1971); it expresses each SCR as a proportion of that subject's largest response. These range-corrected means are shown in figure 1*b*. With this transform, genuine differences between SCRs to familiar and unfamiliar faces will still be evident as a group × familiarity interaction. This interaction was significant for comparisons of the Capgras delusion and psychiatric control groups ($F = 5.26$, d.f. $1, 8$, $p = 0.05$), and the Capgras delusion and normal control groups ($F = 7.46$, d.f. $1, 8$, $p < 0.05$), showing that even when the range of responses was corrected, there was no sign of any differential SCR response to familiar faces by people with Capgras delusion. In contrast, there was no such interaction between the responses of the psychiatric and normal control groups ($F < 1$), indicating that these groups did not differ from each other.

Again, non-parametric tests confirmed this pattern. Binomial tests comparing each group's range-transformed responses to familiar and unfamiliar faces revealed significant differences for the normal group ($p = 0.03$) and the psychiatric control group ($p = 0.03$), with no difference for the Capgras group ($p > 0.2$).

In our SCR test with faces, a post-test was used to establish that the highly-familiar faces could be recognized. Absence of differential SCR to familiar faces was found even though all of the highly familiar faces we used as stimuli in the SCR experiment were overtly recognized by participants in the Capgras delusion group. However, it is known that Capgras delusion can be associated with generalized deficits of face processing (Ellis *et al.* 1993*a*; Young *et al.* 1993, 1994). With this in mind, we also investigated the performance of these patients on standard face processing tasks. Results for familiar face recognition (Young *et al.* 1995), unfamiliar face matching (Benton *et al.* 1983; Young *et al.* 1990), and recognition memory for faces (Warrington 1984) are summarized in table 2. For comparison, table 2 also shows SCR differences (mean amplitude to familiar faces minus mean to unfamiliar faces) from the present investigation. In addition, NART-R estimated IQs (Nelson 1991) and recognition memory for words (Warrington 1984) are included in table 2 to provide further background information.

As table 2 shows, deficits in face-processing tasks were evident in the participants with Capgras delusion, with four people showing deficits on at least one test. We have pointed out previously that some degree of association of Capgras delusion with deficits of overt face processing abilities is likely to happen because neurological abnormalities will usually affect both overt and covert face processing systems (Young *et al.* 1994; Young *et al.* 1993). This does not weaken our prediction, which requires only that covert (autonomic) recognition is the more affected in Capgras delusion. This is entirely consistent with our findings; deficits on overt face-processing tasks were variable in severity and in the tests affected, whereas there was a consistent lack of differential SCR responses to familiar faces.

Table 2 also demonstrates that hyporesponsiveness to faces was found for Capgras patients who remained of normal estimated intelligence using the NART-R, and whose recognition memory for non-facial stimuli was unimpaired (four out of five patients showed good recognition memory for words).

In order to determine whether people with Capgras delusion show generalized hyporesponsiveness to

Table 2. *Facial identity processing by participants with Capgras delusion*

(The table shows recognition of familiar faces (Young *et al.* 1995), matching photographs of unfamiliar faces in the Benton test (Benton *et al.* 1983) or a task using disguised faces (Young *et al.* 1990), and recognition memory for faces (Warrington 1984), together with means and s.d.s for controls. For comparison, the table also shows individual SCR differences to faces (mean amplitude to familiar faces minus mean to unfamiliar faces). Additional background information is provided in the form of NART-R estimated IQs (Nelson 1991) and recognition memory for words (Warrington 1984). NT = not tested.)

						controls	
	HS	VD	IN	VL	SS	mean	s.d.
NART-R estimated IQ		119	97	102	100	NT	
identification of familiar faces							
high familiarity faces							
recognized as familiar (max. = 30):	23[c]	27	30	11[d]	NT	28.80	2.08
ccupation (max. = 30):	23[b]	27	30	11[d]	NT	28.48	2.50
name (max. = 30):	19[b]	20[b]	30	9[d]	NT	26.30	3.72
unfamiliar faces							
correct rejections (max. = 10):	10	9	10	10	NT	9.38	0.74
unfamiliar face matching							
Benton test (max. = 54):	42[a]	45	48	NT	NT	47.95	4.40
disguise test (max. = 24):	NT	NT	NT	NT	24	22.43	2.57
recognition memory (Warrington RMT)							
faces (max. = 50):	41	41	33[d]	39[a]	43	44.50	3.64
words (max. = 50):	48	49	43[b]	46	50	47.65	2.75
SCR difference to faces (familiar–unfamiliar)	0.00[b]	0.00[b]	0.03[a]	0.00[b]	0.00[b]	0.20	0.11

Marked scores are below control mean: [a] $z > 1.29$, $0.1 > p > 0.05$; [b] $z > 1.65$, $p < 0.05$; [c] $z > 2.33$, $p < 0.01$; [d] $z > 3.10$, $p < 0.001$.

all stimuli, we measured habituation of orienting responses to a series of auditory tones. Four of the Capgras patients were able to complete this task; their responses were compared with those of five normal control subjects. Data are summarized in figure 2; the Capgras delusion patients produced responses of the same magnitude as the normal control group, and habituated to the repeated trials at the same rate. Analysis of variance confirmed these impressions; there was no main effect of subject group ($F < 1$), a decline in SCR across trial blocks ($F = 4.61$, d.f. $4, 28$, $p < 0.01$), and no group × blocks interaction ($F = 1.07$, d.f. $4, 28$, $p = 0.39$). The fact that participants with Capgras delusion showed normal SCR habituation to repeated tones also serves to demonstrate that any low responses to faces cannot simply be attributed to poor electrode contact or other types of testing artifact.

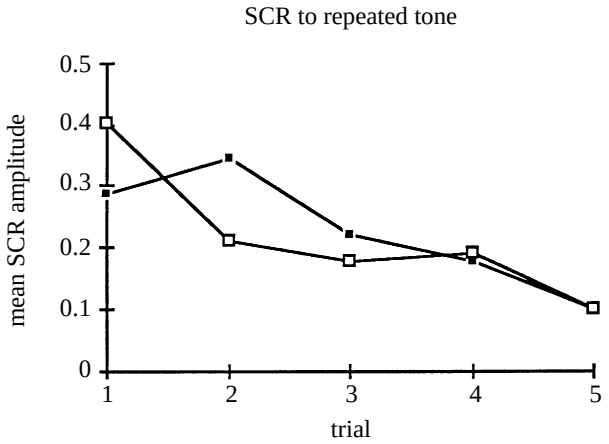

Figure 2. Mean SCR amplitude (μS) across five presentations of an auditory tone for normal controls (filled squares) and people with Capgras delusion (open squares).

4. DISCUSSION

Our findings show that, as predicted by Ellis & Young (1990), there is no differential responding by patients with Capgras delusion to familiar as compared to unfamiliar faces. In addition, Capgras delusion seems to be associated with reduced autonomic responding to facial stimuli, a condition which has been referred to as hypoemotionality (Bauer 1982).

The absence of any differential SCR response to familiar faces in the Capgras group is very striking; higher amplitude SCRs to familiar faces are reliably found for normal subjects (Tranel *et al.* 1985), and were shown by all our normal and psychiatric controls. Moreover, higher SCRs to familiar faces are found even when stimuli are masked to prevent overt recognition, creating an analogue of prosopagnosia in normal subjects (Ellis *et al.* 1993*b*). Yet four of the five Capgras cases showed no differential SCR response to familiar faces whatsoever, and for the fifth patient the size of the SCR effect only entered

the bottom end of the normal range ($p < 0.1$).

The SCRs to repeated tones, however, show that Capgras delusion is not associated with hyporesponsiveness to any stimulus. Note especially that the initial level of SCR to tones (for both Capgras patients and controls) is comparable to the level of normal controls' responses to familiar faces, and that this response to repeated tones rapidly declines to a level comparable with the Capgras patients' responses to (novel) familiar faces.

Whilst we would not seek to claim from these data that the lack of SCR in Capgras delusion is specific to faces (and neither would any such prediction necessarily follow from our theoretical account), it is clear that the hyporesponsiveness is to some extent circumscribed; it ends somewhere in the region between seeing familiar faces and listening to auditory tones.

These findings demonstrate the usefulness of Ellis & Young's (1990) conception of Capgras delusion as a mirror-image of prosopagnosia, in which orienting responses based on the personal and affective significance of faces are lost, but overt recognition is relatively spared. As we have noted, Bauer (1984) had proposed the converse deficit of impaired overt recognition and relatively spared orienting responses to account for his findings of preserved SCRs in prosopagnosia. He (Bauer 1984) proposed that a ventral visuo-limbic route is responsible for overt recognition of the face's identity, and a more dorsal visuo-limbic route for emotional and orienting responses based on the face's personal affective significance. Tranel *et al.* (1995) suggest a different neural substrate. They argue from their work on prosopagnosics and patients with frontal damage that it is damage to the ventromedial frontal cortices that prevents the operation of autonomic control nuclei. Our findings for Capgras delusion support the suggestion of neurologically separable processes responsible for orienting responses and overt recognition, by forming a double dissociation when considered alongside previous results using the same tests in cases of prosopagnosia (Tranel & Damasio 1985; Tranel & Damasio 1988).

An important feature of our results is that abnormal responses to familiar faces were found for people that the Capgras patients had not claimed were duplicates. This is again as predicted by an account in terms of damage to a neurological system involved in orienting responses to seen faces based on their personal significance (Ellis & Young 1990), but would not be expected on other accounts, and especially from psychodynamic hypotheses. We consider that the delusion is held for close relatives because orienting and affective responses are most critical for these people, and hence a more general absence of such responses is most noticeable to the patient for close relatives. There is evidence consistent with this claim. It has often been remarked that, whilst it is initially voiced for close relatives, the Capgras delusion does gradually 'spread' to encompass others (Wallis 1986), as would be expected as the patient gradually becomes aware of the extent of the abnormality in emotional responding.

Unfortunately, given clinical testing constraints, it was not possible in this study to obtain photographs of the people for whom the Capgras patients expressed their delusions. As such, though, the experiment was a more stringent test of the Ellis & Young (1990) prediction. Nonetheless, future studies with a more in-depth analysis of SCRs to a variety of faces, perhaps by a smaller sample of Capgras patients, could be revealing. Equally, functional brain-imaging studies of them looking at familiar and unfamiliar faces could help determine the brain centres and pathways that may be impaired. The results presented here do not speak directly to the neural basis for the Capgras delusion, but they do reinforce the likelihood of dual routes to face recognition. The next step is to determine where these occur and at what stage in the process they separate (Damasio *et al.* 1990; De Haan *et al.* 1992).

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REFERENCES

Alexander, M. P., Stuss, D. T. & Benson, D. F. 1979 Capgras syndrome: a reduplicative phenomenon. *Neurology* **29**, 334–339.

Anderson, D. N. 1988 The delusion of inanimate doubles. Implications for understanding the Capgras phenomenon. *Br. J. Psych.* **153**, 694–699.

Bauer, R. M. 1982 Visual hypoemotionality as a symptom of visual-limbic disconnection in man. *Arch. Neurol.* **39**, 702–708.

Bauer, R. M. 1984 Autonomic recognition of names and faces in prosopagnosia: a neuropsychological application of the guilty knowledge test. *Neuropsychologia* **22**, 457–469.

Benton, A. L., Hamsher, K. S., Varney, N. & Spreen, O. 1983 *Contributions to neuropsychological assessment: a clinical manual.* Oxford University Press.

Berson, R. J. 1983 Capgras' syndrome. *Am. J. Psych.* **140**, 969–978.

Boucsein, W. 1992 *Electrodermal activity.* New York: Plenum.

Brochado, A. 1936 Le syndrome de Capgras. *Ann. Médico-Psychologiques* **15**, 706–717.

Capgras, J. & Carrette, P. 1924 Illusion des sosies et complexe d'Oedipe. *Ann. Médico-Psychologiques* **12**, 48–68.

Capgras, J. & Reboul-Lachaux, J. 1923 L'illusion des 'sosies' dans un délire systématisé chronique. *Bull. Soc. Clinique de Médicine Mentale* **11**, 6–16.

Christodoulou, G. N. 1977 The syndrome of Capgras. *Br. J. Psych.* **130**, 556–564.

Cutting, J. 1991 Delusional misidentification and the role of the right hemisphere in the appreciation of identity. *Br. J. Psych.* **159**, 70–75.

Damasio, A. R., Tranel, D. & Damasio, H. 1990 Face agnosia and the neural substrates of memory. *A. Rev. Neurosci.* **13**, 89–109.

De Haan, E. H. F., Bauer, R. M. & Greve, K. W. 1992 Behavioural and physiological evidence for covert face recognition in a prosopagnosic patient. *Cortex* **28**, 77–95.

de Pauw, K. W. 1994 Psychodynamic approaches to the Capgras delusion: a critical historical review. *Psychopathology* **27**, 154–160.

de Pauw, K. W. & Szulecka, T. K. 1988 Dangerous delusions: violence and the misidentification syndromes. *Br. J. Psych.* **152**, 91–97.

Dennett, D. 1996 *Kinds of minds: towards an understanding of consciousness.* London: Weidenfeld & Nicolson.

Derombies, M. 1935 L'illusion de sosies, forme particulière de la méconnaissance systématique. Thèse de Paris, Paris.

Ellis, H. D. & Young, A. W. 1990 Accounting for delusional misidentifications. *Br. J. Psych.* **157**, 239–248.

Ellis, H. D., de Pauw, K. W., Christodoulou, G. N., Luauté, J.-P., Bidault, E. & Szulecka, T. K. 1992 Recognition memory in psychotic patients. *Behav. Neurol.* **5**, 23–26.

Ellis, H. D., de Pauw, K. W., Christodoulou, G. N., Papageorgiou, L., Milne, A. B. & Joseph, A. B. 1993*a* Responses to facial and non-facial stimuli presented tachistoscopically in either or both visual fields by patients with the Capgras delusion and paranoid schizophrenics. *J. Neurol. Neurosurgery Psychiatry* **56**, 215–219.

Ellis, H. D., Whitley, J. & Luauté, J.-P. 1994 Classic text no. 17. Delusional misidentification. The three original papers on the Capgras, Frégoli and intermetamorphosis delusions. *Hist. Psych.* **5**, 117–146.

Ellis, H. D., Young, A. W. & Koenken, G. 1993*b* Covert face recognition without prosopagnosia. *Behav. Neurol.* **6**, 27–32.

Enoch, M. D. & Trethowan, W. H. 1991 *Uncommon psychiatric syndromes*, 3rd edn. Oxford: Butterworth-Heinemann.

Fleminger, S. & Burns, A. 1993 The delusional misidentification syndromes in patients with and without evidence of organic cerebral disorder: a structured review of case reports. *Biol. Psychiatry* **33**, 22–32.

Förstl, H., Almeida, O. P., Owen, A. M., Burns, A. & Howard, R. 1991 Psychiatric, neurological and medical aspects of misidentification syndromes: a review of 260 cases. *Psychol. Med.* **21**, 905–910.

Joseph, A. B. 1986 Focal central nervous system abnormalities in patients with misidentification syndromes. *Bibliotheca Psychiatrica* **164**, 68–79.

Kaney, S. & Bentall, R. P. 1989 Persecutory delusions and attributional style. *Br. J. Med. Psych.* **62**, 191–198.

Lewis, S. W. 1987 Brain imaging in a case of Capgras' syndrome. *Br. J. Psych.* **150**, 117–121.

Lykken, D. T. & Venables, P. H. 1971 Direct measurement of skin conductance; a proposal for standardization. *Psychophysiology* **8**, 656–672.

Nelson, H. E. 1991 *National Adult Reading Test (NART): test manual*, (revised). Windsor: NFER-Nelson.

Silva, J. A., Leong, G. B., Weinstock, R. & Klein, R. L. 1995 Psychiatric factors associated with dangerous misidentification delusions. *Bull. Am. Acad. Psych. Law* **23**, 53–61.

Staton, R. D., Brumback, R. A. & Wilson, H. 1982 Reduplicative paramnesia: a disconnection syndrome of memory. *Cortex* **18**, 23–26.

Stevens, J. 1990 *Intermediate statistics: a modern approach.* New Jersey: Erlbaum.

Tranel, D. & Damasio, A. R. 1985 Knowledge without awareness: an autonomic index of facial recognition by prosopagnosics. *Science* **228**, 1453–1454.

Tranel, D. & Damasio, A. R. 1988 Non-conscious face recognition in patients with face agnosia. *Behav. Brain Res.* **30**, 235–249.

Tranel, D., Fowles, D. C. & Damasio, A. R. 1985 Electrodermal discrimination of familiar and unfamiliar faces: a methodology. *Psychophysiology* **22**, 403–408.

Tranel, D., Damasio, H. & Damasio, A. R. 1995 Double dissociation between overt and covert recognition. *J. Cognitive Neuroscience* **7**, 425–432.

Venables, P. H. & Christie, M. J. 1980 Electrodermal activity. In *Techniques in psychophysiology* (ed. L. Martin & P. H. Venables). New York: Wiley.

Wallis, G. 1986 Nature of the misidentified in the Capgras syndrome. *Bibliotheca Psychiatrica* **164**, 40–48.

Warrington, E. K. 1984 *Recognition memory test.* Windsor: NFER-Nelson.

Weinstein, E. A. & Burnham, D. L. 1991 Reduplication and the syndrome of Capgras. *Psychiatry* **54**, 78–88.

Wright, S., Young, A. W. & Hellawell, D. J. 1993 Sequential Cotard and Capgras delusions. *Br. J. Clin. Psych.* **32**, 345–349.

Young, A. W. 1994 Recognition and reality. In *The neurological boundaries of reality* (ed. E. M. R. Critchley), pp. 83–100. London: Farrand.

Young, A. W., Aggleton, J. P., Hellawell, D. J., Johnson, M., Broks, P. & Hanley, J. R. 1995 Face processing impairments after amygdalotomy. *Brain* **118**, 15–24.

Young, A. W., Ellis, H. D., Szulecka, T. K. & de Pauw, K. W. 1990 Face processing impairments and delusional misidentification. *Behav. Neurol.* **3**, 153–168.

Young, A. W., Leafhead, K. M. & Szulecka, T. K. 1994 The Capgras and Cotard delusions. *Psychopathology* **27**, 226–231.

Young, A. W., Reid, I., Wright, S. & Hellawell, D. J. 1993 Face-processing impairments and the Capgras delusion. *Br. J. Psych.* **162**, 695–698.

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