

A neural basis for collecting behaviour in humans

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Summary

Collecting behaviour is commonplace in the normal population, but there has been little investigation of its neural basis in humans. The observation that collecting behaviour can assume pathological proportions in patients with certain patterns of brain damage led us to hypothesize that dysfunction in a system encompassing mesial prefrontal cortices accounts for abnormal collecting and may guide normal collecting. We tested the hypothesis in 86 subjects with focal lesions of the telencephalon, by relating the neuroanatomical placement of the lesions to the presence of repetitive and indiscriminate acquisition behaviour and impaired discard behaviour. The subjects had no history of psychiatric disease or abnormal collecting behaviour prior to lesion onset. Lesions were analysed with high-resolution three-dimensional MRI. Collecting behaviour was evaluated with a standardized questionnaire completed by a close relative of each subject.

Thirteen subjects exhibited abnormal collecting, characterized by massive and disruptive accumulation of useless objects. In all cases, the abnormality of collecting behaviour was severe and persisted despite attempted interventions and obvious negative consequences. There were no differences between pathological collectors and non-collectors on tests of executive functions or anterograde memory. All subjects with pathological collecting behaviour had damage to the mesial frontal region (including the right polar sector and the anterior cingulate), but there was no damage to most of the subcortical structures that, in species such as rodents, are known to drive the acquisition and retention of objects. The evidence suggests that damage to the mesial frontal region disrupts a mechanism which normally modulates subcortically driven predispositions to acquire and collect, and adjusts these predispositions to environmental context.

Keywords: collecting behaviour; prefrontal cortex

Abbreviations: AVLT = auditory verbal learning test; BVRT = Benton visual retention test; COWA = controlled oral word association; IRSPC = Iowa rating scale of personality change; OCD = obsessive-compulsive disorder; TMT = trail making test; TOH = tower of hanoi task; WAIS-R = Wechsler adult intelligence scale—revised; WCST = Wisconsin card sorting test

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Introduction

Collecting, the tendency to acquire and retain objects, even when they are not of immediate utility, is commonly seen among both children and adults, in modern as well as primitive societies. Behaviours comparable to those of human collecting have also been described in non-human species and are not limited to food. Some birds (e.g. American Crow, Northern Raven) are known to accumulate aluminium foil and brightly coloured objects; hamsters prefer hoarding glass beads over standard chow or sucrose (Hammer, 1972); food hoarding (also called ‘caching’ or ‘storing’) occurs in at least 12 families of birds, 21 families of mammals and many insects (Smith and Reichman, 1984; Sherry, 1985). The adaptive value of certain forms of collecting behaviour is evident,

e.g. storing food in anticipation of times of scarcity. Anticipated need, however, hardly accounts for most instances of collecting by humans. Collections of art objects, antiques, books and coins can be found in homes throughout the world, no category of objects being without its ardent collectors, and there are conferences and organizations for collectors of items ranging from postage stamps to farm tractors. It appears likely that most humans acquire a collection of some sort at some point in their lives. Pursuing such collections often can be justified on the basis of their aesthetic/emotional value, or monetary value, or both. In some instances, however, collecting behaviour may deviate from the reasonable and acceptable pattern and be directed toward objects that

are not only unnecessary for daily life, but are deprived of discernible aesthetic/emotional and monetary value, not to mention downright useless and inconvenient.

Despite the ubiquity of collecting behaviour, little is known regarding its neurobiological mechanisms. In non-human species, damage to structures such as the ventral tegmental area, lateral hypothalamus, nucleus accumbens, hippocampus, amygdala and thalamus reduces collecting behaviours (e.g. Herberg and Blundell, 1967; Kalsbeek *et al.*, 1988; Stern and Passingham, 1994), suggesting that collecting is supported by subcortical systems involved in biological regulation. In humans, however, it is apparent that the 'collecting drives' are modulated by cognitive processes that take social and other environmental factors into account and require the agency of other neural systems.

Patients with obsessive-compulsive disorder (OCD), autism, schizophrenia, anorexia, Tourette's syndrome and various types of dementia can exhibit abnormal collecting behaviour, but there has been little investigation of the neural correlates of collecting in these disorders. A recent study comparing resting cerebral metabolism ([¹⁸F]fluorodeoxyglucose-PET) of OCD patients with compulsive hoarding with that of OCD patients without hoarding behaviour and normal comparison subjects found that the OCD hoarders had lower glucose metabolism in the posterior cingulate gyrus and cuneus (Saxena *et al.*, 2004). Across all OCD patients, hoarding severity was negatively correlated with glucose metabolism in the dorsal anterior cingulate gyrus. Other suggestions as to possible neural correlates of abnormal collecting are available from reports of patients with focal cerebral lesions. The famous patient Phineas Gage, who sustained damage to mesial prefrontal regions as a result of a tamping bar accident (Damasio *et al.*, 1994), developed a 'great fondness' for animals and souvenirs (Harlow, 1868), and it has been noted that modern Gage-like patients with mesial prefrontal damage exhibit a collecting tendency not present prior to the onset of brain damage (Damasio, 1994). Recently, a small number of case reports have further suggested a link between damage to mesial prefrontal sectors and abnormal collecting behaviour (Anderson *et al.*, 1999; Cohen *et al.*, 1999; Hahm *et al.*, 2001; Volle *et al.*, 2002). To date, however, the neural correlates of these abnormalities have not been investigated systematically.

The purpose of the present study was to investigate the occurrence of abnormal collecting behaviour resulting from focal brain damage, with the goal of identifying a preliminary system-level neuroanatomical correlate of this condition. We hypothesized that abnormal collecting behaviour, characterized by increased, indiscriminate acquisition behaviour and diminished discarding behaviour, would occur following damage to prefrontal regions, but not after damage elsewhere in the brain. We tested this hypothesis in a group of patients with and without involvement of the frontal lobe, the specific prediction being that damage to mesial prefrontal regions (including the anterior cingulate cortex) would be associated with abnormal collecting behaviour.

Subjects and methods

Subjects

The participants in the main study were 63 subjects with focal brain lesions acquired in adulthood and a close relative of each subject. An additional 24 subjects with onset of brain damage during development are considered separately. In the main study, all subjects had normal developmental histories, no history of psychiatric disease and the onset of brain damage after age 18 years. Subjects with a history of neurological disease other than that which caused their focal lesion were not included. No subject had abnormal collecting behaviour prior to onset of brain damage, as ascertained by family members. All subjects were studied only after they became medically stable and their lesions were chronic. The evaluations were carried out at least 2 years following their neurological event. The aetiologies of the lesions included cerebrovascular disease ($n = 48$), surgical resection for the treatment of a meningioma or seizure control ($n = 12$) and herpes simplex encephalitis ($n = 3$). Given the stability and chronicity of the lesions, it is the location of the lesions, rather than their aetiology, that is relevant to the study. All subjects provided informed consent according to the Declaration of Helsinki and the regulations of the Institutional Review Board of the University of Iowa College of Medicine.

Behavioural and cognitive analysis

Each participant and a close relative, usually the spouse, were interviewed to elicit descriptions of all changes in daily activities, social behaviour and personality following the onset of their neurological condition. The interviews included standardized questions that solicited information regarding any collections or unusual accumulations of objects prior to and following the patient's neurological event, and any observed changes in habits related to the acquisition or discarding of items following the onset of brain damage (Collecting Behaviour Questionnaire, Appendix 1).

Abnormal collecting behaviour was defined as the gathering and storing of objects to such an extent that significant interference with normal daily activities resulted from the accumulated clutter or from the act of collecting itself.

Five criteria were used to distinguish abnormal collecting from normal behaviour. (i) Extent: the extent of the accumulated items must be excessive relative to normal behaviour and the individual's circumstances. (ii) Content: the collected items must include objects that are of little or no value. (iii) Interference with normal daily functioning: collecting must interfere with normal daily functioning, either due to the accumulated mass of items or from the act of collecting itself. (iv) Onset following brain injury: the collecting behaviour must have begun after focal damage to the brain. (v) Resistance to change: the collecting behaviour must be present for at least 1 year after onset and persist despite attempts by others to curtail the collecting.

For each of the criteria, the responses of the subjects' relatives to the standardized collecting behaviour interview were

evaluated for evidence of abnormality (criteria 1–3) and time course (criteria 4 and 5). All analyses were based on the descriptions of collecting behaviour provided by the subjects' relatives. The questions elicit descriptions of the presence of any collection—'collection' is defined as the accumulation of any type of object in a greater number than is considered reasonable by peers and is thus noted as 'unusual' or 'remarkable'—as well as the extent and content of the collections. For all subjects who met the criteria of persistent collecting behaviour with onset following their neurological event, the degree of abnormality in the extent and content of the collections was evident from the relatives' descriptions, as was the presence of interference with normal activities (see Appendix 2 for these descriptions). Raters blind to subject identification achieved 100% agreement on judging abnormality on these criteria.

All subjects completed a battery of standardized neuropsychological tests designed to test intellectual abilities [Wechsler Adult Intelligence Scale—Revised (WAIS-R)], memory [Auditory Verbal Learning Test (AVLT) and Benton Visual Retention Test (BVRT)] and executive functions [Wisconsin Card Sorting Test (WCST), Controlled Oral Word Association (COWA) and Trail Making Test (TMT)]. Subjects found to have abnormal collecting behaviour were evaluated further with regard to their planning and organizational abilities. These subjects were administered the Tower of Hanoi task (TOH) as an index of short-term planning ability, and a relative of each subject completed the Iowa Rating Scale of Personality Change (IRSPC) (Barrash *et al.*, 2000). Using this instrument, the daily behaviour of the subjects with pathological collecting was rated on the following dimensions: organization, planning, obsessive tendencies and impulsivity.

Neuroanatomical analysis

The neuroanatomical analysis was based on magnetic resonance (MR) data obtained in a 1.5 Tesla scanner with an SPg sequence of thin (1.5 mm) and contiguous T1-weighted coronal cuts, and reconstructed in three dimensions using Brainvox (Damasio and Frank, 1992; Frank *et al.*, 1997). In a few subjects for whom MR data could not be obtained, the analysis was based on computed axial tomography data. All neuroimaging was obtained at the time of evaluation. Using MAP-3, lesions were transposed and manually warped into a normal 3-D reconstructed brain, so as to permit the determination of the maximal overlap of lesions (voxel by voxel) in subjects with and without collecting behaviour. The method is described in detail by Damasio and colleagues (Damasio, 2000; Damasio *et al.*, 2004). In short, it entails the following: (i) a normal 'template brain' is reconstructed in three dimensions from thin contiguous MR slices; (ii) all major sulci are identified and colour-coded in the template brain; (iii) the lesion boundary is identified in the lesioned brain and the major sulci are identified and colour-coded; (iv) the template brain is resliced to match each of the lesioned brains; (v) the lesion boundary identified in (iii) is manually transferred onto the template

brain for each of the slices in which it is seen, taking into account the relation of this boundary to the identified sulci; (vi) the collection of transferred traces, the regions of interest, defines a volume that can be co-rendered with the template brain; (vii) the volumes of several lesions so transferred intersect in space and create a complex volume that can also be co-rendered with the template brain; and (viii) the overlap of the lesions in this volume is calculated by the sum of lesions overlapping on any single voxel and is colour-coded. Separate maps were created for 'collector' and 'non-collector' groups. A final difference overlap map was created by subtracting the MAP-3 overlap volume for the 'non-collectors' from that obtained for the 'collectors', again on a voxel by voxel basis. The volume so created shows the regions in which there is an excess of lesions in the 'collector' group. The areas considered to be related to abnormal collecting behaviour are those in which there are at least five subjects of the 'collector' group above the number of 'non-collectors', on a voxel by voxel basis.

Results

The subjects were classified as 'collectors' or 'non-collectors' on the basis of the behavioural criteria defined in Subjects and methods, independently of neuroanatomical factors. The behaviour profiles of the subjects fell into two distinct groups. Of the 63 subjects with onset of brain damage in adulthood, nine subjects ('collectors') met all five criteria for abnormal collecting behaviour, i.e. excessive extent of collecting, collection of useless items, collecting interfering with normal activities, onset following brain damage and resistance to change. Fifty-four subjects ('non-collectors') had no abnormalities of collecting behaviour, i.e. these subjects did not meet any of the criteria. Fourteen of these 54 subjects had a collection of some sort; all of these collections featured specific valued objects (e.g. coins, model railroads, figurines), and were acquired prior to the onset of the subjects' neurological disease. The 'non-collectors' showed either diminished (54%) or unchanged collecting behaviour following onset of their neurological disease. The two groups did not differ in age or education (Table 1).

The behavioural findings from the nine 'collectors' are depicted in Tables 2 and 3, and individual behavioural profiles

Table 1 Subjects

	Collectors	Non-collectors
Number	9	54
Men/women	6/3	31/23
Aetiology		
Vascular event	3	45
Neurosurgery	4	8
HSE	2	1
Age, years (range)	59.9 (41–76)	55.2 (25–80)
Education, years (range)	13.4 (8–16)	12.9 (8–20)

HSE = herpes simplex encephalitis.

Table 2 Characteristics of collecting behaviour

	Percentage of subjects with each characteristic	
	Collectors	Non-collectors
Possess any collection	100	26*
Increased acquisition behaviour	100	0
Decreased acquisition behaviour	0	57
Decreased discarding behaviour	89	39
Collecting of food items	56	0
Collecting of useless items	100	0
Selective collecting preferences [†]	23	100
Legal problems from collecting [‡]	33	0

Percentage of subjects in each group ('collectors', $n = 9$; 'non-collectors', $n = 54$) with each characteristic, as reported by their family. *All of the collections of the subjects in the 'non-collectors' group were accumulated prior to the onset of their neurological event. [†]Selective collecting preference was defined as a clear tendency to seek out a specific category of items for collection. Only those subjects in the 'non-collector' group who possessed some collection were considered for specificity of the collected items. [‡]Legal problems secondary to collecting behaviour included citation for fire safety hazard, bankruptcy secondary to uncontrolled purchasing and theft.

Table 3 Examples of collected items

Collected by three or more subjects
Newspapers
Magazines
Junk mail/catalogues
Appliances/components of appliances
Food
Clothing
Collected by at least two subjects
Broken furniture/furniture parts
Televisions/home entertainment items
Scrap metal
Car parts
Tools
Hardware
Grocery bags
Food containers/empty bottles
Cardboard boxes
Matchbooks
Pens/office supplies

Note: this is a partial list, in that all of the collections contained items from a broad range of object categories and were not systematically organized.

are provided in Appendix 2. In all cases, the abnormality of collecting behaviour was severe and persisted despite attempted interventions by family members and obvious negative consequences. All nine 'collectors' showed increased and generally indiscriminate acquisition behaviour, and eight out of nine also showed diminished discarding

Table 4 Neuropsychological findings: 'collectors' and 'non-collectors'

	Collectors	Non-collectors	P
WAIS-R			
VIQ	101.3 (19.2)	98.2 (14.8)	NS
PIQ	105.5 (16.2)	96.4 (16.5)	NS
WCST			
Categories achieved	4.8 (1.9)	4.5 (2.1)	NS
Perseverative errors	16.8 (11.9)	24.9 (19.9)	NS
COWA	34.6 (15.1)	30.4 (13.2)	NS
TMT			
Part A (s)	38.6 (17.4)	54.1 (33.6)	NS
Part B (s)	106.1 (60.0)	129.4 (84.0)	NS
BVRT			
No. correct	5.8 (2.2)	6.3 (1.8)	NS
No. errors	6.9 (4.3)	5.9 (3.4)	NS
AVLT			
Trial 5	8.2 (4.3)	10.0 (2.8)	NS
30 min recall	5.1 (4.9)	7.4 (3.9)	NS

NS = not significant. VIQ = Verbal Intelligence Quotient. PIQ = Performance Intelligence Quotient.

Table 5 Individual neuropsychological profiles of 'collectors'

Test	Subject								
	1	2	3	4	5	6	7	8	9
WAIS-R									
VIQ	42	25	99	10	14	63	25	94	73
PIQ	25	82	99	25	21	66	77	90	37
WCST									
Categories	10	11	20	20	–	20	20	20	20
Pers. errors	34	23	79	42	–	25	90	98	50
COWA	34	96	96	22	15	29	11	99	96
TMT									
Part A	6	73	14	67	–	53	63	53	16
Part B	9	37	37	32	–	70	37	63	53
BVRT									
No. correct	21	50	81	50	2*	6	50	87	19
No. errors	34	30	90	41	19	5	8	84	14
AVLT									
Trial 5	1*	50	86	30	8	16	1*	99	73
30 min recall	1*	63	50	20	1*	1*	1*	99	50

Scores are presented as percentile scores. Each subject's scores are compared with normative data individually matched on age and education. Impairment is defined as a score at least 2 SD below the mean, and is indicated by an asterisk. Pers = Perseveration.

behaviour. Abnormal food acquisition behaviours were also evident in five of the nine 'collectors' (56%).

The neuropsychological test performances of the two groups are presented in Table 4, and individual scores of the 'collectors' are presented relative to age and education matched normative data in Table 5. Both groups had mean verbal and non-verbal intelligence quotients in the average range. There were no significant differences between groups on any of the tests of executive functions or anterograde memory. Although the differences were not statistically significant,

Table 6 TOH test performances

Subject	Trial 1	Trial 2	Trial 3	Trial 4
3	71	95	33	43
4	120*	120	120	120
7	94	78	60	41
8	39	120	95	51
9	120	120	115	44
D1	120	120	120	120
D2	120	60	120	120
D3	79	65	92	94
D4	120	120	120	120

*Maximum number of moves allowed.

Table 7 IRSPC test

Subject	Disorganization	Planning	Obsessiveness	Impulsivity
NPF	3.2–0.3	3.6–0.4	3.7–0.4	2.8–0.2
1	7–3	7–3	0.2–0	3–0
2	5–3	7–3	1–0	7–3
3	6–3	4–1	2–1	3–0
4	3–1	4–2	3–0	5–3
6	6–1	7–3	3–0	7–2
7	1–0	5–2	3–1	5–2
D1	5	5	2	7
D2	7	7	1	7
D3	3	7	1	7

NPF scores are from a comparison group of patients with focal lesions in non-prefrontal regions, from Barrash *et al.* (2000). Scores are presented as 'level-change' for the adult-onset subjects. Scores for the developmental (D) subjects are 'level' scores. Level is rated on a 7-point scale, with 3 indicating an average amount of that characteristic, 5 indicating a problematic level, and 7 indicating severe disturbance. Change is rated on a 3-point scale, with 0 indicating no change, and 3 indicating severe change.

the mean scores of the 'collectors' were superior to those of the 'non-collectors' on all measures of executive functions (number of categories achieved and number of perseverative errors on the WCST, number of words generated on COWA and time to complete TMT Part B). All 'collectors' performed within 2 standard deviations (SD) of the mean on the measures of executive function (one 'collector' was not administered the WCST or TMT). There was a non-significant trend in which the 'non-collectors' obtained better mean scores than the 'collectors' on all measures of memory (BVRT number correct and number of errors, and AVLT Trial 5 and 30 min delayed recall). Four 'collectors' had significant memory impairments (i.e. memory test scores at least 2 SD below the mean for normal subjects), and five performed within normal limits on all of the memory tests. No subject in either group demonstrated abnormal grasp reflex or utilization behaviour. As can be seen in Table 6, five of the nine 'collectors' who completed the TOH had severe impairments on this task, suggesting a significant impairment of planning. On the IRSPC (Table 7) the 'collectors' also were rated as having

acquired deficits in planning, as well as acquired difficulty with organizational abilities and impulse control. In contrast, they were rated as not having significant obsessive tendencies.

Findings from the neuroimaging analysis of the 'collectors' and 'non-collectors' are shown in Fig. 1.

Overlap of lesions in nine 'collectors'

All subjects with collecting behaviour had lesions involving the prefrontal cortex. The area of greatest overlap was in the mesial and inferior prefrontal region bilaterally, slightly favouring the right hemisphere, where it extended anteriorly to involve the frontal pole. There was no evidence in any of these subjects of damage to the subcortical structures associated with acquisition behaviour in rodents, including the ventral tegmental area, lateral hypothalamus and thalamus. Two subjects (two of the three in which the lesion had been caused by herpes simplex encephalitis) had bitemporal damage in addition to the prefrontal damage; their lesions encompassed the nucleus accumbens and the amygdala. In two other subjects, we could not exclude nucleus accumbens damage for certain.

Overlap of lesions in 54 'non-collectors'

These subjects had lesions distributed throughout the left and right cerebral hemispheres. Two of the 'non-collectors' had damage to the nucleus accumbens, and in two others in this group we could not determine with certainty whether the accumbens was damaged. It is of interest that the third herpes simplex encephalitis case was in the group of 'non-collectors', and that his lesion did not involve any portion of the prefrontal cortex. The prefrontal lesion overlaps of the 'non-collectors' were more laterally and posteriorly located than those of the 'collectors'.

Difference overlap map (subtraction of lesions: 'collectors'—'non-collectors')

The area of maximal difference overlap between the two groups was in the right mesial prefrontal sector. More specifically, there is a large cluster of maximal difference overlap (five or more subjects) in the right inferior polar region of the prefrontal cortices, extending posteriorly to the level of the anterior cingulate, in the region immediately anterior to the genu of the corpus callosum. The few voxels of five or more residual overlaps seen in the left hemisphere are not considered to be meaningful given that they are not part of a larger cluster.

Developmental onset of abnormal collecting behaviour

In addition to the subjects reported above with adult-onset brain lesions, we studied four subjects with brain damage acquired prior to age 18 years who developed abnormal

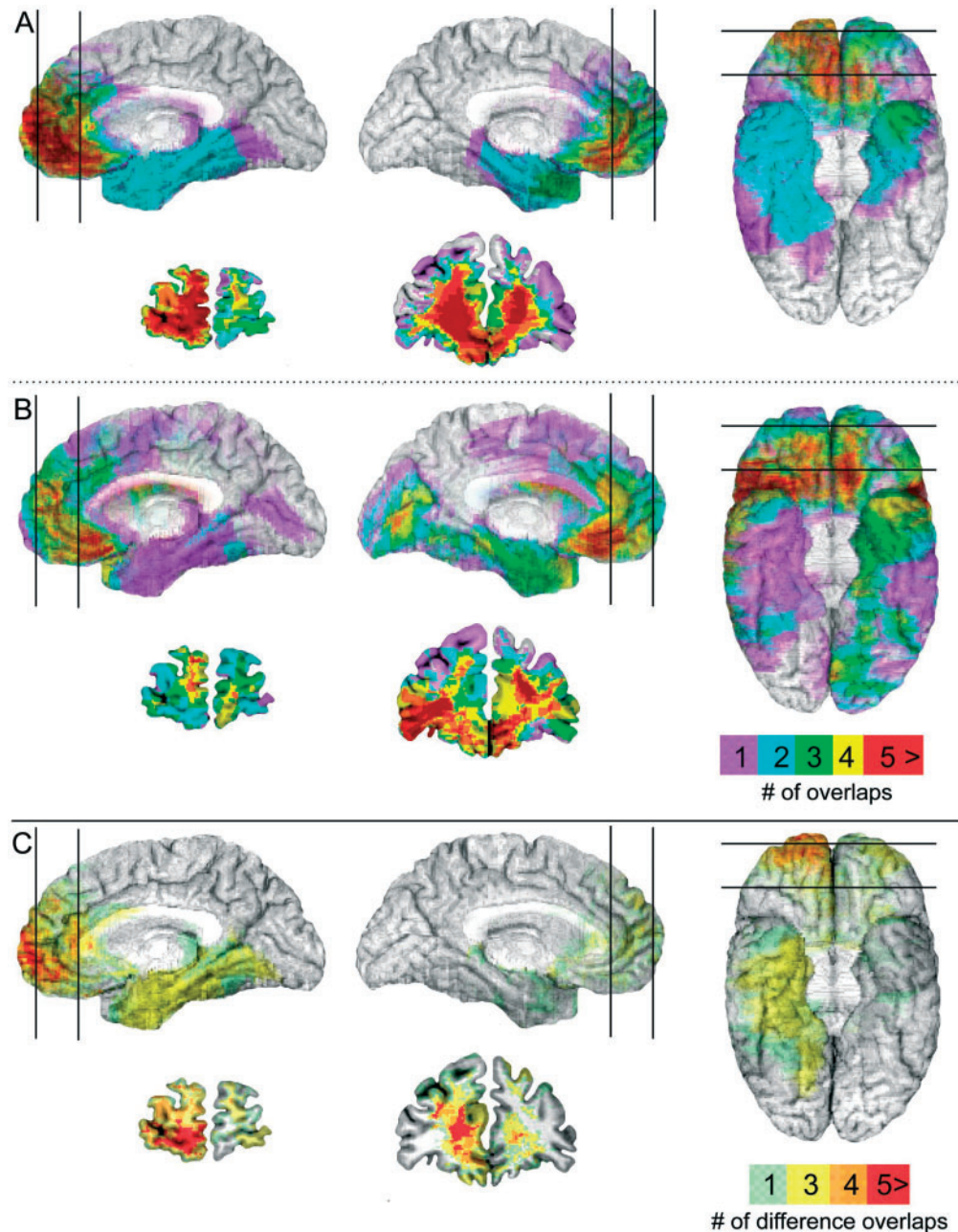


Fig. 1 Overlap of lesions for 'collectors' (A) and 'non-collectors' (B). The colour bar indicates the number of overlapping cases on a voxel by voxel basis. (C) The difference overlap map of volume A minus volume B. The colour bar indicates the number of overlapping cases. Red indicates that in those areas there is an excess of at least five lesions of 'collectors' over 'non-collectors'. The mesial views of the two hemispheres and the basal view of the brain are depicted along with two coronal slices. The vertical and horizontal lines in the mesial and basal views correspond to the placement of the two coronal slices.

collecting behaviour. In all four cases, the disturbed collecting behaviour was pronounced and followed damage to prefrontal cortex. In two cases the damage was largely unilateral, on the right; in two cases it was bilateral. For three of these cases, the prefrontal lesion extended into the anterior cingulate, and one of these also had damage to the nucleus accumbens. Behavioural profiles are presented in Appendix 2. Other than for the earlier age of onset, there were no discernible differences between the collecting behaviour of these subjects and

those with adult-onset lesions. All four early-onset cases had prolific and indiscriminate acquisition behaviour and a near absence of discarding behaviour. To address the question of whether early-onset lesions to other brain areas might also cause abnormal collecting behaviour, we examined the behavioural profiles of 20 additional subjects with focal brain lesions acquired prior to age 18 years and involving varied non-frontal areas. None of these control subjects had abnormal collecting behaviour.

Discussion

These findings support the hypothesis that abnormal collecting behaviour can result from damage to mesial prefrontal regions. The right mesial prefrontal region, at the level of the anterior cingulate and the frontal pole, appears to be most specifically involved. The collecting behaviour of these subjects was blatant, involved repetitive and generally non-selective acquisition behaviour, and a disinclination to discard objects. The behaviours persisted even when the 'collections' led to significant negative consequences.

There was an element of environmental dependency in much of the collecting behaviour, in that objects that were immediately present or easily obtainable were the most likely to be collected. However, the patients showed no inclination to use the objects in the stimulus-bound manner that has been termed 'utilization behaviour', of either the induced (Lhermitte *et al.*, 1986) or incidental (Shallice *et al.*, 1989) varieties. Once objects were in possession, they were ignored. There was no attempt to organize the collections in such a way that an aesthetic pattern might be discerned, and no aesthetic intention was ever voiced. The collections had no conceivable monetary or practical value either. Only when attempts were made by others to reduce or remove the collections did the subjects show some interest in the collected items. The act of collecting was an end in itself, not unlike the behaviour seen in normal rats, whose hoarding is not influenced by depletion of their caches, as would be expected if the hoarding were directed at a particular goal. Morgan (1947) concluded that, 'The rat hoards just to hoard, and it does not care whether it keeps what it hoards or whether it achieves any particular goal. The amount of hoarding seems to depend on an 'urge' to hoard rather than on how much hoarding has actually been accomplished.'

The abnormal collecting was not a consequence of a generalized mental defect, in that the subjects had well-preserved cognitive abilities (e.g. good performances on standard intelligence quotient tests and the WCST). They did, however, have impairments of planning and organization that in all likelihood contributed to the behaviour. Although our subjects with abnormal collecting behaviour were rated as low on obsessive characteristics, there are still interesting parallels between these subjects and OCD patients with compulsive hoarding. It has become evident that hoarding symptoms in the broader population are part of a discrete clinical syndrome that involves symptoms reminiscent of those associated with prefrontal damage, including difficulties with decision-making and organization (Frost *et al.*, 1996; Saxena *et al.*, 2004).

There was some indication that the pattern of collecting behaviour exhibited by two of the subjects may have been influenced by damage to temporal lobe structures in addition to the frontal lobe lesions. These subjects had combined bilateral lesions of posterior prefrontal cortex (including anterior cingulate), the nucleus accumbens and the mesial temporal lobe (involving the hippocampus and amygdala). In a predictable association with temporal lobe damage, the subjects

had marked impairments of learning and memory. In the context of their amnesia, these subjects had collecting profiles that differed slightly from those of the other collectors, in that there was a more compulsive and stimulus-bound elicitation of the habit. For example, they routinely took pens and similar items from our exam rooms, not in order to use them immediately, as seen in utilization behaviour, but simply to keep. One of these subjects performed within normal limits on the measures of executive function (the other did not complete the WCST or TMT due to his amnesia), suggesting this particular deficit is not simply a result of greater executive dysfunction. A third amnesic subject with hippocampal and amygdala damage, but without damage to either the prefrontal cortex or the nucleus, did not exhibit collecting behaviour, and two other 'collectors' who had amnesia due to basal forebrain damage did not have this type of stimulus-bound collecting behaviour. Thus this pattern does not seem to be simply a function of amnesia. It is possible that combined damage to prefrontal and mesial temporal cortices results in greater disruption of collecting behaviour. The significance of the lesion to the nucleus accumbens is uncertain. Although some of our subjects with prefrontal damage and abnormal collecting behaviour had damage to the nucleus accumbens, damage to this structure did not distinguish 'collectors' from 'non-collectors', because such damage was also present in subjects without abnormal collecting behaviour.

A provisional account of collecting behaviour in humans

The results of this study can be considered in the context of other relevant findings to sketch a provisional account of the broader phenomenon of non-pathological collecting behaviour in humans. This account is hypothetical and will require empirical testing and refinement.

We begin with the idea that a behavioural predisposition to acquire caches of food and other potentially useful objects (e.g. tool making supplies) was possibly selected for in evolution because it increased the probability of survival of the individuals who accumulated such caches prior to times of scarcity. The neural foundations of this predisposition are likely to include subcortical and cortical mesolimbic structures involved in homeostatic regulation, as suggested by stimulation and lesion studies in rodents (e.g. Herberg and Blundell, 1967; Kalsbeek *et al.*, 1988; Stern and Passingham, 1994).

With the possible exception of food items, it is unlikely that the targets of acquisition behaviour (i.e. the objects to be collected) are specified at a genomic level. Rather, the individual learning histories and object availability appear to be critical determinants of the objects to be collected. In persons without brain damage, the drive to collect would be initiated from limbic subcortical and mesolimbic cortical structures, but would be modulated by a prefrontal neural system involving anterior and mesial sectors, especially on the right. This

modulating system would direct the drive toward objects that, in an individual's learning history, were associated with significant reward and not with punishing consequences, while also accommodating the expression of the drive to social requirements.

The question of *how* the prefrontal cortex contributes to the modulation of the drive to collect remains open. In all likelihood, multiple interacting mechanisms are involved. The frontal lobes have long been linked to self-regulatory behaviour, and a number of recent functional imaging studies have related self-referential mental activity to medial prefrontal regions (e.g. Gusnard *et al.*, 2001; Kelley *et al.*, 2002; Fossati *et al.*, 2003). This region has been related to top-down control over the processing involved in mentalizing the self relative to others and projecting the self into the future (e.g. Frith and Frith, 2003). This is relevant because our patients had poor self-awareness of their acquired neuropsychological deficits, including the failure to regulate their collecting behaviour.

Both collecting behaviour and its converse, discarding behaviour, are likely to be influenced by anticipated future needs and the potential consequences of acquiring, keeping or discarding items. Damage to prefrontal cortex has been associated with an inability to organize and carry out goal-directed behaviour, particularly in situations with few external constraints (Shallice and Burgess, 1991; Burgess, 2000), and this may contribute to the failure in normal discard behaviour. Also, patients with mesial frontal lobe damage typically have impairments of decision-making, planning and anticipating the future consequences of their behaviour (Bechara *et al.*, 1994; Damasio, 1994, 1996).

It is conceivable that the modulation of the drive to collect would be assisted in part by a weighting system, whereby the neural representation of a stimulus item would be associated with a particular signal value, which would serve as an index of the relative worth of the stimulus. This weighting system would influence the cognitive process of deciding which items would be marked as valuable and thus sought after and retained, and which items with lesser value would be passed over or discarded, along the lines suggested by the somatic marker hypothesis (Damasio, 1994, 1996). The lesions associated with collecting behaviour in our subjects were in the right mesial prefrontal region, a key area in the system proposed by the somatic marker hypothesis, but we are not suggesting that this is either the principal or singular explanation for either normal or pathological collecting behaviour.

Whatever the mechanisms, it is clear that when the normal system is disrupted by damage to mesial prefrontal regions, the drive to collect food and other objects operates without its usual acquired cognitive constraints. The result appears to be a disinhibition of the drive, i.e. the abnormal 'collector's behaviour' observed in our patients, a disinhibited hoarding drive running relatively free. Further study will be needed to address the likely interactions among the various neuropsychological mechanisms discussed above.

In summary, our findings from patients with focal brain damage and abnormal collecting behaviour suggest that activity in mesial prefrontal structures is necessary for regulating the tendency to collect that primarily originates from subcortical bioregulatory nuclei. In the absence of brain injury, this activity guides the reasonable, context-appropriate acquisition of food and other items. The normal operation of this multitiered system probably underlies the ubiquitous tendency of humans to create socially acceptable collections.

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Appendix 1. Collecting behaviour questionnaire

Instructions: For the purposes of this interview, the term ‘collect’ is broadly defined as any accumulation of any type of object in a greater number than is usual (i.e. typical for most people). It does not matter if the ‘collections’ are organized or not.

1. Did he/she have any collections prior to the (*neurological event*)? Yes/No

If yes, describe:

Content:

Extent:

2. Does he/she currently have any collections? Yes/No

If yes, what does he/she currently collect?

Content:

Extent:

3. Has there been any change in his/her tendency to collect objects since the (*neurological event*)?

If yes, does he/she now collect:

(a) more? a little more or a lot more?

(b) less? a little less or a lot less?

4. Does he/she tend to accumulate useless items more than most people? Yes/No

If yes, describe extent and content:

Has this changed since the (*neurological event*)? Yes/No

If yes, does he/she accumulate:

(a) more? A little more or a lot more?

(b) less? A little less or a lot less?

5. Does he/she have any difficulty throwing away things that are not needed? Yes/No

Has this changed since the (*neurological event*)? Yes/

No

If yes, how much has it changed?

(a) a little

(b) a lot

6. Does he/she acquire or store away food in any manner that is unusual? Yes/No

Appendix 2. Abnormal collecting behaviour profiles

Subject 1

A right-handed female homemaker with 12 years of education underwent resection of an olfactory groove meningioma at age 69 years, and subsequently developed severe collecting behaviour. She completely filled a two-car garage with assorted items, primarily broken or otherwise useless items salvaged from others' discard piles (e.g. furniture, appliances, clothing, pet supplies, lawn ornaments). She did not exercise any apparent selection criteria. She made no attempt to use, repair or organize the possessions. Her closets and drawers were overflowing, and more clothing (most of which no longer fit her) was stacked throughout the house. She refused to discard any possessions or allow others to do so, and was very resistant to attempts to manage her collecting behaviour. Her eating behaviour was unchanged.

Subject 2

A right-handed man with 12 years of education underwent clipping of a ruptured anterior communicating artery aneurysm at age 27 years, and subsequently became, in his wife's terms, 'a packrat'. He began collecting assorted tools and materials such as scrap metal and wire, much of which he salvaged from neighbours' garbage. His basement and a garage became filled with acquired items that he did not use. Despite financial difficulties, he engaged in frequent impulsive buying, in which he would purchase unneeded (and often expensive) items that attracted his attention while shopping for something entirely different. He accumulated multiple identical or near identical versions of many tools. Once purchased, he entirely lost interest in the objects, often not even bothering to take them out of the shopping bags. Some items sat in their garage essentially untouched for over two decades, but he refused to consider discarding or selling any of his possessions. He was no longer able to find his tools or other needed items because of the volume and disarray of collected items. His collecting behaviour remained consistent over 35 years following the neurological event. His eating behaviour was not altered.

Subject 3

A right-handed college-educated businessman underwent resection of a large orbitofrontal meningioma at age 31 years. He began to accumulate a vast and diverse collection of items. His home became filled with stacks of old papers and magazines that reached to near the ceiling. He saved every piece of junk mail he received, often writing the date on each piece. The tables, chairs and other furniture in his home were piled with papers and various items. His kitchen counters were covered by multiple toasters, blenders, popcorn poppers and other appliances (e.g. three deep fat fryers), many of which were non-functional. Components from unfinished projects (e.g. automobile parts, boards and tools) and multiple unused televisions, fans, humidifiers and other appliances were scattered throughout the home. To get to his bed, it was necessary to pass through an 18-inch wide path between stacks of boxes and then crawl over another stack of boxes. Traversing most of his home was possible only via such paths. He would argue at length over every item when attempts were made to help him discard items, maintaining that every item was necessary. No significant changes were noted in his eating behaviour, but he generally would not discard food items when they became old. For example, he refused to allow his family to throw out mouldy leftover food from his refrigerator. His collecting behaviour was stable over more than a decade.

Subject 4

A right-handed female homemaker with a high-school education underwent resection of an olfactory groove meningioma at age 74 years. Following the event, her family noted that, 'she never throws anything away'. She began saving every piece of junk mail received. She accumulated stacks of emptied plastic and glass food containers, newspapers and empty boxes. She made many unneeded purchases from salesman, seemingly agreeing with most offers proposed. When in stores to shop for groceries and other necessities, she purchased many unneeded and often expensive items. Her family found it necessary to supervise all of her shopping to prevent purchase of unneeded items. Despite their efforts, her house became filled with useless objects. Her family hired a cleaning woman to help combat her collecting behaviour, but the patient was observed to retrieve old magazines, papers and empty bottles from the trash after the cleaning woman left. She was obese and tended to overeat prior to her surgery, but no significant changes were noted in her eating behaviour following surgery.

Subject 5

A 35-year-old, right-handed salesman with 13 years of education developed herpes simplex encephalitis, resulting in a severe amnesia. In addition, he began to display collecting behaviour. Despite living in a supervised care facility, the behaviour was evident on a daily basis. He consistently collected any small items that were immediately available.

His pockets were regularly filled to capacity with newly acquired items such as napkins, facial tissue, packets of coffee creamer and sugar, and pens. He repeatedly took items from the rooms of other residents; this likely was influenced by his amnesia, which was so severe as to impair recognition of his personal belongings. He did not appear to actively engage in any discard behaviour, but the demands for this were limited in the care facility. He sought out food, coffee and gum from caretakers at frequent intervals, and seemingly would consume as much as was available. His food consumption was controlled by his caretakers.

Subject 6

A 28-year-old, right-handed man with a college education developed herpes simplex encephalitis, which resulted in a severe amnesia. He was cared for by his parents. He developed a pervasive tendency to collect virtually any objects that he could fit in his pockets. He took table settings from restaurants and items from visited bathrooms. He would take his parents' possessions, including items of no value to him, such as their telephone messages and grocery lists. He routinely pocketed other people's pens and other small items left out. His parents always checked his pockets when leaving a host's home, and checked his room when their own possessions were missing. He showed little interest in the items once acquired, and engaged in relatively normal discarding behaviour, i.e. with limited prompting, he would clean his room and throw away much of the accumulated collection. He typically denied taking any items when confronted, but did not object to relinquishing them. He would buy large quantities of soda pop and gum, but generally would not purchase any other items. He developed a voracious appetite, and would eat and drink continuously if left to his own devices. His food consumption was controlled by his caretakers.

Subject 7

A right-handed male welder with 8 years of formal education underwent clipping of a ruptured anterior communicating artery aneurysm at age 47 years. He developed severe impairments of behaviour initiation and organization, rarely completing even routine tasks. Despite his generalized lack of goal-directed activity, he developed collecting behaviour that was highly repetitive and directed primarily toward a specific target, which was the scattered corn left lying in nearby fields after the harvest. He collected corn almost daily when conditions permitted, accumulating large piles of corn and continuing to collect as it rotted and attracted rodents (he used only a small amount to feed his chickens). He also began to bring home found scrap metal and discarded automobile and appliance parts, none of which he would ever touch again after bringing them home. He refused to discard virtually anything. There was no change in his eating behaviour.

Subject 8

A 70-year-old, right-handed, retired bank clerk with 13 years of education underwent resection of an orbitofrontal meningioma. Her husband noted that all of her life she had been reluctant to throw away items with potential value, but that this characteristic was not so prominent as to cause any problems. However, following surgery, she began to collect large quantities of a wide array of items, to the extent that serious space problems arose in their home. She began ordering large quantities of unneeded items, particularly clothes, from mail-order catalogues, most of which her husband would intercept and return. She refused to throw away newspapers, magazines, junk mail or grocery sacks, but rather created large stacks of such items around the house. All drawers, closets and cupboards in the house were filled to overflowing with assorted useless items. She began to write notes to herself and leave these around the house. She also began to clip and save articles from newspapers and magazines. Her food acquisition behaviour was largely controlled by her husband, because when left to her own devices she would purchase unneeded food in quantities which assured it would spoil. She refused to throw away old canned or boxed food goods even when they were several years old. She began eating as much as permitted, and she gained considerable weight.

Subject 9

A 33-year-old, right-handed, college-educated male counsellor underwent clipping of a ruptured anterior communicating artery aneurysm. He had a generally good recovery, but his behaviour became marked by lack of initiation and poor decision-making, and he developed collecting behaviour. Most of his acquisition behaviour took the form of purchasing unneeded items. For example, he purchased large quantities of clothing that he never wore and multiple home entertainment devices that went unused. He appeared easily influenced by advertising, and in response to television and mail advertising he acquired extensive sets of recorded music, movies and books. He quickly exhausted a large disability policy in this manner. He ran up large debts with uncontrolled purchasing, and was forced to declare bankruptcy. He engaged in very little discard behaviour, but he changed residences frequently and often left most of his possessions behind. He began eating large quantities of food with high sugar and fat content, and gained over 80 pounds.

Developmental onset of collecting behaviour
Developmental subject 1

A right-handed girl sustained focal trauma to the prefrontal region at age 15 months. From early childhood she showed a strong tendency to pick up and keep objects that did not belong to her. As a young girl, she took household decorations, her father's tools, silverware and food. She would accumulate the items in her room. As she got older, she

collected empty boxes (e.g. from bathroom products), gum wrappers, pens and pencils, old magazines and catalogues. She repeatedly stole personal effects, jewelry and clothing from family and friends. She virtually never threw anything away. Although her eating behaviour was not abnormal, she routinely would hide food items in her closets and drawers, where they would stay until the smell attracted the attention of her parents.

Developmental subject 2

Following a normal birth, a 3-month-old boy underwent resection of a malignancy in the right frontal region. Surgery was not followed by radiation or chemotherapy, but there has been no indication of recurrence. Developmental milestones were achieved at a normal rate, and he was able to eventually graduate from high school. However, his behaviour throughout life was marked by disrupted social behaviour and impaired decision-making. He also displayed abnormal collecting and eating behaviour from an early age. He collected quantities of useless objects such as food containers and empty matchbooks. His room would repeatedly fill with collected papers and assorted found items. As an adolescent and young adult, he repeatedly would purchase expensive and unneeded items from stores and catalogues. He acquired substantial credit card debt. He had a tendency to pick up items that caught his attention and he engaged in petty thievery. He engaged in virtually no discard behaviour or cleaning, with the result that his living space was severely cluttered and dirty. As a young child, he was observed to pick up and ingest discarded chewing gum. He would eat large quantities of seemingly any food item, such as condiments out of a jar and frozen fish sticks directly out of the freezer.

Developmental subject 3

A right-handed girl had a normal development until age 16 years, when she underwent clipping of an A-com artery aneurysm. Prior to this, she was a good student with no behavioural problems. In the years following the surgery, she developed collecting behaviour characterized by gradual accumulation of useless items. She acquired objects with little regard for their utility. She was constrained in purchasing by limited financial means, but would readily accept any offer of cast-away items and would retrieve items others had thrown away. She had several copies of many items, such as five manual and two electric can openers. Discarding of unneeded objects was severely impaired. She threw virtually nothing away, and did not do any significant cleaning. Her apartment was filled with stacks of boxes full of papers and assorted, unorganized and generally useless items. Large and useless items, such as several old mattresses and broken television sets, took up considerable space in her apartment. She did not appear to place any particular value in the objects she accumulated, and in fact she often gave away her possessions. She allowed her family to clean her apartment on occasion, but would immediately begin

the same pattern of collecting behaviour. There were no significant changes in her eating behaviour. She failed to throw away old food, just as she did with all other items.

Developmental subject 4

This subject was born with a cystic lesion in the right frontal lobe and hydrocephalus; the latter was successfully treated with a shunt, but a shunt revision at age 4 years resulted in an additional small area of damage in the left frontal lobe. His social and emotional behaviour was abnormal throughout his life, and he had a marked impairment of decision-making. Throughout childhood, he would pick up and keep items belonging to his parents or siblings. He repeatedly purchased

expensive and unneeded items from stores and catalogs. Many of the items he purchased were potentially useful or enjoyable (e.g. power tools, electronic games), but he showed no interest in them after bringing them home. Other collected items were related to his actual activities, but in quantities that were unnecessary (e.g. 10 bowling balls). He acquired substantial credit card debt and repeatedly stole money from his parents. He brought discarded items home from his workplace (e.g. a broken leaf-blower, old coffee maker). He took things belonging to others when it was obvious that he would be caught (e.g. using his truck to take a trailer from his church). He engaged in virtually no discard behaviour or cleaning, but allowed his mother to clean his living space so that clutter and dirt were kept under control.