

Eating Habits in Dementia A Descriptive Study

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The eating habits of 33 patients with dementia were studied. They were assessed by semi-structured interview with a relative who was able to act as an informant for the whole period of the dementia. Changes in eating were found to be common and included both increased and decreased food intake, altered food choice, the consumption of inedible substances and disturbances in the process of eating. The clinical significance of these changes and some likely underlying mechanisms are discussed.

The diagnosis of dementia requires demonstration of memory impairment and loss of intellectual abilities sufficient to interfere with social or occupational functioning, and associated with impaired thinking or judgement (American Psychiatric Association, 1987). In addition to these necessary diagnostic features, changes in behaviour are common including aggression, wandering, disturbed sleep and alterations in eating habits. These changes are of theoretical interest since they may be direct expressions of underlying neuropathological changes, and they are of practical importance since they may result in management problems (Fairburn & Hope, 1988*a*). Despite this, they have been the subject of little research.

Among the behavioural changes reported as occurring in dementia are alterations in eating habits (Pilleri, 1966; Sourander & Sjogren, 1970; de Ajiuriaguerra *et al*, 1976; Hope *et al*, 1989; Fairburn & Hope 1988*b*). A number of possible mechanisms have been proposed to account for changes; for example, they may be secondary to cognitive impairment or apraxia, or the result of insufficient care-giving, or the consequence of metabolic or neurochemical abnormalities occurring as part of the dementing process (de Ajiuriaguerra *et al*, 1976; Morley & Silver, 1988; Hope *et al*, 1989; Fairburn & Hope, 1988*b*). The changes in eating habits are clinically important since they may give rise to malnutrition, leading to increased infection rates and longer hospital admissions (Hancock *et al*, 1985; Sandman *et al*, 1987), and since they may present carers with the practical difficulty of how to feed the demented person adequately (Lo & Dornbrand, 1984). It has also been argued that poor nutrition may actually cause or exacerbate the deterioration of cognitive function (Munro, 1982; Goodwin *et al*, 1983; Abalan, 1984; Thomas *et al*, 1986).

Individual case reports have described some of the changes in eating habits that occur in dementia

(Pilleri, 1966; Cummings & Duchon, 1981; Hope *et al*, 1989) but there have been no descriptive studies of the eating habits of a defined population of subjects with dementia. The purpose of this study was to provide a systematic description, using a standard assessment procedure, of the range and nature of the changes in eating habits that occur in dementia.

Method

The principal assessment measure was a semi-structured interview with an informant who was able to give an account of the demented subject's behaviour. Subjects were selected on the basis of having a good informant. The subjects themselves were assessed cognitively and physically by a standard procedure.

Recruitment

The subjects were recruited from a sample of patients who had taken part in an earlier study of behaviour in dementia and were from the case-files of the six psychogeriatric community nurses who serve the Oxford area. The nurses were asked to identify all patients who had been diagnosed by a consultant psychogeriatrician as having multi-infarct dementia or Alzheimer's disease, and who were living at home with a relative able to act as informant for the whole period of the dementia. For the purpose of the study the informants were contacted by telephone and asked if they were willing to help with a further study. If they agreed, an appointment was made to interview them at home. The demented subject was seen at the same time or shortly afterwards.

Assessment of dementia

The diagnosis of dementia was confirmed by examination of psychiatric and medical records, interview with the informant (see below) and examination of the subject. A Mini-Mental State Examination (MMSE) (Folstein *et al*, 1975) was conducted as well as clinical assessment of the subject's mood and a physical examination. The Ischaemic Score (IS) of Hachinski *et al* (1975) was used to subdivide

into those with probable Alzheimer's disease ($IS < 4$), those with probable multi-infarct dementia ($IS > 7$) and those with dementia of mixed type ($IS 5-6$).

Assessment of eating habits

The informant was interviewed by semi-structured pre-coded interview designed for the present study. The interview was an elaboration of the 'eating' section of the sixth edition of the Present Behavioural Examination (PBE). The PBE covers the full range of behavioural changes in dementia and is based on the Eating Disorder Examination, which has been shown to have high reliability (Cooper & Fairburn, 1987). For each item there is a mandatory probe question, followed by one or more optional subsidiary questions. The behaviour to be rated is defined and a rating scheme with specified anchor points is provided. Preliminary work indicates that the reliability of the PBE is high.

The interview had three main sections concerning: (a) basic demographic data about the subject and informant, (b) the medical and psychiatric history of the subject, including the onset and course of the dementia, and (c) any changes in eating habits, weights and related types of behaviour since the onset of the dementia. The third section was the largest and consisted of 32 key items which, if scored positively, were followed by subsidiary items. The interview took between 45 minutes and two hours to administer and was conducted by the first author.

Results

The sample

Of 46 informants approached for possible inclusion in the study, nine refused. Of the 37 subjects, 34 were seen: one refused examination and two others were unavailable. One informant was unable to provide sufficient information for the purposes of the study. Thus 33 pairs of informants and subjects were seen. Of the subjects, 19 were female and 14 male, with an age range of 61-96 years (mean = 77.5 years, s.d. ± 7.7). Twenty-one informants were spouses, ten were children and two were nieces of the subjects. The informant was the main carer in every case.

Diagnoses

All 33 subjects fulfilled DSM-III-R criteria for dementia (American Psychiatric Association, 1987) and scored less than 24 on the MMSE. On the basis of the Ischaemic Score, 27 subjects were classed as having Alzheimer's disease (AD), four as having multi-infarct dementia (MID), and two as having dementia of mixed type. The MMSE scores of the subjects ranged from 0 to 23 (mean = 10, s.d. ± 7.0). Those with a clinical diagnosis of AD are considered first.

Changes in eating habits

The main findings are shown in Table I. All subjects showed changes in eating habits or weight, and many exhibited a number of different and possibly inter-related changes.

Reduced food intake

Two-thirds of the sample (63%) ate significantly less at some stage in their illness than they ate before the onset of dementia. Two subjects had experienced two separate episodes of diminished eating during the course of the dementia. Four subjects had eaten less at an early stage in the dementia at a time when they were living alone. This change in eating habits was one of the first indications of the illness in these cases. Their eating returned to normal when they were looked after by a carer. In the remaining 13 cases (48%) the diminished eating was not accounted for by lack of adequate care. For 13 subjects (48%) currently showing reduced food intake compared with their intake before the onset of dementia, the mean duration of the change was 1.6 years (mean duration of dementia = 5.7 years). Only two subjects had eaten less throughout the dementia. Of these 13 subjects, 10 had lost weight since eating less.

Increased food intake

A quarter of the subjects (26%) had eaten significantly more at some stage since the onset of the dementia than they had eaten previously, by eating both larger meals and eating more between meals. In many cases these subjects requested still more food, or showed evidence of searching for food. In three-quarters of these subjects the hyperphagia was so severe that the carers had had to restrict their access to food. Six of the seven subjects with increased food intake gained weight. One subject lost weight and this may have been accounted for by persistent overactivity.

Altered food choice

Ten subjects (37%) showed a change in their preference for sweet foods compared with their pre-morbid preference (eight showed an increase, two a decrease); four (15%) developed a marked increase in their liking for spicy or 'hot' foods; and eight (30%) exhibited an altered choice of non-alcoholic drinks (for example, a change from drinking coffee to drinking tea).

Altered style of eating

In 16 subjects (60%) their style of eating had altered since the onset of dementia. Eleven used utensils in an abnormal way or were unable to use them at all; nine used their hands to pick up food; and one tried to eat using a pair of scissors.

Oral behaviour

A quarter (26%) of the sample exhibited oral behaviour: four subjects examined or touched objects with their lips (a feature of the Kluver-Bucy syndrome (Terzian & Dalle Ore, 1955)); five tried to eat inedible substances (for example, faeces, Christmas decorations, paper-towels, soap, flowers, tea-bags); and five ate inappropriate foods (for example, an uncooked chicken leg, raw eggs, uncooked potatoes and onions, cat food, a whole jar of marmalade).

TABLE I
Reported changes in eating habits and weight in 33 subjects with dementia

Subject number	IS ¹	Age at onset: years	Duration: years	MMSE ²	Amount eaten		Altered choice of food	Oral behaviour	Exercise change	Weight change
					Increase	Decrease				
1	0	58	9	23	0	+	+	0	↑	↓
2	0	81	1	19	0	+	+	+	↓	↓
3	0	71	9	16	0	(+)	+	0	0	(↓)
4	0	78	6	14	+	0	+	0	↓	↑
5	1	67	5	18	0	+	+	0	↓	↓
6	1	70	2	12	(++)	0	0	0	0	(↑)
7	1	82	14	1	0	+	+	0	↓	(↑)
8	1	77	2	13	0	++	+	0	↓	↓
9	1	79	7	9	0	0	0	0	0	↓
10	1	76	6	10	0	+	+	0	↓	↓
11	1	90	4	19	0	0	+	0	0	0
12	2	73	4	8	+	0	+	+	0	(↓)↑
13	2	66	6	1	++	0	+	++	0	(↓)↑
14	2	73	10	5	+	(+)	+	0	0	(↓)↑
15	2	60	6	3	0	(++)	+	+	0	(↓)↑
16	2	62	10	0	0	+	0	+	↓	↓
17	3	74	3	10	0	(+)+	0	0	(↓)	(↓)↓
18	3	82	4	22	0	+	+	0	↓	↓
19	3	72	3	9	0	+	+	0	↓	↓
20	3	70	6	1	++	0	+	0	0	↑
21	3	54	11	0	+	0	+	+	↑	↓
22	3	77	5	5	0	(+)	0	0	0	(↓)
23	3	72	3	14	0	+	0	0	0	0
24	4	69	4	3	0	+	0	0	↓	↓
25	4	63	8	13	0	0	0	0	↓	↓
26	4	62	4	1	0	+	0	0	↓	0
27	4	77	3	8	0	0	0	+	0	0
28	6	57	4	19	0	0	+	+	0	↓
29	6	76	7	4	0	0	0	0	0	0
30	7	80	5	12	0	+	+	0	↓	↓
31	9	79	2	22	0	0	0	0	0	↓
32	10	75	3	15	0	+	+	0	↓	↓
33	10	72	6	3	+	(+)	0	0	↓	(↓)↑

1. Ischaemic Score (Hachinski *et al.*, 1975).

2. Mini-Mental State Examination total score (Folstein *et al.*, 1975).

0 no change; + moderate change; ++ severe change; () temporary change.

Weight loss

The carers reported a weight loss in over two-thirds of the subjects (70%) at some stage during the dementia. This occurred early in the dementia in seven subjects; in six of these cases the weight lost was regained. One subject went on to lose weight again. For 12 subjects, their weight at the time of the study was lower than their pre-morbid weight.

Weight gain

Six subjects (22%) had gained weight and were heavier at the time of the study than they had been before the onset of dementia. Two others had gained weight temporarily at an early stage.

Subjects with clinical diagnosis of multi-infarct dementia

Both an increase and a decrease in eating and in weight were observed in the four subjects with a clinical diagnosis of multi-infarct dementia. In addition, two of these subjects showed an alteration in food choice.

Relation of the changes in eating to other factors

Functional psychiatric illness

Decreased food intake with weight loss could be attributed to depressive illness in only one subject (subject 17). There were no cases of psychotic phenomena associated with changes in eating.

History of dieting

None of the informants reported that the subjects had dieted significantly in the past.

Physical illness

None of the changes reported here were associated with known or apparent physical illness. Short-term changes associated with brief episodes of physical illness were excluded.

Medication

Increased intake of food was related in time to the taking of medication in two cases (dexamethasone in subject 6 and thioridazine in subject 33). Subject 15 gained weight when first taking chlorpromazine. In no other case was a change in medication associated with a change in eating habits or weight.

Discussion

The aim of this study was to describe the changes in eating habits that occur in Alzheimer's disease and multi-infarct dementia. The subjects were chosen from a sample of those with dementia known to local community psychiatric nurses. All the subjects were living at home with a relative who could act as an informant. Because of these selection factors the sample is likely to be biased towards the physically healthy with dementia of moderate severity but not sufficiently severe to require institutionalisation. Behavioural problems are likely to be over-represented in the sample since they may have resulted in the subject's coming to the attention of the psycho-geriatric team. In no case was a change in eating habits the behavioural problem for which the subject was originally referred. All the subjects were diagnosed clinically as having either Alzheimer's disease or multi-infarct dementia, and the Ischaemic Score (Hachinski *et al*, 1975) was used to distinguish between these diagnoses. Since most of the subjects were diagnosed as having Alzheimer's disease, the results are given for this group separately. The number of those with a diagnosis of multi-infarct dementia was too small for useful conclusions to be drawn. However, both an increase and decrease in the amount eaten was observed in these subjects. In the absence of neuropathological data, definitive diagnosis was not possible.

The semi-structured interview used to assess the changes in eating was developed specifically for this study. It involved a detailed interview with an informant. All the informants had known the subject both before the onset of dementia and throughout the illness. Only clear and substantial reported changes in eating habits and weight have been reported.

The most common change in eating habits observed was a decrease in intake associated with a decrease in weight. Several alternative explanations for this change are discussed below. An increase in eating occurred in a quarter of the subjects and in many cases it was so marked that the carer had to restrict the subject's access to food. In all but one subject the increase was associated with weight gain. Thus there was no evidence that the hyperphagia occurred as a compensatory mechanism for weight loss or gastro-intestinal malabsorption, as suggested by Abalan (1984). Hyperphagia has been reported in dementia as part of the Kluver-Bucy syndrome (Sourander & Sjogren, 1970; Pilleri, 1966). However, in the present study only two subjects with hyperphagia examined objects with their lips, and otherwise there was no association between hyperphagia and the other features of the syndrome in this sample.

The marked changes in food choice observed are, as far as the authors are aware, a novel observation. The commonest change was an increased consumption of sweet foods (in 30% of subjects with Alzheimer's disease). Four subjects ate more spicy foods than they had previously eaten. Seven Alzheimer subjects had eaten substances which are not normally eaten (pica). Some of these substances were foods (but inappropriate either in quantity or because they were uncooked); some were disgusting and dangerous, for example, faeces; and some were neutral, for example, paper. Changes in the physical act of eating were also described. These took the form of using normal utensils abnormally; or using abnormal utensils; or using the hands inappropriately.

Various mechanisms could underlie these changes in eating habits. Five psychological mechanisms merit consideration.

First, self-neglect could account for the weight loss in four of the subjects with Alzheimer's disease. In these cases the weight loss occurred early in the dementing process and while the subjects were living alone. In these subjects the weight lost was regained when they moved in with a carer. This was quite different from the decrease in eating and weight loss observed in other subjects, which was a later feature of the disease and could not be accounted for by neglect.

Secondly, memory impairment might be thought to account both for the decrease in eating in some subjects (i.e. forgetting to eat) and for the increase in eating in others (i.e. forgetting that they had eaten). However, as we have argued elsewhere (Fairburn & Hope, 1988b; Hope *et al*, 1988), such explanations are unsatisfactory both because it seems unlikely that memory impairment would permanently

override sensations of hunger and satiety and because such changes should accompany other disorders associated with memory loss. To our knowledge this is not the case.

Thirdly, a failure to recognise objects might account both for the eating of inedible objects and for the use of odd utensils. With regard to the eating of inedible objects, it would be interesting to investigate experimentally what objects are *not* eaten in order to try to understand the classificatory system that the demented person is using.

Fourthly, Rozin & Fallon (1987) have investigated the acquisition of *disgust*, i.e. "the revulsion at the prospect of oral incorporation of offensive subjects". It is possible that underlying the eating of objects such as faeces (see Ghaziuddin & McDonald, 1985) there is a loss of *disgust* as distinct from a failure of recognition. It would be interesting to explore this possibility and to use the experimental paradigms that Rozin has developed for research with children.

Fifthly, the hyperphagia and changes in food choice could, in principle, develop because the subjects had been dieters in the past, and the dementia had interfered with their ability to diet. However, a history of dieting was specifically denied in all cases by the informants.

Five possible physical mechanisms also merit consideration.

First, the neurochemical or neuropathological changes that occur in dementia result in a disruption of the central control of food intake and weight. Serotonin and noradrenaline are known to be involved in the mechanisms controlling eating and food choice (Leibowitz, 1985; Blundell, 1984) and both are abnormal in AD (Rossor *et al*, 1981; Rossor *et al*, 1984; Crow *et al*, 1984; Whitford, 1986). Similarly, the hypothalamus and medial temporal lobe are severely affected in AD (Ishii, 1966; Herzog & Kemper, 1980; Tomlinson, 1982; Mann *et al*, 1985) and both are involved in the control of food intake (Rolls *et al*, 1979; Gold, 1973; Rabin, 1972). Substance P has been implicated in the recognition of spicy or chilli-containing foods (Morley & Silver, 1988) and a reduction in substance P has been reported in dementia (Davies *et al*, 1982). Thus, the changes in the amount eaten, in weight and in food choice, may be the result of damage to the central mechanisms that control eating.

Secondly, the changes in food choice could result from a change in the sense of taste and of smell. For example, the development of a liking for spicy foods might reflect a desire to taste foods in the presence of impaired taste and smell perception. This is an interesting speculation in the light of recent evidence

that olfactory deficits may occur early in dementia (Rezek, 1987).

Thirdly, malabsorption has been suggested as a cause of the weight loss that occurs in dementia (Abalan, 1984) and it might be expected to account for the increase in food intake. However, as noted earlier, the findings of the present study suggest that the changes in weight reflect a change in food intake rather than vice versa.

Fourthly, neurological deficits leading to dyspraxias probably account for some of the changes in the process of eating.

Fifthly, in three individuals the effects of medication could account for the changes in eating seen.

In conclusion, this study has shown that changes in eating habits are common in dementia and take various forms. They are important clinically because they can have a detrimental effect on health and also because they can pose a major problem for carers. The changes are likely to be the expression of a number of different underlying mechanisms. It will be important to understand the mechanisms in order to develop specific treatments.

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References

- ABALAN, F. (1984) Alzheimer's disease and malnutrition: a new aetiological hypothesis. *Medical Hypotheses*, **15**, 385-393.
- AMERICAN PSYCHIATRIC ASSOCIATION (1987) *Diagnostic and Statistical Manual of Mental Disorders* (3rd edn revised) (DSM-III-R). Washington DC: APA.
- BLUNDELL, J. E. (1984) Serotonin and appetite. *Neuropharmacology*, **23**, 1537-1551.
- COOPER, Z. & FAIRBURN, C. G. (1987) The Eating Disorder Examination: a semi-structured interview for the assessment of the specific psychopathology of eating disorders. *International Journal of Eating Disorders*, **61**, 1-8.
- CROW, T., CROSS, A., COOPER, S., *et al* (1984) New transmitter receptors and monoamine metabolites in the brains of patients with Alzheimer's type dementia and depression. *Neuropharmacology*, **23**, 1561-1569.
- CUMMINGS, J. L. & DUCHEN, L. W. (1981) Kluver-Bucy syndrome in Pick's disease: clinical and pathologic correlations. *Neurology (Nj)*, **31**, 1415-1422.
- DAVIES, P., KATZ, D. A. & CRYSTAL, H. A. (1982) Choline acetyltransferase, somatostatin and substance P in selected cases of Alzheimer's disease. In *Alzheimer's Disease* (ed. S. Corkin). New York: Raven Press.

- DE AJURIAGUERRA, J., RICHARD, J., TISSOT, R., *et al* (1976) Les conduites alimentaires dans le démences dégénératives ou mixtes à prédominance dégénérative du grand âge. *Annales Médico-Psychologiques*, **2**, 213–241.
- FAIRBURN, C. G. & HOPE, R. A. (1988a) Changes in behaviour in dementia: a neglected research area. *British Journal of Psychiatry*, **152**, 406–407.
- & — (1988b) Changes in eating in dementia. *Neurobiology of Aging*, **9**, 28–29.
- FOLSTEIN, M. F., FOLSTEIN, S. E. & McHUGH, P. R. (1975) "Mini-Mental State": a practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, **12**, 189–198.
- GHAZIIDDIN, N. & McDONALD, C. (1985) A clinical study of adult coprophagics. *British Journal of Psychiatry*, **147**, 312–313.
- GOLD, R. M. (1973) Hypothalamic obesity: the myth of the ventromedial nucleus. *Science*, **182**, 488–490.
- GOODWIN, J. S., GOODWIN, J. M. GARRY, P. J. (1983) Association between nutritional status and cognitive functioning in a healthy elderly population. *Journal of the American Medical Association*, **249**, 2917–2921.
- HACHINSKI, V. C., ILIFF, L. D., ZILHKA, E., *et al* (1975) Cerebral blood flow in dementia. *Archives of Neurology*, **32**, 632–637.
- HANCOCK, M. R., HULLIN, R. P., AYLARD, P. R., *et al* (1985) Nutritional status of elderly women on admission to mental hospital. *British Journal of Psychiatry*, **147**, 404–407.
- HERZOG, A. G. & KEMPER, T. L. (1980) Amygdaloid changes in aging and dementia. *Archives of Neurology*, **37**, 625–629.
- HOPE, R. A., FAIRBURN, C. G. & GOODWIN, G. M. (1989) Increased eating in dementia. *International Journal of Eating Disorders*, **8**, 111–115.
- ISHII, T. (1966) Distribution of Alzheimer's neurofibrillary changes in the brain stem and hypothalamus of senile dementia. *Acta Neuropathologica*, **5**, 181–187.
- LEIBOWITZ, S. F. (1985) Brain neurotransmitters and appetite regulation. *Psychopharmacology Bulletin*, **21**, 412–418.
- LO, B. & DORNBRAND, L. (1984) Guiding the hand that feeds: caring for the demented elderly. *New England Journal of Medicine*, **311**, 402–404.
- MANN, D. M. A., YATES, P. O. & MARCYNIAK, B. (1985) Changes in Alzheimer's disease in the magnocellular neurones of the supraoptic and paraventricular nuclei of the hypothalamus and their relationship to the noradrenergic deficit. *Clinical Neuropathology*, **4**, 127–134.
- MORLEY, J. E. & SILVER, A. J. (1988) Anorexia in the elderly. *Neurobiology of Aging*, **9**, 9–16.
- MUNRO, H. N. (1982) Overview: nutritional status of the aged. *Aging*, **19**, 287–294.
- PILLERI, G. (1966) The Kluver-Bucy Syndrome in man. *Psychiatrica Neurologica* (Basel), **152**, 65–103.
- RABIN, B. M. (1972) Ventromedial hypothalamic control of food intake and satiety: a reappraisal. *Brain Research*, **43**, 317–342.
- REZEK, D. L. (1987) Olfactory deficits as a neurologic sign in dementia of the Alzheimer type. *Archives of Neurology*, **44**, 1030–1032.
- ROLLS, E. T., SANGHERA, M. K. & ROPER-HALL, A. (1979) The latency of activation of neurons in the lateral hypothalamus and substantia innominata during feeding in the monkey. *Brain Research*, **164**, 121–135.
- ROSSOR, M. N., IVERSEN, L. L., JOHNSON, A. J., *et al* (1981) Cholinergic deficit in frontal cerebral cortex in Alzheimer's disease is age dependent. *Lancet*, **ii**, 1422.
- , —, REYNOLDS, G. P., *et al* (1984) Neurochemical characteristics of early and late onset types of Alzheimer's disease. *British Medical Journal*, **288**, 961–964.
- ROZIN, P. & FALLON, A. E. (1987) A perspective on disgust. *Psychological Review*, **94**, 23–41.
- SANDMAN, P., ADOLFSSON, R., NYGREN, C., *et al* (1987) Nutritional status and dietary intake in institutionalized patients with Alzheimer's disease and multiinfarct dementia. *Journal of the American Geriatrics Society*, **35**, 31–38.
- SOURANDER, P. & SJOGREN, H. (1970) The concept of Alzheimer's disease and its clinical implications. In *Alzheimer's Disease and Related Conditions* (eds G. E. W. Wolstenholme & M. O'Connor). London: Churchill.
- TERZIAN, H. & DALLE ORE, G. (1955) Syndrome of Kluver and Bucy. *Neurology*, **5**, 373–380.
- THOMAS, D. E., CHUNG-A-ON, K. O., DICKERSON, J. W. T., *et al* (1986) Tryptophan and nutritional status of patients with senile dementia. *Psychological Medicine*, **16**, 297–305.
- TOMLINSON, B. E. (1982) Plaques, tangles and Alzheimer's disease. *Psychological Medicine*, **12**, 449–459.
- WHITFORD, G. M. (1986) Alzheimer's disease and serotonin: a review. *Neuropsychobiology*, **15**, 133–142.

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