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A Comparison of Early-Onset and Late-Onset Depressive Illness in the Elderly

P. W. BURVILL, W. D. HALL, H. G. STAMPFER and J. P. EMMERSON

Elderly patients with early-onset and late-onset depressive illness presenting to psychiatrists for treatment were compared for social, demographic, and clinical measures. For most factors measured no statistically significant differences were found. In the early-onset cases, patients were significantly more severely depressed. There was some evidence for the hypotheses that family history is less important and biological factors more important in late-onset depression. It is suggested that the latter hypothesis should be tested by a range of the newer neuroanatomical and neurophysiological laboratory investigations. The findings indicate that neuroticism is an important underlying factor in both early-onset and late-onset depression in the elderly.

Depressive illness is common in the elderly and has recently become the focus of much research. Its prognosis is relatively poor and it is characterised by frequent and prolonged relapses (Murphy, 1983), although Baldwin & Jolley (1986) have recently reported a more favourable outcome. Various researchers have looked for predictors of a good and poor prognosis (Burvill *et al*, 1986). Some authors (e.g. Kay *et al*, 1955; Post, 1972; Cole, 1983) have drawn a distinction between early-onset depression, that is, the first ever episode of depressive illness occurring before the age of 60 years, and late-onset depression, the first episode occurring after the age of 60 years. Late-onset depressives have been said to have better pre-morbid personalities and less family history of depression than early-onset depressives (Kay, 1959; Hopkinson, 1964; Post, 1972). There is evidence that adverse life events occur with greater than chance frequency where there is depression in younger patients (Paykel *et al*, 1969; Brown *et al*, 1973), and that similar factors contribute to the onset of depression in old age (Murphy, 1982). Post (1978) pointed out that the kind of life events shortly preceding nearly 80% of depressions are the common experiences of all old people, but that since only a few develop depressions there must also be present in the depressives some personality or biological predisposition. After reviewing the literature Cole (1983) concluded that the effect of age of onset on prognosis was unclear.

A relevant question for research is: why do some people develop depression for the first time at the age of 60, 70 or 80 years? Are the same aetiological factors that operate in depression beginning earlier in life relevant to late-onset depression, or are other factors operative? Post (1972) and Jacoby (1981) have raised the question of whether depression in old age may be partly due to a biological ageing process. Although they did not specify what was meant by 'biological ageing process', the reasonable presumption is that this term refers to physiological, biochemical and other cellular changes, including neuronal death, in those areas of the brain thought to be closely associated with affective disorders. Post did not differentiate between early-onset and late-onset depression, although Jacoby was clearly referring to late-onset cases. It is now generally agreed that the relationship of organic brain disease to affective disorder in the elderly is an unresolved question (Jacoby *et al*, 1980). In a comparison of head computerised tomography (CT) scan results in normal, depressed and demented elderly, Jacoby *et al* (1980) suggested that the emergence of depression in old age is facilitated by the biological process of ageing, which is reflected in the central nervous system by ventricular enlargement. Eight out of the nine of their subgroup of in-patient elderly depressed patients who had ventricular enlargement, and subsequently a very poor two-year prognosis, were late-onset depressives.

Studies of elderly people in hospital (Kay & Roth, 1955; Roth & Kay, 1956) and in those living at home (Kay *et al*, 1964; Kay & Bergmann, 1966) have shown that physical illness is important not only in the aetiology of organic syndromes but also in the functional syndromes of old life. Poor prognosis of depression in the elderly has been associated with chronic physical health problems (Murphy, 1983; Roth, 1983; Baldwin & Jolley, 1986) and with cerebral organic illness (Post, 1972). The high incidence of depressive illness following strokes has been well documented by Robinson *et al* (1984), although some of these findings are still controversial (House, 1987).

These findings suggest that biological factors may be more important in late-onset than in early-onset depressives. There is the further possibility that in early-onset depressives biological processes associated with ageing are superimposed upon the clinical state present since an earlier age. Depressive episodes become more frequent with increasing age in those whose depression first developed at an earlier age (Post, 1972). Post (1978) has claimed, on the basis of his long clinical experience and research with elderly depressives, that the failure of elderly depressed patients to respond to adequate treatment, when they have been treated successfully in earlier life, is usually due to the development of a persistent physical disorder.

This study examines whether there were any differences between cases of an early-onset and late-onset depression in a cohort of depressed elderly patients under psychiatric care. On the basis of the relatively scanty evidence in the literature we hypothesised that, compared with early-onset depressives, late-onset patients would have less family history of depression and more stable pre-morbid personalities; major life events would be less important in the onset of the depression; a higher proportion of cases would have endogenous depression, and they would have more cognitive impairment; and both cerebral and non-cerebral physical illness would be more prevalent. There was no strong reason to expect any difference for severity of depression between early- and late-onset cases.

Method

This study formed part of a study of prognosis of depressive illness in the elderly. The cohort consisted of 103 patients who were being treated for a depressive illness by psychiatrists in a number of public psychiatric hospitals in Perth, Western Australia. For entry to the study patients must have been aged 60 years or over, have an adequate command of English to participate in the research process, and have a primary diagnosis of DSM-III major depressive illness (American Psychiatric Association, 1980) that had been present for at least four weeks. The cohort of 103 patients were consecutive patients meeting the criteria.

Each patient was seen by a psychiatrist (PWB or HGS) or a third-year psychiatric registrar (all trained to administer the research instruments), and the following were completed: Geriatric Mental Schedule - Shortened Version (Henderson *et al*, 1983), Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960), Mini Mental Status Examination (MMSE; Folstein *et al*, 1975) and Newcastle Rating Scale (Carney *et al*, 1965). A clinical assessment of pre-morbid personality was performed. Family history of psychiatric illness was obtained only from the patient. Unfortunately, inquiry on this point and about pre-morbid personality was not made of relatives and key informants. An assessment was made of the severity of each patient's physical health, with acute and chronic illness differentiated (Burvill *et al*, 1989). In addition, an OARS Physical Health Rating (Duke University Centre, 1978) and a patient Self-Health Rating were completed. Each patient was assessed by the psychiatrist on whether he/she was an early-onset or late-onset depressive, depending upon whether the first-ever episode of depression began before or after the age of 60 years.

After the clinical assessment, each patient was seen by a psychologist graduate research assistant and the following were assessed: demographic information, Eysenck Personality Inventory (EPI), Major Life Events and Difficulties according to Brown's method (Brown & Harris, 1978) suitably modified for the elderly, and the availability of confidants. As part of the larger study, EPI scores were measured on a randomly selected community sample of 85 non-depressed 'normals', matched with the depressives for age and sex.

The cohort of 103 patients came predominantly from two general hospital psychiatric units in Perth's two largest teaching hospitals. A total of 69 patients came from one of those hospitals, comprising 55 in-patients, five out-patients, and nine subjects of consultations from the Geriatric Service (all in-patients). There were 16 in-patients and two day patients from the second hospital. A further seven in-patients came from a third general psychiatric unit in the latter part of the survey. The remaining nine patients came from mental hospital in-patients (seven) and a psychogeriatric in-patient facility (two).

Data analysis

Two types of analysis were performed to compare characteristics of elderly depressives with early-onset and late-onset illnesses. First, univariate comparisons were performed by means of χ^2 and *t*-tests for each of the individual demographic, life event, psychiatric and physical illness measures. Second, to take account of correlations between the individual measures (and hence the outcomes of the univariate comparisons), the early-onset and late-onset cases were simultaneously compared for introversion, neuroticism, family history, sex of subject, DSM-III severity, and scores on the HRSD, the Newcastle Endogenous Depression Scale, and the Mini Mental State Examination, by means of a discriminant function analysis. This analysis selected that linear combination of these measures which maximally discriminated between the early-onset and late-onset cases. The contribution that each variable makes to the discrimination is indicated by the coefficient that it receives in the linear discriminant function (Harris, 1985).

Results

Demographic

By chance there was an almost equal number of early-onset (52) and late-onset (50) depressed patients in the cohort, with an equal sex distribution (M : F 1:1.8) in each category. The majority of the early-onset patients (85%) were in the

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TABLE I
Age distribution of the early-onset and late-onset depressives

	60–64	65–69	70–74	75–79	80–84	85–89	Total
Early onset	18	13	13	6	1	1	52
Late onset	5	5	11	12	11	6	50

TABLE II
Comparison of mean EPI scores between early-onset, late-onset depressives and community controls

		Depressed		*Community controls*
		Early onset	*Late onset*	
Neuroticism		14.02	12.24	6.64
Extraversion		8.87	10.24	11.28
Early-onset v. late-onset depressives	Neuroticism	$t=1.481$,	d.f. = 90, NS	
	Extraversion	$t=1.420$,	d.f. = 90, NS	
Early-onset v. community controls	Neuroticism	$t=8.413$,	d.f. = 122, $P<0.001$	
	Extraversion	$t=2.842$,	d.f. = 122, $P<0.001$	
Late-onset v. community controls	Neuroticism	$t=6.361$,	d.f. = 122, $P<0.001$	
	Extraversion	$t=1.273$,	d.f. = 122, NS	

60–74-year age group and the majority of the late-onset patients (58%) in the 75–89-year age group (Table I). About half the late-onset depressives had had one or more previous attacks of depression since the age of 60 years, and half were having the first-ever episode of depression.

Life events and personality

There was a higher proportion of late-onset depressives (44%) who had a severe life event in the 12 months before onset of depression than of early-onset depressives (31%). However, this difference was not statistically significant.

As assessed by the EPI, there were no significant differences between the early-onset and late-onset depressives for the degree of neuroticism (Table II), but both had significantly ($P<0.001$) higher neuroticism scores than did the community 'norms'. The early-onset, but not the late-onset, depressives had a significantly ($P<0.01$) lower extraversion score than did the community 'norms'. However, although the extraversion scores were lower for the early-onset than the late-onset depressives, the differences were not significant. A clinical assessment of pre-morbid personality was made by the psychiatrist on a wide spectrum of personality traits, similar to the method used by Mann *et al* (1981). Only three personality traits were found in sufficient numbers for analysis, namely, anxiety, obsessional and dependency traits. A greater proportion (76.9%) of early-onset depressives had pre-morbid anxiety traits compared with late-onset patients (47%) ($\chi^2=9.76$; d.f. = 1; $P<0.002$). There were no significant differences for obsessional and dependency traits.

Psychiatric

The early-onset depressives, as measured both by the DSM–III diagnosis and the HRSD, were more severely depressed. A higher proportion of early-onset depressives (45%) had a DSM–III diagnosis of major depression with psychosis and a lower proportion (22%) major depression without melancholia than the late-onset patients (24% and 41%, respectively) (Table III). The early-onset cases had a HRSD mean of 23.5 and the late-onset cases of 20.4 ($t=2.679$, $P<0.01$).

Of both early-onset and late-onset depressives, 82% had endogenous depression as measured by the Newcastle Scale. Nine (18.4%) early-onset and 13 (27.1%) late-onset depressives scored in the 'organic' (<24) range of the MMSE. Only one patient (late-onset) scored in the severe range (score = 18). These differences were not statistically significant, although the discriminant function analysis (Table III) showed the early-onset depressives to have a significantly higher mean MMSE score. Of the early onset, 56% and of the late-onset depressives, 39% had a family history of depressive illness, but these differences were not statistically significant. Only four early- and two late-onset depressives reported a possible, and two late-onset depressives a definite, family history of other psychiatric illnesses.

TABLE III
DSM–III diagnoses (percentages)

Diagnosis	*Early onset*	*Late onset*
Major depression	22	41
Major depression with melancholia	33	35
Major depression with psychosis	45	24
Total	100	100

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Physical illness

The health status of each group was assessed on ten variables (three of acute illness, three of chronic illness, the OARS, and three of the patients' own health rating). Although there was more chronic illness among the late-onset depressives, a discriminant function analysis failed to detect any statistically significant difference between the early-onset and late-onset depressives for any of these variables. The physical illnesses were predominantly chronic illnesses, of which 92% were non-cerebral. The severity of the small number of cerebral illnesses did not differ between early-onset and late-onset cases.

A CT brain scan was performed on only 26 patients, 13 early-onset and 13 late-onset depressives. There was a higher proportion of organic features in the late-onset (five patients) than the early-onset (one patient) depressives. In addition, one patient from each group showed greater than normal cerebral atrophy (when data were adjusted for age). The one early-onset case (a patient aged 60 years) showed an anterior communicating aneurysm, an *in situ* shunt and an early hydrocephalus from a cerebral episode and neurosurgical operation 12 months earlier. Of the five late-onset patients with organic CT features, three (aged 80, 81 and 85 years) were secondary to strokes, the changes in one (aged 67 years) were consistent with a long history of heavy drinking and one (an 81-year-old) showed signs of congenital hypoplasia of the left cerebellar and inferior vermis.

Multivariate comparisons

Table IV shows the results of a discriminant function analysis comparing early-onset and late-onset depressives for a number of factors. Early-onset depressives were significantly more likely to have a higher MMSE score, and to be more severely depressed when measured both by the DSM-III classification and by the HRSD. The late-onset depressives were significantly more extraverted on the EPI Scale. There were no significant differences for the degree of neuroticism, for the family history of depression, and for the number of endogenous v. neurotic depressives on the Newcastle Scale.

TABLE IV

Discriminant function analysis of a number of factors attempting to discriminate between early-onset and late-onset depressives

Factor	*Canonical coefficients*
Introversion	0.378[1]
Neuroticism	0.043
Family history	0.164
Sex of patient	0.117
DSM-III severity	0.471[1]
HRSD	0.418[1]
Endogenous depression	0.163
MMSE	0.515[1]

1. Statistically significant coefficients.

Discussion

No statistically significant differences were found for the predisposition to become depressed as measured by family history, neuroticism, pre-morbid personality or life events, although the differences in family history were substantial and in the direction predicted. By contrast, the finding for life events was in the opposite direction to that predicted. Clinically, in the early-onset cases, patients were more severely depressed as measured by the HRSD and DSM-III. In both early-onset and late-onset cases, patients were endogenous rather than neurotic depressives, as measured by the Newcastle Scale. Efforts to show a postulated greater biological impairment among the late-onset cases produced mixed results.

Before discussing the significance of these findings we need to consider the possible biases in the selection of the sample, and any limitations these impose upon our conclusions. The initial selection of patients for the study did not take into account the factor of early-onset v. late-onset of depression. It was purely fortuitous that the two groups had an equal sex distribution and were of the same size. The sample represented those elderly depressed patients seen by busy general psychiatrists, in their everyday work in a hospital system, in which very few elderly depressed patients were treated by a small psychogeriatric service. The majority were in-patients. The psychiatric unit, in which over half the study cohort were seen, had close links with an active geriatric unit, and as such, saw a number of late-onset depressives aged 75 years and older, representing about 14% of the late-onset depressives. Rigorous inquiry revealed that very few elderly depressives were treated in private hospitals by psychiatrists during the time of the survey. Thus the cohort seemed a fairly representative sample of severe elderly depressives treated by Perth psychiatrists.

The late-onset group was definitely older than the early-onset group, although patients of both groups were represented in all five-year age groups from 60–64 to 85–89 years of age. It is clear that early-onset depressed patients can continue to become depressed until a later age, and conversely that some people can reach a fairly old age before first becoming depressed. There are several possible reasons for the decreasing frequency of early-onset depressives with increasing age. As early-onset depressives grow older, the frequency of attacks of depression may decrease. However, Post (1978) has expressed the opposite view, that as these patients grow older attacks of depression became more frequent, more protracted, and more difficult to treat. A more likely explanation is that as early-onset

depressives grow older, a high percentage of them will have died, or have been placed in nursing homes or some other form of institution. Accordingly, a much smaller proportion of early-onset depressives will be living in the community. Nearly all the patients in this study lived in a non-institutional setting. By contrast, the older, late-onset depressives came from those who survived into old age, without developing depression at an earlier age, and who were still living in the community rather than in institutions. Thus, they come from a relatively robust group – the survivors of their generation. Hence it is reasonable to postulate that they needed something 'extra' to precipitate depression after having lived for most of their long life without the occurrence of any depressive illness. It is quite likely that the occurrence of these precipitants, especially if they are biological, increases with age.

It was hypothesised that there would be a greater predisposition to depression among the early-onset depressives as indicated by family history, pre-morbid personality, neuroticism and life events. The support for these predictions was weak. There was a difference in the predicted direction for the finding for family history (56% v. 39%) but the sample size was not large enough for this difference to be statistically significant. On the basis of this result it would be unwise to rule out a difference because the confidence intervals (CIs) around the proportion with a family history are consistent with there being a substantial difference in the proportion of elderly depressives (CI for early cases is 0.45–0.67 and 0.28–0.50 for the late-onset cases). A wide range of psychiatric illnesses, especially alcoholism, has been found to be present in excess in the families of depressed patients (Winokur *et al*, 1975) but, in this study, only a very small number of early-onset and late-onset cases gave such a family history.

For pre-morbid personality, there were difficulties in assessing the trait with confidence. We were not fully satisfied with the objectivity and stringency of our attempts to assess pre-morbid personality traits clinically, in spite of this being a long-standing clinical practice which is usually considered to be of major importance in the assessment of patients. Only one significant trait difference was detected, namely, that the early-onset depressives were more anxiety prone. Without accurate confirmatory history from informed relatives or friends, it was difficult to be sure that this trait was a true personality characteristic, or the gradual evolution over time of mild anxiety symptoms which were present for variable periods between the major attacks of depression – a phenomenon often seen clinically in such patients once the depressive illness has been successfully treated. Although a predominance of obsessional personality traits in depressive illness, especially those with more endogenous features, has long been noted by psychiatrists, the presence of this trait was not found to be a differentiating factor between early-onset and late-onset cases.

A related difficulty arises with trait neuroticism. Neuroticism was higher in the depressed than in the community sample, but this may have been because the depressed state coloured the way in which patients completed the EPI, irrespective of whether the depressive illness was of early or late onset. The patients could have been greatly influenced by feelings of negativism, anxiety, apprehension and uncertainty secondary to their depression, and consequently they may have answered the EPI questions as if they had been depressed for many years. The alternative of obtaining information from a reliable informant was not available to the researchers. Only reassessment of their personality, by the EPI when the patients are not depressed, would eliminate this possibility. The authors plan to conduct such an assessment as part of their 12-month follow-up study.

Should it be found that the EPI findings remain stable between the depressed and the non-depressed phases, the finding of a significantly increased neuroticism in both early-onset and late-onset depressives demands explanation. It is possible that excessive neuroticism is a risk factor for the development of depression in this age group, irrespective of whether or not the depression is clinically endogenous in type, or has a predominantly biological base, or is of early or late onset. Stressful life events are more likely to precipitate depression in those with excessive neuroticism. Likewise, minor cerebral organic changes may be more likely to lead to depressive illness in those with excessive neuroticism.

Much has been written of the role of severe life events in the aetiology of depressive illness in the younger age groups, especially by Brown & Harris (1978). In this study severe life events occurred more often in the depressive sample than in the community sample, confirming the earlier finding of Murphy (1982) for elderly depressives. However, there were no significant differences for the proportion of severe life events between early-onset and late-onset depressives. Actually there was a non-significantly greater number of life events in the late-onset cases, which is in the opposite direction to that hypothesised. These findings have been considered in detail by Emmerson *et al* (1989). The fact that more than half of each group did not have a prior severe life event indicates that such events, although

important, are not essential for the development of depression in the elderly.

Both early-onset and late-onset depressive groups showed the full range of severity of depression, but the late-onset group as a whole were clearly less severely depressed, whether judged by HRSD score, or on DSM criteria. This suggests that Post (1978) is right in believing that patients with a long life history of recurrent depression suffer from more severe episodes as they grow older.

Less than one-fifth of patients in either the early-onset or late-onset cases were neurotic depressives, with no differences between the two groups. There are a number of possible interacting explanations of these findings. The differentiation between endogenous and neurotic depression may be of less importance in the elderly. It is possible that depressive illness shows more endogenous features with increasing age, irrespective of whether the depression is of early or late onset. Younger depressed patients are more likely to be neurotically depressed and older patients to be endogenous (Post, 1982; Klerman, 1983). As with most published studies of depressive illness in the elderly, the present study was composed predominantly of in-patients who are at the more severe end of the spectrum of depressive illness, thereby increasing the likelihood of their being diagnosed as suffering from endogenous depression. That is, the cohort is not necessarily representative of depressive illness in the elderly in the wider community, but only of that presenting to psychiatrists for treatment.

It was hypothesised that the late-onset depressives would have a greater degree of biological impairment, as assessed by cognitive impairment and the presence of both cerebral and non-cerebral physical illness. We had too few patients to be able to provide a strong test of this hypothesis. However, there was some evidence in its favour, namely, greater cognitive impairment in the late-onset depressives and CT scan results in the predicted direction.

The possible role of physical illness in the aetiology of depression probably differs for cerebral and non-cerebral illnesses. The mechanism of the relationship is not straightforward and is not well documented in the psychiatric literature. It would be expected that in most non-cerebral illnesses the relationship is more in the nature of a psychological reaction to having the illness rather than to any direct biological connection. However, the close connection of depressive illness with endocrine disorders and with certain malignancies is well known. In the present study two patients had a malignancy diagnosed after the initial diagnosis of definite major depression was established. With cerebral illness, biological factors would be expected to have the predominant role.

A major expectation of the study was that there would be a higher incidence of physical illness, both cerebral and non-cerebral, in the late-onset depressives, which was independent of the age of the patient. The results showed this not to be so. The small non-significant excess of chronic physical disability in the late-onset depressives was easily explainable by the greater age of these patients. Although a greater proportion of the late-onset depressives showed abnormalities on head CT scan, the small number of scans performed (one-quarter of each group) was too small to allow any definite conclusions to be drawn. It was disappointing that CT scans were not able to be done on all patients, as this may have definitely supported or refuted the biological hypothesis. Nevertheless, the findings were in the direction hypothesised. Three of the five late-onset depressives with abnormalities were suffering from depression following strokes. The higher incidence of depression after strokes has been well documented by Robinson *et al* (1984), as have possible neuroanatomical/neurochemical aetiological mechanisms (Lipsey *et al*, 1984). The fourth case showed changes consistent with the patient's history of alcoholism, which could well have been a major factor in the genesis of late-onset depression. The only patient with early-onset depression with a CT abnormality had changes secondary to neurosurgical intervention just before the age of 60 years.

Conclusion

Most of the hypotheses tested failed to be supported. However, for reasons of statistical power consequent upon the number of cases studied, it would be unwise to rule out some of the hypotheses, especially those relating to family history predisposition and biological impairment. In the latter two the differences were in the direction hypothesised, although they did not reach statistical significance. There was no difference between the early-onset and late-onset depressives for the degree of neuroticism and for the proportion with endogenous depression. For severe life events, the differences were in the opposite direction to that hypothesised, even though not statistically significantly so.

There is a great need for more sophisticated laboratory measures of cerebral biological factors to ascertain adequately whether there was a greater degree of biological impairment in the late-onset cases. Such measures would include head CT scans, NMR and PET studies – all of which can, we hope,

detect more subtle changes than the clinical measures adopted in the present study.

Finally, it must be emphasised that many of the findings in this and other published studies of depressive illness in the elderly are based on cases where patients with severe depression present for psychiatric treatment, and do not necessarily apply to the numerically large and clinically wide range of depressive illnesses in the community.

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*P. W. Burvill, MD, FRCPE, FRANZCP, FRCPsych, *Associate Professor of Psychiatry, University of Western Australia, Royal Perth Hospital, GPO Box X2213, Perth, Western Australia 6001*; D. W. Hall, BSc, PhD, *Associate Professor, National Drug and Alcohol Research Centre, University of New South Wales, Sydney, NSW*; H. G. Stampfer, MB, BS, FRANZCPZ, *Senior Lecturer in Psychiatry, University of Western Australia, Sir Charles Gairdner Hospital, Nedlands, Western Australia*; J. P. Emmerson, BSc(Hons), SEN, *formerly Research Assistant, Department of Psychiatry, University of Western Australia, Nedlands, Western Australia*

*Correspondence