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Depression in old age

Is there a real decrease in prevalence? A review

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Abstract The discrepancy between the constancy or increase of the prevalence of depressive symptoms and dysphoria in old age on one hand, and the decrease in the prevalence of the DSM-III diagnoses of major depression and dysthymia on the other, is discussed in light of the most frequent explanatory hypotheses such as memory defects, interpretation of depressive as somatic symptoms, higher risk of institutionalization as well as higher mortality of depressives and a mitigated course of depression in old age. We conclude that higher mortality, mitigation and the rarity of true late-onset depression are arguments for a real decline in prevalence, which occurs in accordance with the decline in all psychiatric disorders that are connected with emotional upheavals and substance ingestion. On the other hand, the connection of depressive states with somatic illness is strengthened, and according to preliminary validation studies, clinically relevant depressive states not reaching the threshold of DSM-III diagnoses may be typical for the depressive psychopathology of old age.

Key words Old age · Decrease in prevalence · Major depressive disorder · Dysthymia · Hypotheses

Introduction

... decline in physical strength, in mental alertness, in earning capacity, in prestige at home and in society, combined with economic deprivation, the loss of significant others, social isolation, and the prospect of death... Why is not every old person in a profound state of depression? (Jarvik 1976)

The above quotation points to the discrepancy between what common sense believes to be the prevalence of depression in old age, and the epidemiological findings of the ECA study (Robins and Regier 1991), of the Edmonton Study (Bland et al. 1988a) and of the Canberra Study (Henderson et al. 1993), which all show a decline of depression. Old age brings multiple losses, and loss is a generally accepted precursor of depression. In addition, a biological depression-proneness increases: Veith and Raskind (1988) and Steiger et al. (1993) showed that old age carries changes in the metabolism of norepinephrine, serotonin and monoamine oxidase (MAO); in growth hormone and cortisol secretion, and in sleep architecture that mimick those associated with depression. The fact that relatives of late-onset depressives are at a lower risk for depression than those of early-onset depressives (Nemeroff and Escalona 1993) could be understood as a consequence of a lifetime-specific higher vulnerability to depression, which is in a certain sense more "exogenous" than at an earlier age.

The epidemiological findings mentioned above, however, contradict an increased vulnerability to depression and dysthymia. Many explanations have been offered for the decline of DSM-III major depressive disorder (MDD) and dysthymia in old age. It is understood as an artefact of loss of memory or the masking of depression by somatic symptoms, or as the consequence of higher mortality and more frequent institutionalization of older depressives. Other hypotheses are the stabilization of surviving depression-prone personalities with age or the possibility that depression and dysthymia lose some of their impact and intensity. Thus, they glide below the threshold of DSM-III major depression and dysthymia, while in many cases remaining clinically valid syndromes worthy of attention (Katona 1994). In this paper the pros and cons of these hypotheses are discussed.

A further popular explanation is left out: a possible cohort effect. Blazer (1994) in his "honest look at the evidence" finds a strong argument for a differential burden of depression by cohort in suicide rates. Even if the prevalence of depression could be deducted immediately from the incidence of suicide, and if we accepted that the contemporary 70- to 80-year-olds are less burdened with depression than earlier and later cohorts, the explanations for this difference remain vague and somehow arbitrary: the postwar economic growth, better health care and better health status. By not entering the discussion of a co-

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hort effect, we do not pretend that it does not exist. We merely do not want to take a stand on very shaky ground (Lewinsohn et al. 1993).

Decline in prevalence depends on diagnostic criteria

All investigators mentioned herein used Diagnostic Interview Schedule (DIS) given by lay interviewers and DSM-III criteria for depression and dysthymia. The ECA Study (Robins and Regier 1991) found in elderly Americans a drop in prevalence of both DSM-III MDD and dysthymia. It appeared for both disorders and for lifetime, 1 year, 6 months and 1 month, and reached the lowest level of all age groups at 65+ years (loc. cit. p. 66; Myers et al. 1984; Regier et al 1988). The Edmonton Study (Bland et al. 1988a) noticed the same sharp drop at age 65+ years for MDD and dysthymia, and both for lifetime and 6-month prevalence. Oliver and Simmons (1985) found current and lifetime prevalence of MDD independent of age. Crowell et al. (1986) in a subsample of the ECA study renounced all exclusion criteria, with the exception of mania, and restricted themselves to a diagnosis of current MDD. In their study the lowest-ever odds ratio (0.6; age 18–24 years: 1.0) appeared at age 65+ years. Thus, two reasons frequently given for the decline of MDD and dysthymia by age are ruled out: that the decrease is entirely due to age-associated memory loss – also point prevalence declines – and that it is due to unjustified exclusions by lay interviewers who take symptoms of depression for somatic disorders.

A memory effect on lifetime prevalence is present at every age. Simon and von Korff (1992) showed with ECA data that both younger and older subjects had a tendency to locate first onset of a disorder at about 10 years before the interview, and that at a reinterview lifetime prevalences of depression went up dramatically if in the meantime there had been an episode.

Interviews and diagnostic criteria different from DIS/DSM-III, however, give a slightly different picture. Carta et al. (1991) used the PSE and the ID-CATEGO procedure (Wing et al. 1973) to investigate a 4-week prevalence of disorders in the population of two Sardinian mining villages: Depressive disorder did not drop sharply at age 65+ years, but remained at a slightly lower level than in middle age.

A similar result was reached by the same method in the Mini-Finland study by Lehtinen et al. (1990): "Neurotic depression", i.e. mild depression, peaked at age 65 years and then decreased to the level found in the 45-years-olds. In a New Zealand female sample the same method resulted in a slight increase after age 65 years (Walton et al. 1990). In the Canadian Stirling County Study (Murphy et al. 1984) where a concept of present "aggregate depression or anxiety" of at least 4 weeks' duration was used, females remained at the same level of prevalence from age 40 to 70+ years, whereas there was a sharp drop for males at age 70+ years.

The main methodological difference between the DIS/DSM-III and the PSE/ID; as well as the Canadian concept, seems to be the number of symptoms and the time

Table 1 Symptom scales: prevalences over cut off (general population)

Scale	Somatic items (%)	Age (years)			
		18–24	25–44	45–64	65+
CES-D (16+)	10				
Markush and Favero 1974 (Southern USA)			23	19	15[a]
Comstock and Helsing 1976 (Washington)		29.0	17.4	14.7	12.3
Frerichs et at. 1981 (California)		11.4	9.0	8.7	8.3
Eaton and Kessler 1981 (U.S. population)		–	17.7	14.9	6.5
Turner and Noh 1988 (Ontario)		14		15	7[b]
SCL-90 (×)	20				
Uhlenhut and Balter 1974 (Oakland)		18	–	–	12

Scale	Somatic items (%)	Age (years)			
		< 30	30–40	40–60	60–70+
GHQ (> 5)					
Lehtinen et al. 1990 (Finland)	29	19	22	34	46
Langner (> 7)					
Gaitz and Scott 1972 (Houston)	54	12.4		14.3	16.8

[a] Ages (years): < 35; 35–54; 54+
[b] Ages (years): 18–44; 45–64; 65+

criterion. DSM-III introduces for MDD and dysthymia a defined minimal duration of 2 weeks and 2 years, and a defined minimal number of key symptoms, whereas the other methods use less stringent definitions and leave more room to clinical judgement.

When number of symptoms as diagnostic criterion is removed, the definition of "depression" is reduced to dysphoria of some duration. Simple dysphoria does not decrease in old age. Leaf et al. (1988) asked for a period of sadness of at least 2 weeks between two ECA interviews that were given at an interval of 1 year. Dysphoria in men and women decreased from age 18–24 years, but increased somewhat after age 65 years, whereas MDD dropped sharply. In a large American southern community sample a current or lifetime history of 2-week dysphoria was found only slightly less often in 65+-year-olds than in younger subjects (Blazer 1989).

Symptom scales give a count of present symptoms with a cut off for a diagnosis of depression without introducing duration (Zung's Self-Rating Depression Scale 1965; Beck's Depressive Inventory 1961; the Center of Epidemiologic Studies Depression Scale by Radloff 1977). Table 1 shows that an association of symptoms with age depends on the content of the scales. An increase in symptom prevalence with age is associated with a larger rate of somatic items, whereas scales mostly consisting of symptoms of psychological distress lead to a moderate drop or a levelling off of depression at age 65+ years.

Jorm's (1987) metaanalysis united by statistical tranformation 25 studies on "depressive states" based on interviews or inventories, and found for both genders a peak in prevalence in early adulthood, and then a very gradual decline to age 80 years.

Blazer et al. (1988) created subtypes of depression by multivariate classification of actual depressive symptoms given in the DIS by a large elderly subsample of the ECA. Of four types of depression, three decreased from adult to old age (MDD, mild dysphoria, mixed anxiety and depression); the last type (organic symptoms with dysphoria) was associated with age over 60 years, but decreased to age 90+ years.

A cautious first conclusion could be that strictly defined depressive states are less frequently found after age 60 or 65 years than in younger groups, that with less stringent definitions prevalence remains more or less unchanged, that "pure dysphoria" somewhat increases, and that a sharp increase in depressive symptoms in old age is connected with the rate of somatic symptoms the scales contain.

Is depression masked by somatic symptoms?

This latter finding leads to the question of whether depression and dysthymia in old age are masked by non-specific somatic symptoms (Katona 1994). Several studies compared symptoms by age and found only slight differences (Winokur et al. 1980; Blazer et al. 1987; Brodaty et al. 1991; Oxman et al. 1990). Somatization in the elderly was restricted to a higher frequency of weight loss and sleeplessness. Berkman et al. (1986) found the same CES-D factors in middle-aged and older populations. Robins and Regier (1991, p. 63) and Koenig and Blazer (1992) showed in a comparison of younger and older ECA subjects by symptom frequency that each kind of depressive symptoms, somatic as well as psychological (with the exception of dysphoria), decreased in old age. The independence of a diagnosis of depression of the presence of nonspecific somatic symptoms is confirmed by two studies of the general population by Lewinsohn et al. (1991) and by Downes et al. (1988). The latter authors showed with hierarchical patterns and Guttman scales that although somatic symptoms increased with age, they were not part of the symptomatology of depression. This finding was confirmed in a longitudinal study by Costa and McCrae (1980): In male volunteers somatic symptoms increased with age, but they were specific for well-defined somatic disorders (e.g. urogenital, cardiovascular). Even in depressed stroke patients nonspecific somatic symptoms were rare (Fedoroff et al. 1991).

It is questionable whether hypochondriasis takes the place of depression. The ECA study presents a "somatization syndrome" as a modern equivalent of hypochondriasis. The prevalenc of 4% was not significantly influenced by age (Robins and Regier 1991). Copeland et al. (1987c) found for the diagnosis of hypochondriasis a prevalence of 0.5 in 65+-year-olds.

We conclude that there is no conclusive evidence that in old age depression is masked to a higher degree by somatic symptoms than in younger people. In old age an increase in somatic symptoms – and in somatic symptoms of depression scales – may not be due to a somatization of depression, but mainly to the presence of frequent and definable somatic disorders.

Are depressed subjects lost by death?

If elderly depressed subjects in the general population are compared with control subjects, they are at an increased risk of death in the follow-up period (Table 2). Mortality is about twice as high in the initially depressed than in the not-depressed persons in the follow-up period. With control of age, gender, and health, in three investigations (Gurland et al. 1988 a; Thomas et al. 1992; Fredman et al. 1989) higher mortality, however, was not associated with depression, but with male gender, older age and ill health. If the group, on the other hand, comprises all affective or all functional disorders (Kay and Bergmann 1966; Bickel 1987; Bruce and Leaf 1989), controlling for gender, health and age still leaves a higher mortality risk attributable to mental illness.

Depression thus seems to be a moderate risk factor for death, mainly because in old age it is associated with somatic illness. Suicide was not an important cause of death in the follow-up of depressed elderlies (Bickel 1987; Kay and Bergmann 1966; Enzell 1984). At a younger age the higher mortality of depressed patients is associated with

Table 2 Mortality in depressed community samples (with exclusion of organic brain syndromes)

Sample	Age (years)	Location	Follow-up time	Controls	Result
Depression measured by symptoms					
Markush et al. (1977; symptom list)	45+	Alahua county	4 years	Not depressed	h.m.[a] Mean annual attributive mortality rate 6.6
Persson (1981) (symptoms in interview)	70+	Gothenburg	5 years	Not depressed	h.m.[a] Males: $P \leq 0.05$ Females: $P \leq 0.01$
Enzell (1984) (symptom questionnaire)	66+	Stockholm	3 years	Not depressed	h.m.[a] $P \leq 0.01$
Thomas et al. (1992; CES-D)	65+	Bronx	3 years	Multivariate analysis (age, gender)	n.s.
Diagnosis of depression					
Murphy (1983; (chronic depression and anxiety)	All ages	Sterling county	16 years	General population	h.m.[a] Standardized mortality ratio men: 2.1
Davidson et al. (1988; GMS/AGECAT)	65+	Liverpool	3 years	Not depressed	h.m.[a] Odds ratio 2.92 (males only)
Gurland et al. (1988a; CARE)	65+	USA/London	1 year	Multivariate analysis (age, gender, health)	n.s.
Fredman et al. (1989; DIS/DSM-III)	60+	Piedmont	2 years	Log regression (age, gender, SES, health)	n.s.
Forsell et al. (1994; DSM-III)	75+	Stockholm	3 years	Not depressed	h.m.[a] $P \leq 0.008$
Functional disorder, affective disorder					
Kay and Bergman (1966; no standard diagnosis)	65+	Newcastle	4 years	General population	h.m.[a] $P \leq 0.02$
Bickel (1987; diagnostic method not specified)	65+	Mannheim	4 years	Not functional (age, gender, health)	h.m.[a] 88%
Bruce and Leaf (1989; DIS/DSM-III)	55+	New Haven	15 months	Log regression (age, gender, health)	h.m.[a] Odds ratio 2.97

[a] h.m. higher mortality in the depressed

suicide and accidents (Tsuang and Simpson 1985; Black et al. 1985; Martin et al. 1985; Ciompi 1969). In Bruce and Leaf's (1989) study, where there was a gross assessment of health, recent – but not lifetime – affective disorder was associated with 15-month mortality, and causes of death were not different for subjects with and without affective disorder. The specific causes of early death in affective disorder seem to disappear with age.

In conclusion, there is a mild increase in the mortality of elderly depressives that is mainly associated with their manifest health status at first investigation. Still, a direct influence of depression on mortality, e.g. by increasing vulnerability to somatic illness, cannot be excluded (O'Brian and Ames 1994). This finding means that some elderly depressives are lost to epidemiological studies because they have worse health and die earlier than subjects who are not depressed. Selective mortality, however, has no large effect on the prevalence of depression (Simon and von Korff 1992).

Are depressed subjects lost by institutionalization?

There is a general agreement that whatever measure is used to assess depression, its prevalence in institutionalized populations always surpasses that found in the community (Abrams et al. 1992). Particularly during old age the exclusion of hospitalized or institutionalized subjects thus could lead to a distortion in prevalence.

The main result of the data in Table 3 is that with the exclusion of severe dementia, the prevalence of relevant depressive symptoms in nursing home populations is 40–50%, and of MDD/DSM-III 15–20%. The prevalences obtained by different authors are not overly disparate. The relative agreement in this context incidentally shows that the decrease of diagnosable DSM-III depression with old age in the noninstitutioalized population is not an artefact of depression being "covered up" by somatic illness. Depression is measurable even in populations where somatic illness is extremely frequent. The relationship of depression measured by symptom scales to depression diagnosed by criteria in the institutionalized elderlies is similar to that found by Boyd and Weissman (1981) for the general population, i.e. about 3 : 1. Although the point-prevalence of institutionalization is low, the inclusion of subjects in institutions appreciably influences the prevalence of depression. Blazer (1989) calculated for the ECA study that the inclusion of older subjects with major depression in nursing homes would have raised the 1-year prevalence for those aged 65+ years from 0.9 to 1.4%, i.e. by 56%.

Is there selective institutionalization of depressed subjects? With the same health status older persons who are

Table 3 Prevalence of depressive syndromes in nursing homes. MDD major depressive disorder

Scale	Location	*n*	Dementia excluded		Prevalence (%)	
Symptom scales						
BAS (Mann 1984)	London	289	Yes		38	
BAS (Harrison et al. 1990)	London	914	Yes		46	
CARE (Gurland et al. 1979)	New York	162	Yes		38–43	
GDS (Snowdon et al. 1986)	Australia	206	Yes		39	
GDS (Katz et al. 1989)	Philadelphia	51	Yes		43–44	
					Depression	
					Major (%)	Minor or symptoms (%)
Diagnoses						
DSM-III (Hyer and Blazer 1982)	North Carolina	156	Yes		15	50
GDS/DSM-III (Katz et al. 1989)	Philadelphia	51	Yes		18–20	27–30
DSM-III (Kafonek et al. 1989)	Baltimore	70	Yes		21	
SADS-DSM-III (Parmelee et al. 1989)	Philadelphia	704	Yes (severe)		13.8–16.8	30.5
DIS/DSM-III (Robins and Regier 1991)	ECA	990	Yes		16.2	
DSM-III (Rovner et al. 1986)	Baltimore	454	Yes		12.6	18.1
DIS/DSM-III (Koenig et al. 1991)	Durban	332	Yes		13	
GMS/DSM-III (Philips and Henderson 1991)	Melbourne	405	Yes	DSM-III	9.7 (MDD) 3.6 (Dysthymia)	
				ICD 10	12.8	6.7
Canberra Interview/DSM-III (Henderson et al. 1993)	Canberra	143	Yes	DSM-III	6.3	
				ICD 10	3.0	4.1
Other diagnoses						
Feighner (Spagnoli et al. 1986)	Milan	237	Yes (severe)		30	
CARE AGE-CAT (Ames 1990)	London	390	Yes (severe)		24	

depressed could be at greater risk of entering institutions, e.g. because of self-neglect and malnutrition, than the not-depressed persons. Depression could promote helplessness in the presence of disability, and severe depression could accelerate the development of brain disorder.

Chronic illness, disability and depression

The association of chronic illness, disability (in the sense of restrictions in daily activity) and depression or depressive symptoms is well known (Turner and Noh 1988; Welz et al. 1989; Cooper and Sosna 1983; Finch et al. 1992; Palinkas et al. 1990; Gurland et al. 1988a, b). The association is genuine, i.e. there is no isolated inflation of somatic symptoms by disability: Somatic and psychological symptoms both increase (Turner and Beiser 1990; Blazer et al. 1991; Berkman et al. 1986). An association does not reveal causal direction. There are, however, five studies on the incidence of depression in elderly populations, which show that it is associated with a change in health. Phifer and Murrell (1986) found in a 55+-year-old population of Kentucky that subjects who over 6 months became new cases (CES-D score 16+) experienced an increase in physical symptoms and disability. Over 4 years a decline in activities of daily living was associated with CES-D depression independently of t_1-depression in a sample of Ontario disabled persons (Turner and Noh 1988). In an elderly Bronx population (Kennedy et al. 1990) the onset of CES-D depression (score 16+) over 2 years was connected not with level of activity and health at t_1, but with a change in activities of daily living. In a Veterans' Administration (VA) elderly sample the incidence of depression as measured by the Zung scale was mainly connected with an "incident somatic diagnosis" (Kukull et al. 1986). The exception is a study by Green et al. (1992), who in a London sample reported onset of depression over 3 years to be connected with smoking, loneliness and life dissatisfaction. Thus, four of five studies resulted in a association of incidence of depression in the elderly with a change in health status. Kennedy et al. (1990) and Turner et al. (1988) showed that the incidence of depression was largely independent of previous depressive symptoms.

In several cross-sectional studies disability appeared as the health aspect most strongly connected with depression. In several elderly samples of the general population regression or path analysis led to the disappearance of other risk factors such as age (Berkman et al. 1986; Blazer et al. 1991), being homebound (Bruce and McNamara

1992), pain and illness (Williamson and Schultz 1992) and left disability as the only risk factor for depression.

It is still possible to maintain that part of the association of incident disability with incident depression is due to disability being caused by depression, and that this association leads to selective institutionalization. In a 1-year longitudinal study of an elderly New York sample Gurland et al. (1988a) found that t_1-disability doubled the rate of incident depression, but that t_1-depression tripled the rate of incident disability.

There are, however, strong arguments against depression being a main cause of disability, although it may have a pathoplastic influence on its severity and course (Turner and Noh 1988; Gurland et al. 1988a, b). Firstly, the prevalence of illness and disability in old age is far higher than of depression (Gurland et al. 1988a, b; Caine et al. 1993; Turner and Beiser 1990). Secondly, the disabilities that in Gurland et al.'s study were preceded by depression comprised heart and respiratory disorder, cognitive impairment and stroke. These disorders and some malign tumours are known for their early association with depression, possibly by subtle organic changes and by side effects of medication (Sunderland et al. 1988, Kerr et al. 1969; Philpot 1990; Ouslander 1982).

A third reason is that depression increases linearly with the number of illnesses and the degree of disability (Welz et al. 1989; Lindesay et al. 1989; Gurland et al. 1988a, b; Hyer and Blazer 1982). A fourth reason is that in very old age the association of disability and depression is loosened. Turner and Noh (1988) found that in their Oklahoma sample the older, as opposed to the younger, disabled did not differ in the prevalence of MDD from a matched nondisabled control group. Gurland et al. (1988 b) noticed an impressive decline in prevalence of depression in disabled males and females from age 65 to 80+ years. Koenig et al. (1991) in a comparison of young and old medically ill patients described depressive symptoms as milder and MDD as less present in the older group. This fact may shed some light on the association of disability with depression: In the older old, disability may be "on time" and to a degree normative, whereas in the younger old it may still cause a severe psychological reaction. Felton and Revenson (1987) showed that with increasing age ways of coping with chronic illness became less dramatic and more accepting.

Still, it is plausible that mainly vulnerable subjects with a history of depression react with depression to disability. Very few investigators have addressed the question of lifetime depression as an indicator of vulnerability to depressive illness in the disabled. Stewart et al. (1965) studied the temporal sequence of illness and depression in young patients afflicted with a chronic life-threatening illness. They found a point prevalence of MDD of 27%. In 5 of 8 cases the onset of depression was connected with the onset of the disorder, and there were neither previous episodes nor a family history of depression. In control cases with milder somatic disorders, on the other hand, depression, if present, was not associated with the onset of the disorder. In the Turner and Beiser (1990) Ontario disabled sample mentioned previously (mean age 56 years); CES-D depression and MDD had a much higher point prevalence than in matched nondisabled controls. An inquiry into lifetime prevalence of depression found a tendency to a higher rate in the disabled ($P \leq 10$, time at risk controlled); but even when previous cases were deducted, actual depression was more frequent among the disabled.

We conclude that disability is a main cause of depression in old age and not vice versa, that the association weakens in very old age and that on the basis of two investigations, depression in disability or severe illness seems relatively independent of antecedent depression. Thus, it is more probable that the surplus of "institutional" depressives arise de novo, because somatic illness leads to depression, which is reinforced by the need to adapt to a new environment, than that the depressives enter institutions and disappear from the community because of disabilities that seem insuperable because of depression. This would mean that the decrease in MDD in the noninstitutionalized population is mainly authentic, and not due to depressives being at an appreciably higher risk for institutionalization.

Are depressed subjects lost by developing organic brain disorder?

The next question concerns the possible association of depression in old age with organic brain disorder, and hence with institutionalization and shortened life expectancy. Two surveys conclude on the basis of treatment data that subjects with senile dementia of the Alzheimer type are at a higher risk of depression than nondemented controls (Teri and Wagner 1992; Wragg and Joste 1989). Because these studies concern selected populations, we turn to epidemiological data in order to answer the question of whether there is a more than random association of dementia and depression (Table 4).

The data show that in the general population incipient dementia is connected with about twice the rate of depression (mainly defined by a cut off on a depression scale) than found among non demented controls. The question immediately arises as to whether depression is a risk factor for dementia or whether incipient dementia leads to depression. There are three methods that may give an answer to this problem: longitudinal studies of elderly depressives in the community, the outcome of hospitalized depressives and the investigation of elderly depressives by new brain-imaging methods.

Investigations of the first type are rare. Bickel and Cooper (1989) gave data on an 8-year follow-up of 65+-year-olds in Mannheim, Germany. Functional mental illness at first investigation did not carry a higher risk of later dementia than the absence of psychiatric disorder. Copeland et al. (1992) investigated the 3-year outcome of depression in Liverpool, England. Among a random sample of 1070 65+-year-olds there were 25 cases of "depressive psychosis". Of the survivors, 8% became organic cases, and of 82 cases of "depressive neurosis", 3.7%. In

Table 4 Studies of the general population on prevalence of depression in dementia

Author	Age (years	Location	Instrument	Result
Welz et al. (1989)	65+	Dunderstadt	Scale of dementia and depression	Beginning dementia doubles rate of cases
Forsell et al. (1994)	75+	Stockholm	DSM-III dementia, depression scale	More symptoms in mildly demented
Fuhrer et al. (1992)	65+	Bordeaux	Scale of dementia and depression	Odds ratio 2.26
Griffiths et al. (1987)	65+	Oxfordshire	Scale of dementia and depression	Rate twice as high as expected
Heeren et al. (1992)	85+	Leiden	DSM-III dementia, depression scale	Rate twice as high as expected
Lindesay et al. (1989)	65+	London	Scale of dementia and depression	Slight correlation
Skoog (1993b)	85+	Gothenburg	DSM-III dementia, depression scale	Rate almost doubled in mild dementia

NOTE: In all studies: not-demented controls

the whole sample the incidence of dementia was 9.2%, and depression did not seem to carry a major risk of dementia.

In longitudinal studies of hospitalized depressed patients, dementia does not occur more often than in the general population. In most follow-up studies dementia is not even mentioned as a separate category of outcome. Murphy (1983) found, over 12 months, 3% of depressed patients demented; Post (1962, 1972) found 26% demented after 8 years, and 3% of another sample after 1 year. Both authors concluded that there is no increased risk for dementia in the hospitalized depressed.

In case-control studies of Alzheimer's disease, previous depression does not appear as a risk factor, but as an early symptom (Jorm 1990). Still, there could be subtypes of old-age depression carrying a higher risk of dementia. The first type is late-onset depression. Its prevalence may have been overrated by clinicians: Data on lifetime incidence of depression given by several large epidemiological studies show that first incidence in old age is rare (Eaton et al. 1989; Burke et al. 1990). In the Edmonton Study (Bland et al. 1988b) at age 55 years, in women 97% of first episodes had taken place, and in men, 92%. In an Oregon sample (Lewinsohn et al. 1986a) the hazard rate for developing RDC major and minor depression dropped sharply after age 65 years, and was zero after age 75 years. Thus, even if late-onset depression carried a high risk of dementia, the impact on prevalence of the disappearance of this type of depression from the older population – because of an association with the organic brain syndrome – would be small.

A main difficulty in comparing early- and late-onset depression in the elderly is the control for age. In elderly depressives the late-onset group tends to be older than the early-onset group (Brodaty et al. 1991; Conwell et al. 1989). There is a general consensus from longitudinal studies of hospitalized patients that late-onset depressives without present cerebral impairment do not develop dementia more frequently than early-onset depressives of the same age (Roth and Kay 1956; Post 1962; Cole 1983; Greenwald and Kramer-Ginsberg 1988; Conwell et al. 1989; Brodaty et al. 1991), and that late-onset depressives have less familial loading and less personality disorder than early-onset depressives (Alexopoulos 1990; Post 1962; Brodaty et al. 1991; Brown et al. 1984; Musetti et al. 1989; Conwell et al. 1989; exception: Greenwald and Kramer-Ginsberg 1988). The latter characteristics seem to be the only ones that differentiate late- from early-onset depression. Brodaty et al. (1991) demonstrated that psychotic symptoms in depression, particularly delusions, were associated with current age and not with age at onset. Roth and Kay (1956) found in late-onset depressives extremely high rates of chronic somatic illness and disability, especially loss of vision, which preceded depression, and concluded that in persons with little genetic vulnerability physical impairment finally overpowered their lifelong resistance to depression. If, however, depression secondary to illness is excluded in the selection of cases, illness and impairment do not seem to be more frequent in late-onset depression (Brodaty et al. 1991; Greenwald and Kramer-Ginsberg 1988; Musetti et al. 1989; Conwell et al. 1989). Thus, the rare cases of genuine primary late-onset depression may not be a group with special symptoms and outcome.

Late-onset depression may still be associated not with the development of dementia, but with the development of subcortical cerebral damage as could be the case with late-onset mania (Young and Klerman 1992; Dhingra and Rabins (1991; Stone 1989; Charron et al. 1991). Comparisons of early- and late-onset depressives by computed tomography (CT) or magnetic resonance imaging (MRI) for white matter lesions, are, however, criticized as inconclusive because of patient selection for chronicity, lack of control for age, no exclusion of present signs of dementia and because of measurements that do not allow the comparison of different studies (Coffey et al. 1988; Krishan et al. 1988; Alexopoulos and Chester 1992; Bird et al. 1986; Baldwin 1993). In addition, nondemented elderlies of the normal population frequently demonstrate white matter changes which, however, are not associated with depression (Skoog 1993a), but with slight memory decrements (Ylikoski et al. 1993; Golomb et al. 1993).

"Depressive pseudodementia" is a second subtype of depression that is associated with dementia. In Copeland et al.'s study (1992) there were 30 cases with a combination of depression with mild or severe organic symptoms. After 3 years 52% had become cases of dementia. Several longitudinal studies of patients with "depressive pseudodementia" show that this subtype in nearly all cases precedes dementia not by causing it, but by revealing it (Stra-

berg 1978 in Stoppe and Staedt 1993; Kral and Emery 1989; Rabins et al. 1984; Emery and Oxman 1992; Mahendra 1985; Reding et al. 1985; McAllister and Price 1982; Alexopoulos et al. 1993). According to an analysis of depressive-symptom clusters in a Stockholm elderly population, the mildly demented when depressed had more in common with the depressed severely demented than with the depressed nondemented (Skoog 1993). It was even maintained that "depressive pseudodementia" is an artefact of an insufficient investigation of the memory defects of depressives, and that better methods would reveal coexisting dementia (O'Connor et al. 1990).

We therefore conclude that there is presently no evidence for depression being a risk factor for dementia, but for dementia being a risk factor for depression, and that elderly depressives thus do not disappear from the community because they are at a higher risk of a dementing illness that leads to earlier death or institutionalization.

Mitigation of severe depression with age

Angst *(unpubl. data)* in a 27-year longitudinal study of hospitalized unipolar depressives found that at death or last follow-up (mean age 71 years) 50% of these previously severely ill patients had been free from acute episodes for at least 5 years (Table 5). During this time half of this group had suffered from severe, and half from slight, residual symptoms.

A similar observation was made by Ciompi (1969) concerning 555 depressives hospitalized before age 65 years and followed up into old age. Of the survivors one third had recovered, generally with residual symptoms, one third had fewer and less severe episodes and one third was unchanged or worse. Particularly self-accusation, agitation and anxiety tended to disappear.

It is tempting to conclude from this data on mitigation of severe depression to the course of the milder disorders in the community. There are some findings that support this hypothesis. Gaitz and Scott (1972) found with the Langner scale that the intensity of expression of both positive and negative feelings declines with age. There are data that older subjects judge their own personality with more leniency (Hare and Shaw 1965; Mirkowsky and Ross 1992). Koenig et al. (1991), comparing younger and older VA hospital patients with depression, found milder symptoms in the older group.

Several other facts outside of psychopathology support the mitigation hypothesis. Various sources of stress are less sharply felt (Schaie 1983; Noack and Weiss 1993). Reported life events dramatically decrease with age (Markush and Favero 1974; Uhlenhut and Balter 1974; Uhlenhut et al. 1983), and while becoming more disruptive (death of a spouse, onset of illness or disability) are more often perceived as normal or natural in old age than at a younger age. Depression becomes more independent of the presence of social support (Blazer et al. 1992), and, while the social network restricts itself, older people are more satisfied with what is left than younger people are with their more extensive contacts (Henderson 1986). Lewinsohn et al. (1991) found in a population sample – admittedly with a strong admixture of volunteers – age negatively correlated with the presence of microstressors, life events, anxiety and hostility, and positively with adequate coping and life satisfaction.

Survivors into old age as a group thus seem to become psychologically more stable. Negative life events, personality, coping and satisfaction with social networks are considered risk factors, respectively protective factors for depression. Their development gives a rationale to a decline in the prevalence of severe depression in the senium.

Table 5 Mitigation of severe depression in old age (UP, $n = 186$ hospitalized, follow-up 27 years)

No recurrence for at least 5 years before death or last observation	*n*	%
GAS > 60 (Slight residual symptoms)	48	25.8
GAS ≤ 60 (Severe residual symptoms)	47	25.3
Total without episodes	95	51.1

From Angst, unpublished

Are there fewer but more severe cases of depression?

Diekstra (1989) and Katona (1994) showed that according to WHO data, in most countries there is a sharp increase in the incidence of suicide in males over age 60 years, which does not occur in females. Comparisons between younger and older male suicides result in fewer previous attempts, more violent methods, less intoxication, more MDD, more chronic somatic illness and more loss of a confidant in elderly suicides (Meehan 1991; Frierson 1991; Conwell et al. 1990). These findings lead to the question of whether, while incidence and prevalence of major depression and dysthymia decrease, the remaining cases take a particularly severe course. Previously in this paper we showed that symptomatology of depression does not change with age. Still, in terms of duration of episodes, chronification and relapse the outcome of depression could be worse in elderly than in younger subjects.

Angst et al. (1973) showed that in hospitalized subjects that continued to have depressive episodes into late life intervals shortened and episodes lengthened. For hospitalized depressives of any age, Angst (1988) found in a survey of longtime follow-up studies that after a first episode lasting recoveries took place in 15–30%. According to the same author (1992) it is probable that in the general population the same low rate of first episodes of MDD lead to a lasting recovery over a lifetime. In a Swiss population that was interviewed four times between ages 20 and 30 years, over 10 years, 70% of major depressive episodes were recurrent. The ECA study found, however, that after 1 year 76% of major depressives of all ages had recovered

Table 6 Rate of recoveries from depression at follow-up in elderly samples

Author	Age (years)	Diagnosis	Interval (years)	Recovered (%)
Hospitalized patients				
Post (1972)	60+	Clinical	3	30 (Lasting recovery)
Murphy (1983)	65+	RDC	1	35
Baldwin and Jolley (1986)	65+	RDC	3–6	22 (Lasting recovery)
Burvill et al. (1991)	Mean age 71	DSM-III	1	47
Hinrichsen (1992)	60+	DSM-III	1	49
Practice populations				
O'Connor et al. (1990)	75+	DSM-III	1	33
Kukull et al. (1986)	Mean age 79	Zung	1	50
General population				
Kennedy et al. (1991)	65+	CES-D	2	54
Copeland et al. (1992)	65+	GMS/AGE-CAT	3	35
Forsell et al. (1994)	75+	DSM-III	3	29

(Sargeant et al. 1990). Table 6 shows the rate at follow-up of recovered depressed elderly patients and depressed elderly subjects in the general population. Follow-up intervals are generally short. A large rate of recurrence/chronicity is associated with longer follow-up periods. None of the samples show the ECA recovery rate mentioned previously. Recovery, however, seems not to be less frequent in older than in younger subjects.

Other authors (Baldwin and Jolley 1986; Murphy 1983; Kennedy et al. 1991) found a chronic course associated with chronic somatic ailments. In Kukull et al.'s (1986) VA clinic sample a chronic course was associated with cataract, chronic pulmonary obstruction and alcoholism. The presence of severe illness may be an obstacle to an efficient treatment of depression (Philpot 1990), and thus a risk for chronicity.

Direct comparisons of the course of depression in younger and older subjects do not find any differences in course and outcome. Between Hinrichsen's (1992) hospitalized elderly and a control sample of mixed age (mean 32 years; Keller et al. 1982) no significant differences in outcome were found. In two studies of the general population with multivariate methods (Lewinsohn et al. 1986 b; Sargeant et al. 1990), duration of episodes and recovery after 1 year were independent of age. A cautious conclusion could be that the question of a worse outcome of depression in elderly than in younger subjects is still open, even if somatic health seems to exert a strong influence not only on incidence, but also on the course of depression (Cole 1990). The increase in male suicide incidence with old age thus remains unexplained. The retrospective analysis of suicide cases points to a connection with incurable disease and disability, bereavement and institutionalization (Katona 1994; Draper 1994). The contributions of personality, earlier vulnerability to depression and incipient organic syndromes to the suicides of older males, still await investigation.

Are major depression and dysthymia replaced by subthreshold depression?

In the absence of longitudinal studies we have to fall back on three cross-sectional comparisons of the prevalence of various levels of depression in general population groups. In a follow-up of the New Haven ECA sample the diagnoses of major and minor depression were given according to RCD criteria (Weissman and Myers 1980). At each age level (26–45 years; 45–65 years; 66+ years) the diagnosis of minor depression was given to 50% of all cases of depression, and there was no relative increase in the rate of minor depression by age. In an analysis of North Carolina ECA data on current dysphoria, Broadhead et al. (1990) created two syndromes below the level of MDD and DSM-III dysthymia: minor depression defined by depressed mood and/or anhedonia and at least one of the DSM-III symptoms of depression, and minor depression without depressed mood and/or anhedonia, but at least one of the symptoms mentioned above. At age 60+ years the prevalence of all diagnostic categories was lower than at an earlier age, with the exception of minor depression without mood disturbance (Table 7a). The latter inflated the total prevalence at age 60+ years to one third, and equalized it to that in middle-aged and younger subjects. The more severe kind of minor depression was reported by about half of each age group of depressives as in the Weissman and Myers (1980) study. Thus, there is no spectacular increase of minor depression and only a shift towards a very mild mood disturbance may be inferred. A somewhat different picture appears in the third study by Romanosky et al. (1992), which also regards an ECA subsample. Here the 65+-year age group in comparison with the younger subjects reported more DSM-III depressive disorders below the MDD level and more being "actively depressed". This diagnosis involved PSE-9 symptoms or the clinical judgement of apathy or mournful appearance. It was given to about one third of the elderly and one fifth

Table 7a Six-month prevalence of subthreshold depression by age (ECA sample, North Carolina)

	Age (years)		
	18–44 %	45–59 %	60+ %
a) MDD	2.3	2.1	0.9
b) Dysthymia	2.3	2.9	1.6
c) Minor depression with mood disturbance	6.8	7.4	4.5
d) Minor depression without mood disturbance	23.0	18.0	25.7
Total	34.4	30.4	32.7
$\frac{c}{a + b + c}$	60	60	64

From Broadhead et al. 1990

Table 7b Point prevalence of subthreshold depression by age (ECA sample, Baltimore)

	Age (years)			
	18–24 %	25–44 %	45–64 %	65+ %
a) MDD	0.0	1.2	2.0	0.5
b) DSM-III depressive disorders below MDD level (dysthymia, adjustment reaction, NOS)	1.7	3.7	2.9	5.0
c) Actively depressed	0.5	1.4	1.4	2.4
Total	2.2	6.3	6.3	7.9
$\frac{c}{a + b + c}$	23	22	22	30

From Romanoski et al. (1992)

of the younger population with a diagnosis of depression. In this study a shift appears towards subclinical forms of depression, which are possibly relevant (Table 7b).

The question immediately arises as to whether subjects with subthreshold depression are "cases", i.e. whether old persons in this category need medical attention.

Validation of subthreshold depression

Broadhead et al. (1990) tried to validate the syndromes of minor depression mentioned previously by assessing comorbidity with anxiety disorders, number of disability days and course over 1 year. Whereas minor depression with mood disturbance was associated with comorbid anxiety disorders, relevant disability and persistence, minor depression without mood disturbance, on the other side, did not appear as a "useful diagnostic category". Dysphoria (anhedonia), one depressive symptom and a duration of 2 weeks seem to be one possible threshold separating cases from noncases.

Other trials at validation also point to a necessary minimum of symptoms and the presence of dysphoria or anhedonia. Blazer and Williams (1980) used a depression scale (OARS) allowing a DSM-III diagnosis of major depressive disorder and one of "substantial dysphoria", which was defined as the presence of at least three symptoms of dysphoria out of seven, and zero to three out of eight DSM-III symptoms of depression. Of a random sample of Durham County elderlies, 11% had substantial dysphoria, and 3.7% had MDD. The diagnosis of "substantial dysphoria" was validated by felt "need for a counselor", "need for medication for nerves" and actually taking such medication. The evidence for "caseness" at this level seems weak, possibly because of the inclusion of "depressives" with mere symptoms of dysphoria.

Later Blazer (1989) and Blazer et al. (1987) diagnosed DSM-III-MDD and dysthymia with the DIS in the ECA North Carolina sample and below this level "symptomatic depression" (at least two of the core symptoms of MDD present) and mixed depression and anxiety (depressive and at least two anxiety symptoms). The latter two groups were significantly different from subjects with mere dysphoria in terms of negative life events, loneliness, poor physical health and use of benzodiazepines.

The following studies regard institutionalized older subjects. Hyer and Blazer (1982) validated a subthreshold syndrome of one to three depressive symptoms and "low life satisfaction" with reports of behaviour. It was associated with nurses' reports of low cooperation, low communication and few social contacts. Parmelee et al. (1989) compared a diagnosis of "minor depression" – corresponding to DSM-III dysthymia and adaptation reaction – with the results of clinical interviews, and found 72% agreement for both presence and level of depression.

The "Gurland-Copeland school" (Copeland et al. 1986; Gurland et al. 1983) introduced a computerized diagnostic system consisting of the interviews of CARE, the Geriatric Mental State (GMS) and the criteria of AGECAT. This procedure resulted in much higher prevalences of depression in elderly subjects than with DSM-III criteria. In 1987, 1070 old people from Liverpool received a diagnosis of "depressive psychosis" in 2.9% and of "depressive neurosis" in 8.3%, totalling 11.3% (Copeland et al. 1987 b). Later, a similar result was obtained with the same population, but prevalences of "neurotic depression" varied considerably between London and New York (Copeland et al. 1987b; Dewey et al. 1993; Saunders et al. in press). When the same data were analysed with both DSM-III criteria and the GMS-AGECAT system, DSM-III did not diagnose a part of GMS-AGECAT milder depressions (Copeland and Griffith-Jones 1990). A validation of GMS-AGECAT depressions with psychiatrists' diagnoses resulted in satisfactory confirmation of caseness (Copeland et al. 1986, 1988). More importantly, at a 3-year follow-up of the earlier Liverpool sample (Copeland et al. 1992), of the cases with depressive neurosis 29% were again diagnosed as cases, and another 11% as subcases of depression, whereas a diagnosis of depression was given to 4% of subjects who at first investigation were neither depressive cases nor subcases. Gurland et al. (1983, 1988a) introduced the concept of "pervasive depression" defining it

as a persistent dysphoria associated with hopelessness and disproportionate worrying. It was found in about 13% of older subjects living in U.S. communities, and in 15.9% of those living in a London ward (Livingston et al. 1990). For validation, subjects were interviewed by psychiatrists and judged to be clinical cases in 80–90%.

Henderson et al. (1993) explored criteria of depression according to draft ICD-10, DSM-III and DSM-III-R in the at least 70-year-old population of Canberra including nursing home residents. ICD-10 mild depression was given a definition similar to DSM-III dysthymia (two items of dysphoria and at least two other depressive symptoms), but with the time criterion of at least 2 years reduced to at least 2 weeks. This definition resulted in a prevalence of mild depression of 1.8% for subjects living at home, and of 4.1% for the institutionalized. These rates are low in comparison with the 15.2% found by Kay et al. (1985) in Hobart with GMS and AGECAT.

Henderson et al.'s (1993) population was presented with a depression scale consisting of all symptoms that are part of the ICD-10 and the DSM-III criteria. Twenty-two percent reported at least four symptoms during the last two weeks (but not necessarily all the time), and 16% at least five. The symptom score was correlated with worse health, more disability, loss of vision and hearing, present pain, earlier depressive states (according to an informant) and neuroticism (+0.51). A threshold separating cases from noncases was not given.

These are preliminary and relatively unsophisticated validation data. Still, they show that subthreshold depression in old age may be a relevant category, and that many subjects who suffer from a variety of depressions, which are all at a symptom and duration level below DSM-III depression and dysthymia, are different from controls in associated psychopathology, impairment, somatic health and risk of later psychopathology.

Context of the decline in prevalence of major depression and dysthymia

Table 8 shows that according to ECA data (Robins and Regier 1991), most psychiatric disorders are at their highest for 1-year and lifetime prevalence either at age 18–29 years or at age 20–44 years, and then decrease to a minimum prevalence at age 65+ years. The disorders with highest prevalence at age 18–29 years are alcoholism (1-year prevalence), generalized anxiety, obsessive compulsive disorder, antisocial disorder and drug abuse (not presented). Phobias in females and OCD decline less from late-middle into old age than the other disorders.

An early decline in prevalence was found in other epidemiological surveys as well. As in the ECA, the Edmonton study (Bland et al. 1988a) used the DIS/DSM-III method and found a strong gradual decline in the 6-month prevalence of antisocial disorder and alcoholism in men up to age 65+ years, but a very modest decrease in female phobias, and even an increase in female OCD from age group 55–64 years to 65+ years. The National Comorbid-

Table 8 *General decrease in prevalence of nonorganic psychiatric disorder with age (ECA study). y 1-year prevalence; LT lifetime prevalence*

	MDD		Dysthymia		Schizophrenia		Bipolar I		Alcoholism (*men*)		Antisocial *personality*		Panic (*women*)		Generalized anxiety		Phobia (*women*)		Obsessive-compulsive disorders	
	y (%)	LT (%)	y (%)	LT (%)	y (%)	LT (%)	y (%)	LT (%)	y (%)	LT (%)	y (%)	LT (%)	y (%)	LT (%)	y (%)	LT (%)	y (%)	LT (%)	y (%)	LT (%)
Rate of decline from age 30–44 years to 45–64 years	41	47	–	5	60	56	83	79	43	25	87	62	42	39	(+5)[a]	–	28	24	52	33
Rate of decline from age 45–64 years to 65+ years	61	65	–	53	66	70	50	66	62	33	100	79	64	70	61	–	24	20	16	45
Age group with highest prevalence (years)	30–44		–	30–44	30–44		30–44		18–29 (y) 30–44 (LT)		18–29		30–44		18–29	–	30–44		18–29	

From Robins and Regier (1991)
[a] Increase

ity Survey used the CIDI interview to generate DSM-III-R diagnoses up to age 54 years, and whereas higher lifetime prevalences resulted than in the ECA study, a decline in lifetime prevalence for affective disorder took place from age 35–44 years to 45–54 years, for anxiety disorder from age 25–34 years to age 35–44 years, and for asocial disorder from age 15–24 years to age 35–44 years (Kessler et al. 1994).

As in depression, also in other disorders, many causes in different combinations lead to a decline in prevalence by age. Patients with schizophrenia and bipolar illness have reduced life expectancies, and in survivors as a group the course of the illness is usually mitigated (Angst 1988; Bleuler 1972; Ciompi 1969; Tsuang et al. 1985). Both factors, heightened mortality and mitigation, also influence the course of alcoholism and antisocial behavior (Ernst 1989). In addition, institutionalization may reduce the prevalence of these disorders in the general population. Häfner (1992) showed the change in the symptoms of schizophrenia with age: severe emotional upheavals give way to fixed delusional systems, rigid attitudes and stereotyped habits. The comparison of the prevalence of disorders in the general populations by age reveals the same trend: The emotionally intense disorders of panic and general anxiety become less frequent, while there is a much slower decline or even an increase in phobias and OCD, which are interpreted as habits acquired to ward off emotions. Thus, the falling off of DSM-III major depression and dysthymia seem to be a part of a general psychological development associated with age. The relative constancy of OCD and phobias is an argument against it being shame or lack of psychological mindedness that keep back older subjects from reporting depression. It is certainly easier to admit to the ubiquitous depressive symptoms than to the more exotic compulsive thoughts and actions.

Conclusion

Let us try to answer our initial question: Is there a real decrease in prevalence of DSM-III major depressive disorder and dysthymia in old age? The hypotheses of the decline being entirely an artefact of forgetting is refuted because of a decrease in point prevalence of MDD in old age. The hypothesis that lay interviewers wrongly ascribe depressive symptoms to somatic illness is refuted as well, because the decline persists in the absence of all exclusion criteria. The hypothesis that depression in old age is masked by hypochondriasis is not confirmed by the comparison of symptoms of older and younger depressives. The hypothesis that depressives are at a considerably higher risk for institutionalization than the nondepressed lacks plausibility, because according to longitudinal studies disability and illness are the most important risk factors that precede depression. The same is true for dementia that triggers depression, but not vice versa. Thus, in our opinion the high rates of depressives in institutions is mainly due to the selection of this population for somatic illness and organic brain disorder that cause depression, and not to a selection for depression. These rates incidentally prove the ability of the DIS/DSM-III method to diagnose depression also in the presence of somatic illness.

Older depressives have about twice the mortality of nondepressed controls, mainly because of the association of depression with somatic illness. Selective death thus contributes to some degree to a disappearance of depressed elderlies from the community. A mitigation of the course of depression with age was shown in hospitalized samples by Angst (1988) and Ciompi (1969). As in the ageing general population life events become more rare and social relationships more satisfactory, there is reason to believe that in the community the course of depression is mitigated in the senium. According to epidemiological data, the first incidence of MDD and dysthymia in old age seems to be a rare event. It is probable, however, that subthreshold depression frequently arises de novo in the context of somatic illness, and that former major depression and dysthymia glide below a diagnostic threshold by becoming more transient (Gurland 1976; Gianturco and Busse 1978) and losing some of their symptomatic variability. The rare investigations where the prevalence of subthreshold depression can be compared by age in part confirm this hypothesis. Efforts are presently made to validate subthreshold depression in old age, but as yet with little methodological stringency as far as reliability and validation at symptom level is concerned. According to the criteria of impairment, course and clinical judgement of caseness, it is possible that the lasting presence or frequent recurrence of two depressive symptoms together with substantial dysphoria delimit clinically relevant subthreshold syndromes from mere mood swings. That subthreshold depression may be a relevant diagnosis in old age is confirmed by the discrepancy between the prevalence of dysphoria and depressive symptoms, and of major depression and dysthymia (Katona 1994; Katona and Bell 1990).

The decline of the prevalence of DSM-III major depressive disorder and dysthymia thus, in our view, is mainly authentic and not an artefact, the more so as all psychiatric disorders decline with age. The decrease is most visible in those associated with strong emotions, aggression or substance ingestion. In accordance with this development, intermittent and less colorful depressive states strongly associated with somatic illness may accumulate in old age at a subthreshold level (Blazer 1994). The challenge of extending the diagnosis of depression with well-defined criteria into this field should be taken up by those in charge of developing a common diagnostic language in psychiatry.

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