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# Depression and Cognitive Impairment in Institutionalized Older Adults

José Antonio Camacho-Conde<sup>a</sup> José Manuel Galán-López<sup>b</sup>

<sup>a</sup>Center of Medical and Health Research, University of Málaga, Málaga, Spain; <sup>b</sup>Department of Nursing, University Hospital of Ceuta, Ceuta, Spain

#### Keywords

 $\label{eq:cognitive} \begin{array}{l} \mbox{Cognitive decline} \cdot \mbox{Cross-sectional study} \cdot \mbox{Depression} \cdot \\ \mbox{Prevalence} \cdot \mbox{Psychosocial factors} \end{array}$ 

## Abstract

Background: In the last three decades, the relationship between depression and cognition in geriatric patients has been a popular topic among researchers and clinicians. Clinical and epidemiological research has focused on the identification of risk factors that could be modified in pre-dementia syndromes, at a preclinical and early clinical stage of dementia disorders, with specific attention to the role of depression. The objective of this work was to determine the relationship between depressive disorder and cognitive deterioration in institutionalized older adults. Methods: In this descriptive, correlational study, data were gathered from two nursing homes in the province of Jaen (Spain), from a random sample of 140 older adults (70 nondependent and 70 dependent). The variables were measured using comprehensive geriatric assessment, the Cambridge Cognitive Test (CAMCOG), and the Geriatric Depression Scale (GDS). Results: Depression was correlated with cognitive level in the nondependent older adult sample (r = -0.471, p = 0.004).

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karger@karger.com www.karger.com/dem Age was inversely associated with the score obtained in the CAMCOG of the nondependent older adult sample (r = -0.352, p = 0.038). The functional capacity in several activities of daily living was correlated with the score obtained in the CAMCOG in each of the two groups. Depression was more prevalent in the dependent than in the nondependent older adults (82.85 vs. 57.14%). No association was observed between institutionalization time and the score obtained on the cognitive and affective scales (GDS and CAMCOG) in both groups (GDS-nondependent, r = -0.209, p = 0.234; CAMCOG-nondependent, r = 0.007, p = 0.967; GDS-dependent, r = 0.907). **Conclusion:** Depressive symptomatology is associated with cognitive deterioration. Depression is prevalent in institutions that care for older, more dependent adults.

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## Introduction

In the last three decades, the relationship between depression and cognition in geriatric patients has been a popular topic among researchers and clinicians [1–6]. Despite the growth of research, significant questions re-

José Antonio Camacho-Conde

Center of Medical and Health Research (CIMES), University of Málaga c/Marqués de Beccaria, 3 (Campus de Teatinos), MemoryLab, 2nd Building ES-29010 Málaga (Spain) jacamacho@uma.es

main unanswered in the cognition-affect relationship. Clinical and epidemiological research has focused on the identification of risk factors that could be modified in pre-dementia syndromes, at a preclinical and early clinical stage of dementia disorders, with specific attention to the role of depression [7]. Research in multiple disciplines has been conducted to determine the relationship between depression and cognition, including neuropsychology, gerontology, psychiatry, and neurobiology. Most studies are cross-sectional [1, 8-13] and are based on samples of patients with dementia, either with or without depressive disorders [1, 14-16]. A recent longitudinal study showed that neuropsychiatric symptoms - including depression - are risk factors or clinical indicators of preclinical dementia syndrome [6] and that depressive symptoms can be predictors of visual memory deficits in middle age [17].

Depressive mood is a broad construct and includes many clinical subtypes. However, the current classifications use a single definition to describe a major depressive episode [18]. Affective disorders, and more specifically major depressive disorder, are characterized by a depressed mood and/or a loss of interest or pleasure for at least 2 weeks [19], which might be accompanied by sudden changes in weight, sleep problems (insomnia or hypersomnia), agitation or psychomotor delay, feelings of guilt and worthlessness, lack of energy, thoughts of death, and decreased cognitive abilities, such as concentrating or making decisions [20].

Cognition depends on functions specialized for the acquisition of information about goals and the means to achieve them. These functions exert a top-down influence on the lower-level automatic processes that mediate sensory analysis, memory storage, and motor outputs, orchestrating and directing them toward a given goal. The prefrontal cortex is a brain structure that reaches its greatest complexity in the primate brain and seems to have a central role in cognitive control [21]. Two major types of cognitive dysfunction can be identified in depressive disorders: cognitive biases, understood as distorted information processing leading to depressive thinking errors, and cognitive deficits encompassing areas such as attention, memory and learning, executive functions, and drive [22]. In addition to affective symptoms, it has also been found that people with major depression have a decrease in various cognitive processes [23], such as processing speed [24-26], sustained attention [27], executive functions [28-32], visual memory [33], working memory [34], verbal fluency [35], episodic memory [12], visuospatial memory [35], selective attention or inhibitory

control [27, 36], and attentional arousal [37], among others. In addition, the existence of two different patterns of attention deficit in clinical depression has been suggested: some depressives have a disorder of inhibitor distraction, and others show abnormalities in the processing of resources [38].

The term "pseudodementia" was coined by Kiloh [39] to describe those cases that closely mimic the picture of dementia. Since then, the term has been used to describe the cognitive profile of various psychiatric disorders, especially depression [40] in old age [41], which present with cognitive deterioration in dementia. Since the term came into academic use, there have been several arguments against its usage [42, 43] as well as in favor of it [44]. Pseudodementia is not an irreversible deficit since when there is an improvement in neuropsychological tests, the patient's mood is normalized. The patients suffer mainly from inattention disorders, as they find themselves withdrawn and engrossed in depressive thoughts. The use of the term "pseudodementia" has been successful and is commonly used to refer to these cases, but we think it can be misleading. The difficulty in the diagnostic assessment of pseudodementia and pervasive developmental disorder is particularly evident in older adults because of the additional confusion created by age-related cognitive deficits [45]. A meta-analysis study conducted by Kang et al. [45] addressed the important issue of diagnostic confusion between dementia and pseudodementia. A clearer picture of this diagnostic dilemma was provided by Dufouil et al. [46], who delineated four clinically important scenarios that complicate the differentiation between these two clinical states: (1) cognitive changes in older adults blur the distinction between normal aging and early signs of pervasive developmental disorder; (2) cognitive impairment frequently accompanies depression and can be severe enough to cause confusion between dementia and depression; (3) signs of some neurologic diseases associated with the progressive decline (e.g., Alzheimer's disease [AD] and Parkinson's disease [PD]) have symptoms that overlap with depression; (4) dementia and depression can coexist. Once depression has been diagnosed, in the absence of the marked cognitive deficits typical of dementia, it should simply be assigned the term depressive disorder, and no other diagnosis is necessary [47].

Memory, perception, verbal fluency, and nominal tasks were identified as sensitive to the effects of depression in several studies [6, 17, 48]. In all studies, those with depression scored significantly below normal controls, but significantly above the group of AD patients [49].

However, studies of older people affected by psychiatric or neurologic pathologies lack representativeness and, therefore, do not allow for extrapolating their results to the general population of older adults. Longitudinal studies examining depression over time provided a variety of findings associated with reducing depression following treatment [50], oscillating between no effect and complete change of cognitive deficits [1].

Some of the drawbacks of studies that have found memory deficits associated with depression are due to the fact that they were performed in patients with severe depression compared to normal controls and used tests that require a great effort of memory and that are dependent on the patient's motivation [51–53]. Although there are more studies that matched their depressed and nondepressed groups in some geographical characteristics, others did not directly control the effects of variables such as age, sex, and education, which made it impossible to directly assess the relationship between demographic and cognitive variables.

Likewise, this is also a problem when studies compare the extremes of the continuum (severely depressed versus not severely depressed) and do not directly control the effects of variables such as age, sex, and education. To minimize these issues, depression and cognition should be used as continuous variables, thus making it easier to identify relationships between the variables. In this way, the effects of sociodemographic variables on cognition could be directly controlled [1].

Studies that do not find associations between depression and memory are characterized by comparing people with less severe depressive symptoms and using simpler memory measures, such as recognition or immediate memory measures [54, 55]. These results are consistent with the hypothesis formulated by Hasher and Zacks [56] and supported by findings indicating that depressed people exhibit memory deficits in processes that require effort, but not in automatic memory processes [57].

Longitudinal studies provide data that can be used to determine whether older people who are depressed have a higher risk of cognitive impairment than those who are not depressed, controlling the effect of their cognitive condition at the start of the study and other characteristics such as age, sex, and level of instruction. In other words, we can estimate the relative risk of cognitive impairment associated with depression. Although longitudinal population studies are scarce [6, 46, 58–61], in Europe, we can find the studies of Zunzunegui et al. [52] and Paterniti et al. [62], and more recently, in North America, the study of Ezzati et al. [63], who considered depression as a predictive factor of cognitive impairment in older adults.

Cross-sectional studies on depressive symptomatology in Spanish older adults [64, 65] consistently estimate a prevalence two to three times higher than in other European populations, despite using different measuring instruments. On the contrary, Spanish studies on the prevalence of moderate to severe cognitive deficits obtain a prevalence similar to those of other countries: between 6 and 14% of the population over 65 years old [66–70].

Depression is one of the most frequent problems among Spanish older adults [71], as revealed by the different epidemiological studies on the prevalence of depression in the population of older adults, with rates ranging from 5 to 20% in the community population, figures that can double or triple among the institutionalized older adult population [72–74]. Depressive disorders in residential centers for the older adults are a frequent problem, but are underdiagnosed and undertreated, with a consequent decrease in quality of life. Its high recurrence rate as well as the lack of proper recognition and treatment contribute to greater morbidity and mortality, being a frequent cause of suicide [75–78].

The fact that older adults were away from home and subject to the specific routine of the nursing home might suggest some predisposition to depression. To this is probably added a late result in the diagnosis of depression, and above all, the routine of institutionalization, which is substantially different from that of the family and the home settings [79]. However, we should keep in mind that institutionalized older adults usually have low levels of social and family support and a high prevalence of other comorbidities, which is likely to aggravate the depressive symptoms preexisting at the time of institutionalization [80]. Various studies have reported factors - when associated with institutionalization - that can increase the prevalence of depression, including feelings of abandonment in the nursing home [81], economic difficulties or limitation of the possibility of using money [82], and isolation or distance from one's usual social network, which leads to a relevant uprooting and adaptation effort [73, 83, 84], significant changes in lifestyle with increased stress [85], forced adaptation to a regulation and norms that can negatively affect privacy and autonomy and give rise to feelings of disability, loss of freedom, or low level of life satisfaction [85], lack of objectives and activities [86], and increased selfperception of health deficits and increased anxiety about death [85, 87]. To this, we should add the various reasons that lead to the institutionalization of older adults, such as the existence of a chronic disease that determines a signif-

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Table 1. Affiliation data of institutionalized older adults

Indicators	Nondependents	Dependents			
Voluntarity level upon admission					
Voluntary income	54 (77.14%)	44 (62.86%)			
Involuntary income	16 (22.86%)	26 (37.14%)			
Marital status prior to admission					
Widower	28 (40%)	44 (62.86%)			
Married	24 (34.28%)	6 (8.56%)			
Separated/divorced	6 (8.57%)	18 (25.72%)			
Single	12 (17.14%)	2 (2.86%)			
Current marital status					
Widower	54 (77.15%)	48 (68.57%)			
Married	8 (11.43%)	2 (2.86%)			
Separated/divorced	4 (5.71%)	18 (25.71%)			
Single	4 (5.71%)	2 (2.86%)			
Residence before admission	1				
Institution	6 (8.57%)	4 (5.71%)			
Sons	8 (11.42%)	10 (14.28%)			
Spouse	12 (17.14%)	22 (31.43%)			
Alone	30 (42.85%)	26 (37.14%)			
Other family	12 (17.14%)	8 (11.43%)			
Others	2 (2.86)	- (-)			
Values are presented as	n (%)				

icant disability with loss of autonomy, a high age, and a lack of socio-family support in some cases due to the death of the spouse [73], which results in a direct increase in depressive disorder rates [88], as well as the negative value burden and the level of voluntary income in nursing homes [89]. Knowing the effects that institutionalization might have on older adults, daytime outpatient care would help families who do not wish for their older relative to be institutionalized. In line with this, health care professionals should also contribute to decreasing the need for institutionalization by providing preventive, interdisciplinary, and community cares aimed at promoting the necessary self-sufficiency and independence for older adults to accomplish self-care at home, close to their relatives, in their usual and family environment.

Also, the usual frequency of somatic problems in institutionalized older adults and the increase in the use of drugs with depressive capacity contribute to the increase in the prevalence rates of cognitive impairment in this population group [90]. This is combined with the comorbidity of diseases and decreased health in the older adults [91], which is also associated with the onset of depression, especially disorders related to the deficiency of some sense [74, 92–96] or urinary incontinence [97]. Also, hearing loss increases the risk of neurocognitive impairment [98]. Finally, it should be noted that late-onset depression has a lower hereditary component and is more strongly influenced by the social, family, and vital environment [99].

Here we investigate the relationship between depressive symptomatology and cognitive impairment in a population of institutionalized older adults. Institutionalized older adults were chosen as the focus of study for three reasons: (1) Research has consistently shown that the prevalence of depression in older adults is two to three times higher than among older people living in the community. (2) Depression in institutionalized seniors is often not as severe as in psychiatric patients, and similarly, antidepressants are less likely to be used. (3) Assessment of depression and cognition in the institutionalized population is useful and has clinical applicability for gerontology professionals.

## **Subjects and Methods**

## Participants

The sample was made up of 140 older adults of Spanish nationality – 70 nondependent (60% female and 40% male) and 70 dependent (54.29% female and 45.71% male). The nondependent group was 60% female (63–99 years old, mean age 78.51  $\pm$  6.69 years) and the dependent group was 54.29% female (67–99 years old, mean age 82.63  $\pm$  6.60 years). All participants were recruited from two residential institutions for older adults in the province of Jaen. The length of institutionalization was longer in the older dependents versus the older nondependent – means of 4.65 years (SD 56.53 months) and 4.75 years (SD 57.82 months), respectively. The average widowhood times of the nondependent and dependent groups were 12.69 and 8.47 years, respectively.

To counteract the strange effects of psychiatric variables and to facilitate the administration of tests and the validity of the results, residents with the following pathologies were not included: epileptic disorders, schizophrenia, unspecified psychotic disorders, mental retardation to varying degrees, aphasia, and very severe dementia. Older adults with mild hearing loss and mild vision disorders were not excluded since these do not interfere with the reliability of the tests. Two participants were unable to complete the entire evaluation because of severe dementia, and their data were excluded from the study.

The sociodemographic variables (sex, age, education level, socioeconomic level, origin, and marital status) were collected through the center's database, all under the guidelines dictated by Organic Law 15/1999, of December 13 [100], Protection of Personal Data, and controlled by randomization. The most relevant variables are shown in Table 1.

## Measuring Instruments

The scales used to assess depression and cognitive impairment were the Geriatric Depression Scale (GDS) [101] and the Cambridge Cognitive Test (CAMCOG) subscale of the Cambridge Mental Disorders of the Elderly Examination (CAMDEX) [102] and are described below.

#### Validity and Reliability of the Scales

The CAMCOG subscale [102] of the CAMDEX interview is a cognitive battery that objectively evaluates a wide range of superior functions: orientation, language, memory, praxis, attention, abstract thinking, perception, and calculation. This scale consists of 60 items (63 in the Spanish version) and includes all of the Mini-Mental State Examination (MMSE) [103], and in the Spanish version, three others have been added to include the Spanish adaptation of the Cognitive Miniexam [104]. The effectiveness of the CAMDEX in both clinical [105–107] and epidemiological studies [108, 109] led to translation, adaptation, and validation in the Spanish population [102].

The CAMCOG [102] and its revised version (CAMCOG-R) [110] have been shown to have high sensitivity and specificity to discriminate between organic and nonorganic cases. Its main advantages over the MMSE [103] are that it (1) covers a wider range of cognitive functions, (2) detects middle stages of cognitive impairment, and (3) avoids the "roof" effect [9]. However, due to a "floor" effect, test yields overlap between patients with mild dementia and those with moderate/severe dementia [109]. Setting the cutoff point at 70/71, the sensitivity is 86.7, and the specificity is 73.3. The global predictive value is 80. When applying the 66/67 cutoff point, the sensitivity is 86.7 and the specificity is 86.7. Its predictive value is 86.7 [102]. The internal consistency of the Spanish version of the CAMCOG-R [110, 111] total score was 0.81 (measured by Cronbach's alpha coefficient). Elimination of any of the items of the CAMCOG-R scale did not reduce the reliability to <0.80. Split-half reliability with Spearman-Brown correction for equal length forms was 0.83. As the difference between forms was observed in variance distribution, an additional Guttman split-half coefficient for reliability correction was calculated and yielded a similar value of 0.81 [112]. The convergent validity of the CAM-COG-R was estimated as 0.75 (p < 0.001), and the divergent validity of the CAMCOG-R was estimated as 0.30 (p < 0.001) [112].

The GDS [101] is the only self-report built specifically for the assessment of depression in older adults. It is composed of 30 items, without somatic items. In this way, a problem present in most self-reports - the confusion of depressive symptoms with the common physical symptoms in older adults - was avoided [76]. Later empirical studies confirmed these ideas. This shows that the GDS of a sample of older adults suffering from a physical illness distinguished between depressed and nondepressed elders and also between depressed older adults and nondepressed older adults [78]. If the cutoff point is set to 11, a sensitivity of 0.84 and a specificity of 0.95 is obtained [79, 80]. Applying a cutoff point of 14 gives a sensitivity of 0.80 and a specificity of 1 [69]. The Spanish adaptation [113] of the GDS [114] shows internal consistency, high temporal stability, and high convergent validity ( $\rho_{x,y} = 0.86$ ), and its 15-item abbreviated version [115] shows high internal consistency (Cronbach's alpha = 0.99) and intraobserver reliability (weighted kappa = 0.95), acceptable interobserver reliability (weighted kappa = 0.65), and appropriate values for convergent ( $\rho = 0.61$ ), divergent ( $\rho = 0.23$ ), and concurrent validities.

#### Procedure

The collection of psychological, medical, and social data was carried out through a cross-sectional investigation in the two residences, following the protocol above and measured by the study's psychologist.

Cognitive function was measured by the CAMCOG and depressive symptomatology was assessed by the GDS (both Spanish

adaptations) [102, 116]. The psychological data concerning cognitive and affective evolution were offered by the psychologists of the centers. The sociodemographic data were mostly extracted through the Psychosocial Variables Questionnaire of the author, corroborated and complemented with the files of social workers. The clinical data were provided by the doctors at each center. To avoid the twilight effect after lunch in the gerontological population, the tests were applied in the morning.

#### Statistical Analyses

Statistical analyses were done using the statistical program IBM SPSS Statistics version 25.0 for Windows, and results are presented as descriptive and correlational statistics (sample size, percentage, and Pearson's correlation). *p* values <0.05 were considered statistically significant.

## Results

When applying the Pearson test, we observed no interaction between age and the GDS score of the nondependent older adults (r = 0.142, p = 0.415) nor with cognitive impairment (r = -0.234, p = 0.177).

We detected no significant relationship between age and GDS score in the sample of dependents (r = 0.133, p = 0.447), but there was a significant inverse relationship with the CAMCOG score (r = 0.352, p = 0.038); the CAM-COG score tends to decrease (indicating greater cognitive impairment) with advancing age.

No association was observed between institutionalization time and the score obtained on the cognitive and affective scales (GDS and CAMCOG) in both groups (GDSnondependent, r = -0.209, p = 0.234; CAMCOG-nondependent, r = 0.007, p = 0.967; GDS-dependent, r = 0.251, p = 0.152; CAMCOG-dependent, r = -0.021, p = 0.907).

Among the 6 subjects who had depressive symptoms without cognitive impairment, it was subsequently found that 4 presented cognitive impairment. Among the 11 who presented neither depression nor cognitive impairment, we found that 27.27% presented cognitive impairment and depression combined.

Functional capacity was analyzed through activities of basic (BADL) and instrumental activities of daily living (IADL). The results obtained in both are shown together (normal nondependent versus impaired nondependent), specifying the mean, the average criterion (without help, with difficulty, needs help, and cannot), and the percentage (Tables 2, 3).

On the other hand, there was no significant relationship between the functional capacity in the BADL and the score obtained in the GDS of the nondependents (r = 0.159, p = 0.361). There was a direct correlation between

Table 2. H	BADL	of the non	depende	nt elderly	accord	ing to	degree	of
autonom	y							

**Table 3.** IADL of the dependent elderly by degree of autonomy

Indicators	Autonomous	Some physical impairment
To get up/to lay down		
Without help	32 (100%)	36 (94.74%)
With difficulty	- (-)	- (-)
Need help	- (-)	2 (5.26%)
Dressing/undressing		
Without help	32 (100%)	36 (94.74%)
With difficulty	- (-)	- (-)
Need help	- (-)	2 (5.26%)
Cleanliness		
Without help	32 (100%)	36 (94.74%)
With difficulty	- (-)	- (-)
Need help	- (-)	2 (5.26%)
Bath/shower		
Without help	32 (100%)	30 (78.94%)
With difficulty	- (-)	4 (10.53%)
Need help	- (-)	4 (10.53%)
Toilet use		
Without help	32 (100%)	34 (89.47%)
With difficulty	- (-)	4 (10.53%)
Need help	- (-)	- (-)
Up/down stairs		
Without help	28 (87.50%)	26 (68.42%)
With difficulty	4 (12.50%)	10 (26.32%)
Need help	- (-)	2 (5.26%)
To walk and to stroll		
Without help	28 (87.50%)	24 (63.16%)
With difficulty	4 (12.50%)	12 (31.58%)
Need help	- (-)	2 (5.26%)
Eating		
Without help	32 (100%)	34 (89.47%)
With difficulty	- (-)	- (-)
Need help	- (-)	4 (10.53%)

Values are presented as n (%). BADL, basic activities of daily living.

the functional capacity in BADL and the score obtained in the CAMCOG of the nondependent group (r = 0.463, p = 0.005), which means that the higher the functional capacity score in the BADL, the higher the CAMCOG score. According to the above, the functional capacity in these types of activities results in a better cognitive level and vice versa. There were no significant relationships between functional capacity in BADL in dependents and the scores obtained using the GDS (r = -0.109, p = 0.532) and the CAMCOG (r = 0.089, p = 0.611).

No significant relationship was found between the functional capacity in the IADL and the resulting scores in the GDS (r = -0.312, p = 0.068), but a significant rela-

Indicators	Autonomous	Some physical impairment
To read		
Without help	26 (81.25%)	22 (57.80%)
With difficulty	2 (6.25%)	6 (15.78%)
Need help	4 (12.50%)	10 (26.32%)
To write		. ,
Without help	22 (68.75%)	22 (57.80%)
With difficulty	8 (25.00%)	6 (15.78%)
Need help	4 (5.26%)	10 (26.32%)
To use the phone		
Without help	30 (93.75%)	34 (89.47%)
With difficulty	- (-)	- (-)
Need help	2 (5.26%)	4 (10.53%)
Shopping		
Without help	32 (100%)	18 (47.36%)
With difficulty	- (-)	2 (5.26%)
Need help	- (-)	18 (47.36%)
To use medication		
Without help	32 (100%)	24 (63.16%)
With difficulty	- (-)	2 (5.26%)
Need help	- (-)	12 (31.58%)
To use public transport		
Without help	30 (93.75%)	18 (47.36%)
With difficulty	2 (6.25%)	2 (5.26%)
Need help	- (-)	18 (47.36%)
To handle money		
Without help	28 (87.50%)	30 (78.95%)
With difficulty	4 (12.50%)	2 (5.26%)
Need help	- (-)	6 (15.79%)
To visit the doctor		22 (24 210)
Without help	28 (87.50%)	32 (84.21%)
With difficulty	4 (12.50%)	-(-)
Need help	- (-)	6 (15./9%)
10 trim one s toenails	22 (69 750/)	12(21 = 00/)
with difficulty	22(68.75%)	12(31.58%)
With difficulty	0 (23%) 2 (6 25%)	4(10.33%)
Need neip	2 (0.25%)	22 (37.89%)

Values are presented as *n* (%). IADL, instrumental activities of daily living.

tionship was found between the functional capacity in the IADL and the resulting score in the CAMCOG (r = 0.682, p = 0.000) in the sample of nondependents. The latter result indicates that the existing direct relationship expresses a better cognitive capacity when greater functional capacity is preserved in these activities, and a better cognitive level facilitates the development of these activities.

This conjunction was also presented in the sample of dependents, i.e., the relationship between the functional capacity in the IADL and the CAMCOG score was significant (r = 0.631, p = 0.000), but this relationship was

not significant when using the GDS score (r = -0.144, p = 0.409).

The comorbidity associated with depression in nondependent older adults was 45.71%, compared to 74.28% in dependent older adults. The most representative pathologies in both groups were hypertension in 40%, diabetes in 22.86%, hip fractures in 20%, depression in 17.14%, chronic obstructive pulmonary disease in 17.14%, stroke in 11.43%, heart disease in 8.57%, AD in 8.57%, PD in 8.57%, vertiginous syndrome in 8.57%, mild hearing loss in 8.57%, varicose syndrome in 5.71%, and headaches and/or migraines in 5.71%.

In the analysis of the drugs associated with depression, we found that 26.66% of a subsample of nondependents take a medication with possible depressive action, and among these, 100% have depression. Among the subsample of dependents, 53.33% are prescribed medication with possible depressive action, and also 100% of these have depression.

The prevalence of depression in nondependent older adults was 57.14%, compared to 82.85% in dependent older adults. The mean age of those with depression was 79.55 years among nondependents and 82.72 years among dependents. In the nondependent older adults, 72.22% of women and 41.18% of men were depressed. In the dependent older adults, 89.47% of women and 75% of men were depressed.

The mean scores obtained in the CAMCOG were  $66.80 \pm 14.25$  for the sample of nondependent older adults and  $34.62 \pm 16.18$  for the sample of dependent older adults. In the nondependent older adults, the mean GDS score was  $11.60 \pm 6.00$ , compared to  $16.94 \pm 6.05$  among dependent older adults. The mean scores in the CAM-COG were  $77.66 \pm 4.90$  for the sample of nondependent and nonimpaired older adults and  $55.35 \pm 11.63$  in the sample of nondependent and impaired older adults. The average GDS scores were: nondependent, nondepressed (mean = 5.93, SD = 2.52); nondependent, depressed (mean = 15.85, SD = 3.94); dependent, nondepressed (mean = 7.16, SD = 1.47); and dependent, depressed (mean = 18.96, SD = 4.40).

We found that among nondependents who scored  $\leq 69$ on the CAMCOG, 94.11% (16/17) had depression; with a CAMCOG score <58, 100% (7/7) had depression. In the group of dependents with a CAMCOG score  $\leq 69$ , 82.85% (29/35) had depression; among those scoring <58, 87.10% (27/31) had depression; among those scoring <45, 88.46% (23/26) had depression; and among those scoring  $\leq 30$ (17/17), 100% had depression.

In both groups, we observed an increase in cognitive impairment as the GDS score increased (in the nonde-

Depression and Cognitive Impairment in Institutionalized Older Adults pendent group, when the GDS score was  $\leq 11, 78.95\%$  had cognitive impairment; when the GDS score was  $\geq 14$ , 85.71% had cognitive impairment; and with a score < 20, 100% had cognitive impairment). In contrast, this difference disappeared in the group of dependents, in which, with a score of  $\geq 11, 100\%$  had cognitive impairment.

In the nondependent residents, the degrees of deterioration in the two groups (none, minimum, slight, moderate, or severe) were: no deterioration, 51.42% (n = 18); minimum deterioration, 2.86% (n = 1); slight deterioration, 22.86% (n = 8); moderate deterioration, 17.14% (n = 6); and severe deterioration, 5.71% (n = 2). Conversely, the dependent residents showed a higher percentage of deterioration in the higher-grade criteria: no residents with no deterioration and minimal deterioration, 11.43% (n = 4) with slight deterioration, 22.86% (n = 23) with severe deterioration.

When comparing the GDS and CAMCOG scores of nondependent older adults, we detected a significant relationship (Pearson's correlation test, r = -0.471, p = 0.004). We detected no such CAMCOG-GDS relationship for the dependent older adults (Pearson's correlation test, r = 0.156, p = 0.372).

The Student *t* test was applied in the two independent samples, obtaining a bilateral significance of 0.005, so that the null hypothesis was rejected. This indicates that there is a likely relationship between depression, cognitive impairment, and the development of dementia.

#### **Discussion and Conclusion**

Our results indicate that depressive symptomatology correlates with cognitive impairment, and through a retrospective analysis, in a subsample, we detected considerably impaired development in one in three older adults. This finding is consistent with longitudinal studies of large samples that predict the incidence of dementia [49, 52, 63]. Our findings are in line with those of other studies [82, 83] that have categorically demonstrated a cognitive decline associated with depressive states in various forms. A recent meta-analysis by Rock et al. [30] revealed moderately significant cognitive deficiencies in executive functions, memory, and attention in patients with depression compared to patients in the control group (without depression). In addition, moderately significant cognitive impairments in executive functions and attention tasks, and minor nonsignificant impairments in memory, were found to persist in patients whose depres-

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sive symptoms had remitted, indicating that cognitive impairment occurs separately from episodic depression to major depressive disorder. Some recent studies showed that depression predicts conversion to cognitive impairment over time [117, 118] or the development of AD [119, 120].

Our findings indicate high depressive symptoms in institutionalized older adults. The prevalence of depressive disorder exposed in the present work is high and similar to that noted in other studies [113, 116, 121, 122], and depressive disorder was more common in the dependent group of older adults. These findings are consistent with those of other studies that have reported an incidence of depression four times higher among institutionalized than noninstitutionalized older people [123].

There are significant differences in the BADL and IADL between nondepressed and depressed nondependents and between nondepressed and depressed dependents. Both depressive symptomatology and cognitive impairment are related to a greater deficit in executive functions [124]. The basic and instrumental functional capacity is significantly related to the cognitive level of the nondependent group, and the advanced functional disability situation in the dependent group correlates with the cognitive level of impairment, since the degree of limitation and dependence of the dependents is higher [72, 125].

Also, this is facilitated by the connection between the structural and/or environmental obstacles that the older adults face when they are institutionalized and assume the role of dependents. It is necessary to carry out an efficient assessment with a general evaluation technology and measuring devices that offer us the possibility of determining the degree of limitation in ADL. Graphic representation of the functional state can facilitate the compression and global vision of the disability areas, with the aim of rehabilitating them more effectively and efficient-ly [125, 126].

The wide variety of instruments used to measure depression and cognition might also help explain the inconsistent results found in some studies [12]. Assessment tools should be used that are more sensitive to the effects of subtle alteration in older adults. For example, La Rue et al. [127] reported that the Fuld Object Memory Evaluation distinguished depressants from nondepressants, while the Benton Visual Retention Test and the English Paired-Associate Learning Test did not. Also, many studies have used cognition and brief screening tests (Neurocognitive Status Exam, MMSE) that might not be sensitive to the more subtle effects of cognition and depression. Depression is a frequent symptom in neurodegenerative diseases (AD, PD, dementia with Lewy bodies, frontotemporal and cerebrovascular dementia), both from the initial and more advanced stages of the disease, which can accompany cognitive symptoms [15, 16, 74, 128, 129].

Attributing a series of symptoms to aging and considering them normal, when sometimes they are manifestations of a depressive condition, is referred to by Butler and Lewis [130] as gerontophobia, accounting for the finding that although the real frequency of geriatric depression is high, it is not detected operatively. The progressive aging of the population and the increased risk of depression in recent generations means that in the future, the frequency of depression in older adults will be even greater and will become a public and/or mental health problem of great relevance. Given this health problem, it is necessary to make availability early detection instruments [124] as well as to initiate an early therapeutic intervention in residential institutions by gerontology professionals [124] in the same way as it has been carried out from the centers of social or health care [131].

A better understanding of the relationship between depression and alterations in cognitive function can be useful for the early diagnosis of cognitive impairment and, in case a causal relationship is established between depression and subsequent cognitive impairment, for eventual prevention through the adequate treatment of depressive symptoms, which results in greater life expectancy and better quality of life [72].

Also, due to the gradual aging of the population and the increase in the institutionalization of older adults. further research should be carried out to assess how it affects traditional models compared to other more innovative models [132]. The relationships between depressive symptoms and institutionalization should be further investigated, since in our study it was not possible to determine whether institutionalization alone favors depression or whether there are a greater number of depressed older people institutionalized by their families. In a recent study conducted by Frade et al. [133], based on a sample of institutionalized and noninstitutionalized older adults, the high prevalence of depressive symptoms among the institutionalized aged group was confirmed. The same relationship can be considered when thinking about cognitive impairment and institutionalization [124]. Recently, Hoffmann et al. [134] showed that more than half of nursing home residents suffer from dementia, which is about 19-fold higher than for those living in the community. Further studies should focus on whether prevalences also

increase in higher age groups in nursing homes and on regional differences.

Finally, it is important to consider that depressive symptoms might be an early manifestation of rather than a risk factor for dementia and AD, arguing that the underlying neuropathological condition that causes cognitive impairment or dementia also causes depressive symptoms [135]. Cognitive impairment is estimated to occur in 94% of patients in the acute phase of depression, but to persist in as many as 44% of patients in remission [136]. In this scenario, at least in certain subsets of older patients, end-of-life depression, mild cognitive impairment, and dementia could represent a possible clinical continuum [7].

In our study, the presence of a high prevalence of depression coincides with the results of other studies conducted in the institutionalized gerontological population [53, 99–101]. Depressive symptomatology has not been able to be associated with functional capacity in either group, but this has been proven in longitudinal studies. However, it remains unclear whether there is a significant relationship between basic and instrumental functional capacity at the cognitive level of the nondependent group with the cognitive level of impairment [22, 102– 106].

Nondependent older adult patients who enter with depression seem to have a greater tendency to develop cognitive impairment and dementia than those who enter without depressive symptoms [22, 28, 30]. The comorbidity of pathologies associated with cognitive impairment is greater in the group of dependents, and depressive symptoms might go unnoticed in both groups when attributed to other coexisting processes [45, 107, 108]. The appearance of depressive symptoms might be the result of the side effects of drugs with a possible depressive capacity [45, 53].

The high frequency of worries and fears might favor the appearance of depressive symptomatology in nondependent and dependent patients, with the latter likely being greater due to disability devices (e.g., dysfunctionality in ADL, adoption of the role of dependence, distance from the social network, infrequent visits) [53]. There are significant differences in the BADL and IADL of life between nondepressed and depressed nondependents and between nondepressed and depressed dependents. The GDS maintains its diagnostic validity when there is no cognitive impairment. Also, our results confirm that the GDS scale loses validity when we apply it to a population that presents moderate to severe and severe cognitive impairment or AD [77]. Depression is significantly and in-

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versely related to cognitive impairment, i.e., the higher the GDS score, the lower the CAMCOG score. Therefore, the greater the degree of depression, the greater the degree of cognitive impairment.

The generalizability of the study is limited by the small number of older adults in the sample. We did not attempt to make exhaustive conclusions and believe that this work should stimulate future, more extensive and longitudinal cross-sectional research with a national scope. The findings were extrapolated to older adults who are able to complete cognitive tests and answer the GDS questions. Taken together, such data indicate that cognitive impairments resulting from depression cannot be considered an epiphenomenon entirely secondary to low mood symptoms. On the contrary, such deficiencies should be considered as a "target" for future interventions.

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#### Statement of Ethics

The Ethics Committee of the Equality and Social Welfare Council of the Andalusian Government (Spain) and the University of Jaen Ethical Committee (IRB approval number JAE1234) approved the study. The study was carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki) for human experiments. All patients provided written informed consent to participate in the study.

#### **Conflict of Interest Statement**

The authors declare no conflict of interest.

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#### **Author Contributions**

J.A. Camacho-Conde drafted the manuscript and participated in the study concept and design, the acquisition of subjects and/or data, the analysis and interpretation of data, and the preparation of the manuscript. J.M. Galán-López participated in the preparation of the manuscript.

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