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Emotional Decoding Abilities in Alzheimer's Disease: A Meta-Analysis

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Abstract. Studies on emotional processing in Alzheimer's disease (AD) have reported abnormalities in emotional decoding. However, it remains unclear whether the impairment depends on a general cognitive decline that characterizes these patients or is an independent deficit. We conducted a comprehensive meta-analysis of existing studies that compared AD patients with age-matched healthy older adults (HOA) on measures of emotional decoding abilities. Our first goal was to quantify the magnitude of the AD patients' deficit. The second goal was to identify variables that may modulate the deficit, including emotional task design and participants' characteristics. The random-effects model analysis on 212 effect sizes indicated that AD patients showed significant impairment in emotional decoding abilities. This deficit is consistent regardless of the emotional task, stimuli, type of emotion considered, or disease severity. After we controlled for cognitive status, the emotional performance in AD patients was still poorer than that in HOA. The effect size of emotional performance was significantly lower when the cognitive status was considered than when it was not. Thus, our results suggest that impaired emotion processing in AD patients cannot be solely explained by the cognitive deficit. These findings provide evidence that progressive neuropathological changes characterizing the disease could affect emotional processing, which may suggest that clinicians should be sensitive to the emergence of impairments in emotional decoding. Further research that addresses the limitations of existing studies is needed to draw conclusions about methodological issues and the impact of the AD patient's depression symptoms on emotional decoding.

Keywords: Alzheimer's disease, emotion, meta-analysis

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INTRODUCTION

Alzheimer's disease (AD) is a neurodegenerative disorder characterized by neuronal cell loss, neurofibrillary tangles, and senile plaques that first develop within the entorhinal cortex and hippocampus [1]. In parallel with progressive impairment of global cognitive functions, particularly episodic memory [2], clinical reports suggest massive emotional disturbances [3]. Several authors have hypothesized that emotional disorders may be the expression of a more global emotional processing deficit [4, 5]. This assumption is supported by neuroimaging findings showing an alteration of the emotional brain in AD patients [1, 6–9].

Consequently, an extensive literature this last decade suggests a decline in emotional processing in AD patients [10–18]. Compared with healthy older adults

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(HOA), AD patients are impaired in recognizing emotion, especially facial expressions, as revealed by numerous studies [15, 19, 20]. Although 61% of studies show a deficit of emotion recognition, 39% show similar performances for emotion decoding in AD and in HOA [19]. This discrepancy raises questions about a possible global deficit in emotional decoding abilities in AD. Consequently, the current meta-analysis aims to determine whether there is a deficit in emotional decoding abilities in AD and to identify factors that potentially modulate the deficit. In particular, the influence of such factors related to task design and changes in participant characteristics associated with the disease remain to be better understood.

Traditionally, researchers have investigated the performance of AD patients with tasks such as emotion identification (labeling or naming) [10, 15], discrimination [21], selection, and matching [14, 22, 23], partly by using the Florida Affect Battery (FAB) [24]. Even for a specific type of task, both significant deficits and preserved abilities to decode emotions have been observed among studies (Table 1) [19]. Several cognitive components that are engaged in such emotional decoding tasks are thought to be implicated in the problems with performing these tasks in AD patients [20]. Although most researchers assessed the performance of emotional decoding by using facial stimuli, a wide variety of situations and other stimuli have also been used: cartoon drawings [16], prosody [25], audiotaped stories depicting emotion-provoking situations [26], and videotaped vignettes that include body movements, gestures, faces, and prosody [15, 16]. Thus, emotional decoding abilities have been assessed in visual (e.g., facial expressions) [5, 27], auditory [12, 22], and audiovisual modalities [15]. The variability of emotional stimuli could lead to discrepancies, as some stimuli could be decoded more easily than others. Indeed, stimuli depicting more realistic emotional situations may enrich, both temporally and structurally, the input properties and enhance the perception of emotion [28]. Studies based on emotional decoding from prosody [12] and video clips [15] found that performance was similar in the AD patient group to that in the HOA group, whereas a deficit was observed for decoding tasks in which facial expression was used [12, 15]. Pictures with standardized facial expressions are thought to poorly reflect the context of the natural environment, preventing AD patients from correctly identifying and interpreting the emotion [15]. In contrast, prosodic language adds supplementary dynamic information that allows easier identification of emotion [12].

An important issue is to determine whether the impairment of AD patients is related to a specific type of emotion. Two hypotheses have been proposed in the literature. First, the ability to decode positive emotions (i.e., happiness) is preserved in AD, whereas the ability to decode negative emotions is impaired [5, 20, 29]. Second, the deficit occurs only for several specific emotions. For instance, a large part of the AD literature demonstrated a decline in the ability to identify negative emotions such as fear [4, 14, 15, 20, 23, 30, 31], but a preservation of the ability to identify disgust [15]. For other emotions, the results are less consistent. Compared with HOA, AD participants may be impaired for decoding sadness, anger, and surprise, as revealed in several studies [14, 20] but not shown in others [4, 22]. In the present meta-analysis, we aim to shed further light on these issues by examining whether people with AD are impaired in decoding specific emotions (e.g., fear) and not impaired in decoding others (e.g., happiness, disgust).

Another classical assumption is that AD patients' poor performance on emotional tasks does not necessarily indicate impairment in emotional decoding abilities, but rather a global cognitive deficit that impacts general processes, including emotional processing [10, 12, 16, 21–23, 29, 32, 33]. Consequently, the emotional deficit in AD may result from cognitive declines in memory and visuospatial skills, rather than being representative of a primary deficit of emotional abilities [10, 16, 22]. Indeed, when the emotional decoding performances are adjusted for general cognitive functions, there are no significant differences between AD and HOA for tasks such as emotional selection and matching [22] or emotional naming [15]. Deficits in emotional decoding also seem to mirror language deficits [10, 25, 34]. Moreover, numerous researchers have examined impairments in perceptual abilities as a potential cognitive deficit suspected to interfere with emotional decoding scores [10, 12, 16, 21–23, 29, 33]. When visuospatial abilities were controlled for, some authors still reported significantly poorer performance in AD patients [14] for the decoding of facial emotion, whereas others did not find such an effect [10]. Overall, declines in visual perception have been suspected to contribute to difficulties in emotion processing in AD in some studies [10, 12, 16], although other studies have refuted this suggestion [11, 14].

The relationship between the level of emotion decoding abilities and the level of dementia severity in AD patients also remains unclear. The progression of AD pathology is generally measured by means of

Table 1
Description and main results of studies investigating emotional decoding abilities in patients with Alzheimer's disease (AD) and age-matched healthy older adults (HOA)

Authors	Number of participants	Emotions considered in the study	Stimuli	Emotional tasks	Main results[a]
Albert, Cohen & Koff [10]	AD = 19 HOA = 19	Happy, surprise, neutral, sadness, disgust, anger, fear	Cartoon drawing, face, story listening	Discrimination	AD < HOA (face)
				Matching	AD < HOA (cartoon-story listening)
				Naming	AD < HOA (face)
				Selection	AD < HOA (face)
Allender & Kaszniak [11]	AD = 13 HOA = 10	Interest, happy, shame, sadness, disgust, anger, contempt	Face, prosody	Naming	AD < HOA (face)
				Naming/Discrimination	AD < HOA (prosody)
Bediou et al. [43]	AD = 10 HOA = 50	Happy, fear, anger, disgust	Face	Naming	AD < HOA
Bucks & Radford [22]	AD = 12 HOA = 12	Happy, sadness, fear, anger, neutral	Face, prosody	Discrimination	AD = HOA (face)
					AD < HOA (prosody)
				Matching	AD = HOA (face)
				Matching face to prosody	AD < HOA
				Matching prosody to face	AD < HOA
				Naming	AD = HOA (face)
					AD < HOA (prosody)
				Selection	AD < HOA (face)
Burhnam & Hogervorst [23]	AD = 13 HOA = 13	Happy, sadness, fear, surprise, anger, disgust	Face	Matching	AD = HOA
				Naming	AD < HOA (fear, happy, sadness)
				Naming	AD = HOA (anger, disgust, surprise)
Drapeau et al. [30]	AD = 7 HOA = 16	Happy, sadness, fear, surprise, anger, disgust	Face, prosody, music	Naming	AD = HOA (prosody, music, faces: happy, anger, surprise)
					AD < HOA (faces: fear, sadness, disgust)
Fernandez-Duque & Black [38]	AD = 8/9 HOA = 10	Happy, surprise, disgust, sadness, fear, anger, neutral	Face	Discrimination	AD < HOA
				Naming	AD = HOA
Granato et al. [13]	AD = 12 HOA = 12	Happy, anger, surprise, sadness, fear, disgust, neutral	Face	Naming/Matching	AD < HOA
Hargrave, Maddock & Stone [14]	AD = 22 HOA = 14	Happy, sadness, fear, surprise, anger, disgust	Face	Discrimination	AD < HOA
				Matching	AD < HOA
				Naming	AD < HOA

Table 1
(*Continued*)

Authors	Number of participants	Emotions considered in the study	Stimuli	Emotional tasks	Main results[a]
Henry et al. [15]	AD = 24 HOA = 30	Happy, surprise, fear, sadness, disgust, anger, neutral	Face, video clip	Naming	AD < HOA (Face) AD = HOA (Video clip)
Horley et al. [54]	AD = 20 HOA = 20	Anger, happy, sad, surprise	Prosody	Naming	AD < HOA
Koff et al. [16]	AD = 23 HOA = 19	Neutral, happy, sadness, anger, fear	Prosody, cartoon drawing, face, gestures, body movements	Matching	AD = HOA (cartoon drawing)
				Match prosody to face	AD < HOA (cartoon drawing)
				Naming	AD = HOA (prosody)
				Naming	AD < HOA (face, gestures, body movements)
Lavenu et al. [4]	AD = 20 HOA = 12	Happy, sadness, fear, surprise, anger, disgust, contempt	Face	Discrimination	AD = HOA
				Naming	AD < HOA (fear and contempt)
Ogrocki, Hills & Strauss [33]	AD = 17 HOA = 15	Anger, happy, sadness, neutral	Face	Naming	AD = HOA
Phillips et al. [20]	AD = 27 HOA = 30	Happy, sadness, fear, surprise, anger, disgust	Face	Discrimination	AD < HOA
				Naming	AD < HOA
Roberts et al. [25]	AD = 20 HOA = 11	Surprise, anger, sadness	Prosody	Naming	AD < HOA
Roudier et al. [21]	AD = 31 HOA = 14	Happy, anger, sadness, neutral	Face	Discrimination	AD = HOA
				Selection	AD < HOA
				Naming	AD < HOA
Shimokawa et al. [17]	AD = 25 HOA = 12	Anger, happy, sadness, surprise	Cartoon drawing	Matching/Selection	AD < HOA
Spoletini et al. [31]	AD = 50 HOA = 50	Happy, sadness, anger, fear disgust, neutral	Face	Naming	AD < HOA
Taler et al. [36]	AD = 10 HOA = 10	Anger, happy, sad	Prosody	Naming	AD < HOA
Testa et al. [34]	AD = 27 HOA = 20	Happy, surprise, sadness, anger, neutral, disinterested	Prosody	Discrimination	AD < HOA
				Naming	AD < HOA
Werheid et al. [69]	AD = 18 HOA = 18	Happy, neutral, anger	Face	Naming	AD = HOA
Zaitchik et al. [26]	AD = 20 HOA = 20	Sadness, happy	Stories listening	Naming	AD < HOA

Note: [a]AD < HOA means that AD patient's emotional performances were significantly lower than HOA. AD = HOA means that AD patients emotional performances were similar from those of HOA.

an index that considers the cognitive impairment level as assessed by the Mini-Mental State Examination (MMSE) [35]. Despite a few studies suggesting no strong relationship between MMSE scores and emotional performance in AD [21, 25, 36], a significant correlation was revealed by other authors [14, 20].

Finally, a wide panel of affective disturbances characterizing AD patients [3, 37] was suspected to be associated with the ability to decode emotion [25, 34, 38]. In particular, depression symptoms were suggested to interfere with the ability to process emotional information [25], as confirmed in studies of patients with major depression and bipolar disorder [39].

In the present study, we therefore conducted a comprehensive meta-analysis of existing studies on emotional decoding abilities in AD. We included 23 studies in which the performances of AD patients were quantitatively compared with those of HOA, and we calculated effect sizes corresponding to the change in emotional decoding ability. Our first goal was to quantify the magnitude of the deficit by dealing with the statistical power issue (small effect size used in clinical studies) [40]. The second goal was to examine whether or not the presence and size of a deficit depends on the characteristics of the tasks used to assess emotion decoding (i.e., task design, stimuli, emotion) and on the characteristics of the AD samples (i.e., cognitive status, depression symptoms). Regarding task features, we expected that AD patients would be significantly impaired regardless of the task used (i.e., matching, selection, discrimination, naming), as the underlying cognitive processes involved in each task are thought to be affected in the disease. However, as processing demands could vary between tasks, we were also interested in assessing the extent to which the task used could moderate the size of the deficits in AD patients. Another task feature that could moderate the size of these deficits is the kind of stimuli involved. For this purpose, we compared the more widely used stimuli in the literature (i.e., audiotaped story, face, cartoon drawing, video clip, and prosody) with each other. We expected to observe a lower deficit for decoding stimuli that are more likely involved in the realistic situations an individual may encounter in the natural environment [12]. Stimuli such as prosody, video clips, or audiotaped stories are potentially easier to decode than cartoon drawings or standardized faces that may present less realistic information. In addition, the richness of the emotional stimuli could potentially influence the findings. Stimuli providing a context in which additional emotional cues are available could modulate the ease of decoding emotions [15]. Thus, we hypothesized that the effect size could be modulated by the stimuli being considered. Regarding the specific emotions involved in the decoding tasks, we expected to confirm findings from the existing literature indicating that the ability to decode some emotions are preserved in AD (e.g., happiness, disgust) and that patients are particularly impaired in decoding fear emotions.

In addition, we attempted to assess to what extent the expected lower emotional decoding performances of AD patients compared with those of HOA could be attributed to changes in the global cognitive system that characterizes AD patients, as well as to their depression symptoms. We expected that AD patients' deficits would be lower when between-group[1] differences in cognitive status are controlled for, and when between-group differences in depression symptoms are controlled for. Moreover, consistent with the hypothesis of a primary impairment in emotion decoding abilities, we also expected AD patients' emotional decoding performance to remain significantly lower than those of HOA when the influence of the two potentially confounded variables is controlled. Finally, we tested the hypothesis that AD patients' deficits in emotion decoding significantly correlate with the progression of the pathology, as indexed by the MMSE (Fig. 1).

METHODS

Literature search procedure

Studies were identified through a computerized literature search of the PubMed, PsycARTICLES, PsychINFO, Dissertation Abstracts International, and Psychology and Behavioral Sciences Collection databases from 1989 through September 2011. We used the most inclusive combination of terms, as follows: "emotion* AND Alzheimer." More than 1,400 references were retrieved. A brief review of the abstracts for all the references indicated that 79 articles and nine dissertations reported at least one experiment in which the performance of emotion decoding abilities in AD patients might have been compared with the performance in HOA. None of the dissertations could be obtained from electronic databases or from their author following our request by email. We simultaneously sent a request for unpublished studies by email to the mailing list for the International

[1] In this article, between-group differences refer to differences between AD patients and HOA.

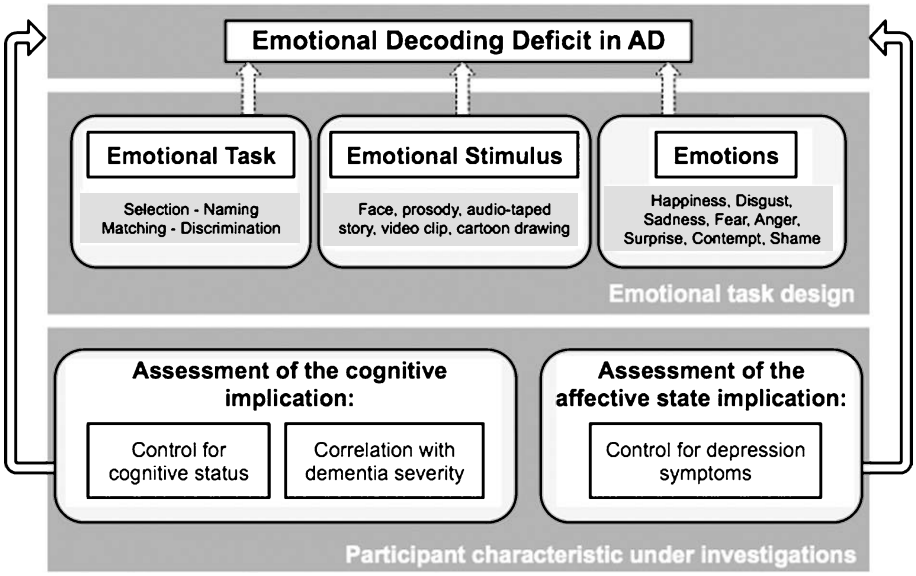

Fig. 1. A framework of potential factors modulating emotional decoding performances in AD. Emotional decoding abilities were tested to discover whether there is a deficit in AD for emotional decoding performance compared with HOA. The emotional task design was tested across the type of emotional task, the stimuli, and the emotion used. To assess the extent to which the expected lower emotional decoding performance is modulated by the characteristics of AD participants, we investigated the affect of cognitive impairments by controlling for the deficit in cognitive status in AD patients and measuring the correlation of emotional performance with dementia severity. The assessment of the influence of the affective state in emotional performance was measured by controlling for depression symptoms.

Society for Research on Emotion and to the French list of researchers and clinicians in psychology on the Psy-16 mailing list. Our call for unpublished studies gave us access to three additional references (a dissertation, two master's degree dissertations, and an oral communication). We then read the 79 reports available in French or English.

Complete coverage of relevant studies was ensured by checking whether the papers referenced in a synthesis of the literature on emotion perception in AD [19], as well as in every study we included in the meta-analysis, have already been located during the computerized search. No additional reference emerged, supporting the validity of our literature search procedure.

Inclusion criteria

We examined all of the references to determine which studies were eligible for inclusion in the meta-analysis. To be included, every study had to meet the following criteria: (a) report original empirical data; (b) include a pathological sample of AD patients (diagnosed using the criteria recommended either by the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (NINCDS/ADRDA) [41], or by the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) [42], patients with mild cognitive impairment were not included); (c) compare AD with age-matched HOA; (d) include at least one measure of explicit emotional decoding performances (given the memory deficits that characterize AD patients, we were interested in emotional decoding tasks involving only minimal short-term memory load; thus, only emotional decoding tasks requiring the participant to respond during or immediately after stimuli presentation were included [38, 43]; (e) include at least one of the six basic emotions [44]; and (f) contain enough statistical information to compute or reconstruct an effect size of the difference between AD patients and HOA in the performance obtained on emotional decoding tasks. Useful information includes means and standard deviations, t tests, F tests, r values, χ^2 values, proportions, and exact p values derived from one-way tests comparing the performances of AD patients and HOA. It excludes statistics obtained from analyses that include additional variables, such as multiple regressions and factorial analyses of variance, when one or more of the nonfocal factors are individual difference factors [45, 46].

When a study met all the inclusion criteria but the paper lacked some of the statistical information

necessary to compute an effect size, the missing data were requested from the authors. We also asked for the statistical information necessary to compute an effect size for each emotion separately, whenever the information was not already communicated in the research report. Information concerning the population characteristics was also requested (i.e., score related to MMSE or dementia severity, depression symptoms, and perceptive abilities), as well as the information necessary to compute an effect size controlling for between-group differences on any of these variables.

Recorded variables and coding

Many studies involved several tasks and several emotional stimuli on which the performance of AD patients and HOA were compared. As a result, several effect sizes could be obtained from each study. For the purposes of the meta-analysis, each effect size was coded along with information describing the population characteristics, as well as the kind of emotional decoding task used, the stimulus and modality used, and the emotions involved.

Population characteristics

Each effect size was recorded along with several characteristics of the HOA and AD samples. Population characteristics included (a) sample sizes, (b) age, (c) information related to cognitive status, (d) information related to depression symptoms, and (e) information related to the severity of the disease.

Concerning the information related to cognitive status, two cognitive variables were considered: global cognitive functions and perceptual abilities. We used the MMSE as an index of global cognitive function. Perceptual abilities were measured through nonemotional matching and discrimination tasks [19]: the Benton Test of Facial Recognition [47], the facial gender discrimination task [38, 43], and the identity discrimination tasks of the FAB [24].

Concerning the information related to depression symptoms, several indicators of mood disorders were considered. The Hamilton Rating Scale for Depression [48], the Geriatric Depression Scale [49], the Cornell Scale for Depression in Dementia [50], depression severity derived from the Cambridge Examination for Mental Disorders of the Elderly [51], or the Beck Depression Inventory [52] were coded.

The severity of the disease was assessed using AD patients' MMSE scores. Other indicators of dementia severity (e.g., CDR scores) were too rarely reported in the studies to be used as moderator variable in the present meta-analysis.

Control for cognitive impairment and depression

Each effect size was recorded along with a variable representing whether it was derived from a design which did versus did not control for cognitive status. Between-group differences in cognitive status were considered to be controlled for when an indication was made of nonsignificant between-group differences in either perceptual skills or global cognition in the study the effect size came from. In addition, between-group differences were considered to be controlled for each effect size that resulted from a statistical design where the impact of either perceptual skills or global cognition on emotional decoding performance was controlled by treating that variable as a covariate. Between-group differences in cognitive status were considered as not being controlled when two conditions were met: (i) an indication was made of significant between-group differences in perceptual skills and global cognition favoring HOA in the study the effect size came from, and (ii) no action was made during statistical analyses to control the impact of these cognitive variables on emotional decoding performance.

In addition, each effect size was recorded along with a variable representing whether or not between-group differences in depression symptoms were controlled for when the authors examined the between-group differences in emotional decoding performances corresponding to that effect size. We used the same procedure we used for the control of cognitive status.

The indication that nonsignificant differences were found between AD patients and HOA on one variable (i.e., global cognition, perceptual abilities, or depression) required special consideration related to statistical power issues. In the present meta-analysis, studies showing nonsignificant differences on a variable were considered as controlling for between-group differences on that variable only when enough power was reached to detect potentially significant differences. To determine whether or not each study had enough power to detect existing between-group differences on a variable, a first step was to determine the mean effect size of between-group differences on this variable. To our knowledge, no meta-analysis exists that provide a global standardized mean difference between AD patients and HOA on depression symptoms, perceptual skills, or global cognition. Consequently, we used the data recorded in the present meta-analysis to compute the three required mean

effect sizes. A second step consisted in computing the statistical power of each study to detect differences between AD patients and HOA corresponding to the computed mean effect sizes. When a study indicated that nonsignificant differences were found between AD patients and HOA on one variable (e.g., depression), we used the G*power program [53] to determine the power achieved by this study to detect differences between two independent means (e.g., AD patients and HOA's depression scores) given the computed mean effect size (e.g., standardized mean difference between AD patients and HOA on depression symptoms), the study sample size, and $\alpha = 0.05$. In cases in which the statistical power was not sufficient, this information was not coded.

Emotional decoding tasks

Inspired by the FAB [24], we classified four main emotional tasks: naming (with or without emotional label), selection, matching, and discrimination. In the emotional naming task, the participant is presented with an emotional stimulus (e.g., prosody, face) and has to name it. Depending on the specific task being considered, the name has to be either spontaneously and verbally produced, or selected from a set of labels. When selected, the name is given either orally or by pointing to its label. In the emotional selection task, subjects are given the name (orally) or a printed label (visually) of an emotion and asked to select, from a set of various emotional stimuli (e.g., face, prosody), the stimuli that corresponds to the target emotion. In the emotional matching task, subjects are shown an emotional target and asked to match it with one of several alternatives. Emotional matching tasks most often involve identical stimuli (e.g., both the target and the alternatives are faces). Emotional matching tasks also encompass matching emotional prosody to emotional faces tasks, which requires the participant to listen to an audiotaped sentence spoken with an emotional prosody; they are asked at the same time to point to the emotional face that corresponds to the emotional prosody of the speaker. Tasks that involved matching faces to emotional prosody were excluded, as they entail some memory load [24]. Finally, in the emotion discrimination task, a discrimination task requires the participant to look at pairs of visual stimuli or to listen to two emotional prosodies and to indicate whether the emotions shown or listened to are the same or different. We also considered as emotion discrimination tasks those tasks in which the subject is presented with an emotional and a neutral stimulus and must indicate which stimulus is emotional.

As semantic relatedness could affect performances on emotional prosody tasks using either congruent or incongruent semantic content [22, 36, 54], we coded only prosody tasks in which the emotional content was semantically neutral, which is recommended in order to minimize the impact of the semantic on the emotional prosody detection.

Tasks necessitating the repetition or elicitation of an emotional prosody have also been used in one study [25]. Being hardly ever used in the literature, these two tasks were not coded in the present meta-analysis.

Stimuli

We chose to separate emotional stimuli into five groups: face, prosody, cartoon drawing, audiotaped story, and video clip. The stimuli were also coded as being auditory, visual, or audiovisual.

Emotions

Only the emotions included in Paul Ekman's list were included in the present meta-analysis and were coded as such (i.e., fear, happiness, anger, surprise, sadness, disgust, shame, contempt, amusement, satisfaction, discomfort, excitation, guilt, pride in the success, relief and sensory pleasure) [44].

Publication form

We recorded the form in which each study reporting one or more effect sizes was published (published article or unpublished study).

Individual effect sizes

The effect size used in our meta-analysis was the standardized mean difference in scores between HOA and AD patients on measures of emotional decoding abilities. It is indexed by Cohen's standardized difference, g, which corresponds to the difference between the two sample means divided by the pooled standard deviation [45]. To compute g-values, we recorded the mean scores and standard deviation obtained by AD patients and HOA that were reported by the authors. Some studies comparing the emotional decoding performances of AD patients and HOA did not report means and standard deviations. In these cases, t-, F-, or χ^2-values were used to compute g using the formulae described by Johnson and Eagly [45]. A positive g-value indicates better decoding performances in HOA compared with those in AD (i.e., when error scores were used, the g-value was reversed, so that a positive g-value still indicates better performance in HOA).

Finally, when reported, the significance status associated with each effect size estimate was recorded.

All the studies were coded independently by the first two authors. Agreement for the categorical moderators was indexed by kappa coefficients, which ranged from 0.95 to 1.00, indicating a high level of agreement [55]. The coders' initial ratings of effect size values were different for only three effect sizes of 212. All disagreements were resolved through discussion.

Analytical procedures

Effect size calculations

As recommended in the literature [45, 56–59], each *g*-value was corrected to provide an unbiased estimate of the population effect size using the *J(m)* correction [56]. The corrected effect size index is referred to as Hedges' *d*.

In order to preserve the independence of the effect sizes involved in our analyses, we applied the shifting unit method [60], which involves shifting the unit of analysis (samples, moderator modalities) according to the hypothesis being tested. The global mean effect size analysis involved one effect size per independent sample, whereas each moderator analysis involved one effect size per moderator modality within each sample. This method provides a good compromise between preserving the independence of the effect sizes and retaining a maximum amount of information from each study.

In addition, several studies included two independent groups of AD patients that were compared with a common group of HOA [25]. In order to preserve the independence of the effect sizes, for each analysis, one AD sample was randomly selected from the AD samples having control subjects in common. Only the effect sizes contributed by the selected sample were included in the analyses. This procedure was also used for an article that described a study that was partly conducted on AD and HOA samples used in a study reported in another article [61].

Data analyses

We assumed there would be some variability in the individual effect sizes because of random differences among the participants in each study and systematic (due to the moderators) and random differences between the studies. Consequently, we tested the statistical significance of the mean effect size using random-effects model, and we performed the moderator analyses using a mixed-effects model. Although conservative, these statistical models allowed us to extend our inferences to the universe of studies from which the study sample was drawn, rather than just to the studies included in the sample [56, 58]. To ensure that effect-size estimates resulting from large sample sizes had a greater weighting than effect-size estimates from smaller samples, each *d*-value was weighted by multiplying its value by the inverse of its variance, which is strongly correlated with sample size [57].

The effects of categorical moderators were analyzed by using analysis of variance (ANOVA) analog analyses in which the Q_B statistic is used to test whether the individual effect sizes associated with each modality differ significantly in their mean. Q_B has a chi-squared distribution and is analogous to an *F* test. The effect of continuous moderators was analyzed using weighted generalized least square regressions with the method-of-moments estimation method. All analyses were carried out by using Wilson's SPSS for Windows Meta-Analysis Macros [62].

For each analysis, the leverage statistic was used to identify outliers within that set of effect sizes [63, 64]. Any outliers detected were Windsorized to their nearest effect size [58] and their weight recomputed. As well, we looked for extreme cases within the set of effect size weights. Any effect size whose weight was identified as being an outlier had its weight replaced with the nearest weight given the actual effect size value.

Power analyses

In order to retrospectively estimate the statistical power of the moderator analyses, we used the procedures for mixed-effects tests of moderators described by Hedges and Pigott [65]. These calculations were based on two-tailed inferential tests, observed sample sizes and between-studies variance components, and prespecified effect size. We computed the power of the moderator analyses to detect a small, medium, and large effect [66]. The statistical power of the moderator analyses that did not have sufficient power to detect small effects (i.e., power < 0.80) are indicated in the text.

Assessment of potential publication bias

A funnel plot was used to assess the possibility that selection biases due to publication biases affected our results. A funnel plot is a plot of effect size against sample size. If there is no bias, the distribution resembles a symmetrical inverted funnel. Bias against the selection of unexpected findings leads to an asymmetric distribution in which unexpected effect sizes (i.e., negative in the case of the present meta-analysis) are lacking. Biases against the selection of nonsignificant

findings are indicated by distributions in which small-sample studies are lacking in the region representing small effect sizes [67].

RESULTS

Descriptive data

The literature search yielded 24 papers presenting 23 studies in which the emotion decoding performances of AD patients were compared with those of age-matched HOA. The 23 studies reported 212 separate effect sizes based on 24 AD patient samples matched with HOA. One study included two independent groups of AD patients that were compared with a common group of HOA. The meta-analysis database thus contained 23 independent matched pairs of AD/HOA samples. The 212 individual effect sizes are presented in our Supplementary Material (available online: http://www.j-alz.com/issues/32/vol32-1.html #supplementarydata03), along with the characteristics that we coded. In total, 435 AD patients (mean age = 76 years, mean MMSE scores = 17.91) participated in these studies and were compared with 394 HOA (mean age = 74 years). Individual effect sizes were reported for samples sizes ranging from 6 to 50 AD patients ($Mdn = 17.5$) and 10 to 50 HOA ($Mdn = 14$).

The presence of a potential selection bias against either nonsignificant or negative effect sizes was assessed by using a funnel plot. With the exception of three extreme individual effect sizes on the right side of the distribution, which contributed to independent effect sizes that were diagnosed as outliers in every subsequent analysis and were consequently Windsorized, the distribution resembled the expected inverted funnel. The funnel plot did not indicate that nonsignificant findings had been excluded. In fact, many small effect sizes were reported by studies on the basis of small sample sizes. Moreover, the symmetry of the funnel plot indicates that a bias toward the nonpublication of results showing better performance of AD patients compared with that of HOA is unlikely. Thus, selection bias is not likely to have influenced the results.

Global mean effect size

Two outliers were detected and Windsorized to their nearest neighbors. The 23 independent effect sizes ranged from 0.26 to 2.00. The weighted mean d was 0.98, with a 95% confidence interval (CI) from 0.80 to 1.17, indicating that AD patients are significantly impaired in emotion decoding compared with HOA. Moreover, this is a large effect size according to Cohen's guidelines for the magnitude of d [68]. The homogeneity statistic Q indicated a marginally significant heterogeneity of the individual effect sizes, $Q(22) = 31.67$, $p = 0.08$.

Moderators related to the emotional task design

Concerning the emotional task, we hypothesized that the AD deficit would be apparent regardless of the kind of task used to assess emotional decoding abilities (i.e., naming, selection, matching, and discrimination). In addition, we were interested in exploring whether the kind of task used could account for some of the heterogeneity detected among the effect sizes included in the global mean effect size analysis. For this purpose, we performed an ANOVA-analog analysis on the effect sizes associated with the four kinds of tasks. As expected, the results indicated that the mean effect sizes were significant regardless of the task (Table 2). Moreover, the kind of task used did not moderate the size of the mean effect sizes (Table 2). It should be noted that the ANOVA-analog analysis had sufficient

Table 2
Number of effect sizes, weighted mean effect size, confidence interval, and ANOVA analog test of effect size homogeneity for each moderator

Moderator	k	Weighted mean d	95% CI Lower	95% CI Upper	Q_B
Task					1.81
Selection	3	1.11	0.67	1.55	
Matching	4	1.03	0.64	1.42	
Naming	19	0.95	0.78	1.12	
Discrimination	7	0.80	0.52	1.08	
Modality					1.96
Visual	18	1.04	0.83	1.24	
Auditory	8	0.76	0.44	1.09	
Stimulus					1.85
Drawing	2	1.25	0.62	1.88	
Face	16	1.01	0.78	1.24	
Prosody	7	0.79	0.42	1.15	
Emotion					5.64[a]
Fear	10	0.81	0.56	1.06	
Sadness	9	0.79	0.53	1.05	
Disgust	6	0.67	0.35	0.99	
Anger	12	0.65	0.43	0.87	
Happiness	13	0.52	0.31	0.73	
Surprise	5	0.43	0.08	0.78	
Control of cognitive status					3.80*
No	5	1.16	0.87	1.44	
Yes	5	0.75	0.44	1.05	

[a]Omnibus test statistic; *$p < 0.05$.

statistical power for detecting medium to large effects, although it lacked the statistical power for detecting small effects (<0.22).

Concerning the stimulus, only one study (yielding one independent effect size) included in the present meta-analysis assessed the participants' decoding abilities by using a story as the stimulus. As well, only one study (one independent effect size) used a videotaped emotional stimulus. Consequently, these two modalities were excluded from the present moderator analysis, leaving the moderator variable stimulus with three modalities: face, prosody, and drawing. The independent effect sizes associated with these three modalities were subjected to an ANOVA-analog analysis. The results indicated that the mean effect sizes were significant regardless of the stimulus used in the emotional decoding tasks and were not significantly different from each other (Table 2). It should be noted, however, that this analysis did not have enough statistical power to detect small to medium effects (<0.77). Concerning the modality, only one study (yielding one independent effect size) included in the present meta-analysis assessed the participants' decoding abilities by using an audiovisual modality as the stimulus, and only the visual and auditory modalities were tested. The results of the ANOVA analog analysis indicated that the mean effect sizes were significant regardless of the modality involved and were not significantly different from each other (Table 2). However, the statistical analysis lacked the statistical power to detect even large effects (0.72).

Concerning emotions, we hypothesized that AD patients would be particularly impaired in decoding fear, whereas the ability to decode disgust and happiness would be relatively preserved. These hypotheses were tested using planned contrasts. The first contrast compared Fear to Anger, Surprise, Sadness, Happiness, and Disgust considered together (C1, 5/6 −1/6 −1/6 −1/6 −1/6 −1/6). The second contrast compared, within C1, Happiness and Disgust to the other emotions within C1 (C2, 0 2/5 2/5 2/5 −3/5 −3/5). Three additional orthogonal contrasts were created to complete the representation of the Emotion moderator (C3, 0 2/3 −1/3 −1/3 0 0; C4, 0 0 1/2 −1/2 0 0; C5, 0 0 0 0 1/2 −1/2). As expected, the standardized mean difference between AD and HOA in decoding fear was significant, and it corresponded to the largest mean effect size (Table 2). However, despite sufficient statistical power, it was not significantly higher than the effect sizes associated with the other five emotions considered together. Contrary to our expectations, neither the decoding of happiness nor the decoding of disgust corresponded to the smallest effect sizes. Moreover, the decoding of happiness and disgust were significantly impaired, as was the case for the other three emotions.

Moderators related to the control of confounded variables

For the control of between-group differences in cognitive status, we expected the mean effect sizes to be larger when obtained from methodological or statistical designs in which between-group differences in cognitive status are present and significant rather than controlled for. As explained in the Recorded variables and coding section, the indication that nonsignificant differences were found between AD patients and HOA on one indicator of cognitive status (i.e., global cognition and perceptual abilities) required special consideration related to statistical power issues. To determine whether or not each study had enough power to detect existing between-group differences on each indicator of cognitive status, we first determined the mean effect size of between-group differences on each indicator. We then computed the statistical power of each study to detect differences between AD patients and HOA corresponding to the mean effect sizes just determined. Studies showing nonsignificant differences on an indicator were considered as controlling for between-group differences on that indicator only when enough power was reached to detect potentially significant differences. Studies lacking statistical power were not coded as either controlling or not controlling cognitive status.

Statistical power to detect differences in perceptual abilities

Eleven studies included in the present meta-analysis provided enough data to compute standardized mean differences in scores between HOA and AD patients on measures of perceptual abilities. A random-effects model meta-analysis of the 11 independent effect sizes indicated a significant weighted mean effect size favoring the HOA, $d = 1.03$, 95% CI [0.76, 1.29]. Among the three studies reporting no significant between-group differences in perceptual abilities, only one study (one independent effect size) had enough statistical power to detect the computed mean effect size and was coded as controlling for between-group differences in perceptual skills. Three other independent effect sizes were coded as such because they were derived from analy-

ses in which scores obtained on perceptual tasks were entered as covariates.

Statistical power to detect differences in global cognitive function

Seventeen studies included in the present meta-analysis provided enough data to compute standardized mean differences between the MMSE scores of HOA and AD patients. A random-effects model meta-analysis of the 17 independent effect sizes indicated a significant weighted mean effect size favoring the HOA, $d = 2.54$, 95% CI [2.19, 2.90]. One study reported no significant between-group differences in MMSE scores, and the power analysis that we conducted indicated that this study had enough power to detect the computed mean effect size. Thus, this study was coded as controlling for between-group differences in global cognition.

The five effect sizes that we coded as controlling between-group differences in either global cognition function or perceptual abilities were compared with the five independent effect sizes coded as controlling between-group differences in neither global cognition nor perceptual abilities. As the results presented in Table 2 indicate, the effect sizes obtained from designs controlling for between-group differences in cognitive status were, on average, significantly smaller than the effect sizes obtained from designs in which AD patients' perceptual performances and MMSE scores were significantly lower than those of HOA.

Concerning the control of between-group differences in depressive symptoms, we expected the mean effect sizes to be larger when obtained from methodological or statistical designs in which between-group differences in depressive symptoms are present and significant rather than controlled for. Unfortunately, we were not able to test this hypothesis, as the database contained only one independent effect size obtained from a design in which AD patients had significantly higher depression scores than those of HOA [69].

Relation with dementia severity

Concerning the correlation with dementia severity, we expected the individual effect sizes to increase with decreasing AD patients' MMSE scores. The results of the meta-regression analysis indicated that the standardized mean differences between AD patients and HOA in decoding performances were negatively correlated, although not significantly so, with AD patients' MMSE scores, $\beta = -0.27$, $p = 0.22$.

DISCUSSION

Emotional disturbances present in dementia are of considerable interest [70]. Everyday social life requires correct perception and interpretation of different emotional cues for adequate behavior in social contexts. The ability to interpret nonverbal emotional cues thus plays an important role in maintaining successful relationships and healthy psychological functioning [71]. In the context of AD, this ability may also influence important indicators of well-being and predict the quality of life [20]. Our meta-analysis confirms that the deficit in emotional decoding ability in AD patients is large and significant. Moreover, the results indicate that the deficit is significant in all the conditions that we considered. AD patients are significantly impaired in decoding emotions regardless of the task used, the stimuli involved, the emotion to be processed, and the severity of the disease. This emotional disturbance seems therefore to constitute a major and robust disorder in AD, providing a potential diagnostic value that can be used in existing clinical settings. A deficit in emotional decoding is in fact suspected to contribute to social function impairment, including poor communication, decreased interpersonal relatedness, or inappropriate social behavior in AD patients [14, 72]. Therefore, the deficit in AD patients' emotional decoding abilities deserves better comprehension and more detailed explanation. In this study, we examined two main factors that may modulate the findings: one related to the task design and the other to participants' characteristics (Fig. 1).

Four classical paradigms are used to measure emotional decoding (i.e., emotional naming, discrimination, selection, matching). Despite the suggestion by several authors that emotional tasks could vary in their complexity level (for discussion, see [22]), the results from our meta-analysis suggest that patients show impaired performances independently of the nature of the task. Emotional tasks involve underlying processes that are thought to be varied across emotional decoding tasks [20]. Indeed, an emotional naming task may involve a higher executive load in order to make a decision about the name of the emotion presented, whereas an emotion discrimination or matching task may require more visuospatial abilities [12, 20]. Given the massive changes affecting several cognitive domains (e.g., memory, visuospatial abilities, executive functions) and the relationship between cognitive processes and emotional decoding [73], completing these emotional tasks may place AD participants in

a cognitively vulnerable position that is independent of the task considered. Additionally, given the overlap between the neural structures affected by AD pathology and the areas thought to be implicated in emotional decoding tasks, it is not surprising to observe that deficits are not restricted to one kind of emotional task. Severe lesions in the right anteromedial temporal lobe [74], amygdala [75], frontal regions (e.g., orbitofrontal cortex) [76], and anterior cingulate region [77] are followed by a decline in emotional decoding performance. As a result, the neuroanatomical changes in the frontal area and, more specifically, the limbic system in AD [1, 78–80] could lead to a deficit across all of the emotional tasks that assess this area of emotion processing.

Regarding the type of stimuli and modalities, no significant differences emerge. To date, static photographs of faces have been thought to lack sufficient realistic information to allow AD patients to accurately decode emotions [12]. The ability of AD patients to decode emotions should be better when they can integrate information about emotional cues from several modalities (e.g., visual and auditory) within the context of environment cues [11, 15]. Nonetheless, such differences were not observed in our meta-analysis. One considerable limitation is that the statistical analysis lacked sufficient power to detect an effect. Indeed, the existing literature lacks data about how AD participants decode emotions from stimuli with greater ecological value, such as stimuli that encompass several emotional cues and/or dynamic stimuli [15].

A main finding from our work is that the decline in emotional decoding abilities was significantly reported in AD for all emotions. Recent neuroimaging findings support the hypothesis that the emotional network is impaired in dementia. In particular, there is converging data in favor of neuropathological and functional amygdala lesions in AD [9, 78, 79, 81–87]. One classical approach has associated amygdala functioning with fear processing (see [88–91] for meta-analysis). Consequently, in the context of AD, some authors have hypothesized that fear could be altered more than other emotions [31]. However, another suggestion has been defended in which relevance detection in the sequential cognitive appraisals that induce emotion has been attributed to the amygdala [92, 93]. In this approach, the amygdala is involved in the elicitation of a wide range of emotions, which can explain why the deficits in emotional decoding in AD patients are not restricted to the emotion of fear. These recent findings lend support to the hypothesis that amygdala lesions could be related to a deficit in AD patients to extract salient information from an emotional stimulus rather than to a deficit in the processing of fear only [93]. Future studies should confirm this hypothesis.

In the second part of this study, we examined the implication of cognitive skills in the emotional decoding performances of AD patients. We made the assumption that a decline in performance on emotional tasks in AD patients does not necessarily indicate a primary impairment in emotional decoding abilities, but rather indicates cognitive impairments that impact global performances [10, 12, 16, 21–23, 29, 33]. The present meta-analysis demonstrates that a deficit in emotional decoding in AD remains present even when the cognitive status of AD patients and HOA is equivalent. However, it is possible that the deficit in emotional decoding in AD patients is related to other cognitive impairments that were not taken into account or only partially taken into account in our control analysis for cognitive status. It could also be supposed that AD patients are impaired in emotional decoding abilities as a result of specific emotional impairments. In addition, we found that there is a significant difference between the mean effect sizes with control for cognitive status and the mean effect size without such control. This suggests that the cognitive deficits of AD patients contribute to the deficit in emotional decoding abilities and shows how important it is to control for cognitive skills when exploring emotional decoding abilities in AD.

Several studies have repeatedly highlighted the presence of depression symptoms in AD [94–96], and these affective disturbances constitute a risk factor for its development [94, 97, 98]. We assumed that emotional decoding performance in AD might be accounted for by their depressive symptoms [39]. However, because of a lack of studies investigating this question, we were unable to find sufficient evidence to draw any conclusions. Nevertheless, the association between affective states (e.g., depression symptoms, agitation, and irritability) and emotional decoding abilities are of considerable interest and should be addressed more systematically.

The hypothesis that an emotional deficit is present regardless of the stage of the disease has been questioned in previous work. Across emotional tasks and procedures, a significant correlation was found between the MMSE and AD patients' emotional decoding performance [14, 20]; three studies, however, found no significant correlation [21, 25, 36]. In the present analysis, we found no significant correlation between emotional decoding performance and increasing dementia severity, as indexed by MMSE scores. Consequently, our result suggests that the

ability to decode emotion is poorly predicted by MMSE scores. This finding allows us to suppose that instead of there being a primary impairment in the perception of emotion in AD, the instrument that measures dementia severity may not be adequate for measuring the relationship to emotional decoding performances. Disease progression is a function of deficits in cognitive abilities, which are themselves expected to contribute to disturbances in emotional performance [10, 21]. Our result could question the ambiguous information that results from the MMSE score. The MMSE score is used to measure the progression of dementia severity. However, the MMSE is also a screening instrument for global cognition. Clinical tests developed to specifically measure the severity of the AD, such as the Clinical Dementia Rating Scale, should be considered in future to assess the relationship to emotional decoding performance. Integrating an affective dimension into the MMSE subtests could be relevant for fine-tuning the clinical diagnosis as our results have demonstrated a significant deficit in the ability of AD patients to decode emotion.

In conclusion, to our knowledge, this is the first comprehensive meta-analysis demonstrating a large deficit in emotional decoding abilities in AD. This decline is consistent regardless of the emotional decoding task, the stimuli, the emotion considered, or the severity of the disease. Our data permit us to strongly point out the need to control for cognitive deficits when exploring emotional decoding abilities in AD. Researchers who investigate this question in the future should be particularly aware of this issue. To date, the number of studies using comparison criteria regarding healthy aging remains limited, sometimes resulting in issues of low statistical power. Thus, it will be interesting to confirm our results in future. This meta-analysis may motivate interest in standardizing the methods used to explore emotional processing in AD. Further investigations that address the limitations of existing studies are needed to clarify the association between emotion decoding abilities and the clinical aspects of AD patients, including the implications of their affective state and the extent of control of cognitive skills in several domains (e.g., language abilities, executive components, and verbal memory).

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