1. Our results clearly show that the encoding task strongly affected participants’ ability to retrieve conceptual information. We now discuss this point and the limitation associated with the lack of new items in the manuscript, on pages X and Y, respectively.

2. First, we have taken the reviewer’s suggestion and added descriptive and statistical data on confidence (as well as accuracy and RT) for the parity judgment on Odd/Even trials (see page X).

3. As shown in Figure 5, the MDD group was less confident than the controls in response to the Side but not the Question cue, but this was not specific to either encoding task. This does not mirror the accuracy data, which showed a *Group* x *Cue* interaction for words from the mobility task, and which did not show a significant controls > MDD effect under the Side cue (or the Question cue). Thus, the reviewer’s point about lower confidence not tracking lower accuracy is well-taken. Consequently, on page X we now offer a more careful discussion of the confidence data.

4. As precedent, we note that when Dobbins & Wagner (2005) found slower encoding RTs for pleasant/unpleasant judgments than for animacy judgments, they suggested “that comparison of the former to the latter encoding trials would identify regions differentially engaged during sustained conceptual analysis” (p. 1773). Here, we are similarly arguing that the longer RTs observed for mobility versus animacy judgments implies “sustained conceptual analysis” (i.e., deeper processing) for the former relative to the latter trials. We have attempted to clarify this point on page X.

5. *The authors indicate that “observed ERP differences over parietal areas meant that ‘recollection was strongest under the Question cue and reduced in MDD’”. This is problematic because: (1) only correct responses were analyzed; (2) “I thought the behavioral data did not show an accuracy deficit for the MDD participants and thus claims that the ERPs correspond to group differences in the quality of memory evidence seem strained”; and (3) there is no evidence that individual differences in ERP amplitude correspond to individual differences in source accuracy or confidence.*

We appreciate the reviewer’s comments and have re-phrased the problematic passages—please see the new text about X on page Y in the Discussion.

6. Third, in the revised manuscript (page X) we note that there were positive relationships between left parietal Question minus Side ERP difference waves in the 400-800 ms and 800-1400 ms time windows, on the one hand, and Question minus Side source accuracy and confidence difference scores on the other.

7. We also added a note about the exploratory nature of the subsequent ERP analyses on page X of the Discussion.

8. As noted in response to point 2 (above), the absence of new items is a clear limitation, and we now discuss that on page X.

9. Finally, we note that prior studies of multidimensional source monitoring tasks also offer the “guess” response option (e.g., Starns & Hicks, 2005), and we have added references to those studies on page X.

10. *“The sentence ‘In short, memory retrieval is impaired in depression and enhancing it can bring lasting relief’ is unsubstantiated and needs clarification and references*.

We thank the reader for their careful reading of the manuscript. In response to this critique, we have edited the sentence (p. X) by adding references to work by Mark Williams, Tim Dalgleish, (and anyone else?), Filip Raes, and their colleagues. This work touches on the two topics mentioned in the sentence. First, several studies (i.e., X, Y, Z) provide evidence of retrieval deficits in depression, primarily in the context of autobiographical memory.

11. Instead, it seems that enhancing the specificity of memory retrieval may involve enhancements in executive control that have broad benefits on mood and cognitive function in depression. We have attempted to clarify these points in the manuscript (see page X), and we thank the reviewer for highlighting this need.

12. In the revision we devote more space to discussing about the lack of stronger negative effects of depression on memory accuracy (see page X). Speaking directly to the reviewer’s point, in the same place we specifically suggest that future neuroscientific studies of retrieval use emotional stimuli to see the expected memory deficit in MDD. In fact, such studies are underway in our laboratory.

13. As described in the manuscript (page X), we used a screening procedure to identify individuals who were in the midst of a Major Depressive Episode and who had a BDI-II score of at least 14 (the published cut-off for minimal depression).

14. It was on the basis of this statement that we adopted a criterion of 50% contaminated trials for rejection, and we have added a reference to Luck’s book on page X to indicate this.

15. We felt that loss of up to ~15% of the data (i.e., 18 of 128 channels) was a reasonable threshold for rejection. We now explain this on page X of the revised manuscript.

16. On page X of the revision, we now indicate that the first mechanism—additional cue elaboration—is more likely, based on a recent functional magnetic resonance imaging fMRI paper by Han and colleagues.

17. As noted in our response to Reviewer 5’s fifth critique (see above), we now put greater emphasis on the link between relatively intact source memory and increased left parietal ERPs in the Question/mobility condition for the MDD group (see page X)—this seems to be consistent with what the reviewer is driving.

18. We appreciate this comment and can see how early introduction of neural systems adds complexity to an already complicated discussion of the psychological constructs that mediate encoding and retrieval. In the revision, we have tried to take a more sequential approach of the kind the reviewer suggests—please see page X.

19. Therefore, seeing accuracy under the Question cue vary so strongly by encoding task was striking. We regret superficial treatment of this result, and in the revised manuscript it is now clearly emphasized (see pages X and Y).

20. As described on page X, we found that “Question minus Side”/*mobility* difference scores for accuracy and confidence were significantly correlated across the groups, *r*(46) = 0.42, *p* = 0.002, indicating that it may be difficult to tease apart these two factors.

21. Putting these two analyses together, we have advanced a cautious argument in the revision (see page X). Specifically, we indicate that the ERPs track accuracy more closely than confidence, but we also note that the correlations link the ERPs to both accuracy and confidence. A more definitive separation of accuracy and confidence in MDD will have to accomplished in a follow-up study better designed to tease apart these two factors.

22. Thus, we cannot advance a strong argument but we have added a reference to mental imagery on page X to reflect the reviewer’s interest, which we share.

23. A major shortcoming of the original manuscript was that our discussion of the PSQI data was very truncated due to space limitations. In the revision we devote more attention to this result (see page X).

24. Thus, we interpret the reviewer as suggesting that more drowsy depressed participants unhelpfully generate high amplitude alpha rhythms over the left parietal cortex, which might explain the negative correlation between chronic sleep disruption (as measured by PSQI) and the amplitude of the Question minus Side ERP effects (for words from the mobility task) seen over left parietal scalp. This is a fascinating suggestion, and we briefly acknowledge it on page X in the revision.

25. Thank you for this careful reading—we avoid referring to ERPs as indexing “activation” in the revised manuscript.

26. *On page 13, ‘no significant effects were seen in any time window’ is ambiguous, in the Abstract, ‘slasting’ is a typo, and on page 4, ‘loses’ is a typo*. Make these corrections or cut the relevant sentences.

27. We did this and found significant *Group* x *Cue* x *Task* interactions for 400-800 ms, *F*(1, 46) = 8.23, *p* = 0.006, and for 800-1400 ms, *F*(1, 46) = 5.09, *p* = 0.03. As described in the manuscript on page X, in both cases the *Group* x *Cue* interaction was significant for words from the mobility task, *Fs* > 14.3, *p*s < 0.0005.

28. When we considered the data across both groups, we found modest correlations between source accuracy, confidence, and left parietal ERP amplitudes for the “Question minus Side” contrast for words from the mobility task (**400-800 ms**; accuracy, *r* = 0.18, *p* = 0.21; confidence, *r* = 0.27, *p* = 0.06; **800-1400 ms**; accuracy, *r* = 0.28, *p* = 0.05; confidence, *r* = 0.28, *p* = 0.05), as described on page X. However, when we restricted these correlations to the MDD group, nothing approached significance, all *ps* > 0.12 (see page X).

29. Indeed, when we ran stepwise regressions predicting “Question minus Side”/*mobility* accuracy with BDI-II entered in Step 1 and either MASQ-GDA or MASQ-AA entered in Step 2, neither regression yielded a significant effect for the anxiety measures (χ2 < 2.7, *p*s > 0.11). This is not terribly surprising, as these three self-report measures were highly correlated (*rs* > 0.68, *p*s < 0.0002). We describe these new analyses in the manuscript on page X; although they do not support an argument for a selective effect of anxiety on memory, they certainly indicate that anxiety should be investigated in future studies of memory in psychopathology.

30. Unfortunately, space limitations made it difficult for us to describe our rationale for including the PSQI in the original manuscript. We do so in the revision on page X.

31. *In the Introduction, the authors hint at the Group* x *Cue* x *Task interaction for accuracy but do not explicitly describe it until later—it is worth stating it clearly here as well*.

Thank you for this suggestion; we now clarify the nature of the key interactions in the Introduction, on page X.

32. *On page 12 of the Results, the statement that “These data suggest that recollection was strongest under the Question cue and reduced in MDD*” *combines two main effects and reads as though there was an interaction, which there is not*.

The reviewer is correct and we apologize for the lack of clarity. This passage has been revised, please see page X.

33. Regarding **e**, we now report on correlations between source accuracy and confidence—please see page X.

34. For instance, if the reviewer compares the ERP bar graph at the bottom of Figure 9 with the accuracy data in Figure 4A, he or she will see the resemblance: in the MDD group there is a Question > Side effect, but in controls the pattern is reversed (Question < Side). By contrast, the confidence data in Figure 5A do not show such a pattern—for confidence, the pattern is Question > Side for both groups. Clearly this is not rock solid evidence and we do not wish to make too much of it, but on page X we point to this exercise as our reason for slightly favoring an accuracy account of the ERP effects.

35. Regarding point **f**, the reviewer’s comment (as well as a similar comment from Reviewer 5) made it clear that we did not plainly articulate our idea about RT and depth of encoding. We describe our thinking in more detail on page X of the revision.

36. In the revision, we devote significantly more space to the topic of rote memory difficulty in depression (see page X), and strive to be clear about the precise aspect of memory we are discussing (e.g., see line X on page Y).

37. Nonetheless, we now explicitly mention low power as limitation of the study (see page X).

38. *The manuscript does not carefully distinguish between recall and recognition within the broader concept of cued/uncued retrieval*.

We apologize for this and now provide important background information on page X.

39. On page X of the revision, we devote space to thinking about why the MDD group performed as well as it did.

On the nature of (non-emotional) memory impairments in depression

You could spend more time pointing to the relative deficit in Side in MDD, as this is consistent with expectations (not going to do well on an arbitrary relationship b/w word and position).

1. **Burt et al. (1995)**

* Meta-analysis of 99 recall and 48 recognition studies
* In their Table 2, they report extremely strong negative relationships between memory and free recall (Cohen’s *d* = 0.56, fail-safe Ns > 300 depending on the number of variables included in the study), with especially strong relationships for visual (as opposed to verbal) stimuli and for immediate (versus delayed) tests. There are many fewer studies of recognition memory that examine discriminability (*n* = 9 for “one-level” studies, as opposed to 54 such studies for recall), but these also support a negative relationship with depression, with *d* = 0.33 and fail-safe *N*s > 11. Regarding patient characteristics (Table 2), the effects are stronger with younger, unmedicated, inpatients. In terms of manipulations (Table 5), there is a stronger effect of depression on positive vs. neutral vs. negative memory.
* For recognition, some of this changes . . . although many studies only looked at hit rate, not discriminability. But with recognition you see stronger effects of depression on verbal vs. visual stimuli, and after a delay relative to immediate test. Again, the effects are stronger on younger inpatients relative to older outpatients.
* They make the point that many of these effects are not specific to depression and may be associated with “one or more overarching factors common to various forms of psychopathology (e.g., severity of illness, motivational deficits, effortful processing deficits)”

2. **Zakzanis et al. (1998)**

* Meta-analysis of 22 neuropsychological studies encompassing 726 adults with unipolar depression and 795 healthy controls. A nice aspect of this review is that it includes multiple measures of memory and other cognitive functions as well, so you have a shot at specificity.
* They find a median effect size of 0.52 (patients < controls), with several measures of episodic memory (and attention) showing effects greater than the median, but measures of semantic memory, primary memory, working memory, reasoning, and motor speed yielding weaker group differences (i.e., effect sizes below the median).
* They also find near-complete separation between the groups (only 10% overlap b/w distributions) on tasks characterized as “effortful”, versus substantial overall (73%) for tasks characterized as demanding only “superficial” processing.
* “depression had the largest effects on tests of episodic, declarative memory”
* Interestingly, they note that the negative effect of depression on declarative memory varies according to the amount of support provided at encoding—for instance, the California Verbal Learning Test, which uses lists of words sorted into categories, yielded weaker effects than the Ray Auditory Verbal Learning Test, which involves encoding 16 unrelated words. They regard this and similar evidence as consistent with an encoding deficit in depression than can be counteracted by external support (e.g., provision of categories that can be used to sort the lists and relate items to one another).

3. **Rock et al. (2014)**

* This is a meta-analysis of studies over 30+ years (1980-2012) that used the Cambridge Automated Neuropsychological Test Automated Battery (CANTAB), which is desirable as it ensures that everyone had the same tests, administered in the same way.
* Includes data from 784 currently depressed adults (vs. 727 controls) and 168 adults with remitted depression (vs. 178 controls).
* The study reveals moderate deficits in current depression across all domains tested (memory, attention, and executive function), with the exception of simple reaction time. Following remission, the memory deficits were small to moderate. They conclude that broad cognitive deficits are common in depression, with deficits in executive function and attention particularly persistent even in remission.
* A valuable point: this study found no relationship between cognitive deficits and depressive symptoms, and they note that even when such associations are found, they typically account for no more than about 10% of the variance in the cognitive disturbance. Thus, the cognitive side of depression is clearly not just about mood disturbance (or cognitive disturbance accessible to consciousness, such as rumination).
* An additional motivation supported by this paper and Zakzanis, and Snyder (2012)—there are executive functioning deficits in MDD and those may compromise retrieval, even if there is no emotion at stake. See the next two papers . . .

4. **Snyder (2012)**

* Points to DP’s 2002 paper as suggesting that PFC dysfunction in MDD could be the cause of broad EF impairments.
* This is a meta-analysis of 113 EF studies (3,936 MDD; 3,771 controls) with emotionally neutral materials only, so any EF deficits seen here could be in play in SOURCE.
* The bottom line is that the analysis reveals significant depression-related impairments in every aspect of EF, with different aspects of EF each associated with effect sizes between d = 0.45 and d = 0.58. There is little evidence that any one EF sub-domain is worse than the others, although there is some such evidence for inhibition.

5. **Williams et al. (1996)**

* This paper examined the relationship between AMS and ability to imagine the future.
* They begin with a review of the reduced AMS phenomenon in suicide and depression, again relating it to poor problem solving (**look up**). In this context they mention “mnemonic interlock”—the tendency to get stuck at a categorical level of description—and they note that this issue is not specific to emotional memories but extends to neutral memories as well. This is very clinically relevant because if inability to draw up specific memories is directly related to inability to imagine a concrete (and hopefully better) future, that could lead directly to hopelessness and to suicidality. Note that the “interlock” refers to the idea that the subject stops searching at an intermediate level, and then that the products of this intermediate-level search just activate representations at the same level of specificity, so the subject keeps getting categorical memories back.
* *Study 1:*
  + Group 1 = 24 hospital patients who survived a suicide attempt by poisoning; Group 2 = 24 hospital patients in for minor medical issues; Group 3 = 24 healthy controls. Nice to have both control groups . . .
  + The suicide group is more depressed (BDI) and hopeless than both control groups, which do not differ. There are no group differences on verbal fluency, and in fact the mean is higher in the suicide group relative to the hospital controls, so that suggests that we’re not seeing a general cognitive deficit.
  + Because the two control groups did not differ on any measure, they were combined into a single group for subsequent analyses.
  + Regarding the AMT, there was a main effect of group, as the parasuicide group recovered fewer specific memories than the controls. This did not interact with the valence of the memories, although—strangely enough—the neutral cue prompted the most specific memories (main effect of valence).
  + The same pattern was observed for prospection: specificity was lower in the suicide group relative to the controls, and although this did not interact with valence, there was a main effect of valence due to increased specificity in response to the neutral cues.
  + Across both groups, there are reliable positive correlations between specificity of memories and specificity of imagined future events, *r*s > 0.5, *p*s < 0.001. There was no modulation by valence.
  + Relationships between either overly general memories or overly general prospection and BDI scores were small (*r*s< 0.23) and non-significant, with the exception of a correlation with prospection in the *controls*.
* *Study 2*:
  + The goal is to see whether the basic pattern of results from Study 1 can be reproduced by inducing a negative or specific cognitive style.
  + Participants. 40 healthy undergraduates, 20 each assigned to specific vs. generic induction groups.
  + Method. In the induction phase, participants in both groups see positive, negative, and neutral cue words and are asked to reply with either a specific memory from their past (specific induction) or a description of the type of event the cue reminds them of (generic induction). Then, in the test phase, they are cued with more positive, negative, and future words and are asked to describe the kind of event that might happen to them.
  + Results. As expected, the specific group generates much more specific events than the generic group during the induction phase. Valence does not have any effect here. More importantly, there is the expected group difference (specific > generic) in the specificity of imagined future scenarios. Valence does not interact with group on this, but negative cues elicit less specific scenarios.
* *Study 3*:
  + The goal of this experiment is to see whether the Study 2 results can be obtained when demand characteristics are minimized. Rather than ask for specific vs. generic responses, the experimenters use cue words of high vs. low imageability to elicit them.
  + Participants. Healthy undergraduates (*n* = 34).
  + Method. Half the participants (*n* = 17) are assigned to the specific and generic induction groups, in which they are told to generate event descriptions in response to high vs. low imageability cues, respectively. Importantly, everyone is given an example of a specific and a generic response (without them being called that) in the instruction period. Following this, they generate responses to the same group of 12 test words, which (I think) are of intermediate imageability.
  + Results. The inductions work, in that more specific memories are produced in response to the high vs. low imageability cues. Critically, this carries over to the test phase, with participants who received the specific induction generating more specific responses. Everyone is also more specific to neutral vs. positive or negative cues (i.e., main effect of *Valence*, no interaction).
  + The overall discussion begins with the conclusion that there must be some link between the specificity of retrieval and prospection.
  + The interpretation is that you get mnemonic interlock in psychopathology either because the participants find it too upsetting to retrieve specific memories, or because there is some basic deficit in EF that prevents the subject from moving down to a more precise level of retrieval.

6. **Raes et al. (2005)**

* A paper examining reduced memory specificity as mediating the link between rumination and poor problem-solving in depression.
* Participants. 24 depressed women.
* Method. Participants complete the AMT (which provides a measure of Autobiographical Memory Specificity [AMS]), the Means-Ends Problem Solving task (MEPS), the BDI, two measures of rumination, and the Letters Numbers Sequencing task (LNS), which is a measure of working memory.
* Results. Replicating prior work, they find negative correlations between AMS and rumination, plus a positive correlation between AMS and MEPS (more specific memories = better problem-solving). Rumination and MEPS are also negatively correlated. Using the Baron-Kenny approach, they find evidence that AMS mediates the relationship between rumination and MEPS. In other words, the relationship b/w AMS and MEPS is stronger than the relationship between rumination and MEPS. Moreover, the relationship b/w AMS and MEPS is not affected when they control for BDI, so it’s not just a reflection of depression severity or some non-specific psychopathology. Also, they find no relationship b/w MEPS and LNS (working memory).

7. **Dalgleish et al. (2007)**

* Focused on deficits in executive function as a cause of the autobiographical memory abnormalities in depression. This paper includes 8 studies!
* Begin by noting: (a) a relationship between reduced autobiographical memory specificity (AMS) and clinical course in depression; (b) that reduced AMS is observed in remitted as well as acute depression; (c) that at least one study has found a link between reduced AMS and poor problem-solving; and that (d) there is a relationship between AMS and ability to visualize the future.
* Interesting discussion of the process of memory retrieval here. The idea is that to retrieve a specific memory in response to a cue, you first generate a set of “categorical descriptors” that constrain the search space, and then you repeat that processes iteratively until you land on an event that satisfies your search criteria (it’s not entirely clear here why the categorical descriptors are needed—seems like you could just rely on the cue for that). The proposal is that (a) subjects generate more categorical descriptors than are necessary, (b) unnecessary descriptors must be inhibited, (c) depressed adults (or adults with PTSD, or survivors of childhood abuse) have difficulty achieving the inhibition and so (d) they are stuck at the level of the categorical descriptors. This serves (or is driven by?) a need to avoid the emotional pain that a truly specific memory of a negative event would elicit, or so the thinking goes. But here they emphasize an alternative hypothesis: that reduced AMS in depression is simply a consequence of poor EF/attentional control.
* They cite Hertel’s cognitive-initiative model as helpful, noting that it would predict poor performance on the AMT in depression because depressed adults have trouble initiating, maintaining, and re-engaging strategic performance unless external support is provided.
* No need to review all 8 studies in detail here, but suffice it to say that they consistently find negative correlations b/w BDI and AMS that can be explained by poor performance on measures of executive control (controlling for depressed mood). The strongest evidence for this basic relationship comes from Study 8, where they reverse the instructions so that participants are asked to produce general, “categorical” memories (summaries that span several events) rather than specific memories—when they do this, there is a positive relationship b/w BDI and too much specificity! In other words, the problem appears to be related to EF and not specificity per se: MDDs simply have trouble coming up with answers that meet the multiple constraints imposed by the task (must be a memory, must be specific, must relate to the cue). This is interesting for us because, again, it suggests that you might see retrieval deficits in other tasks.

8. **Airaksinen et al. (2005)**

* Meta-analysis of neuropsychological studies of episodic memory in anxiety disorders
* Not huge samples, n = 112 with anxiety vs. n = 175 healthy. The anxiety group includes panic, GAD, SAD, OCD, phobia. But note that all the participants come from a broad sample taken in Sweden, which likely makes the results generalizable (i.e., the anxiety disorder group is living in the community). A nice point is that everyone was tested individually, and all the neuropsych stuff was done before the interview used to assign diagnosis.
* Hah, from the abstract can see that they will conclude that GAD (together with specific phobia) does not affect episodic memory.
* One immediate point: much higher proportion of females (72% vs. 49%) in the anxiety disorder group. On the other hand, the anxious group is modestly but significantly younger (~40 years vs. ~44 years).
* The episodic task involves 32 common neutral words: 4 from 8 different categories. At encoding the participants are warned that their memory will be tested but their attention is not drawn to the semantic organization of the word list. At encoding, the entire list is read aloud at a rate of 3 s per word; free recall immediately follows. After that, there is a cued recall session in which the participants are given the 8 category titles and asked to produce as many words as they can recall from each category.
* The participants also complete the Word Association Test (F, A, S: say as many words beginning with this letter as you can in one minute) to test verbal fluency, and the complete the Trail Making Test versions A (1-2-3-4) and B (1-A-2-B-3-C) as measures of perceptual-motor speed and EF, respectively.
* Given the small samples for some diagnoses (e.g., n = 7 for GAD), I am inclined to focus on the “controls vs. all anxiety disorder” comparisons, which show deficits in (a) free recall, (b) cued recall, and (c) Trails B, for speed but not accuracy. This is evidence for a deficit in episodic memory and EF. I wish they’d tested recognition so we could tell whether this deficit was due to encoding vs. retrieval—I suspect, but can’t know for sure, that it’s encoding-related.
* They account for the fact that 34 adults with anxiety also have depression, and they find no difference in the results. But I note that that’s a relatively small n given the overall sample size.
* Ah, they also suggest that the episodic memory deficit in anxiety stems from problems at encoding rather than retrieval, because the anxious and healthy patients show a similar benefit when going from free to cued recall. This implies that they are getting out whatever’s in there in a similar way (and presumably, similarly effectively) as is seen in controls. The implication is that less got in there in the anxious group (and by “got in there” I mean “was encoded successfully”).
* Turns out this group did similar work focused on MDD . . . let’s look at that next as it’d be great to bring up one paper and then the other in the Introduction.

9. **Airaksinen et al. (2004)**

* These data are from the same sample as used in their paper on anxiety, and the controls are the same, I think (*n* = 175). But here the patient groups are major depression (*n* = 68), dysthymia (*n* = 28), mixed anxiety-depression (*n* = 25) and “minor depression” (*n*  = 66), where minor depression is
* As a group, the patients show impaired episodic memory and impaired mental flexibility (I’m thinking they’re referring to Trails B). But their sub-group analysis suggests that the episodic deficit is specific to major and mixed depression, which corresponds to our group.
* Yes, the pattern of results is exactly the same as in their paper with anxiety disorders: the depressed group as a whole shows deficits in free and cued recall, as well as in Trails B speed. Because both groups improve going from free to cued recall, they surmise that they’re looking at an encoding deficit. As noted above, for free and cued recall the deficits are significant in major and mixed depression considered alone. They find no evidence of a medication effect on memory.
* They take this study (along with their study in anxiety) as primarily pointing to encoding deficits, but they point to Ilsley et al. (1995) and Massman et al. (1992) as arguing for retrieval deficits in MDD. Let’s look at Ilsley next . . .

10. **Ilsley et al. (1995)**

* This is a small study (15 MDD [11 medicated] vs. 15 controls), and the basic result is that they find no group differences in recognition (or working memory, implicit memory, or semantic memory), but they find a difference in immediate and delayed recall. Consequently, they argue that the problem in their group is with retrieval, not encoding: “Depressed patients may have particular difficulty in organizing the cognitive operations necessary for successful search and retrieval” (p. 8).
* They find no correlations with depressive severity, and they find no effect of emotion (pos, neg, neu) on implicit or explicit memory.
* I think the most parsimonious way to look at all this is to say that, across depression and anxiety, there is a cognitive impairment that is liable to affect both encoding and retrieval depending on which is more demanding and/or less well-supported.

11. **Zlomusica et al. (2014)**

* Examines recent evidence for disrupted episodic memory in anxiety disorders.
* The bottom line is that evidence for impaired neutral, episodic memory in various anxiety disorders is, at best, mixed. The Airaksinen et al. (2005) study is a positive example but there are many negative examples, especially from studies that used experimental methods.

12. **Brown et al. (2012)**

* Interesting study in which 33 healthy undergraduates undergo either a high self-efficacy induction (n = 17) or a low self efficacy induction (n = 16) and then are asked to recall/imagine a past/future experience in response to negative and positive cue words. The key results are that (a) memories and imaginings are more specific following the high vs. low self-efficacy induction, and (b) memories are more specific than imaginings in response to the negative cues, but (c) imaginings are more specific than memories in response to the positive cues.
* The *t*-values for the between-groups differences are pretty large, mostly 3s and 4s.
* Regarding problem-solving, they find that overgeneralized positive imaginings predict worse scores on the MEPS.

13. **Sumner et al. (2010)**

* Meta-analysis on the links b/w overgeneral memory and prognosis for depressed adults; this is from Sue Mineka’s group (you should re-read Werner-Siedler & Dalgleish as well)

Maybe save for the Discussion

0. A key point is that Hertel predicts a depression deficit only in cases where one could use a strategy but it’s not well-signaled . . . if there’s little need for (or use of) a strategy in the controls, then you won’t see a deficit in MDD. Thus, I’m not sure she would expect a deficit in Side because that’s not a condition that naturally suggests a strategy for controls.

0. Might want to add correlation with BDI since you’ll say MASQ anxiety measures don’t improve on that . . .

A parsimonious way to accommodate all these results is to point out that depression is associated with deficits in executive function important for effective encoding and retrieval. In fact, impaired executive function in depression was highlighted in two of the neuropsychological reviews already mentioned (Airaksinen, Larsson, & Forsell, n.d.; Rock et al., 2014), and a meta-analysis of over 100 studies confirmed the presence of broad deficits in executive function in depression (Snyder & Snyder, 2012).

The relevance of executive function to memory retrieval has been made strikingly clear in work on the overgeneral autobiographical memory phenomenon (Williams et al., 2007). Overgeneral autobiographical memory refers to the fact that when depressed adults are cued to retrieve specific (i.e., lasting one day or less) autobiographical memories they often struggle to do so, instead offering “categorical” memories that span several episodes and thus exceed the 1 day time limit (e.g., cued to retrieve a positive memory, a depressed adult might reply, “When we went on family vacations” rather than “When my parents took me to see the Red Sox in 3rd grade”). Overgeneral autobiographical memory is important: it predicts a longer course of illness (Brittlebank, Scott, Williams, & Ferrier, 1993; Peeters, Wessel, Merckelbach, & Boon-Vermeeren, 2002; Sumner, Griffith, & Mineka, 2010), and increasing retrieval specificity can decrease hopelessness and brooding rumination, improve problem solving, and lead to sustained remission (Neshat-Doost et al., 2012; Raes, Williams, & Hermans, 2009). With this in mind, a new “memory therapeutics” approach is focused on increasing the precision of autobiographical memory retrieval as a means of alleviating depression (Dalgleish & Werner-Seidler, 2014). Given these facts, a recent manuscript describing a reliable relationship between impaired autobiographical memory retrieval and decreased executive function over a series of eight separate experiments is particularly noteworthy (Dalgleish et al., 2007). The most striking result emerged from the eighth experiment, in which participants were cued to retrieve *non-specific* memories extending over more than one day. Reasoning that the typical deficit observed in depression reflects difficulty satisfying multiple criteria simultaneously (retrieve cue-related memory, must be autobiographical, cannot last more than one day) rather than anything about specificity *per se*, the authors predicted that depressed adults would now show *more* specificity than controls—and indeed, this is what was observed: relative to healthy controls, depressed adults had more difficulty generating categorical memories on demand, consistent with the idea that diminished exee