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## ***Interactive comment on “Carbon isotopes support Atlantic meridional overturning circulation decline as a trigger for early deglacial CO<sub>2</sub> rise” by A. Schmittner and D. C. Lund***

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The questions of what drove the CO<sub>2</sub> rise during HS1, and what AMOC variability does to CO<sub>2</sub> in general, are both really interesting, and really difficult. Published models disagree on the impact of an AMOC shutdown (as pointed out by Referee #2), which suggests a real need to bring in tighter observational constraints to test model predictions.

In this light, it is great to see the global array of foraminiferal carbon isotope measurements being brought to bear on these problems. This paper uses the carbon isotopes in the right way, in comparing to a good ocean model that accounts for both the phys-

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ical processes (including air-sea exchange) and the biological respiration. As such, I think this is a very useful set of simulations and comparisons with data.

However, I'd like to raise two points with regard to the conclusions.

1. The model simulations start from pre-industrial boundary conditions (also pointed out by Referee #2). As shown by Schmittner et al. (2007), the effect of water-hosing on the UVic model in a pre-industrial state is much larger (27 ppmv) than on an LGM state (5 ppmv). Since the real deglaciation started from the LGM state, it seems the impact of the AMOC shutdown on CO<sub>2</sub> should be overestimated by something like a factor of five in the model experiments shown here. This contrasts with marine isotope stage 3, when the intermediate ocean state would have presumably still left the AMOC with a larger amount of leverage on CO<sub>2</sub>. So if Schmittner et al. (2007) is still right, the actual direct effect of the AMOC shutdown during HS1 should have been much smaller, according to the UVic model (nevermind disagreement with other models, which show equivocal impacts of AMOC shutdown on CO<sub>2</sub>).

2. The comparison with data stops short just before the B-A. Prior experience with the UVic model shows clearly that the ocean will take up CO<sub>2</sub> once again when the AMOC resumes (e.g. Schmittner and Galbraith, 2008). However, this did not happen during the deglaciation - instead, there was a permanent, net increase in CO<sub>2</sub> between the LGM and the B-A. The mechanism behind the HS1 CO<sub>2</sub> increase was therefore either a) did not include an input from the AMOC shutdown, or b) did include an input from the AMOC shutdown, which was followed immediately by a compensatory subsequent source of CO<sub>2</sub> that masked the AMOC re-uptake of CO<sub>2</sub> during the B-A.

Together, these two points suggest to me that the AMOC variability played only a minor role in the CO<sub>2</sub> rise during HS1. That's not to say it didn't have any role, nor that it didn't have a big impact on the redistribution of carbon isotopes within the ocean during HS1 - it probably did, and these results provide very useful support to the idea that NADW formation really did shut down at the time. But I think there's enough wiggle

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room between the model  $\delta^{13}\text{C}$  and the foraminiferal  $\delta^{13}\text{C}$  observations to allow other processes, such as iron fertilization, changes in sea ice, changes in Southern Ocean convection, and changes in the marine ecosystem to have done the heavy lifting of atmospheric  $\text{CO}_2$  during HS1.

If the authors agree with me (at least partially) on these points, perhaps it would be helpful to somehow quantify the model disagreement in a way that would suggest the non-AMOC processes that contributed to the net  $\text{CO}_2$  rise during HS1? In other words, could the model-data mismatch help to diagnose any other processes that were behind the net  $\text{CO}_2$  rise?

Best regards, Eric Galbraith

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