Associations between delay discounting and expectancies for alcohol analgesia excluding influential cases (N=47)

The overall EAA model was non-significant and accounted for 13.9% of the variance in expectancies for alcohol analgesia [F(4, 42)=1.69, p=.17]. Delay discounting rates (*b*=-7.32, p=.28, 95% CI [-20.78-6.13]), total QFI *(b*=2.31, p=.70, 95% CI [-9.77-14.39]), and sex *(b*=-3.02, p=.39, 95% CI [-10.00-3.95]) did not significantly predict EAA scores. The sex x delay discounting interaction was significant (*b*=-18.10, p=.02, 95% CI [-32.50--3.69]).

For AE VAS 1, the regression model was non-significant and accounted for 19% of the variance in expectancies for alcohol analgesia [F(4, 42)=2.45, p=.06]. The main effects of delay discounting rates (*b*=-2.23, p=.75, 95% CI [-16.48-12.01]), and sex (*b*=-5.30, p=.15, 95% CI [-12.68-2.08]) were non-significant, while total QFI (*b*=14.04, p=.03, 95% CI [1.25-26.83]) was a significant predictor of AE VAS 1 scores. The sex x delay discounting interaction was also significant (*b*=-16.45, p=.04, 95% CI [-31.70--1.20]).

The AE VAS 2 model was non-significant and accounted for 12.7% of the variance in expectancies for alcohol analgesia [F(4, 42)=1.53, p=.21]. The main effects of delay discounting rates (*b*=-6.02, p=.24, 95% CI [-16.29-4.26]) and total QFI (*b*=1.90, p=.68, 95% CI [-7.32-11.12]) were non-significant. Sex was a significant predictor of AE VAS 2 scores (*b*=-5.49, p=.04, 95% CI [-10.81 - -.17]), such that women reported higher average scores than men. The sex x delay discounting interaction was not significant (*b*=-8.15, p=.14, 95% CI [-19.15-2.85]).

Decomposition of interaction effects indicated differential associations between delay discounting rates and alcohol analgesia expectancies for men and women, while controlling for average alcohol consumption. For women, positive associations between delay discounting rates and EAA (*b*=8.51, p=.26), AE VAS 1 (*b*=12.40, p=.18), and AE VAS 2 (*b*=1.36, p=.82) were

non-significant. For men, delay discounting rates were a significant negative predictor of EAA (*b*=-31.67, p=.03) and AE VAS 1 (*b*=-24.61, p=.02). Delay discounting rates did not significantly predict AE VAS 2 scores among men (*b*=-17.23, p=.09).

Exploratory analyses without inclusion of typical drinking as covariate

For the EAA, delay discounting rates and sex remained non-significant predictors of alcohol analgesia expectancies (ps>.05), and their interaction remained significant (*b*=-14.85, p=.02, 95% CI [-26.91 - -2.79]). The same pattern of results for AE VAS 1 was observed, such that the sex x delay discounting interaction remained significant (*b*=-15.48, p=.02, 95% CI [-28.67--2.30]), while main effects of delay discounting rates and sex were non-significant (ps>.05). For the AE VAS 2 model, non-significant main effects of sex and delay discounting rates were observed (ps>.05), and the sex x delay discounting interaction was also non-significant (*b*=7.22, p=.10, 95% CI [-1.48-15.93]).

Post-hoc multiple linear regressions were also conducted to decompose significant sex x delay discounting interactions without adjustment for typical alcohol consumption. For women, positive associations between delay discounting rates and EAA (*b*=11.49, p=.09) and AE VAS 2 (*b*=1.49, p=.77) were non-significant. Delay discounting rates were a significant positive predictor of AE VAS 1 scores (*b*=15.74, p=.05). For men, negative associations between delay discounting rates and EAA (*b*=-18.22, p=.09), AE VAS 1 (*b*=-15.23, p=.15), and AE VAS 2 (*b*=-12.96, p=.08) attenuated and were non-significant.