Results of the LNPT analysis showed a significant difference in the GAF scores over all three assessment points between the BD patients with higher and lower SCZ-PRS load (p<0.01). When comparing BD patients at the lower and upper 33.33% of the SCZ-PRS distribution, the observed group difference was even more pronounced (p<0.004).

Figure 1. SCZ-PRS was significantly associated with both (1) premorbid (R2=0.02) and (3) current level (R2=0.013) of functioning in BD patients, (though the association with current GAF lost significance after correcting for multiple testing). No association with (2) worst ever GAF was detected.

Figure 2. No significant correlation was seen between SCZ-PRS and (1) premorbid,
(2) worst ever or
(3) current GAF at any of the pTs.

Figure 3. SCZ-PRS significantly predicted (1) age at onset in BD (though not after correction for multiple testing), but (2) not in SCZ.

Discussion
This study was the first to investigate the influencing role of SCZ-PRS on the level of functioning and age at onset in SCZ and BD. The observed negative correlation between SCZ-PRS and premorbid functioning and age at onset in BD suggest, that having a higher polygenic load on SCZ, a condition with pronounced premorbid impairment in all levels of life, might contribute to a similar premorbid clinical picture in BD. The analysis of the longitudinal course of GAF gives further evidence for the influencing role of SCZ-PRS on BD functioning as BD patients with higher SCZ polygenic load tended to have a more impaired functioning across all measurement points compared to those with less SCZ polygenic risk.

We are currently working on a replication study in more than 700 longitudinally followed patients with SCZ and BD, featuring detailed GAF assessments to see if our results also stand in an independent population.

Acknowledgements
We thank all of the patients for participating in this study. This research was funded by the Deutsche Forschungsgemeinschaft (DFG): Klinische Forschergruppe (KFO) 241: TPI (SCHU 1603/5-1), FKZ RO4076/1-1 and FKZ RO4076/3-1. Thomas G. Schulze was supported by the Lisa-Dehler-Foundation.