15 showed ratios of lipid-adjusted hepatic to visceral fat lipid-adjusted concentrations exceeding 1. However, given the modest sample size here, no definitive conclusions can be drawn regarding the possibility of a specific binding mechanism for BDE 47 in human liver.

These data provide for the first time data on concentrations of brominated dioxins and furans as well as PBDEs in paired liver and adipose tissue samples. Based on the provisional TEF values assigned to PBDD/F compounds, these data suggest that brominated dioxins and furans constitute a relatively low fraction of total TEQ. Chlorinated TEQ, but not brominated TEQ or PBDEs, show a positive trend with BMI, with an increase of 4 BMI units associated with an increase in lipid-adjusted TEQ in visceral fat of approximately 1 ng/kg lipid. While the chlorinated TEQ concentrations display a positive trend with age typically observed for these compounds, no such trend is apparent for the brominated TEQ or for PBDE compounds. Two possible reasons may contribute to this observation. First, the notable temporal trend of declining exposure levels with time since the 1970s that has been observed for chlorinated TEQ constituents may not apply to the brominated compounds. Thus, older individuals would not carry an increased body burden due to the higher exposures in earlier time periods. Second, even if a declining trend in exposure exists, if the half-lives of elimination are not as extended for the brominated compounds, differences in exposure levels from earlier time periods would not leave as distinct of a residual signature in tissue concentrations.

Further work with this population will examine changes in tissue concentrations and body burdens over time for these compounds following weight loss.

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