comorbidity (AD+D group) (age 41.2 (SD 9.903), 22% females) and 112 healthy controls (age 35.5 (SD 8.286), 15% females). rs1108580 and rs1611115 were detected by RT-PCR.
Results: For rs161111580, frequencies of minor T allele (p=0.031) and TT genotype (p=0.017) was higher, CC genotype (p=0.042) was lower in AD group vs. controls. rs161111580 T allele and TT genotype increases the risk of AD (OR=3.715, 95%CI [1.728-7.986], P=0.001 and OR=4.009, 95%CI [1.502-10.699], P=0.006). For rs161111580, frequency of TT genotype (p=0.009) was higher in AD+D group vs. controls. For rs1108580, frequency of major A allele (p=0.059, trend) was higher in AD+D, then in AD group. Major A allele rs1108580 increases the risk of depression in alcohol-dependent patients (OR=2.74, 95%CI [1.283-5.855], P=0.001).
Conclusions: It was shown that the DBH rs1108580 increases the risk of depression in patients with alcohol dependence.

Disclosure: No significant relationships.
Keywords: Alcohol dependence; Dopamine; Genetics; Depression

EPP0501

Symptoms of diabetes distress, depression, and anxiety in people with type 2 diabetes: identifying central and bridge symptoms using network analysis

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Introduction: People with diabetes are vulnerable to diabetes-related distress and are more likely to experience depressive and anxiety symptoms than the general population. Diabetes distress, depressive, and anxiety symptoms also tend to commonly co-occur.
Objectives: This study aimed to apply network analysis to explore the associations between diabetes distress, depressive, and anxiety symptoms in a cohort of adults with type 2 diabetes.
Methods: Data were from the baseline (2011) assessment of the Evaluation of Diabetes Insulin Treatment (EDIT) study ($N = 1,796$; 49% female; mean age = 60, $SD = 8$) from Quebec, Canada. A first network using the 17 items of the diabetes distress scale (DDS-17) was estimated. A second network was estimated using the 17 items of the DDS-17, the 9 depressive items of the PHQ-9, and the 7 anxiety items of the GAD-7. Symptom centrality, network stability, and bridge symptoms were examined.
Results: Regimen-related and physician-related distress symptoms were amongst the most central (highly connected) in the diabetes distress network. *Worrying too much* (anxiety), *Not feeling motivated to keep up diabetes self-management* (diabetes distress), and *Feeling like a failure* (depression) were the most central symptoms in the combined network. *Feeling like a failure* (depression) was highly connected to diabetes distress symptoms, representing a potential bridge between diabetes distress and depression.
Conclusions: Identifying central and bridge symptoms may provide new insights into diabetes distress, depressive, and anxiety symptom maintenance and comorbidity in people with type 2 diabetes.

Disclosure: No significant relationships.
Keywords: Network Analysis; comorbidity; diabetes; diabetes-distress

EPP0502

Evaluation of the role of lisdexamfetamine on attention-deficit/hyperactivity disorder common psychiatric comorbidities: mechanistic insights on binge eating disorder and depression

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Introduction: Attention-deficit/hyperactivity disorder (ADHD) is a psychiatric condition in which children suffer from inattentiveness, hyperactivity, and or impulsivity. ADHD patients frequently present comorbid psychiatric disorders: in adults, the most common are depression, substance-related disorders, anxiety, and eating disorders. Children and adolescents present conduct disorders, learning disorders, anxiety and depression. Since ADHD and its psychiatric comorbidities share similarities, a partial overlap of their pathophysiological mechanisms has been suggested. ADHD, can be treated with lisdexamfetamine (LDX), a prodrug indicated by the FDA as treatment for binge eating disorder (BED) and ADHD.
Objectives: To evaluate, through a systems biology-based *in silico* method, the efficacy of LDX as first-line ADHD treatment to improve ADHD psychiatric comorbidities. Furthermore, we explored the molecular mechanisms behind LDX's action.
Methods: We used the systems biology- and artificial intelligence-based Therapeutic Performance Mapping System (TPMS) technology to characterise and model ADHD comorbidities. Artificial neural networks (ANNs) algorithms were used to identify specific relationships between protein sets. Finally, we modelled the mechanisms of LDX for the most relevant comorbidities by using sampling methods and comorbidity-specific virtual patients in each case.
Results: This study predicts a strong relationship between LDX's targets and proteins involved in BED and depression (Fig 1). Our results could be explained not only by LDX role in neurotransmitter regulation, but also by modulation of neuroplasticity (BDNF/NTRK2, GSK3), neuroinflammation (interleukins, inflammasome), oxidative stress (NOS2, SOD), and the hypothalamic-pituitary-adrenal (HPA) axis (CRH, CRHR1).