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Screen Time and Depression

According to the results of a longitudinal study, time spent viewing social media and watching TV is associated with depression in adolescents, possibly via a reinforcing spiral of upward social comparisons. Time spent playing video games or in general computer use did not increase depression and may have even had positive effects on self-esteem.

Background: Several hypotheses could explain the association of screen time with adolescent depression. The displacement hypothesis presumes that irrespective of content, screen time displaces time spent in healthier activities. The upward social comparison hypothesis presumes that negative comparisons with others with idealized bodies, better lives, etc. occur with social media or TV exposure, but not other types of content. Finally, the reinforcing spirals hypothesis proposes that individuals seek out information consistent with their cognitions, such as violent or political content. The present study was undertaken to test all 3 hypotheses.

Methods: This analysis was part of a larger 4-year study of drug and alcohol prevention in students in Montreal. Adolescents were assessed every year from grades 7 through 11 using a confidential web-based survey of screen time spent on video games, social networks, watching shows or movies on TV or the computer, and other activities on the computer. Depression symptoms were assessed using the Brief Symptoms Inventory, which rates the severity of 7 specific depression symptoms on a scale from 0 (not at all) to 4 (very much). Self-esteem and exercise were also measured every year. Effects of screen time on depression were analyzed between persons, indicating a main effect of media exposure, and within persons, indicating self-reinforcing effects.

Results: The analysis was based on nearly 37,000 adolescents (47% girls) with a baseline mean depression severity score of 4.3 (maximum possible score, 28). Higher average social media use was associated with an increase in depressive symptom severity over the 4 years, with both a between-person and a within-person effect. (See table, next page.) Increased computer use was also associated with between-person increased depression severity. Between persons, TV watching was associated with less severe depression symptoms, but within individuals,

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increases in TV watching were associated with increases in depression. No associations were found for video gaming.

A post-hoc analysis examined the relationship of screen time and depression with self-esteem and exercise. No effects were found for exercise. All 4 types of exposure were associated with lower self-esteem in the within-person analysis. Between persons, video gaming and computer use were associated with decreases in self-esteem.

Depression severity change for each 1-hour increase in screen time		
Type of screen time	**Between-person**	**Within-person**
Social media	0.64	0.41
Video gaming	0.15	0.02
Television	-0.22	0.18
Computer use	0.69	0.09

Severity measured on a scale from 0 to 28, with higher scores indicating greater depression.

Discussion: The results of this study do not support the displacement hypothesis. The lack of association between depression and video gaming reflects other research and suggests video gamers are not socially isolated; rather, they tend to play with friends or online groups, and playing can generate positive feelings. Increased general computer use in adolescents may reflect increases in computer experience, skills, and self-efficacy. High use of social media contributes to depression, possibly by providing the opportunity for negative social comparisons and lowering self-esteem; and there is evidence of a reinforcing spiral. High levels of TV watching were associated with less depression, but increases over the 4 years were associated with increased depression.

Boers E, Afzali M, Newton N, Conrod P: Association of screen time and depression in adolescence. *JAMA Pediatrics* 2019; doi 10.1001/jamapediatrics.2019.1759. From the University of Montreal, Canada; and other institutions. **Funded by the Canadian Institutes of Health Research; and other sources. The authors declared no competing interests.**

Brain Connectivity and Self-Injury

Differences in baseline brain network connectivity distinguished patients with nonsuicidal self-injury (NSSI) from healthy controls and also predicted patients' response to psychotherapy. These observations may contribute to the personalized treatment of NSSI, as well as advancing understanding of the mechanisms involved in the disorder.

Background: Connectivity of regions examined in the study—the amygdala and the medial prefrontal cortex (mPFC)—is known to be involved in emotional regulation and to be sensitive to the effects of psychological treatments. The present study was undertaken to evaluate functional connectivity between frontolimbic regions in adolescents with NSSI, compared with healthy adolescents, and to investigate baseline intrinsic neural connectivity patterns that may be associated with response to psychological interventions.

Methods: Study participants were 24 adolescents (aged 12–18 years) who had engaged in repetitive NSSI over the past 12 months and 16 healthy controls. Adolescents with NSSI were enrolled from an outpatient child and adolescent psychiatry clinic and randomly assigned to receive 16 weeks of treatment as usual or a dialectical behavior therapy (DBT)-based psychosocial intervention. Structural and functional MRI images were obtained at baseline from all participants, and NSSI was assessed using the Columbia Suicide Severity Rating Scale (CSSRS).

Results: Patients randomly assigned to DBT or treatment as usual had similar rates of response and dropout, and the 2 groups were combined for the functional connectivity analysis. MRI found functional connectivity was reduced in patients with NSSI compared with controls in 4 networks: amygdala–anterior cingulate cortex, subcallosal cortex, paracingulate cluster;

amygdala–right planum temporale and right insula cluster; mPFC–precentral and postcentral gyri; and mPFC–left insula.

On average, adolescents with NSSI experienced episodes at least monthly but less than weekly at study entry. After treatment, 12 of the 24 patients reported improvement, with less frequent episodes of NSSI. Of the remaining 12 patients, 6 had reported no current NSSI at baseline, 4 reported unchanged frequency at study end, and 2 patients worsened during treatment. Improvements in NSSI correlated negatively with connectivity of the amygdala with the anterior cingulate cortex (correlation coefficient* [r]=-0.642; p=0.001) and positively with amygdala–brainstem connectivity (r=0.747; p<0.001). Greater improvement in NSSI was also associated with reduced positive connectivity and greater negative connectivity between the mPFC and amygdala (r=-0.630; p=0.001) and between the mPFC and intracalcarine cortex (r=-0.471; p=0.002). Background medications, in the 18 patients taking them, were not associated with baseline differences in connectivity or with changes in NSSI.

Discussion: These observations suggest the anterior cingulate cortex has a reduced ability to inhibit amygdala activity in adolescents with NSSI, resulting in impaired regulation of emotional behavior. Other observed findings could explain altered pain perception and aberrant perception of bodily sensations. Psychotherapy may improve emotion regulation skills associated with the mPFC and improvement in prefrontal connectivity might be associated with response to psychotherapy.

Santamarina-Perez P, Romero S, Mendez I, Leslie S, et al: Fronto-limbic connectivity as a predictor of improvement in nonsuicidal self-injury in adolescents following psychotherapy. *Journal of Child and Adolescent Psychopharmacology* 2019;29:456–465. doi 10.1089/cap.2018.0152. From the Institute of Neurosciences, Hospital Clinic, Barcelona, Spain; and other institutions. **Funded by the Alicia Koplowitz Foundation. Two of 13 study authors disclosed potentially relevant financial relationships; the remaining authors declared no competing interests.**

***See Reference Guide.**

Methylphenidate and Psychosis Risk

According to a population–based study, starting methylphenidate (*Ritalin*) does not increase risk of psychosis in adolescents and young adults, including those with a history of psychosis. This observation contradicts the widely held clinical opinion that methylphenidate should be avoided or restricted in patients with a history of psychosis.

Background: Central nervous system stimulants act as indirect dopamine agonists and have been presumed to amplify neuronal signaling by increasing synaptic dopamine, a mechanism also believed to be implicated in psychosis onset. Psychotic episodes or disorders have been reported as adverse reactions to methylphenidate. Some clinicians believe stimulants are contraindicated in patients with a history of psychosis, leading to prescription of less effective medications for ADHD. Previous observational studies of the question have been inconclusive.

Methods: Study subjects were nearly 24,000 new users of methylphenidate, aged 12–30 years, who were identified from Swedish population-based registries. The main analysis was a within-individual comparison of the incidence of psychosis during 4 separate 12-week time intervals: period 1–beginning 1 year before the first prescription of methylphenidate; period 2–immediately before initiation; period 3–immediately after initiation; and period 4–beginning 1 year after initiation. Patients taking other ADHD medications were excluded. Psychosis was defined as any hospital visit for a psychotic event, or a chronic psychotic condition. Separate analyses were carried out for patients with a history of psychosis (2%) and those without.

Results: A total of 304 patients (1.3%) experienced a psychotic event during the study period. The incidence of psychosis was similar in the 12 weeks immediately before and immediately after the start of methylphenidate (periods 2 and 3), both for patients with and

those without a history of psychosis. (See table). In period 4 (i.e., beginning 1 year after starting methylphenidate), the incidence of psychosis was reduced by 36% in young persons with a history of psychosis. When examining onset of psychosis week by week, the incidence of events was similar across the year before and the year after starting methylphenidate treatment.

Risk of Psychosis after Methylphenidate Initiation		
12-Week Period	**% With a Psychotic Event**	**Relative Risk***
Persons with a history of psychosis (n=479)		
Period 1: 1 year before	11.5%	—
Period 2: immediately before	9.2%	reference
Period 3: immediately after	9.4%	0.95
Period 4: 1 year after	6.3%	0.64
Persons with no history of psychosis (n=23,419)		
Period 1: 1 year before	0.1%	—
Period 2: immediately before	0.3%	reference
Period 3: immediately after	0.3%	1.04
Period 4: 1 year after	0.3%	0.82

In a secondary analysis comparing onset in period 1 with period 4 (i.e., 1 year before starting treatment vs 1 year after) the incidence was reduced by 49% in individuals with a history of psychosis and increased by 72% in those with no history. The investigators attribute this increase to the background pattern of psychosis onset, uninfluenced by methylphenidate. When the analysis was stratified by age (adolescents aged 12–17 years versus young adults), similar patterns were observed.

Discussion: This study found no evidence that initiation of methylphenidate increases the risk of psychotic events in adolescents or young adults, regardless of their history of psychosis. The results challenges a widely held view that methylphenidate should be avoided or restricted in patients with a history of psychosis.

Hollis C, Chen Q, Chang Z, Quinn P, et al: Methylphenidate and the risk of psychosis in adolescents and young adults: a population-based cohort study. *Lancet Psychiatry* 2019;6 (August):651–658. doi 10.1016/S2215-0366(19)30189-0. From the University of Nottingham, U.K.; and other institutions. **Funded by the Swedish Research Council; and other sources. Four of 9 study authors disclosed potentially relevant financial relationships; the remaining authors declared no competing interests.**

***See Reference Guide.**

Developmental Trajectories of Depression

Five trajectories of depression symptoms from late childhood into young adulthood were identified in a longitudinal study. Two of the trajectories were associated with genetic and environmental risk factors.

Methods: The study cohort consisted of participants in the Avon (UK) Longitudinal Study of Parents and Children (ALSPAC)—more than 14,000 individuals born in 1991 or 1992. Depression symptoms were measured on 9 occasions when participants were aged 10–24 years, using the 13-item self-reported Short Mood and Feelings Questionnaire. These data were analyzed to identify latent trajectories of depression symptoms. Risk factors for depression including sex, a polygenic risk score based on a genome-wide association study of depression, maternal postnatal depression, partner abuse of the mother during early childhood, childhood anxiety, and childhood bullying, were evaluated for each identified trajectory.

Results: Nearly 9400 individuals with ≥1 measurement of depressive symptoms were included in the analysis; complete data on risk factors and confounders was available for 3525 individuals. The factor analysis identified 5 trajectories of depressive symptoms. The majority of young people, 71%, had consistently low depressive symptoms. An early-onset adult trajectory, with initially low symptoms that increased during adolescence and young adulthood, was

present in 11% of the cohort. An adolescent-limited trajectory, with elevated symptoms only during adolescence, was present in 9%. Nearly 6% demonstrated a childhood-limited trajectory with high depressive symptoms in childhood that decreased throughout adolescence. Finally, in nearly 3% of individuals a childhood persistent trajectory was evident with moderate symptoms during childhood that increased to high levels in adolescence and early adulthood.

Compared with the stable low symptom trajectory, odds ratios* were increased for nearly all risk factors across the depression trajectories. (See table.) Being female and being bullied were both consistently associated with depression across all trajectories.

Adjusted Odds Ratios for Depression Trajectories by Risk Factor

	Childhood persistent	Early-adult onset	Adolescent limited	Childhood limited
Being female	6.45	1.96	6.04	1.81
Polygenic risk score	1.47	1.29	1.04	1.01
Postnatal depression	2.37	2.39	1.12	1.70
Partner abuse	1.60	1.78	2.30	1.23
Childhood anxiety	1.30	1.12	1.09	1.08
Childhood bullying	4.91	1.73	1.56	8.05

Discussion: These findings highlight several possible mechanisms, both genetic and environmental, that may contribute to the development of depression and could help identify groups with chronic and severe depression that should be prioritized for intervention.

Kwong A, López-López J, Hammerton G, Manley D, et al: Genetic and environmental risk factors associated with trajectories of depression symptoms from adolescence to young adulthood. *JAMA Network Open* 2019; doi 10.1001/jamanetworkopen.2019.6587. From the University of Bristol, U.K.; and other institutions. **Funded by the U.K. Medical Research Council. The authors declared no competing interests.**

***See Reference Guide.**

Metabolic Syndrome in Bipolar Disorder

In adolescents and young adults with bipolar disorder the prevalence of metabolic syndrome is nearly 4-times higher than in the general adolescent population, according to a cross-sectional study. Moreover, metabolic syndrome is associated with increased burden of depressive symptoms. Contrary to expectations, metabolic syndrome was not associated with use of antimanic medications or atypical antipsychotics in the study population.

Methods: Components of metabolic syndrome were assessed in 162 young people enrolled in the NIMH Course and Outcome of Bipolar Youth (COBY) study. Participants with bipolar disorder were enrolled in the study between the ages of 7 and 17 years and continue to be followed longitudinally. For the present analysis, patients completed a metabolic syndrome evaluation an average of 8.5 years after enrollment in COBY (mean age, 21 years; range 13–28 years). Metabolic syndrome was defined using the International Diabetes Federation criteria: central obesity (based on waist circumference) plus ≥2 of the following: high triglycerides, low HDL-cholesterol, high systolic or diastolic blood pressure, and high fasting glucose. Psychiatric symptoms over the 6 months before the evaluation were assessed using the Longitudinal Interval Follow-Up Evaluation (LIFE).

Results: The overall prevalence of metabolic syndrome was nearly 20%. (See table, next page.) Many individuals had components of metabolic syndrome but did not meet the full criteria. Individuals with metabolic syndrome had small but statistically significant increases in the

percentage of weeks in the past 6 months with any full-threshold mood state (odds ratio,* 1.05; p=0.04), weeks with full-threshold pure depression (odds ratio, 1.07; p=0.02), and weeks receiving antidepressant medication (odds ratio, 1.06; p=0.001). Other than antidepressants, metabolic syndrome was not associated with any class of medications.

Prevalence of metabolic syndrome components in adolescents and young adults with bipolar disorder	
Metabolic syndrome: full criteria	19.8%
Individual criteria	
Low HDL-C	56.5%
Abdominal obesity	46.9%
High blood pressure	24.2%
High triglycerides	15.4%
High glucose	15.4%
Number of components	
0	21.3%
1	30%
2	28.1%
3+	20.6%
At least 1 component	78.8%
At least 2 components	48.8%

Discussion: The prevalence of metabolic syndrome in this study was higher than the 5.5% occurring in the U.S. adolescent population, but lower than rates reported in adults with bipolar disorder. If replicated, these observations suggest a need to implement early screening, prevention, and intervention strategies for metabolic syndrome in youth with bipolar disorder. In addition to reducing cardiovascular risks, it is possible that addressing metabolic syndrome could reduce the burden of depression in bipolar disorder.

Li C, Birmaher B, Rooks B, Gill M, et al: High prevalence of metabolic syndrome among adolescents and young adults with bipolar disorder. *Journal of Clinical Psychiatry* 2019; doi 10.4088/JCP.18m12422. From Sunnybrook Health Sciences Centre, Toronto, Canada; and other institutions. **Funded by the NIMH. Fifteen of 16 study authors disclosed potentially relevant financial relationships; the remaining author declared no competing interests.**

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Correlation Coefficient (r): A measure of the closeness of the relationship between two variables. The value of r can range from -1 to 1. An r value near 1 indicates a strong positive relationship. An r-value close to zero indicates no relationship, and a negative r-value indicates a negative relationship.

Odds Ratio: A comparison of the probability of an event in 2 groups. An odds ratio of 1 implies that the event is equally likely in both groups. An odds ratio greater than 1 indicates that the event is more likely to occur in that group than in the comparison group.

Relative Risk: The risk of an event (or of developing a disease) relative to exposure. Relative risk is a ratio of the probability of the event occurring in the exposed group versus the control (non-exposed) group.