



**Developmental changes in brain function linked with
addiction-like social media use two years later**

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3 **Developmental changes in brain function linked with addiction-like social media**
4 **use two years later**
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43

44 **ABBREVIATIONS:** ASMU: Addiction-like social media use; PDS: Pubertal Development
45 Scale; SID: social incentive delay; DSM-5: Diagnostic Statistical Manual V; SMFQ: Short
46 Mood and Feelings Questionnaire; GLMs: general linear models; ROIs: regions of
47 interest; vmPFC: ventral media prefrontal cortex; mPFC: medial prefrontal cortex; PCC:
48 posterior cingulate cortex rIFG; right inferior frontal gyrus; rSTS: right superior temporal
49 sulcus.
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Abstract

Background. Addiction-like social media use (ASMU) is widely reported among adolescents and is associated with depression and other negative health outcomes. We aimed to identify developmental trajectories of neural social feedback processing that are linked to higher levels of ASMU in later adolescence.

Methods. Within a longitudinal design, 103 adolescents completed a social incentive delay task during 1-3 fMRI scans (6th-9th grade), and a 4th self-report assessment of ASMU and depressive symptoms ~2 years later (10th-11th grade). We assessed ASMU effects on brain responsivity to positive social feedback across puberty and relationships between brain responsivity development, ASMU symptoms, and depressive symptoms while considering gender effects.

Results. Findings demonstrate decreasing responsivity, across puberty, in the ventral media prefrontal cortex, medial prefrontal cortex, posterior cingulate cortex, and right inferior frontal gyrus associated with higher ASMU symptoms over 2 years later. Significant moderated mediation models suggest that these pubertal decreases in brain responsivity are associated with increased ASMU symptoms which, among adolescent girls (but not boys), is in turn associated with increased depressive symptoms.

Conclusions. Results suggest initial hyperresponsivity to positive social feedback, before puberty onset, and decreases in this response across development, may be risk factors for ASMU in later adolescence.

KEYWORDS: social media addiction, social feedback, adolescence, puberty, depression

INTRODUCTION

Social media serves many functions and is often part of healthy adolescent development (Deters & Mehl, 2013; Ellis et al., 2020; Flannery et al., 2023; Leung, 2011). However, addiction-like social media use (ASMU) is becoming increasingly reported (Kuss et al., 2014). Despite debate regarding the diagnostic utility of ASMU (Panova & Carbonell, 2022), use of addiction terminology to discuss social media use behaviors has permeated popular culture (Adorjan & Ricciardelli, 2021). Because of this, it is of interest to explore how behaviors reflecting craving for, and difficulty abstaining from, social media occur in adolescence. Accumulating work suggests that ASMU may share some of the same characteristics as other addictive disorders such as sustained preoccupation with cues, use for mood modification, tolerance following repeated use, and withdrawal symptoms following abstinence (Goldberg, 2004). Importantly, ASMU is not necessarily equated with the degree to which one uses social media, but instead captures the degree to which one feels a loss of control over their social media use or experiences negative effects (emotional or circumstantial) due to their use (Baumer et al., 2015; Turel et al., 2018). As ASMU is characterized by maintained or increased social media use despite negative impacts on other aspects of life, and difficulty reducing use despite intentions to do so (Baumer et al., 2015), it is unsurprising that a quickly growing body of work indicates that ASMU may become disruptive to other aspects of life and have detrimental impacts on health and wellbeing (Turel et al., 2018). Indeed, research shows that ASMU symptoms are associated with depressive symptoms (Robinson et al., 2019), especially among adolescent girls (Raudsepp & Kais, 2019). As adolescence is a period of vulnerability for both the onset of internalizing psychopathology (McLaughlin & King,

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2
3 2015) and ASMU symptoms (Stavropoulos et al., 2018), understanding links between
4
5 ASMU and depressive symptoms across this period is vital.
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8 The types of social feedback delivered via social media may be especially relevant
9
10 to promoting addiction-like social media use behavior. Social feedback is both frequent
11
12 and quantifiable (e.g., number of likes, number of followers) via social media. Such social
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14 feedback is also usually reinforcing and delivered on a variable ratio reinforcement
15
16 schedule which is highly resistant to behavior extinction and is thus particularly addictive
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18 (Greenfield, 2007). Social feedback delivery may be especially salient to adolescents, as
19
20 adolescence is thought to be a period of heightened neurobiological and behavioral
21
22 sensitivity to social stimuli, as well as prioritization of social connection and peer
23
24 acceptance (Somerville, 2013). Additionally, adolescence is characterized by a peak in
25
26 reward sensitivity (Lamm et al., 2014) and reward seeking behaviors (Galván, 2013),
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28 particularly in social contexts (Smith et al., 2015). This reward sensitivity is thought to
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30 stem from normative changes in brain structure and function that begin around the onset
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32 of puberty (Padmanabhan et al., 2011). Specifically, neuroimaging and preclinical work
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34 has repeatedly demonstrated links between higher levels of pubertal hormones and
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36 increased reward-related striatum activity among adolescents (Forbes et al., 2011; Op de
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38 Macks et al., 2011). The onset of puberty may thus elicit normative developmental
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40 increases in neural responsivity to reinforcing social feedback (Smith et al., 2015;
41
42 Somerville, 2013). Given these sensitivities, the continuous stream of highly salient and
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44 reinforcing social information dispensed via social media may have a uniquely powerful
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46 impact on adolescents.
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3 Yet, all adolescents may not be equally prone to ASMU due to various individual
4 predispositions, including possible biological vulnerabilities that increase sensitivity to
5 social media cues. For example, individual differences in sensitivity to reinforcing social
6 feedback may determine how adolescents navigate social media environments and the
7 impact those environments have on adolescents' mental health (Sherman et al., 2016).
8 Specifically, adolescents who are more sensitive to social reward may be particularly apt
9 to seek out social media incentives and thus may also be more susceptible to the
10 provocation of continued use or even ASMU (Sherman et al., 2016). While development
11 of adaptive incentive processing during adolescence is important for healthy
12 development, hypersensitivity to rewards has been linked to externalizing and risk-taking
13 behaviors (Bjork & Pardini, 2015) and blunted reward sensitivity has been linked to
14 depression (O'Callaghan & Stringaris, 2019). Further, recent research has shown that, in
15 the context of negative social experiences, neural hypersensitivity to social feedback is
16 associated with an increased susceptibility to depressive symptoms (Pagliaccio et al.,
17 2023) and externalizing behaviors (Turpyn et al., 2021) among adolescents. However,
18 relationships between this neural hypersensitivity and ASMU across development are still
19 not fully understood.

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Prior work shows pubertal increases in neural responsivity to social feedback in
brain regions typically involved in social processing including the medial prefrontal cortex
(mPFC), posterior cingulate cortex (PCC), precuneus, inferior frontal cortex, fusiform
gyrus, and hippocampus (Gunther Moor et al., 2010). However, it is unclear whether
individual differences in this development might constitute risk for future ASMU. We
hypothesize that an initial hypersensitivity to such stimuli may drive some adolescents to

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3 increase their social media use more than others. However, recurrent over-exposure to
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5 social rewards via social media may, in turn, contribute to desensitization to social
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7 rewards across development. This hypothesis is based on prior observations of tolerance-
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9 build up after repeated administrations of an addictive drug (Albrecht et al., 2007; Miller
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11 et al., 1987). Specifically, we hypothesize that adolescents reporting high ASMU in later
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13 adolescence may display initially heightened social reward responsivity that decreases
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15 over years of increasing social media use. This decrease may reflect a desensitization to
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17 rewarding social feedback and a need for more social reward exposure to get the same
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19 reinforcing effects (Griffiths et al., 2014).
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24 The current study investigates individual differences in pubertal trajectories of brain
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26 responsivity to positive social feedback across 6th-9th grade that are related to ASMU ~2
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28 years later. Interactive effects of gender identity were considered given prior work
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30 showing gender effects on social media use behaviors (Nesi & Prinstein, 2015), ASMU
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32 prevalence (Hawi & Samaha, 2019), and relationships between social media use and
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34 wellbeing (Booker et al., 2018). Further, we examine indirect effects of this differential
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36 brain function development on depressive symptoms in later adolescence through
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38 associations with ASMU symptoms, while again, considering gender effects. We
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40 hypothesize that initial hypersensitivity to positive social feedback, and longitudinal
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42 decreases across pubertal development, will be associated with higher ASMU symptoms
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44 in later adolescence. Further, we expect that, higher ASMU symptoms will, in turn, be
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46 associated depressive symptoms, particularly among girls.
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54 **METHODS**

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Participants. Two cohorts of adolescent participants were recruited from 3 public middle schools across 2 years as part of a larger longitudinal study of 6th and 7th grade students (**Figure S1**). The current study examines 103 adolescents that completed a social incentive delay (SID) task across 1 to 3 annual fMRI scan sessions (6th-9th grade; 256 data points), as well as a self-report assessment 2.27±0.21 years following their final fMRI scan (10th-11th grade). The sample of 103 adolescents did not differ from the larger sample from which they were recruited on baseline gender, race/ethnicity, income, pubertal development, or depressive symptoms. Of these 103 adolescents that had data at the final wave, 80 had data at the 1st timepoint, 90 had data at the 2nd timepoint, and 86 had data at the 3rd timepoint (**Table 1**).

Table 1. Demographic information by timepoint.

| Timepoint | 1 st | 2 nd | 3 rd | 4 th |
|------------------------|-----------------|-----------------|-----------------|-----------------|
| | n=80 | n=90 | n=86 | n=103 |
| Gender Identity | | | | |
| Girls | 46.3% | 46.7% | 46.5% | 51.5% |
| Boys | 51.2% | 51.1% | 51.2% | 45.6% |
| Nonbinary | 2.5% | 2.2% | 2.3% | 2.9% |
| Age | | | | |
| | 12.8±0.5 | 13.7±0.6 | 14.7±0.6 | 17.0±0.6 |
| Grade | | | | |
| 6 th | 47.5% | 0 | 0 | 0 |
| 7 th | 52.5% | 51.1% | 0 | 0 |

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|------------------|---|-------|-----|-------|
| 8 th | 0 | 48.9% | 50% | 0 |
| 9 th | 0 | 0 | 50% | 0 |
| 10 th | 0 | 0 | 0 | 53.4% |
| 11 th | 0 | 0 | 0 | 46.6% |

NOTE. Gender identity (% of total), age (mean \pm standard deviation), and grade (% of total) are presented for each data collection timepoint. The multiple cohort structure of the study resulted in planned missing data across timepoints. Specifically, 62 of the total 103 participants had 3 fMRI timepoints, 29 had 2, and 12 had 1. All participants had a 4th self-report timepoint.

Procedures. All participants provided informed consent/assent and were compensated for each completed session. The University's Institutional Review Board approved all aspects of the study. Adolescent participants and their primary caregiver attended annual data collection sessions across 3 timepoints of a longitudinal fMRI study, at which adolescents completed an fMRI scan lasting approximately 1.5 hours. During each scan participants completed a social feedback task called the Social Incentive Delay (SID) task, as well as an anatomical scan and four other tasks that are not the focus of the current manuscript. Following the scan, adolescents and caregivers completed several self-report measures. Adolescents and their caregivers returned about 2.3 years after their final fMRI scan timepoint and completed a 4th self-report assessment.

Measures. Pubertal development. Adolescents' primary caregivers completed the Pubertal Development Scale (PDS) (Petersen et al., 1988) at each of the three fMRI scan

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3 timepoints. Scores ranged from (0): “puberty has not yet started” to (3): “puberty is
4 complete” (Icenogle et al., 2017). Depressive symptoms. Depressive symptoms were
5 assessed at the final timepoint (~2.3 years after the final fMRI scan) in 10th-11th grade,
6 using total scores on the unidimensional, 13-item Short Mood and Feelings Questionnaire
7 (SMFQ; $\alpha=.93$) (Messer et al., 1995), designed to measure depressive symptomology in
8 children and adolescents aged 6-17 years old.
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12 Addiction-like social media use symptoms. ASMU symptoms were also measured
13 at the final timepoint with a novel 7-item questionnaire ($\alpha=.90$) based on selected items
14 from the Diagnostic Statistical Manual V (DSM-5) substance use disorder checklist.
15 Example items include, “Does social media use ever get in the way of things you are
16 supposed to be doing (e.g., sleep, exercise, schoolwork)?”, “Do you ever have a craving
17 or strong desire to use social media?”, and “Have you ever been away from social media
18 and felt like you were missing it too much to engage in normal day to day activities?”.
19 Participants rated each symptom on a 4-point scale: (0): “I don’t have social media/not
20 applicable”, (1): “never”, (2): “sometimes”, and (3): “often”. ASMU symptom endorsement
21 was operationalized as responding “sometimes” or “often” for a given symptom and
22 ranged from 0 to 7 symptoms endorsed. Participants were classified into severity levels
23 based on the following DSM criteria: none: 0-1 symptom endorsed, mild: 2-3 symptoms
24 endorsed, moderate: 4-5 symptoms endorsed, and severe: 6 or more symptoms
25 endorsed (Hasin et al., 2013). Participants meeting “severe” criteria (i.e., 6 or more
26 symptoms endorsed) were classified into a high ASMU group ($n=52$) and all other
27 participants, with 5 or less symptoms endorsed, were classified into a low ASMU group
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3 While additional validation of this novel ASMU measure in other samples is
4 warranted, the measure demonstrates evidence of important convergent validity with
5 constructs that have been previously tested in the literature with other problematic and
6 social media addiction/addiction-like social media use measures (with some minor
7 deviations, as to be expected with variations across recruited samples). For example,
8 past research has demonstrated correlations of $r = 0.17$ and $r = -0.15$ with neuroticism
9 and conscientiousness, respectively (Huang, 2022); the correlations observed in our data
10 are similar, at 0.30 (neuroticism) and -0.26 (conscientiousness). In both our data and past
11 research (Huang, 2022), correlations with other Big Five traits are trivial and/or null ($r <$
12 0.12 in our study; $r < 0.10$ in a meta-analysis conducted by Huang, 2022). Past meta-
13 analytic work has also found a moderate correlation of $r = 0.29$ with depressive symptoms
14 (Cunningham et al., 2021); the bivariate correlation observed in our data is quite similar
15 at $r = 0.34$. There is meta-analytic evidence that the correlation between the fear of
16 missing out and social media addiction is quite high ($r = 0.47$; (Yali et al., 2021)); our data
17 similarly demonstrates a high correlation ($r = 0.68$). Past research has found a moderate
18 association between pathological social media use and poorer self-regulation ($r = -0.26$;
19 (Coyne et al., 2017)), and our data finds similar evidence for moderate associations
20 between ASMU and various facets of poorer self-control and greater impulsivity (r 's =
21 $0.24 - 0.45$; (Marino et al., 2018)). In addition to evidence of convergent validity, there
22 was also evidence of discriminant validity. Past research has demonstrated that
23 problematic Facebook use is only moderately associated with time spent on Facebook
24 ($r = 0.34$); likewise, we find a moderate association between ASMU and frequency of
25 checking social media ($r = 0.42$). Overall, these findings support the validity of our ASMU
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measure, given the comparable associations observed with various constructs in relation to past research utilizing alternative measures.

Social incentive delay (SID) task. During MRI scanning, participants completed two 6.5 min runs of a Social Incentive Delay (SID) task (**Figure 1**) designed to measure neural sensitivity to anticipation (cue) and receipt (outcome) of positive social feedback (smiling face) and negative social feedback (scowling face). A total of 24 adolescent faces were used from the NIH faces dataset (12 female, 12 male). In this task, participants first see a cue indicating what type of trial will follow (happy, angry, or neutral). Participants must press their right index finger as fast as they can after seeing the target to receive positive social feedback and avoid negative social feedback. To ensure sufficient and comparable exposure to all feedback types, task difficulty was individually and dynamically adapted based on prior performance by increasing or decreasing the target duration.

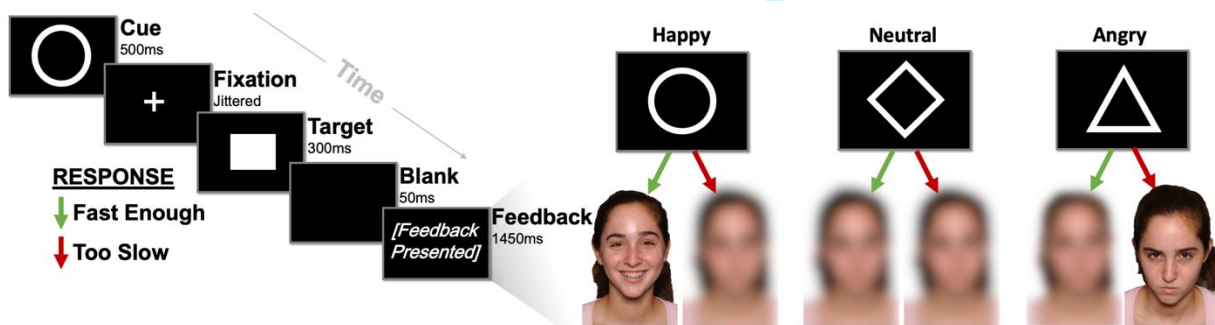


Figure 1. Social incentive delay (SID) task. During MRI scanning, participants completed two 6.5 min runs of a Social Incentive Delay (SID) task designed to measure neural sensitivity to anticipation (cue) and receipt (outcome) of social rewards (smiling face) and punishments (scowling face). Participants completed two 6.5 min runs

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3 consisting of 58 trials, resulting in a total of 116 trials (48 reward trials, 48 punishment
4 trials, and 20 neutral trials). In the task, participants see a cue (circle, square, or diamond,
5 500 ms) indicating what type of trial will follow. Then, following a fixation cross (duration
6 jittered ~509-4249 ms), they see a target (white square, 160-500 ms). Participants are
7 trained to press their right index finger as fast as they can after seeing the target, but not
8 before. Following a delay (50 ms), participants receive social feedback (1450 ms) based
9 on both the trial type and whether they pressed fast enough. The social feedback is
10 photographs of adolescent faces taken from the NIH faces dataset (Egger et al., 2011).
11 In the task, there were 24 faces shown (12 female, 12 male). Participants are explicitly
12 told that the circle is a happy cue, the square is an angry cue, and the diamond is a neutral
13 cue, meaning if they press fast enough after seeing the happy cue (Reward cue), they
14 will see a smiling face (Reward hit); if they press too slow after the happy cue, they will
15 see blurred face (Reward miss). If they press fast enough after seeing the angry cue
16 (Punishment cue), they will see a blurred face (Punishment hit); if they press too slow
17 after the angry cue, they will see a scowling face (Punishment miss). Following the Neutral
18 cue, they will see a blurred face whether they press fast enough (Neutral hit) or too slow
19 (Neutral miss). To ensure sufficient exposure to all feedback types, task difficulty was
20 individually and dynamically adapted based on prior performance by increasing or
21 decreasing the target duration (starting at 300ms) by 20 ms intervals unless reaching a
22 minimum of 160 ms or maximum of 500 ms duration.
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51 **MRI data analysis.** MRI data were collected on a Siemens Prisma MRI, 3-Tesla
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3 and preprocessing. Following preprocessing, SID functional data were entered into
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5 whole-brain participant-level general linear models (GLMs; SPM12) including eight event-
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7 related task regressors as impulse functions time-locked to stimulus onset and convolved
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9 with a hemodynamic response function. Task regressors included three anticipation
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11 conditions (happy, angry, or neutral), two feedback conditions (i.e., hit and miss) for both
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13 the positive and negative conditions, and one neutral feedback condition. Six motion
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15 parameters were modeled as regressors of no interest. Whole-brain contrast images
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17 comparing conditions of interest were calculated for each participant. As we were
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19 interested in examining individual differences in how the brain responds to the reinforcing
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21 and social properties of social media, positive social feedback vs. neutral feedback (i.e.,
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23 smiling face feedback following a hit in positive conditions vs. blurred out face feedback)
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25 was the primary contrast of interest for this study.

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31 **Statistical analysis.** Next, we examined the effect of AMSU symptom
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33 endorsement on developmental brain responsivity to positive social feedback. To assess
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35 within-individual effects we utilized multilevel statistical modeling that allowed for data
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37 missing at random. Within a linear multilevel framework (3dImr; AFNI) (Cox, 1996),
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39 modeling the random intercept and slope, we assessed a 3-way GENDER \times ASMU \times
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41 PUBERTY cross-level interaction on the positive social feedback vs. neutral feedback
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43 contrast. As ASMU research is still in its infancy, whether it is best to characterize ASMU
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45 as an all-or-none diagnosis or as a spectrum of severity is still an open question. Thus, in
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47 follow-up analyses, we also considered high (6 or more symptoms endorsed; $n=52$) and
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49 low (5 or less symptoms endorsed; $n=51$) ASMU-groups. For full transparency, we report
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51 results from analyses in which ASMU symptoms are treated as a continuous variable and
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3 analyses in which ASMU symptoms are used to group participants. Given prior
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5 neuroimaging evidence of brain function during the SID task (Martins et al., 2021), we
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7 expected feedback delivery in the SID to recruit brain regions involved in social
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9 processing. As such, this analysis was conducted within a small volume corrected (SVC)
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11 brain mask (36,386 voxels) defined via NeuroSynth's meta-analysis of the term "social"
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13 **(Figure S2)**. Three participants reporting nonbinary gender identities were not included
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15 in analyses comparing effects of self-reported girl and boy identities (N=100). For
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17 graphical examination and follow-up analyses, β coefficients associated with the positive
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19 social feedback contrast were extracted by averaging across voxels within identified
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21 significant clusters/regions of interest (ROIs).
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27 Next, we examined gender effects on ASMU symptom endorsement and
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29 depressive symptoms with independent-samples *t*-tests and assessed gender effects on
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31 ASMU-group counts with a cross tabulation Chi-squared test. Second, to assess both
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33 ASMU and gender effects on pubertal development across age, an ASMU-group \times
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35 GENDER \times AGE cross-level interaction was modeled while including random intercepts
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37 and slopes (*lmer*, *lme4* R package) (Bates et al., 2015). Third, we examined the effect of
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39 gender on associations between ASMU symptom endorsement and depressive
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41 symptoms with GENDER \times ASMU symptom ANCOVAs. Finally, we formally tested
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43 relations between variables in a moderated mediation model (PROCESS v.4, model 14)
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45 (Hayes, 2017) in which developmental changes in positive social feedback brain
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47 responsivity across pubertal development (slope coefficients associated with pubertal
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49 development) were associated with ASMU symptoms at the final timepoint that, in turn,
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51 were associated with depressive symptoms. Gender moderated the path from ASMU
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3 symptoms to depressive symptoms. In these models, positive social feedback slopes
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5 across pubertal development were entered as the predictor. Slope coefficients for each
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7 participant were extracted from linear multilevel models of pubertal development on
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9 positive social feedback β coefficients from identified significant clusters/ROIs, in which
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11 the random intercept and slope of puberty as well as their correlation were modeled within
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13 participant (**Figure S3**).
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19 RESULTS

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21 **Descriptives.** Gender identity, race/ethnicity, and annual household income did
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23 not significantly differ between high and low ASMU-groups (**Table 2**, p 's>0.1). As
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25 expected, the high ASMU-group had significantly higher depressive symptoms than the
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27 low ASMU-group (**Table 2**). We examined pubertal development trajectories in our
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29 sample of adolescents. A significant GENDER \times AGE interaction on pubertal
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31 development was observed such that girls had higher pubertal development scale scores
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33 at an earlier age than boys (est.=0.2, $t[68.4]=3.2$, $p=0.002$). Nonsignificant ASMU-group
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35 \times GENDER \times AGE interaction effects on pubertal development (p 's>0.2) indicated that
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37 ASMU-groups did not significantly differ in their pubertal development by age.
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45 **Table 2. Demographic, socioeconomic, and symptom information by ASMU-group.**

| | N=103 | High ASMU (n=52) | Low ASMU (n=51) | Group Differences |
|------------------------|-------|---------------------|--------------------|----------------------|
| Gender Identity | | | | $p=0.1$ |
| Female | 51.5% | 55.8% | 47.1% | |

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|--------------------------------|---------|---------|---------|------------------|
| Male | 45.6% | 38.5% | 52.9% | |
| Nonbinary | 2.9% | 5.7% | 0 | |
| Race/Ethnicity | | | | <i>p</i> =0.6 |
| White | 37.9% | 36.6% | 39.2% | |
| Hispanic/Latinx | 32.0% | 34.6% | 29.4% | |
| Black | 23.3% | 19.2% | 27.4% | |
| Multi-Racial | 4.9% | 7.7% | 2.0% | |
| Asian | 1.9% | 1.9% | 2.0% | |
| Household Annual Income | | | | <i>p</i> =0.5 |
| \$0-29,999 | 28.0% | 27.5% | 28.6% | |
| \$30,000-59,999 | 31.0% | 37.2% | 24.5% | |
| \$60,000-99,999 | 26.0% | 21.6% | 30.6% | |
| >\$100,000 | 15.0% | 13.7% | 16.3% | |
| Symptoms | | | | |
| Depressive symptoms | 7.0±6.5 | 8.3±6.9 | 5.6±6.0 | <i>p</i> =0.04* |
| ASMU symptoms endorsed | 4.5±2.6 | 6.8±0.4 | 2.5±1.7 | <i>p</i> <0.001* |

NOTE. Gender identity (% of total), and race/ethnicity (% of total) were measured at each participant's first timepoint (6th or 7th grade). Household annual income (% of total) and depressive symptoms (mean ± standard deviation) were measured at the final timepoint in 10th-11th grade. Addiction-like social media use (ASMU) symptoms (mean ± standard deviation) were also measured at the follow-up timepoint. ASMU symptoms endorsed is

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3 a count of the total number endorsed. ASMU-Group differences in gender identity, and
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5 race/ethnicity were assessed with cross tabulation Chi-squared tests. ASMU-Group
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7 differences in household annual income were assessed with a Kruskal-Wallis test for
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9 ordinal variables which is similar to a one-way ANOVA but ranks of the data values are
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11 used in the test rather than the actual data points. ASMU-Group differences in depressive
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13 symptoms, ASMU symptoms were assessed with independent samples *t*-tests.
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19 **Changes in social feedback brain responsivity across puberty linked to**
20 **ASMU**. We examined an ASMU symptom endorsement \times PUBERTY \times GENDER
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22 interaction on positive social feedback brain responsivity. While we did not observe any
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24 significant effects for the 3-way interaction, we did observe ASMU \times PUBERTY
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26 interactions on positive social feedback responsivity in the PCC (130 voxels) and two
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28 clusters in the ventral media prefrontal cortex (vmPFC; 90 and 36 voxels; $p_{\text{voxel-}}$
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30 $\text{level} < 0.005$). However, these results did not pass cluster-extent thresholding (minimum
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32 cluster size=213 voxels) determined via AFNI's ACF 3dclustsim (Cox et al., 2017). In
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34 follow-up analyses, we also examined an ASMU-Group \times PUBERTY \times GENDER
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36 interaction on positive social feedback brain responsivity. While again we did not observe
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38 any significant effects for the 3-way interaction, we did observe significant ASMU-Group
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40 \times PUBERTY interactions (**Figure 2; Table S1**) on positive social feedback responsivity in
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42 the vmPFC (358 voxels), mPFC (393 voxels), PCC (295 voxels), and right inferior frontal
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44 gyrus (rIFG; 226 voxels). To probe this interaction, β coefficients were averaged across
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46 voxels in each of these four significant clusters/ROIs. In all four clusters, adolescents in
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48 the low ASMU-group displayed relatively lower responsivity to positive social feedback
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before puberty onset that increased with pubertal development. In contrast, adolescents in the high ASMU-group displayed hyper-responsivity before puberty onset that decreased with pubertal development. These results did not significantly change after removing brain responsivity outliers. However, given potential for spurious detection of disordinal interactions with whole-brain ANOVAs, we caution against over-interpretation of differences in ASMU-group trajectories (Chavez & Wagner, 2017). While no significant interactive effects of GENDER were detected, we observed a significant GENDER main effect on positive social feedback responsivity in the right superior temporal sulcus (rSTS 350 voxels) such that girls displayed increased rSTS responsivity compared to boys. No significant PUBERTY main effects or PUBERTY \times GENDER interactions were observed. In follow-up exploratory analyses we also assessed brain responsivity to anticipation of positive social feedback vs. anticipation of neutral feedback but did not observe any significant effects of interest.

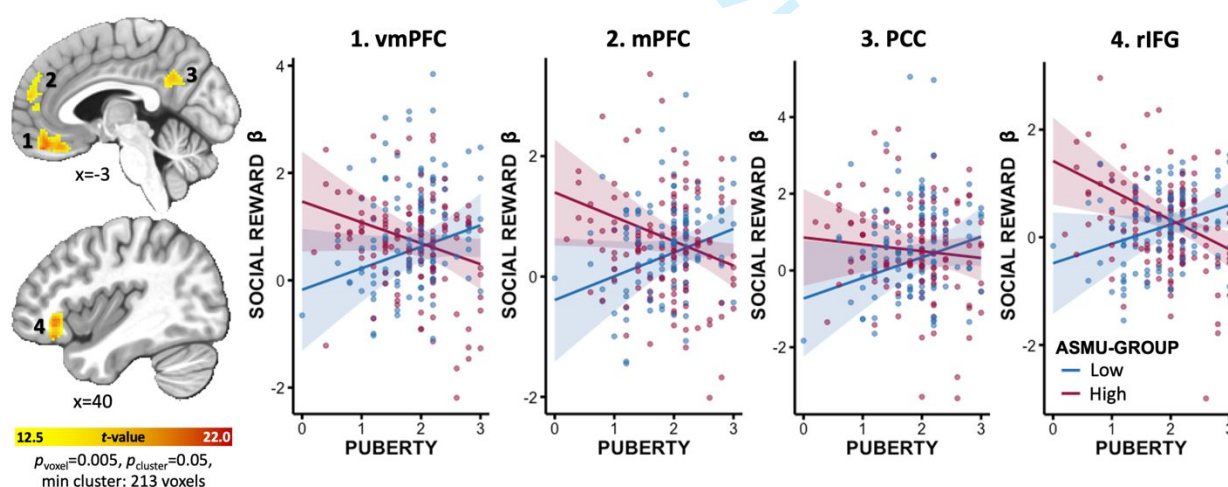


Figure 2. Changes in social feedback brain responsivity across puberty linked to ASMU. Significant ASMU-Group \times PUBERTY interactions were observed in the ventral media prefrontal cortex (1. vmPFC; 358 voxels), medial prefrontal cortex (2. mPFC; 393 voxels), posterior cingulate cortex (3. PCC; 295 voxels), and right inferior frontal gyrus (4. rIFG; 226 voxels) when controlling for GENDER effects. In all four significant clusters, adolescents in the low ASMU-group displayed relatively lower responsivity to positive social feedback before puberty onset that increased with pubertal development, whereas adolescents in the high ASMU-group displayed hyper-responsivity before puberty onset that decreased with pubertal development.

Gender effects. At the final timepoint in 10th-11th grade, we did not observe significant gender differences in ASMU symptom endorsement ($t[98]=1.1$, $p=0.2$) or ASMU-group counts ($\chi^2[1, 100]=2.7$, $p=0.3$). However, girls reported significantly higher depressive symptoms ($t[98]=2.9$, $p=0.004$, Cohen's $D=0.58$) than boys. (**Figure 3A**). Further, higher ASMU symptom endorsement was significantly associated with higher depressive symptoms among girls, but not among boys ($F[100]=6.3$, $p=0.014$, $\eta_p^2=0.06$; **Figure 3B**).

ASMU symptoms differentially mediate the effect of brain responsivity development on depressive symptoms among adolescent girls and boys. A significant moderated mediation model (Index of Moderated Mediation: 2.1, 95% CI=[0.1, 4.8]) indicated that, higher ASMU symptom endorsement mediated the association between decreasing vmPFC responsivity across pubertal development and increased depressive symptoms over two years later; this effect was only significant amongst girls

(indirect effect among girls: $ab = -2.0$, 95% CI = [-4.3, -0.1]; **Figure 3C**). Specifically, while decreases in vmPFC responsivity to positive social feedback across puberty was not directly related to depressive symptoms in later adolescence, it was related to increased ASMU symptom endorsement which was, in turn, related to higher depressive symptoms, but only among girls. This same model was also significant for the rIFG (Index of Moderated Mediation: 6.8, 95% CI = [2.3, 12.8]; indirect effect among girls: $ab = -5.0$, 95% CI = [-9.8, -1.6]; **Figure S4A**), but was not for the PCC (Index of Moderated Mediation: 3.0, 95% CI = [-1.0, 7.3]; **Figure S4B**) nor the mPFC (Index of Moderated Mediation: -8.4, 95% CI = [-77.1, 70.8]; **Figure S4C**). Follow up analyses indicated that the unmoderated mediation was also not significant for the PCC nor the mPFC.

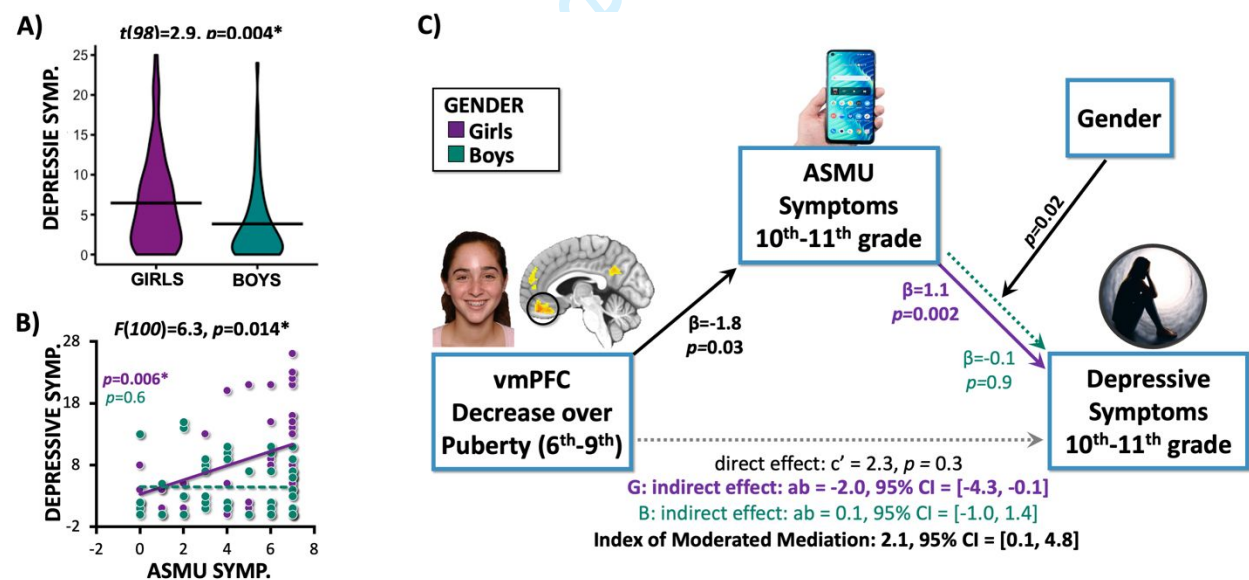


Figure 3. Addictive social media use symptoms mediate the effect of vmPFC change across puberty on depressive symptoms among girls. **(A)** Significantly higher depressive symptoms in 10th-11th grade, among adolescent girls compared to boys. **(B)** Higher addiction-like social media use (ASMU) symptoms were significantly

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3 associated with higher depressive symptoms among girls but not among boys. **(C)** A
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5 significant moderated mediation model (PROCESS v.4, model 14) demonstrated that
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7 decreasing ventral media prefrontal cortex (vmPFC) responsivity across puberty was
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9 related to increased ASMU symptoms ~2.3 years later (10th and 11th grade) which, among
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11 adolescent girls (but not boys), was in turn, associated with increased depressive
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13 symptoms.
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19 DISCUSSION

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21 **Changes in social feedback brain responsivity across puberty linked to**
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23 **ASMU**. Our results identify differential changes in social feedback brain responsivity
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25 across puberty in four brain regions (i.e., vmPFC, rIFG, mPFC, and PCC) among
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27 adolescents that subsequently reported more addiction-like social media use. Prior work
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29 has demonstrated developmental changes (e.g., childhood to adolescence, and early
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31 adolescence to late adolescence) in responsivity to positive social outcomes (Smith et
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33 al., 2015; Somerville, 2013). For example, striatal and anterior cingulate cortex activity to
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35 positive social feedback anticipation increases from childhood to adolescence (Gunther
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37 Moor et al., 2010). As such, we expected brain responsivity to social feedback to increase
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39 with pubertal development. However, across the full sample this was not observed.
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41 Instead, we observed that this trajectory diverged for high and low ASMU-groups. While
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43 the low ASMU-group displayed expected increases in positive social feedback
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45 responsivity across pubertal development, adolescents in the high ASMU-group
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47 displayed elevated social feedback responsivity in the vmPFC, rIFG, mPFC, and PCC
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49 before puberty onset that then decreased with pubertal development.
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3 These results could suggest that some adolescents with premature elevations in
4 neural social feedback sensitivity may initially be more sensitive to the delivery of social
5 feedback via media. However, observed pubertal decreases in social feedback vmPFC,
6 rIFG, PCC, and mPFC responsivity may reflect desensitization to such feedback, possibly
7 through mechanisms similar to those driving tolerance-build up after repeated
8 administrations of an addictive drug. As we did not have data on participants' amount of
9 social media exposure over pubertal development this hypothesis could not be further
10 explored in this sample. Nonetheless, our findings indicate that developmental changes
11 in brain function previously implicated in social information processing may be associated
12 with individual differences in ASMU susceptibility. Such information could help target
13 prevention efforts aiming to mitigate maladaptive social media use and its influence on
14 adolescent mental health.

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31 **ASMU symptoms mediate the effect of brain responsivity development on**
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33 **depressive symptoms.** Prior work suggests that increased brain responsivity to social
34 feedback may impact adolescent mental health through effects on their social
35 experiences. Specifically, anterior insula, cingulate, amygdala, and striatum responsivity
36 to social feedback among young adolescents (12-14 years old) impacts associations
37 between peer interpersonal stress and depression years later (Pagliaccio et al., 2023).
38 Further, prior findings from our research team similarly demonstrate that heightened
39 amygdala, and striatum activity to social feedback among young adolescents (11-14
40 years old) impacts associations between family conflict and externalizing behavior
41 (Turpyn et al., 2021). Our current findings extend this prior work showing that elevations
42 in neural responsivity to social feedback in early adolescence and subsequent
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3 developmental decreases into later adolescence may also impact social media use
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5 behaviors, and that this impact may increase risk for depressive symptoms.
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8 Further, our results highlight the importance of vmPFC and rIFG social feedback
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10 responsivity development. While all identified brain regions are implicated in social
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12 information processing (Spreng & Andrews-Hanna, 2015), the vmPFC, in particular, has
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14 been distinguished from other regions for its role in processing positively valenced or
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16 rewarding social information (van den Bos et al., 2007). Further, other work has
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18 suggested that the vmPFC and PCC may both be involved in processing self-referential
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20 social information (like social feedback) while more dorsal medial frontal regions are
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22 involved in thinking about the mental states of others (Wagner et al., 2012). Additionally,
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24 while the rIFC is known to be recruited when processing affective social information
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26 evidence suggests that it may be specifically involved when reappraising this information
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28 (Grecucci et al., 2013). Encoding valence, affective information, and referencing the self
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30 are likely all important when processing social feedback delivered on social media (e.g.,
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32 likes or comments). Taken together, these results suggest that development of vmPFC,
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34 and rIFG responsivity to social feedback are likely important targets for understanding
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36 how adolescents process social feedback delivered via social media and the impact it has
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38 on their media use behaviors and mental health.
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45 **Gender differences in mediating effect of ASMU symptoms on relationship**
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47 **between brain responsivity development and depressive symptoms.** Our results
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49 suggest gender effects in ASMU consequences by demonstrating a path through which
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51 divergent developmental trajectories of vmPFC and rIFG function may indirectly lead to
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53 depressive symptoms among girls through associations with more ASMU symptoms. This
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3 finding corresponds with prior work indicating higher social media use at age 10 is
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5 associated with declines in well-being into early and mid-adolescence for girls but not for
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7 boys (Booker et al., 2018). One possible explanation may be, as proposed by social role
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9 theory (Eagly & Wood, 2012), girls are socialized to value social connections and social
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11 standing more so than boys are. If this is the case, it is perhaps unsurprising that girls'
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13 neural sensitivity to social feedback might carry more influence on their mental wellbeing
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15 in instances of regular over-exposure to such information on social media.
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19 Another possible explanation of these results may be that gender is associated
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21 with differential susceptibility to different types of media use (i.e., social media vs. non-
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23 social digital media) and the respective consequences. Emergent meta-analytic research
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25 suggests that ASMU may be more prevalent among women while addiction-like internet
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27 gaming is more prevalent among men (Su et al., 2020). Social media may particularly
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29 foster certain maladaptive use experiences among adolescents, that are not as prevalent
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31 when using other non-social media, including social comparison, fear of missing out,
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33 feedback seeking, and body image insecurities (Nesi & Prinstein, 2015). Therefore,
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35 adolescent girls may be especially at risk. Future work examining various media types
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37 and their unique impacts on mental health could more fully depict gender-based
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39 susceptibility to ASMU.
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45 **Limitations.** Certain limitations should be considered. First, additional validation
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47 of the ASMU measure in other samples is warranted and as social norms surrounding
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49 digital media use shift, what constitutes problematic digital media behavior will also likely
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51 need to shift. Second, several statistical considerations should be noted regarding our
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53 observed ASMU-group by puberty interaction on social reward brain responsivity. First,
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3 while we observed significant interaction effects when dichotomizing the ASMU variable,
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5 results did not pass cluster-extent threshold corrections before dichotomizing. This
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7 discrepancy may be related to dichotomization reducing error in the measurement of the
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9 ASMU construct and thus increasing power. Further, we also note the potential for
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11 spurious interactions in whole-brain ANOVAs and thus the nature of each ASMU-group's
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13 developmental trajectory should be interpreted with caution (Chavez & Wagner, 2017).
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15 Third, while the Pubertal Development Scale is a widely used indicator of pubertal
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17 maturation that corresponds with other measures of puberty, including physician Tanner
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19 ratings and hormone levels (Herting et al., 2021), puberty is a highly individualized
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21 process and future work examining bias in caregiver-reports is warranted. Finally,
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23 information on social media exposure and use behaviors before and across pubertal
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25 development was not available. As such, we could not explore hypotheses regarding
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27 neural desensitization to social feedback due to accumulating exposure via social media
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29 use.
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35 **Conclusions.** Our findings suggest that before puberty onset, hyper-responsivity
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37 to positive social feedback, in four brain regions associated social information processing
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39 (vmPFC, rIFG, mPFC, and PCC), may represent a risk factor for ASMU in later
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41 adolescence. In contrast, decreases in this neural response over pubertal development
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43 could suggest atypical development of social feedback processing that is also linked with
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45 ASMU. Results suggest that these developmental differences in social feedback brain
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47 responsivity are associated with depressive symptoms among adolescent girls through
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49 their relationship with increased ASMU symptom endorsement in later adolescence.
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51 Taken together, this study identifies developmental individual differences (i.e., brain
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responsivity to social feedback and gender identity) that may be important for understanding ASMU susceptibility and related mental health outcomes.

DISCLOSURES: The authors have no conflicts to declare.

DATA AVAILABILITY: The authors have released all code associated with this manuscript. Code is available on GitHub https://github.com/Flanneryg3/ASMU_ProjectCode and group-level statistical brain maps are stored in the following NeuroVault collection: <https://identifiers.org/neurovault.collection:14537>

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For Peer Review

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3 **Developmental changes in brain function linked with addiction-like social media**
4 **use two years later**
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43

44 **ABBREVIATIONS:** ASMU: Addiction-like social media use; PDS: Pubertal Development
45 Scale; SID: social incentive delay; DSM-5: Diagnostic Statistical Manual V; SMFQ: Short
46 Mood and Feelings Questionnaire; GLMs: general linear models; ROIs: regions of
47 interest; vmPFC: ventral media prefrontal cortex; mPFC: medial prefrontal cortex; PCC:
48 posterior cingulate cortex rIFG; right inferior frontal gyrus; rSTS: right superior temporal
49 sulcus.
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Abstract

Background. Addiction-like social media use (ASMU) is widely reported among adolescents and is associated with depression and other negative health outcomes. We aimed to identify developmental trajectories of neural social feedback processing that are linked to higher levels of ASMU in later adolescence.

Methods. Within a longitudinal design, 103 adolescents completed a social incentive delay task during 1-3 fMRI scans (6th-9th grade), and a 4th self-report assessment of ASMU and depressive symptoms ~2 years later (10th-11th grade). We assessed ASMU effects on brain responsivity to positive social feedback across puberty and relationships between brain responsivity development, ASMU symptoms, and depressive symptoms while considering gender effects.

Results. Findings demonstrate decreasing responsivity, across puberty, in the ventral media prefrontal cortex, medial prefrontal cortex, posterior cingulate cortex, and right inferior frontal gyrus associated with higher ASMU symptoms over 2 years later. Significant moderated mediation models suggest that these pubertal decreases in brain responsivity are associated with increased ASMU symptoms which, among adolescent girls (but not boys), is in turn associated with increased depressive symptoms.

Conclusions. Results suggest initial hyperresponsivity to positive social feedback, before puberty onset, and decreases in this response across development, may be risk factors for ASMU in later adolescence.

KEYWORDS: social media addiction, social feedback, adolescence, puberty, depression

INTRODUCTION

Social media serves many functions and is often part of healthy adolescent development (Deters & Mehl, 2013; Ellis et al., 2020; Flannery et al., 2023; Leung, 2011). However, addiction-like social media use (ASMU) is becoming increasingly reported (Kuss et al., 2014). Despite debate regarding the diagnostic utility of ASMU (Panova & Carbonell, 2022), use of addiction terminology to discuss social media use behaviors has permeated popular culture (Adorjan & Ricciardelli, 2021). Because of this, it is of interest to explore how behaviors reflecting craving for, and difficulty abstaining from, social media occur in adolescence. Accumulating work suggests that ASMU may share some of the same characteristics as other addictive disorders such as sustained preoccupation with cues, use for mood modification, tolerance following repeated use, and withdrawal symptoms following abstinence (Goldberg, 2004). Importantly, ASMU is not necessarily equated with the degree to which one uses social media, but instead captures the degree to which one feels a loss of control over their social media use or experiences negative effects (emotional or circumstantial) due to their use (Baumer et al., 2015; Turel et al., 2018). As ASMU is characterized by maintained or increased social media use despite negative impacts on other aspects of life, and difficulty reducing use despite intentions to do so (Baumer et al., 2015), it is unsurprising that a quickly growing body of work indicates that ASMU may become disruptive to other aspects of life and have detrimental impacts on health and wellbeing (Turel et al., 2018). Indeed, research shows that ASMU symptoms are associated with depressive symptoms (Robinson et al., 2019), especially among adolescent girls (Raudsepp & Kais, 2019). As adolescence is a period of vulnerability for both the onset of internalizing psychopathology (McLaughlin & King,

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3 2015) and ASMU symptoms (Stavropoulos et al., 2018), understanding links between
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5 ASMU and depressive symptoms across this period is vital.
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8 The types of social feedback delivered via social media may be especially relevant
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10 to promoting addiction-like social media use behavior. Social feedback is both frequent
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12 and quantifiable (e.g., number of likes, number of followers) via social media. Such social
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14 feedback is also usually reinforcing and delivered on a variable ratio reinforcement
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16 schedule which is highly resistant to behavior extinction and is thus particularly addictive
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18 (Greenfield, 2007). Social feedback delivery may be especially salient to adolescents, as
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20 adolescence is thought to be a period of heightened neurobiological and behavioral
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22 sensitivity to social stimuli, as well as prioritization of social connection and peer
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24 acceptance (Somerville, 2013). Additionally, adolescence is characterized by a peak in
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26 reward sensitivity (Lamm et al., 2014) and reward seeking behaviors (Galván, 2013),
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28 particularly in social contexts (Smith et al., 2015). This reward sensitivity is thought to
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30 stem from normative changes in brain structure and function that begin around the onset
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32 of puberty (Padmanabhan et al., 2011). Specifically, neuroimaging and preclinical work
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34 has repeatedly demonstrated links between higher levels of pubertal hormones and
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36 increased reward-related striatum activity among adolescents (Forbes et al., 2011; Op de
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38 Macks et al., 2011). The onset of puberty may thus elicit normative developmental
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40 increases in neural responsivity to reinforcing social feedback (Smith et al., 2015;
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42 Somerville, 2013). Given these sensitivities, the continuous stream of highly salient and
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44 reinforcing social information dispensed via social media may have a uniquely powerful
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46 impact on adolescents.
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3 Yet, all adolescents may not be equally prone to ASMU due to various individual
4 predispositions, including possible biological vulnerabilities that increase sensitivity to
5 social media cues. For example, individual differences in sensitivity to reinforcing social
6 feedback may determine how adolescents navigate social media environments and the
7 impact those environments have on adolescents' mental health (Sherman et al., 2016).
8 Specifically, adolescents who are more sensitive to social reward may be particularly apt
9 to seek out social media incentives and thus may also be more susceptible to the
10 provocation of continued use or even ASMU (Sherman et al., 2016). While development
11 of adaptive incentive processing during adolescence is important for healthy
12 development, hypersensitivity to rewards has been linked to externalizing and risk-taking
13 behaviors (Bjork & Pardini, 2015) and blunted reward sensitivity has been linked to
14 depression (O'Callaghan & Stringaris, 2019). Further, recent research has shown that, in
15 the context of negative social experiences, neural hypersensitivity to social feedback is
16 associated with an increased susceptibility to depressive symptoms (Pagliaccio et al.,
17 2023) and externalizing behaviors (Turpyn et al., 2021) among adolescents. However,
18 relationships between this neural hypersensitivity and ASMU across development are still
19 not fully understood.

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Prior work shows pubertal increases in neural responsivity to social feedback in brain regions typically involved in social processing including the medial prefrontal cortex (mPFC), posterior cingulate cortex (PCC), precuneus, inferior frontal cortex, fusiform gyrus, and hippocampus (Gunther Moor et al., 2010). However, it is unclear whether individual differences in this development might constitute risk for future ASMU. We hypothesize that an initial hypersensitivity to such stimuli may drive some adolescents to

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3 increase their social media use more than others. However, recurrent over-exposure to
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5 social rewards via social media may, in turn, contribute to desensitization to social
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7 rewards across development. This hypothesis is based on prior observations of tolerance-
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9 build up after repeated administrations of an addictive drug (Albrecht et al., 2007; Miller
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11 et al., 1987). Specifically, we hypothesize that adolescents reporting high ASMU in later
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13 adolescence may display initially heightened social reward responsivity that decreases
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15 over years of increasing social media use. This decrease may reflect a desensitization to
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17 rewarding social feedback and a need for more social reward exposure to get the same
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19 reinforcing effects (Griffiths et al., 2014).
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24 The current study investigates individual differences in pubertal trajectories of brain
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26 responsivity to positive social feedback across 6th-9th grade that are related to ASMU ~2
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28 years later. Interactive effects of gender identity were considered given prior work
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30 showing gender effects on social media use behaviors (Nesi & Prinstein, 2015), ASMU
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32 prevalence (Hawi & Samaha, 2019), and relationships between social media use and
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34 wellbeing (Booker et al., 2018). Further, we examine indirect effects of this differential
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36 brain function development on depressive symptoms in later adolescence through
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38 associations with ASMU symptoms, while again, considering gender effects. We
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40 hypothesize that initial hypersensitivity to positive social feedback, and longitudinal
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42 decreases across pubertal development, will be associated with higher ASMU symptoms
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44 in later adolescence. Further, we expect that, higher ASMU symptoms will, in turn, be
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46 associated depressive symptoms, particularly among girls.
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54 **METHODS**

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Participants. Two cohorts of adolescent participants were recruited from 3 public middle schools across 2 years as part of a larger longitudinal study of 6th and 7th grade students (**Figure S1**). The current study examines 103 adolescents that completed a social incentive delay (SID) task across 1 to 3 annual fMRI scan sessions (6th-9th grade; 256 data points), as well as a self-report assessment 2.27±0.21 years following their final fMRI scan (10th-11th grade). The sample of 103 adolescents did not differ from the larger sample from which they were recruited on baseline gender, race/ethnicity, income, pubertal development, or depressive symptoms. Of these 103 adolescents that had data at the final wave, 80 had data at the 1st timepoint, 90 had data at the 2nd timepoint, and 86 had data at the 3rd timepoint (**Table 1**).

Table 1. Demographic information by timepoint.

| Timepoint | 1 st | 2 nd | 3 rd | 4 th |
|------------------------|-----------------|-----------------|-----------------|-----------------|
| | n=80 | n=90 | n=86 | n=103 |
| Gender Identity | | | | |
| Girls | 46.3% | 46.7% | 46.5% | 51.5% |
| Boys | 51.2% | 51.1% | 51.2% | 45.6% |
| Nonbinary | 2.5% | 2.2% | 2.3% | 2.9% |
| Age | | | | |
| | 12.8±0.5 | 13.7±0.6 | 14.7±0.6 | 17.0±0.6 |
| Grade | | | | |
| 6 th | 47.5% | 0 | 0 | 0 |
| 7 th | 52.5% | 51.1% | 0 | 0 |

Social media & brain development

Flannery *et al.*

| | | | | |
|------------------|---|-------|-----|-------|
| 8 th | 0 | 48.9% | 50% | 0 |
| 9 th | 0 | 0 | 50% | 0 |
| 10 th | 0 | 0 | 0 | 53.4% |
| 11 th | 0 | 0 | 0 | 46.6% |

NOTE. Gender identity (% of total), age (mean \pm standard deviation), and grade (% of total) are presented for each data collection timepoint. The multiple cohort structure of the study resulted in planned missing data across timepoints. Specifically, 62 of the total 103 participants had 3 fMRI timepoints, 29 had 2, and 12 had 1. All participants had a 4th self-report timepoint.

Procedures. All participants provided informed consent/assent and were compensated for each completed session. The University's Institutional Review Board approved all aspects of the study. Adolescent participants and their primary caregiver attended annual data collection sessions across 3 timepoints of a longitudinal fMRI study, at which adolescents completed an fMRI scan lasting approximately 1.5 hours. During each scan participants completed a social feedback task called the Social Incentive Delay (SID) task, as well as an anatomical scan and four other tasks that are not the focus of the current manuscript. Following the scan, adolescents and caregivers completed several self-report measures. Adolescents and their caregivers returned about 2.3 years after their final fMRI scan timepoint and completed a 4th self-report assessment.

Measures. Pubertal development. Adolescents' primary caregivers completed the Pubertal Development Scale (PDS) (Petersen et al., 1988) at each of the three fMRI scan

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3 timepoints. Scores ranged from (0): “puberty has not yet started” to (3): “puberty is
4 complete” (Icenogle et al., 2017). Depressive symptoms. Depressive symptoms were
5 assessed at the final timepoint (~2.3 years after the final fMRI scan) in 10th-11th grade,
6 using total scores on the unidimensional, 13-item Short Mood and Feelings Questionnaire
7 (SMFQ; $\alpha=.93$) (Messer et al., 1995), designed to measure depressive symptomology in
8 children and adolescents aged 6-17 years old.
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12 Addiction-like social media use symptoms. ASMU symptoms were also measured
13 at the final timepoint with a novel 7-item questionnaire ($\alpha=.90$) based on selected items
14 from the Diagnostic Statistical Manual V (DSM-5) substance use disorder checklist.
15 Example items include, “Does social media use ever get in the way of things you are
16 supposed to be doing (e.g., sleep, exercise, schoolwork)?”, “Do you ever have a craving
17 or strong desire to use social media?”, and “Have you ever been away from social media
18 and felt like you were missing it too much to engage in normal day to day activities?”.
19 Participants rated each symptom on a 4-point scale: (0): “I don’t have social media/not
20 applicable”, (1): “never”, (2): “sometimes”, and (3): “often”. ASMU symptom endorsement
21 was operationalized as responding “sometimes” or “often” for a given symptom and
22 ranged from 0 to 7 symptoms endorsed. Participants were classified into severity levels
23 based on the following DSM criteria: none: 0-1 symptom endorsed, mild: 2-3 symptoms
24 endorsed, moderate: 4-5 symptoms endorsed, and severe: 6 or more symptoms
25 endorsed (Hasin et al., 2013). Participants meeting “severe” criteria (i.e., 6 or more
26 symptoms endorsed) were classified into a high ASMU group ($n=52$) and all other
27 participants, with 5 or less symptoms endorsed, were classified into a low ASMU group
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3 While additional validation of this novel ASMU measure in other samples is
4 warranted, the measure demonstrates evidence of important convergent validity with
5 constructs that have been previously tested in the literature with other problematic and
6 social media addiction/addiction-like social media use measures (with some minor
7 deviations, as to be expected with variations across recruited samples). For example,
8 past research has demonstrated correlations of $r = 0.17$ and $r = -0.15$ with neuroticism
9 and conscientiousness, respectively (Huang, 2022); the correlations observed in our data
10 are similar, at 0.30 (neuroticism) and -0.26 (conscientiousness). In both our data and past
11 research (Huang, 2022), correlations with other Big Five traits are trivial and/or null ($r <$
12 0.12 in our study; $r < 0.10$ in a meta-analysis conducted by Huang, 2022). Past meta-
13 analytic work has also found a moderate correlation of $r = 0.29$ with depressive symptoms
14 (Cunningham *et al.*, 2021); the bivariate correlation observed in our data is quite similar
15 at $r = 0.34$. There is meta-analytic evidence that the correlation between the fear of
16 missing out and social media addiction is quite high ($r = 0.47$; (Yali *et al.*, 2021)); our data
17 similarly demonstrates a high correlation ($r = 0.68$). Past research has found a moderate
18 association between pathological social media use and poorer self-regulation ($r = -0.26$;
19 (Coyne *et al.*, 2017)), and our data finds similar evidence for moderate associations
20 between ASMU and various facets of poorer self-control and greater impulsivity (r 's =
21 $0.24 - 0.45$; (Marino *et al.*, 2018)). In addition to evidence of convergent validity, there
22 was also evidence of discriminant validity. Past research has demonstrated that
23 problematic Facebook use is only moderately associated with time spent on Facebook
24 ($r = 0.34$); likewise, we find a moderate association between ASMU and frequency of
25 checking social media ($r = 0.42$). Overall, these findings support the validity of our ASMU

measure, given the comparable associations observed with various constructs in relation to past research utilizing alternative measures.

Social incentive delay (SID) task. During MRI scanning, participants completed two 6.5 min runs of a Social Incentive Delay (SID) task (**Figure 1**) designed to measure neural sensitivity to anticipation (cue) and receipt (outcome) of positive social feedback (smiling face) and negative social feedback (scowling face). A total of 24 adolescent faces were used from the NIH faces dataset (12 female, 12 male). In this task, participants first see a cue indicating what type of trial will follow (happy, angry, or neutral). Participants must press their right index finger as fast as they can after seeing the target to receive positive social feedback and avoid negative social feedback. To ensure sufficient and comparable exposure to all feedback types, task difficulty was individually and dynamically adapted based on prior performance by increasing or decreasing the target duration.

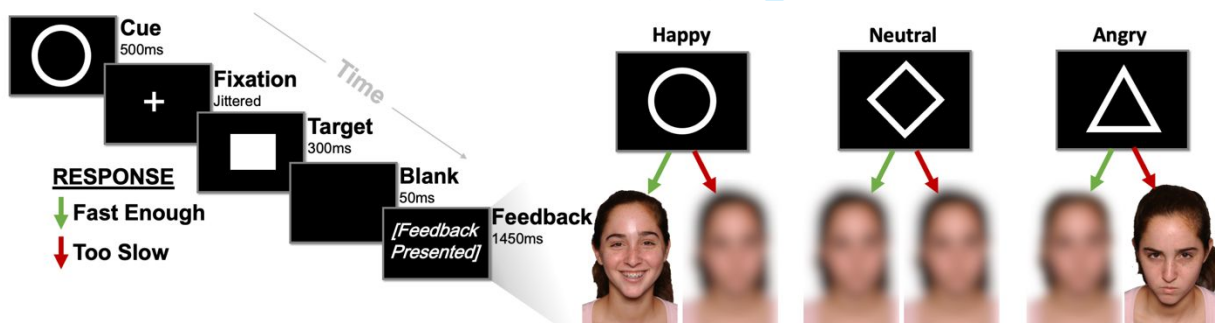


Figure 1. Social incentive delay (SID) task. During MRI scanning, participants completed two 6.5 min runs of a Social Incentive Delay (SID) task designed to measure neural sensitivity to anticipation (cue) and receipt (outcome) of social rewards (smiling face) and punishments (scowling face). Participants completed two 6.5 min runs

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3 consisting of 58 trials, resulting in a total of 116 trials (48 reward trials, 48 punishment
4 trials, and 20 neutral trials). In the task, participants see a cue (circle, square, or diamond,
5 500 ms) indicating what type of trial will follow. Then, following a fixation cross (duration
6 jittered ~509-4249 ms), they see a target (white square, 160-500 ms). Participants are
7 trained to press their right index finger as fast as they can after seeing the target, but not
8 before. Following a delay (50 ms), participants receive social feedback (1450 ms) based
9 on both the trial type and whether they pressed fast enough. The social feedback is
10 photographs of adolescent faces taken from the NIH faces dataset (Egger et al., 2011).
11 In the task, there were 24 faces shown (12 female, 12 male). Participants are explicitly
12 told that the circle is a happy cue, the square is an angry cue, and the diamond is a neutral
13 cue, meaning if they press fast enough after seeing the happy cue (Reward cue), they
14 will see a smiling face (Reward hit); if they press too slow after the happy cue, they will
15 see blurred face (Reward miss). If they press fast enough after seeing the angry cue
16 (Punishment cue), they will see a blurred face (Punishment hit); if they press too slow
17 after the angry cue, they will see a scowling face (Punishment miss). Following the Neutral
18 cue, they will see a blurred face whether they press fast enough (Neutral hit) or too slow
19 (Neutral miss). To ensure sufficient exposure to all feedback types, task difficulty was
20 individually and dynamically adapted based on prior performance by increasing or
21 decreasing the target duration (starting at 300ms) by 20 ms intervals unless reaching a
22 minimum of 160 ms or maximum of 500 ms duration.
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51 **MRI data analysis.** MRI data were collected on a Siemens Prisma MRI, 3-Tesla
52 scanner Supplemental materials contain information on neuroimaging data acquisition
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3 and preprocessing. Following preprocessing, SID functional data were entered into
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5 whole-brain participant-level general linear models (GLMs; SPM12) including eight event-
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7 related task regressors as impulse functions time-locked to stimulus onset and convolved
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9 with a hemodynamic response function. Task regressors included three anticipation
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11 conditions (happy, angry, or neutral), two feedback conditions (i.e., hit and miss) for both
12
13 the positive and negative conditions, and one neutral feedback condition. Six motion
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15 parameters were modeled as regressors of no interest. Whole-brain contrast images
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17 comparing conditions of interest were calculated for each participant. As we were
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19 interested in examining individual differences in how the brain responds to the reinforcing
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21 and social properties of social media, positive social feedback vs. neutral feedback (i.e.,
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23 smiling face feedback following a hit in positive conditions vs. blurred out face feedback)
24
25 was the primary contrast of interest for this study.

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31 **Statistical analysis.** Next, we examined the effect of ASMU symptom
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33 endorsement on developmental brain responsivity to positive social feedback. To assess
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35 within-individual effects we utilized multilevel statistical modeling that allowed for data
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37 missing at random. Within a linear multilevel framework (3dLmer; AFNI) (Cox, 1996),
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39 modeling the random intercept and slope, we assessed a 3-way GENDER \times ASMU \times
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41 PUBERTY cross-level interaction on the positive social feedback vs. neutral feedback
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43 contrast. As ASMU research is still in its infancy, whether it is best to characterize ASMU
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45 as an all-or-none diagnosis or as a spectrum of severity is still an open question. Thus, in
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47 follow-up analyses, we also considered high (6 or more symptoms endorsed; $n=52$) and
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49 low (5 or less symptoms endorsed; $n=51$) ASMU-groups. For full transparency, we report
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51 results from analyses in which ASMU symptoms are treated as a continuous variable and
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3 analyses in which ASMU symptoms are used to group participants. Given prior
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5 neuroimaging evidence of brain function during the SID task (Martins et al., 2021), we
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7 expected feedback delivery in the SID to recruit brain regions involved in social
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9 processing. As such, this analysis was conducted within a small volume corrected (SVC)
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11 brain mask (36,386 voxels) defined via NeuroSynth's meta-analysis of the term "social"
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13 (Figure S2). Three participants reporting nonbinary gender identities were not included
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15 in analyses comparing effects of self-reported girl and boy identities (N=100). For
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17 graphical examination and follow-up analyses, β coefficients associated with the positive
18
19 social feedback contrast were extracted by averaging across voxels within identified
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21 significant clusters/regions of interest (ROIs).
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27 Next, we examined gender effects on ASMU symptom endorsement and
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29 depressive symptoms with independent-samples *t*-tests and assessed gender effects on
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31 ASMU-group counts with a cross tabulation Chi-squared test. Second, to assess both
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33 ASMU and gender effects on pubertal development across age, an ASMU-group \times
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35 GENDER \times AGE cross-level interaction was modeled while including random intercepts
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37 and slopes (*lmer*, *lme4* R package) (Bates et al., 2015). Third, we examined the effect of
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39 gender on associations between ASMU symptom endorsement and depressive
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41 symptoms with GENDER \times ASMU symptom ANCOVAs. Finally, we formally tested
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43 relations between variables in a moderated mediation model (PROCESS v.4, model 14)
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45 (Hayes, 2017) in which developmental changes in positive social feedback brain
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47 responsivity across pubertal development (slope coefficients associated with pubertal
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49 development) were associated with ASMU symptoms at the final timepoint that, in turn,
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51 were associated with depressive symptoms. Gender moderated the path from ASMU
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3 symptoms to depressive symptoms. In these models, positive social feedback slopes
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5 across pubertal development were entered as the predictor. Slope coefficients for each
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7 participant were extracted from linear multilevel models of pubertal development on
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9 positive social feedback β coefficients from identified significant clusters/ROIs, in which
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11 the random intercept and slope of puberty as well as their correlation were modeled within
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13 participant (**Figure S3**).
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19 RESULTS

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21 **Descriptives.** Gender identity, race/ethnicity, and annual household income did
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23 not significantly differ between high and low ASMU-groups (**Table 2**, p 's>0.1). As
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25 expected, the high ASMU-group had significantly higher depressive symptoms than the
26
27 low ASMU-group (**Table 2**). We examined pubertal development trajectories in our
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29 sample of adolescents. A significant GENDER \times AGE interaction on pubertal
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31 development was observed such that girls had higher pubertal development scale scores
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33 at an earlier age than boys (est.=0.2, $t[68.4]=3.2$, $p=0.002$). Nonsignificant ASMU-group
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35 \times GENDER \times AGE interaction effects on pubertal development (p 's>0.2) indicated that
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37 ASMU-groups did not significantly differ in their pubertal development by age.
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45 **Table 2. Demographic, socioeconomic, and symptom information by ASMU-group.**

| | N=103 | High ASMU (n=52) | Low ASMU (n=51) | Group Differences |
|------------------------|-------|---------------------|--------------------|----------------------|
| Gender Identity | | | | $p=0.1$ |
| Female | 51.5% | 55.8% | 47.1% | |

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| | | | | |
|--------------------------------|---------|---------|---------|------------------|
| Male | 45.6% | 38.5% | 52.9% | |
| Nonbinary | 2.9% | 5.7% | 0 | |
| Race/Ethnicity | | | | <i>p</i> =0.6 |
| White | 37.9% | 36.6% | 39.2% | |
| Hispanic/Latinx | 32.0% | 34.6% | 29.4% | |
| Black | 23.3% | 19.2% | 27.4% | |
| Multi-Racial | 4.9% | 7.7% | 2.0% | |
| Asian | 1.9% | 1.9% | 2.0% | |
| Household Annual Income | | | | <i>p</i> =0.5 |
| \$0-29,999 | 28.0% | 27.5% | 28.6% | |
| \$30,000-59,999 | 31.0% | 37.2% | 24.5% | |
| \$60,000-99,999 | 26.0% | 21.6% | 30.6% | |
| >\$100,000 | 15.0% | 13.7% | 16.3% | |
| Symptoms | | | | |
| Depressive symptoms | 7.0±6.5 | 8.3±6.9 | 5.6±6.0 | <i>p</i> =0.04* |
| ASMU symptoms endorsed | 4.5±2.6 | 6.8±0.4 | 2.5±1.7 | <i>p</i> <0.001* |

NOTE. Gender identity (% of total), and race/ethnicity (% of total) were measured at each participant's first timepoint (6th or 7th grade). Household annual income (% of total) and depressive symptoms (mean ± standard deviation) were measured at the final timepoint in 10th-11th grade. Addiction-like social media use (ASMU) symptoms (mean ± standard deviation) were also measured at the follow-up timepoint. ASMU symptoms endorsed is

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3 a count of the total number endorsed. ASMU-Group differences in gender identity, and
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5 race/ethnicity were assessed with cross tabulation Chi-squared tests. ASMU-Group
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7 differences in household annual income were assessed with a Kruskal-Wallis test for
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9 ordinal variables which is similar to a one-way ANOVA but ranks of the data values are
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11 used in the test rather than the actual data points. ASMU-Group differences in depressive
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13 symptoms, ASMU symptoms were assessed with independent samples *t*-tests.
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19 **Changes in social feedback brain responsivity across puberty linked to**
20 **ASMU**. We examined an ASMU symptom endorsement \times PUBERTY \times GENDER
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22 interaction on positive social feedback brain responsivity. While we did not observe any
23
24 significant effects for the 3-way interaction, we did observe ASMU \times PUBERTY
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26 interactions on positive social feedback responsivity in the PCC (130 voxels) and two
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28 clusters in the ventral media prefrontal cortex (vmPFC; 90 and 36 voxels; $p_{\text{voxel-}}$
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30 $\text{level} < 0.005$). However, these results did not pass cluster-extent thresholding (minimum
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32 cluster size=213 voxels) determined via AFNI's ACF 3dclustsim (Cox et al., 2017). In
33
34 follow-up analyses, we also examined an ASMU-Group \times PUBERTY \times GENDER
35
36 interaction on positive social feedback brain responsivity. While again we did not observe
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38 any significant effects for the 3-way interaction, we did observe significant ASMU-Group
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40 \times PUBERTY interactions (**Figure 2; Table S1**) on positive social feedback responsivity in
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42 the vmPFC (358 voxels), mPFC (393 voxels), PCC (295 voxels), and right inferior frontal
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44 gyrus (rIFG; 226 voxels). To probe this interaction, β coefficients were averaged across
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46 voxels in each of these four significant clusters/ROIs. In all four clusters, adolescents in
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48 the low ASMU-group displayed relatively lower responsivity to positive social feedback
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before puberty onset that increased with pubertal development. In contrast, adolescents in the high ASMU-group displayed hyper-responsivity before puberty onset that decreased with pubertal development. These results did not significantly change after removing brain responsivity outliers. However, given potential for spurious detection of disordinal interactions with whole-brain ANOVAs, we caution against over-interpretation of differences in ASMU-group trajectories (Chavez & Wagner, 2017). While no significant interactive effects of GENDER were detected, we observed a significant GENDER main effect on positive social feedback responsivity in the right superior temporal sulcus (rSTS 350 voxels) such that girls displayed increased rSTS responsivity compared to boys. No significant PUBERTY main effects or PUBERTY \times GENDER interactions were observed. In follow-up exploratory analyses we also assessed brain responsivity to anticipation of positive social feedback vs. anticipation of neutral feedback but did not observe any significant effects of interest.

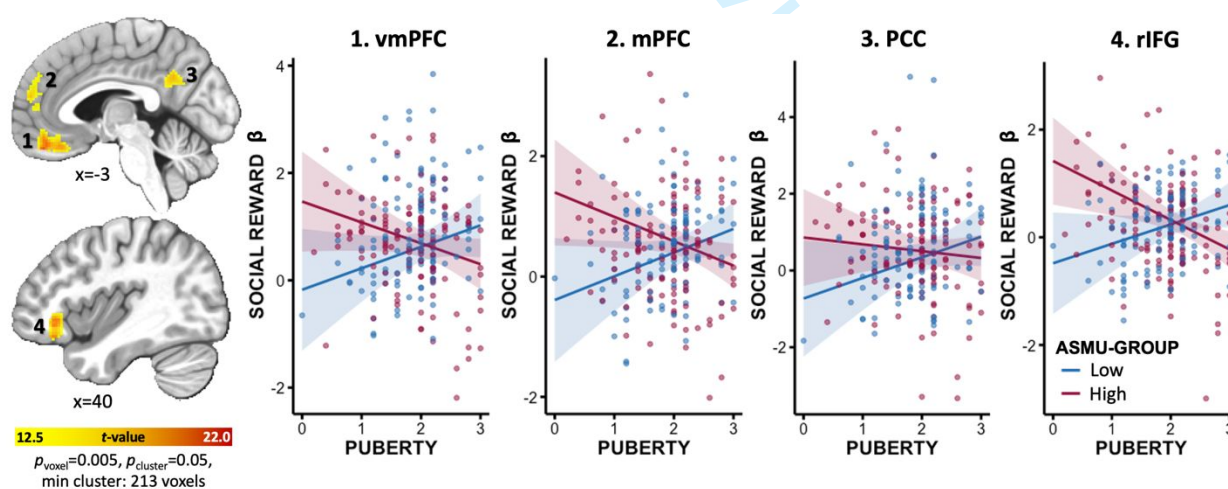


Figure 2. Changes in social feedback brain responsivity across puberty linked to ASMU. Significant ASMU-Group \times PUBERTY interactions were observed in the ventral media prefrontal cortex (1. vmPFC; 358 voxels), medial prefrontal cortex (2. mPFC; 393 voxels), posterior cingulate cortex (3. PCC; 295 voxels), and right inferior frontal gyrus (4. rIFG; 226 voxels) when controlling for GENDER effects. In all four significant clusters, adolescents in the low ASMU-group displayed relatively lower responsivity to positive social feedback before puberty onset that increased with pubertal development, whereas adolescents in the high ASMU-group displayed hyper-responsivity before puberty onset that decreased with pubertal development.

Gender effects. At the final timepoint in 10th-11th grade, we did not observe significant gender differences in ASMU symptom endorsement ($t[98]=1.1$, $p=0.2$) or ASMU-group counts ($\chi^2[1, 100]=2.7$, $p=0.3$). However, girls reported significantly higher depressive symptoms ($t[98]=2.9$, $p=0.004$, Cohen's $D=0.58$) than boys. (**Figure 3A**). Further, higher ASMU symptom endorsement was significantly associated with higher depressive symptoms among girls, but not among boys ($F[100]=6.3$, $p=0.014$, $\eta_p^2=0.06$; **Figure 3B**).

ASMU symptoms differentially mediate the effect of brain responsivity development on depressive symptoms among adolescent girls and boys. A significant moderated mediation model (Index of Moderated Mediation: 2.1, 95% CI=[0.1, 4.8]) indicated that, higher ASMU symptom endorsement mediated the association between decreasing vmPFC responsivity across pubertal development and increased depressive symptoms over two years later; this effect was only significant amongst girls

(indirect effect among girls: $ab=-2.0$, 95% CI=[-4.3, -0.1]; **Figure 3C**). Specifically, while decreases in vmPFC responsivity to positive social feedback across puberty was not directly related to depressive symptoms in later adolescence, it was related to increased ASMU symptom endorsement which was, in turn, related to higher depressive symptoms, but only among girls. This same model was also significant for the rIFG (Index of Moderated Mediation: 6.8, 95% CI=[2.3, 12.8]; indirect effect among girls: $ab=-5.0$, 95% CI=[-9.8, -1.6]; **Figure S4A**), but was not for the PCC (Index of Moderated Mediation: 3.0, 95% CI=[-1.0, 7.3]; **Figure S4B**) nor the mPFC (Index of Moderated Mediation: -8.4, 95% CI=[-77.1, 70.8]; **Figure S4C**). Follow up analyses indicated that the unmoderated mediation was also not significant for the PCC nor the mPFC.

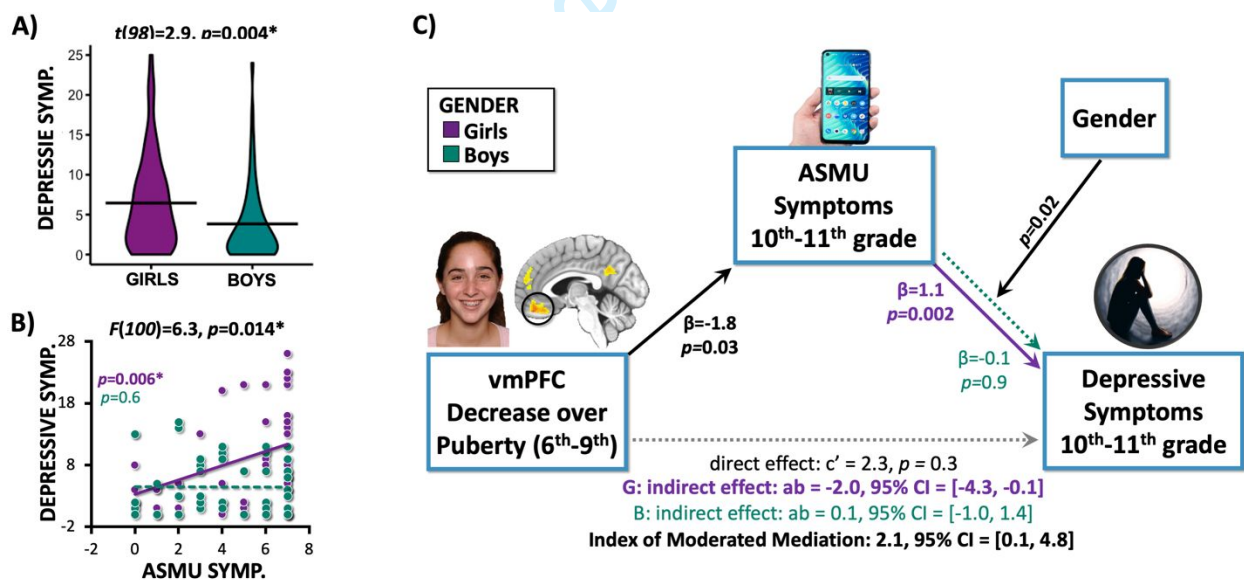


Figure 3. Addictive social media use symptoms mediate the effect of vmPFC change across puberty on depressive symptoms among girls. (A) Significantly higher depressive symptoms in 10th-11th grade, among adolescent girls compared to boys. (B) Higher addiction-like social media use (ASMU) symptoms were significantly

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3 associated with higher depressive symptoms among girls but not among boys. **(C)** A
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5 significant moderated mediation model (PROCESS v.4, model 14) demonstrated that
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7 decreasing ventral media prefrontal cortex (vmPFC) responsivity across puberty was
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9 related to increased ASMU symptoms ~2.3 years later (10th and 11th grade) which, among
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11 adolescent girls (but not boys), was in turn, associated with increased depressive
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13 symptoms.
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19 DISCUSSION

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21 **Changes in social feedback brain responsivity across puberty linked to**
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23 **ASMU**. Our results identify differential changes in social feedback brain responsivity
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25 across puberty in four brain regions (i.e., vmPFC, rIFG, mPFC, and PCC) among
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27 adolescents that subsequently reported more addiction-like social media use. Prior work
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29 has demonstrated developmental changes (e.g., childhood to adolescence, and early
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31 adolescence to late adolescence) in responsivity to positive social outcomes (Smith et
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33 al., 2015; Somerville, 2013). For example, striatal and anterior cingulate cortex activity to
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35 positive social feedback anticipation increases from childhood to adolescence (Gunther
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37 Moor et al., 2010). As such, we expected brain responsivity to social feedback to increase
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39 with pubertal development. However, across the full sample this was not observed.
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41 Instead, we observed that this trajectory diverged for high and low ASMU-groups. While
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43 the low ASMU-group displayed expected increases in positive social feedback
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45 responsivity across pubertal development, adolescents in the high ASMU-group
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47 displayed elevated social feedback responsivity in the vmPFC, rIFG, mPFC, and PCC
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49 before puberty onset that then decreased with pubertal development.
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3 These results could suggest that some adolescents with premature elevations in
4 neural social feedback sensitivity may initially be more sensitive to the delivery of social
5 feedback via media. However, observed pubertal decreases in social feedback vmPFC,
6 rIFG, PCC, and mPFC responsivity may reflect desensitization to such feedback, possibly
7 through mechanisms similar to those driving tolerance-build up after repeated
8 administrations of an addictive drug. As we did not have data on participants' amount of
9 social media exposure over pubertal development this hypothesis could not be further
10 explored in this sample. Nonetheless, our findings indicate that developmental changes
11 in brain function previously implicated in social information processing may be associated
12 with individual differences in ASMU susceptibility. Such information could help target
13 prevention efforts aiming to mitigate maladaptive social media use and its influence on
14 adolescent mental health.

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31 **ASMU symptoms mediate the effect of brain responsivity development on**
32 **depressive symptoms.** Prior work suggests that increased brain responsivity to social
33 feedback may impact adolescent mental health through effects on their social
34 experiences. Specifically, anterior insula, cingulate, amygdala, and striatum responsivity
35 to social feedback among young adolescents (12-14 years old) impacts associations
36 between peer interpersonal stress and depression years later (Pagliaccio et al., 2023).
37 Further, prior findings from our research team similarly demonstrate that heightened
38 amygdala, and striatum activity to social feedback among young adolescents (11-14
39 years old) impacts associations between family conflict and externalizing behavior
40 (Turpyn et al., 2021). Our current findings extend this prior work showing that elevations
41 in neural responsivity to social feedback in early adolescence and subsequent
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3 developmental decreases into later adolescence may also impact social media use
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5 behaviors, and that this impact may increase risk for depressive symptoms.
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8 Further, our results highlight the importance of vmPFC and rIFG social feedback
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10 responsivity development. While all identified brain regions are implicated in social
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12 information processing (Spreng & Andrews-Hanna, 2015), the vmPFC, in particular, has
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14 been distinguished from other regions for its role in processing positively valenced or
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16 rewarding social information (van den Bos et al., 2007). Further, other work has
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18 suggested that the vmPFC and PCC may both be involved in processing self-referential
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20 social information (like social feedback) while more dorsal medial frontal regions are
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22 involved in thinking about the mental states of others (Wagner et al., 2012). Additionally,
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24 while the rIFC is known to be recruited when processing affective social information
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26 evidence suggests that it may be specifically involved when reappraising this information
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28 (Grecucci et al., 2013). Encoding valence, affective information, and referencing the self
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30 are likely all important when processing social feedback delivered on social media (e.g.,
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32 likes or comments). Taken together, these results suggest that development of vmPFC,
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34 and rIFG responsivity to social feedback are likely important targets for understanding
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36 how adolescents process social feedback delivered via social media and the impact it has
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38 on their media use behaviors and mental health.
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45 **Gender differences in mediating effect of ASMU symptoms on relationship**
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47 **between brain responsivity development and depressive symptoms.** Our results
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49 suggest gender effects in ASMU consequences by demonstrating a path through which
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51 divergent developmental trajectories of vmPFC and rIFG function may indirectly lead to
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53 depressive symptoms among girls through associations with more ASMU symptoms. This
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3 finding corresponds with prior work indicating higher social media use at age 10 is
4 associated with declines in well-being into early and mid-adolescence for girls but not for
5 boys (Booker et al., 2018). One possible explanation may be, as proposed by social role
6 theory (Eagly & Wood, 2012), girls are socialized to value social connections and social
7 standing more so than boys are. If this is the case, it is perhaps unsurprising that girls'
8 neural sensitivity to social feedback might carry more influence on their mental wellbeing
9 in instances of regular over-exposure to such information on social media.

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19 Another possible explanation of these results may be that gender is associated
20 with differential susceptibility to different types of media use (i.e., social media vs. non-
21 social digital media) and the respective consequences. Emergent meta-analytic research
22 suggests that ASMU may be more prevalent among women while addiction-like internet
23 gaming is more prevalent among men (Su et al., 2020). Social media may particularly
24 foster certain maladaptive use experiences among adolescents, that are not as prevalent
25 when using other non-social media, including social comparison, fear of missing out,
26 feedback seeking, and body image insecurities (Nesi & Prinstein, 2015). Therefore,
27 adolescent girls may be especially at risk. Future work examining various media types
28 and their unique impacts on mental health could more fully depict gender-based
29 susceptibility to ASMU.
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45 **Limitations.** Certain limitations should be considered. First, additional validation
46 of the ASMU measure in other samples is warranted and as social norms surrounding
47 digital media use shift, what constitutes problematic digital media behavior will also likely
48 need to shift. Second, several statistical considerations should be noted regarding our
49 observed ASMU-group by puberty interaction on social reward brain responsivity. First,
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3 while we observed significant interaction effects when dichotomizing the ASMU variable,
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5 results did not pass cluster-extent threshold corrections before dichotomizing. This
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7 discrepancy may be related to dichotomization reducing error in the measurement of the
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9 ASMU construct and thus increasing power. Further, we also note the potential for
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11 spurious interactions in whole-brain ANOVAs and thus the nature of each ASMU-group's
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13 developmental trajectory should be interpreted with caution (Chavez & Wagner, 2017).
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15 Third, while the Pubertal Development Scale is a widely used indicator of pubertal
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17 maturation that corresponds with other measures of puberty, including physician Tanner
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19 ratings and hormone levels (Herting et al., 2021), puberty is a highly individualized
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21 process and future work examining bias in caregiver-reports is warranted. Finally,
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23 information on social media exposure and use behaviors before and across pubertal
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25 development was not available. As such, we could not explore hypotheses regarding
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27 neural desensitization to social feedback due to accumulating exposure via social media
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29 use.
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35 **Conclusions.** Our findings suggest that before puberty onset, hyper-responsivity
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37 to positive social feedback, in four brain regions associated social information processing
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39 (vmPFC, rIFG, mPFC, and PCC), may represent a risk factor for ASMU in later
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41 adolescence. In contrast, decreases in this neural response over pubertal development
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43 could suggest atypical development of social feedback processing that is also linked with
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45 ASMU. Results suggest that these developmental differences in social feedback brain
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47 responsivity are associated with depressive symptoms among adolescent girls through
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49 their relationship with increased ASMU symptom endorsement in later adolescence.
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51 Taken together, this study identifies developmental individual differences (i.e., brain
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responsivity to social feedback and gender identity) that may be important for understanding ASMU susceptibility and related mental health outcomes.

DISCLOSURES: The authors have no conflicts to declare.

DATA AVAILABILITY: The authors have released all code associated with this manuscript. Code is available on GitHub https://github.com/Flanneryg3/ASMU_ProjectCode and group-level statistical brain maps are stored in the following NeuroVault collection: <https://identifiers.org/neurovault.collection:14537>

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For Peer Review

SUPPLEMENTAL INFORMATION**Developmental changes in brain function linked with addiction-like social media use two years later**

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SUPPLEMENTAL CONTENT

- Retention and demographic info for data collection waves (pp. 2)
- Figure S1. Retention across data collection waves (pp. 2)
- MRI data acquisition and analysis (pp. 3)
- Figure S2. Small Volume Corrected Mask. (pp. 4)
- Figure S3. Social reward brain responsivity across pubertal development (pp. 5)
- Figure S4. Addiction-like social media use symptoms mediate the effect of rIFG change across puberty on depressive symptoms among girls. (pp. 6)
- Table S1. Cluster Coordinates for GENDER × ASMU-Group × PUBERTY effects on positive social feedback vs. neutral feedback brain activity. (pp. 7)
- Supplemental references (pp. 8)

Retention and demographic info for data collection waves. Participants were recruited from a larger study of 873 students in 6th and 7th grade in a small, diverse, rural community in the southeast United States. Data collection took place from December 2016 to January 2022. Some of the demographic, descriptive, and fMRI data from this sample is also reported elsewhere^{1,2}. At timepoint 1, five participants were excluded due to exclusionary criteria being met after recruitment (e.g., major claustrophobia during the fMRI session). These participants were not invited back for subsequent study participation. Out of the 143 remaining timepoint 1 participants, 8 were excluded due to impeded task data collection (e.g., acute participant anxiety, overwritten E-prime file). The final timepoint 1 sample size included 135 adolescents. At timepoint 2, 116 participants from cohort 1, and 30 new participants from cohort 2, participated. 17 participants were excluded due to impeded task data collection, resulting in 129 adolescents included in timepoint 2. Finally, at timepoint 3, 119 participants from cohort 1, and 26 participants from cohort 2, participated. 20 participants were excluded due to impeded task data collection bringing the final timepoint 3 sample size to 125 adolescents. 103 additional self-report data points were collected at a 4th timepoint. A schematic of participant recruitment at each wave is presented in **Figure S1**. Ultimately, we only included participants that had addiction-like social media use symptom data at the 4th and final timepoint ~2.3 years after the final fMRI data collection timepoint (n=103). This sample (Mage = 17.0 ± 0.6; 51.5% female) included 53.4% in 10th grade, and 46.6% in 11th grade and did not significantly differ from the larger non-participating sample on age, gender, race/ethnicity, depression symptoms, or pubertal development. Of these 103 adolescents that had data at the final timepoint, 80 had data at the 1st timepoint, 90 had data at the 2nd timepoint, and 86 had data at the 3rd timepoint (see **Table 1** in main text).

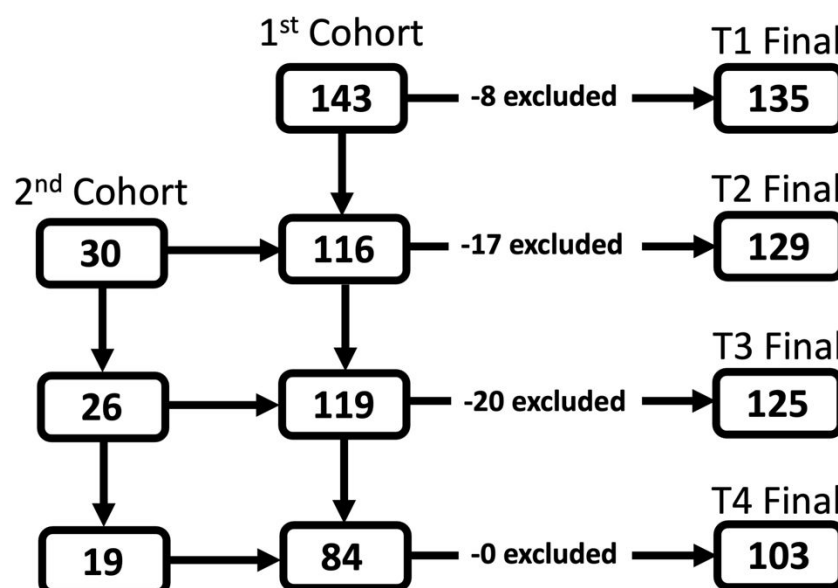


Figure S1. Retention across data collection waves. 143 adolescents participated in timepoint 1 however, 8 were excluded due to impeded task data collection (e.g., acute participant anxiety, overwritten E-prime file) resulting in 135 adolescents in the final

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2
3 sample at timepoint 1. At timepoint 2, 116 participants from cohort 1, and 30 new
4 participants from cohort 2, participated. 17 participants were excluded due to impeded
5 task data collection, resulting in 129 adolescents included in timepoint 2. Finally, at
6 timepoint 3, 119 participants from cohort 1, and 26 participants from cohort 2, participated.
7 20 participants were excluded due to impeded task data collection bringing the final
8 timepoint 3 sample size to 125 adolescents. 103 additional self-report data points were
9 collected at a 4th timepoint with data from 84 participants from cohort 1 and 19 participants
10 from cohort 2.
11
12

13 **MRI data acquisition and analysis.** MRI data were collected on a Siemens Prisma MRI,
14 3-Tesla scanner. For the two functional SID runs, 37 slices (3 mm thick; voxel size 2.5 x
15 2.5 x 3 mm) were obtained using a T2*-weighted, single-shot, gradient-echo, echo-planar
16 imaging (EPI) sequence sensitive to blood oxygenation level-dependent (BOLD) effects
17 (195 volumes/run, repetition time [TR] = 2000 ms, echo time [TE] = 25 ms, field of
18 view = 230 mm, 92 × 92 matrix). The orientation for the EPI scans was oblique axial to
19 maximize brain coverage and to reduce noise. T1-weighted structural images were
20 obtained in the sagittal plane using a magnetization-prepared rapid gradient-echo
21 (MPRAGE) sequence (TR = 2400 ms; TE = 2.22 ms; 208 slices; voxel size = 0.8mm³).
22

23 Preprocessing was conducted using FSL (FMRIB's Software Library, version 6.0;
24 www.fmrib.ox.ac.uk/fsl) and included the following steps: Skull stripping using BET;
25 motion correction with MCFLIRT; spatial smoothing with a Gaussian kernel of 6 mm, full-
26 width-at-half maximum; high-pass temporal filtering with a filter width of 128 s (Gaussian-
27 weighted least-squares straight line fitting, with sigma = 64.0 s); grand-mean intensity
28 normalization of the entire 4D dataset by a single multiplicative factor; and individual level
29 ICA denoising for artifact signal using MELODIC (version 3.15), combined with an
30 automated signal classifier³; Neyman-Pearson threshold =0.3). For the spatial
31 normalization, the EPI data were registered to the T1 image with a linear transformation,
32 followed by a white-matter boundary-based transformation using FLIRT, linear and non-
33 linear transformations to standard Montreal Neurological Institute (MNI) 2-mm brain using
34 Advanced Neuroimaging Tools, and then spatial normalization of the EPI image to the
35 MNI.
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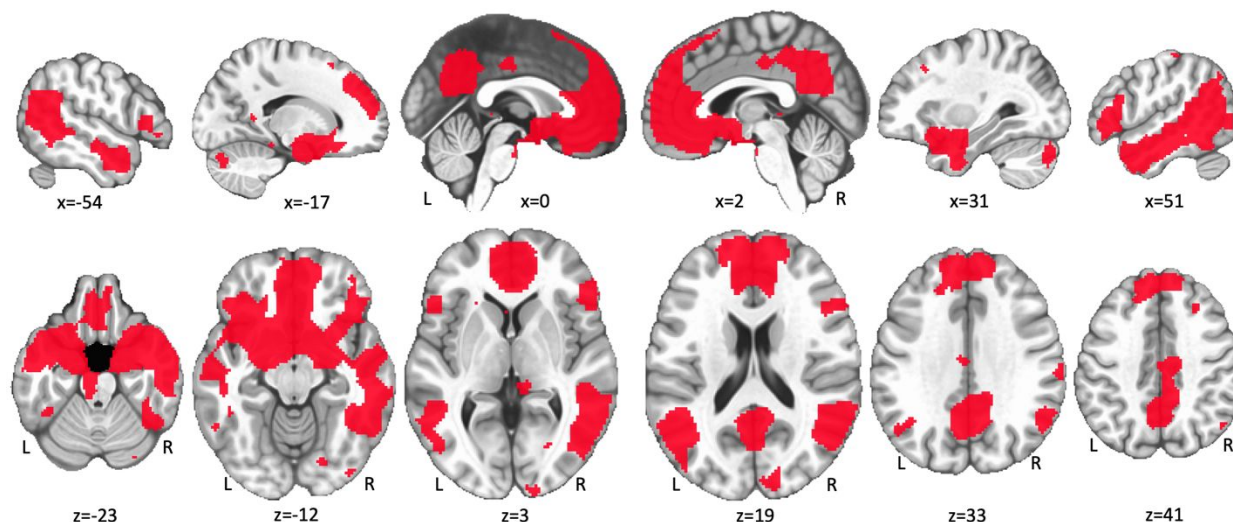


Figure S2. Small Volume Corrected Mask. We defined a small volume corrected (SVC) brain mask via NeuroSynth's meta-analysis of the term "social" (thresholded at false discovery rate=0.01) derived from 1,302 studies. This map was registered to the functional data, binarized, dilated by two units, and subsequently eroded by 2 units using AFNI's 3dmask_tool to connect little speckled parts around big regions. We then removed small areas of the mask that did not overlap with the sample-specific gray-matter mask. The final mask included 36,386 voxels.

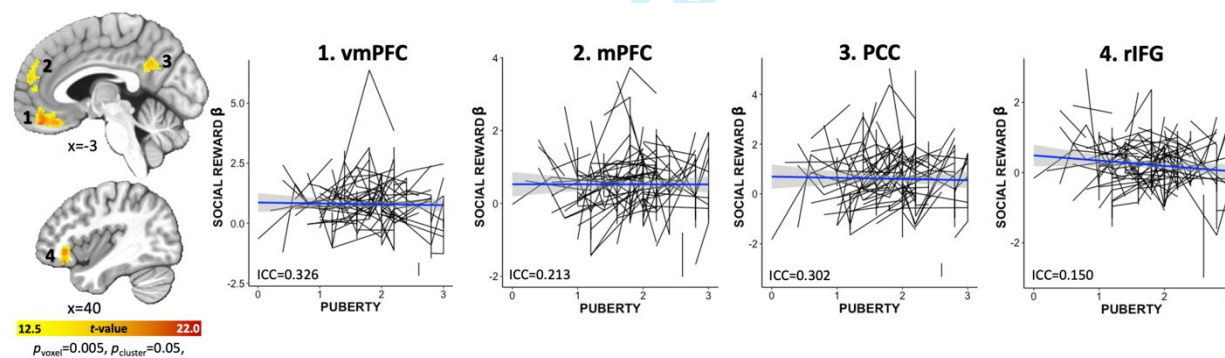


Figure S3. Social reward brain responsivity across pubertal development. To examine developmental changes in responsivity to social reward feedback, we assessed the effect of pubertal development total score on task contrast β coefficients within linear mixed-effect models (*lmer*, *lme4* R package). The random intercept and slope of puberty as well as their correlation were modeled within subject. We did not observe any significant main effects of puberty across the full sample (p 's>0.3). To interrogate variability versus stability of social reward feedback processing brain responsivity in identified regions, over time we assess the interclass correlation (ICC), across the full sample, for each brain region. **(1)** 32.6% of the variance in ventral media prefrontal cortex (vmPFC) social reward feedback responsivity is associated with between-participant differences. Thus, within a given adolescent, vmPFC responsivity across pubertal

development has a 0.326 correlation. **(2)** Within a given adolescent, medial prefrontal cortex (mPFC) responsivity has a 0.213 correlation across pubertal development. Thus 21.3% of the variability in mPFC social reward feedback responsivity is associated with between-participant differences. **(3)** Within a given adolescent, posterior cingulate cortex (PCC) responsivity has a 0.302 correlation across pubertal development and 30.2% of the variance is associated with between-participant differences and finally, **(4)** within a given adolescent, right inferior frontal gyrus (rIFG) social reward feedback responsivity has a 0.15 correlation across pubertal development and 15.0% of the variance is associated with between-participant differences.

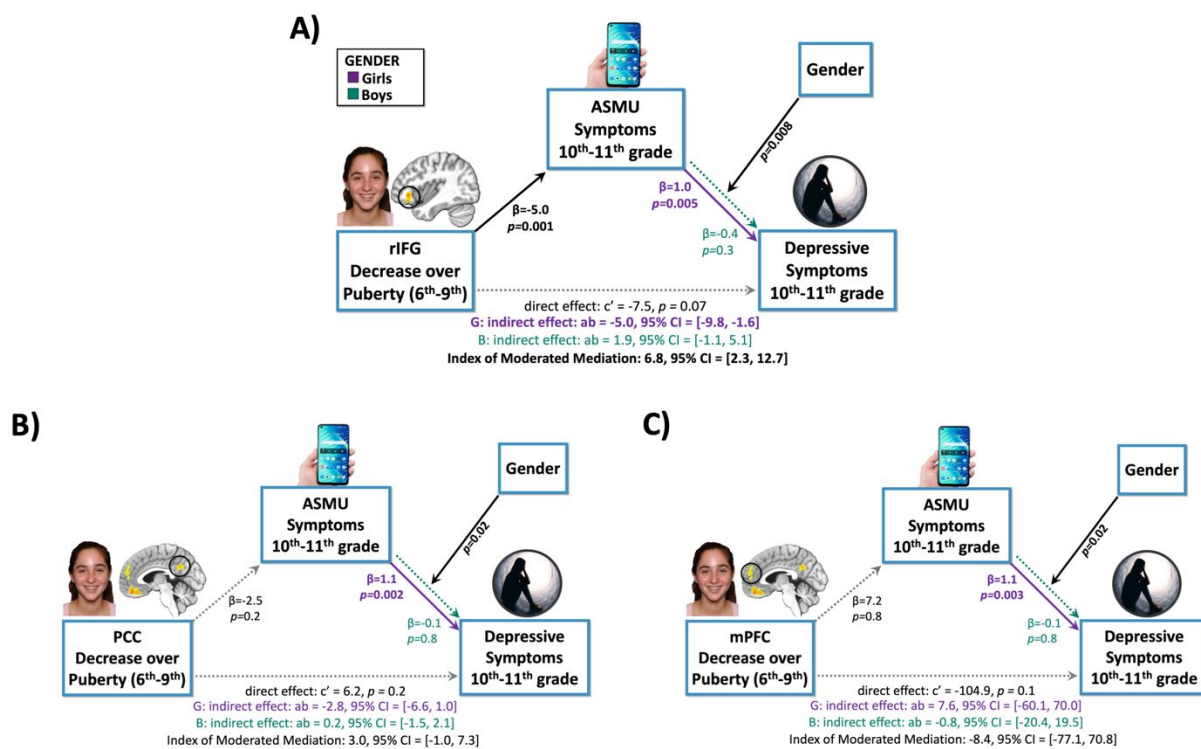


Figure S4. Addiction-like social media use symptoms mediate the effect of rIFG change across puberty on depressive symptoms among girls. (A) A significant moderated mediation model (PROCESS v.4, model 14)⁴ demonstrated that decreasing right inferior frontal gyrus (rIFG) responsivity across puberty was related to increased addiction-like social media use (ASMU) symptoms ~2.3 years later (10th and 11th grade) which, among adolescent girls (but not boys), was in turn, associated with increased depressive symptoms. **(B)** This same model was not significant for the posterior cingulate cortex (PCC) nor for the **(C)** medial prefrontal cortex (mPFC).

Table S1. Cluster Coordinates for GENDER × ASMU-Group × PUBERTY effects on positive social feedback vs. neutral feedback brain activity. Within a linear multilevel model framework (3dimer; AFNI), modeling the random intercept and slope, we assessed a 3-way GENDER × ASMU-Group × PUBERTY cross-level interaction on the positive social feedback vs. neutral feedback contrast within a small volume corrected brain mask (36,386 voxels) defined via NeuroSynth's meta-analysis of the term "social" (3dLMER⁵, $p_{\text{voxel-level}} < 0.005$, $p_{\text{cluster-level}} < 0.05$, cluster extent threshold = 213 voxels, determined via ACF 3dclustsim)⁶. Three participants reporting nonbinary gender identities were not included in analyses comparing effects of self-reported female and male identities (N=100).

| Positive Social Feedback > Neutral Feedback | | Hemisphere | Center Coordinates (MNI, LPI) | | | Cluster Size (# of Voxels) |
|---|--|------------|----------------------------------|-----|-----|-------------------------------|
| | | | X | Y | Z | |
| ASMU × PUBERTY | | | | | | |
| 1 | Superior medial frontal gyrus (BA 9) | B | -7 | 49 | 28 | 393 |
| 2 | Ventral media frontal gyrus/orbital gyrus/rectal gyrus | B | -3 | 37 | -20 | 358 |
| 3 | Posterior cingulate gyrus/precuneus (BA 31) | B | 42 | 31 | -8 | 295 |
| 4 | Inferior frontal gyrus (BA 11) | R | 42 | 31 | -8 | 226 |
| GENDER Main Effect | | | | | | |
| 1 | Superior temporal gyrus (BA 13) | R | 53 | -47 | 22 | 350 |

NOTE. Voxel size: 2 x 2 x 2 mm³. X: Left (-), Right (+); Y: Posterior (-), Anterior (+); Z: Inferior (-), Superior (+). Region labels informed by the AFNI Talairach daemon atlas. See Main Text Figure 1 for graphical representation.

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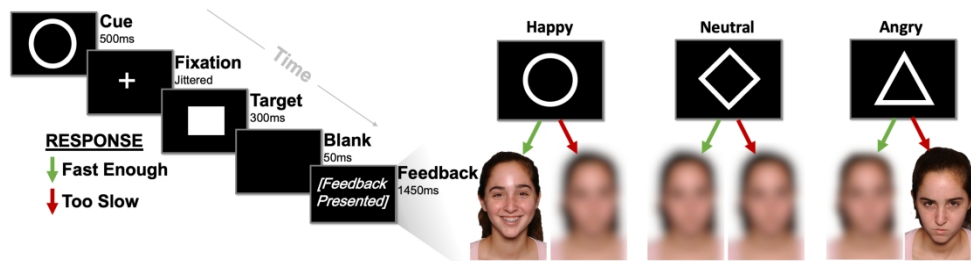


Figure 1. Social incentive delay (SID) task. During MRI scanning, participants completed two 6.5 min runs of a Social Incentive Delay (SID) task designed to measure neural sensitivity to anticipation (cue) and receipt (outcome) of social rewards (smiling face) and punishments (scowling face). Participants completed two 6.5 min runs consisting of 58 trials, resulting in a total of 116 trials (48 reward trials, 48 punishment trials, and 20 neutral trials). In the task, participants see a cue (circle, square, or diamond, 500 ms) indicating what type of trial will follow. Then, following a fixation cross (duration jittered ~509-4249 ms), they see a target (white square, 160-500 ms). Participants are trained to press their right index finger as fast as they can after seeing the target, but not before. Following a delay (50 ms), participants receive social feedback (1450 ms) based on both the trial type and whether they pressed fast enough. The social feedback is photographs of adolescent faces taken from the NIH faces dataset (Egger et al., 2011). In the task, there were 24 faces shown (12 female, 12 male). Participants are explicitly told that the circle is a happy cue, the square is an angry cue, and the diamond is a neutral cue, meaning if they press fast enough after seeing the happy cue (Reward cue), they will see a smiling face (Reward hit); if they press too slow after the happy cue, they will see a blurred face (Reward miss). If they press fast enough after seeing the angry cue (Punishment cue), they will see a blurred face (Punishment hit); if they press too slow after the angry cue, they will see a scowling face (Punishment miss). Following the Neutral cue, they will see a blurred face whether they press fast enough (Neutral hit) or too slow (Neutral miss). To ensure sufficient exposure to all feedback types, task difficulty was individually and dynamically adapted based on prior performance by increasing or decreasing the target duration (starting at 300ms) by 20 ms intervals unless reaching a minimum of 160 ms or maximum of 500 ms duration.

253x71mm (300 x 300 DPI)

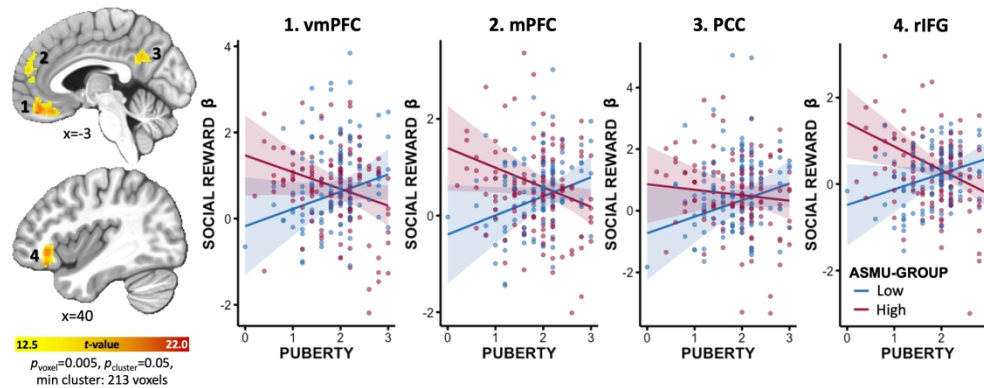


Figure 2. Changes in social feedback brain responsivity across puberty linked to ASMU. Significant ASMU-Group \times PUBERTY interactions were observed in the ventral media prefrontal cortex (1. vmPFC; 358 voxels), medial prefrontal cortex (2. mPFC; 393 voxels), posterior cingulate cortex (3. PCC; 295 voxels), and right inferior frontal gyrus (rIFG; 226 voxels) when controlling for GENDER effects. In all four significant clusters, adolescents in the low ASMU-group displayed relatively lower responsivity to positive social feedback before puberty onset that increased with pubertal development, whereas adolescents in the high ASMU-group displayed hyper-responsivity before puberty onset that decreased with pubertal development.

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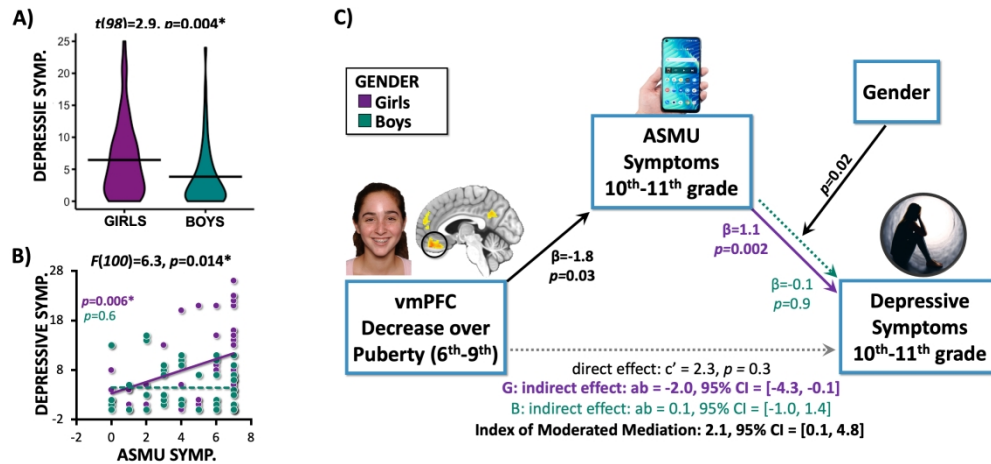


Figure 3. Addictive social media use symptoms mediate the effect of vmPFC change across puberty on depressive symptoms among girls. (A) Significantly higher depressive symptoms in 10th-11th grade, among adolescent girls compared to boys. (B) Higher addiction-like social media use (ASMU) symptoms were significantly associated with higher depressive symptoms among girls but not among boys. (C) A significant moderated mediation model (PROCESS v.4, model 14) demonstrated that decreasing ventral media prefrontal cortex (vmPFC) responsivity across puberty was related to increased ASMU symptoms ~2.3 years later (10th and 11th grade) which, among adolescent girls (but not boys), was in turn, associated with increased depressive symptoms.

253x116mm (300 x 300 DPI)

Table 1. Demographic information by timepoint.

| Timepoint | 1st | 2nd | 3rd | 4th |
|------------------------|-----------------------|-----------------------|-----------------------|-----------------------|
| | n=80 | n=90 | n=86 | n=103 |
| Gender Identity | | | | |
| Girls | 46.3% | 46.7% | 46.5% | 51.5% |
| Boys | 51.2% | 51.1% | 51.2% | 45.6% |
| Nonbinary | 2.5% | 2.2% | 2.3% | 2.9% |
| Age | | | | |
| | 12.8±0.5 | 13.7±0.6 | 14.7±0.6 | 17.0±0.6 |
| Grade | | | | |
| 6 th | 47.5% | 0 | 0 | 0 |
| 7 th | 52.5% | 51.1% | 0 | 0 |
| 8 th | 0 | 48.9% | 50% | 0 |
| 9 th | 0 | 0 | 50% | 0 |
| 10 th | 0 | 0 | 0 | 53.4% |
| 11 th | 0 | 0 | 0 | 46.6% |

NOTE. Gender identity (% of total), age (mean ± standard deviation), and grade (% of total) are presented for each data collection timepoint. The multiple cohort structure of the study resulted in planned missing data across timepoints. Specifically, 62 of the total 103 participants had 3 fMRI timepoints, 29 had 2, and 12 had 1. All participants had a 4th self-report timepoint.

Table 2. Demographic, socioeconomic, and symptom information by ASMU-group.

| | N=103 | High ASMU (n=52) | Low ASMU (n=51) | Group Differences |
|--------------------------------|---------|---------------------|--------------------|---------------------------|
| Gender Identity | | | | <i>p</i> =0.1 |
| Female | 51.5% | 55.8% | 47.1% | |
| Male | 45.6% | 38.5% | 52.9% | |
| Nonbinary | 2.9% | 5.7% | 0 | |
| Race/Ethnicity | | | | <i>p</i> =0.6 |
| White | 37.9% | 36.6% | 39.2% | |
| Hispanic/Latinx | 32.0% | 34.6% | 29.4% | |
| Black | 23.3% | 19.2% | 27.4% | |
| Multi-Racial | 4.9% | 7.7% | 2.0% | |
| Asian | 1.9% | 1.9% | 2.0% | |
| Household Annual Income | | | | <i>p</i> =0.5 |
| \$0-29,999 | 28.0% | 27.5% | 28.6% | |
| \$30,000-59,999 | 31.0% | 37.2% | 24.5% | |
| \$60,000-99,999 | 26.0% | 21.6% | 30.6% | |
| >\$100,000 | 15.0% | 13.7% | 16.3% | |
| Symptoms | | | | |
| Depressive symptoms | 7.0±6.5 | 8.3±6.9 | 5.6±6.0 | <i>p</i>=0.04* |
| ASMU symptoms endorsed | 4.5±2.6 | 6.8±0.4 | 2.5±1.7 | <i>p</i><0.001* |

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3 NOTE. Gender identity (% of total), and race/ethnicity (% of total) were measured at each
4 participant's first timepoint (6th or 7th grade). Household annual income (% of total) and
5 depressive symptoms (mean \pm standard deviation) were measured at the final timepoint
6 in 10th-11th grade. Addiction-like social media use (ASMU) symptoms (mean \pm standard
7 deviation) were also measured at the follow-up timepoint. ASMU symptoms endorsed is
8 a count of the total number endorsed. ASMU-Group differences in gender identity, and
9 race/ethnicity were assessed with cross tabulation Chi-squared tests. ASMU-Group
10 differences in household annual income were assessed with a Kruskal-Wallis test for
11 ordinal variables which is similar to a one-way ANOVA but ranks of the data values are
12 used in the test rather than the actual data points. ASMU-Group differences in depressive
13 symptoms, ASMU symptoms were assessed with independent samples *t*-tests.
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