Developmental trajectories to reduced activation of positive valence systems: A review of biological and environmental contributions

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ABSTRACT

Reduced activation of positive valence systems (PVS), including blunted neural and physiological responses to pleasant stimuli and rewards, has been shown to prospectively predict the development of psychopathology. Yet, little is known about how reduced PVS activation emerges across development or what implications it has for prevention. We review genetic, temperament, parenting, and naturalistic and laboratory stress research on neural measures of PVS and outline developmentally-informed models of trajectories of PVS activation. PVS function is partly heritable and appears to reflect individual differences in early-emerging temperament traits. Although lab-induced stressors blunt PVS activation, effects of parenting and naturalistic stress on PVS are mixed and depend on the type of stressor, developmental timing, and interactions amongst risk factors. We propose that there may be multiple, dynamic developmental trajectories to reduced PVS activation in which combinations of genes, temperament, and exposure to severe, prolonged, or uncontrollable stress may exert direct and interactive effects on PVS function. Critically, these risk factors may alter PVS developmental trajectories and/or PVS sensitivity to proximal stressors. Distinct factors may converge such that PVS activation proceeds along a typical, accelerated, chronically low, or stress-reactive trajectory. Finally, we present directions for future research with translational implications.

1. Positive valence systems (PVS) and mental and physical health

In October 2012, Superstorm Sandy struck the coastal regions of New York and New Jersey, with devastating consequences. Our group has been following a cohort of children in the region as part of the Stony Brook Temperament Study (Klein and Finsaas, 2017), and had the unique ability to examine pre-existing vulnerabilities that predicted responses to natural disaster-related stress. Although we anticipated that heightened neural reactivity to threatening images, as measured by event-related potentials (ERPs), would increase risk for psychiatric symptoms, reduced neural reactivity to positively-valenced stimuli also emerged as a unique predictor of the development of psychiatric symptoms in combination with hurricane-related stress (Kujawa et al., 2016). That is, children who showed reduced sustained attention to pleasant images—assessed at the neural level—exhibited elevated symptoms when exposed to an acute stressor.

Indeed, growing evidence indicates that reduced activation of positive valence systems (PVS), including low positive emotionality and reduced reward responsiveness, is a key predictor of both mental and physical health problems (Danner et al., 2001; Keren et al., 2018; Kujawa and Burkhouse, 2017; Salovey et al., 2000; Tugade et al., 2004). PVS, a domain of the National Institute of Mental Health Research Domain Criteria (RDoC), includes behavioral and physiological processes involved in anticipating, obtaining, and responding to positive stimuli and rewards (National Institute of Mental Health, 2019; Olino, 2016). Particularly relevant to the current review, reduced activation of PVS predicts, and therefore may be a vulnerability for, some forms of psychopathology, particularly depression (Keren et al., 2018; Kujawa and Burkhouse, 2017). Reduced PVS function has been proposed both as a mechanism of the development of psychiatric disorders (Hanson et al., 2017b; Keren et al., 2018) and a moderator of the effects of stress and other risk factors on the emergence of psychiatric symptoms (Corral-Frías et al., 2015; Dennison et al., 2016; Goldstein et al., 2019; Kujawa

Table 1
Genetics: Overview of studies of genes/heritability and PVS function.

First Author, Year	Predictor	PVS Measure	Sample Size (N)	Sample Gender (% Female)	Age Range or M (Years)	PVS Finding	Estimated Effect Size (r)
Child and Adolescent Studies							
Jia, 2016	VPS4A	BOLD response to reward anticipation	1544	53	12.9–16.4	VPS4A C associated with ↓ striatum	.14
Moser, 2018	Heritability (family study)	RewP to reward feedback	145 children, 130 parents	53	3–13	Father-child RewP positively correlated; mother-child RewP negatively correlated	—
Silverman, 2014	Heritability (twin study)	BOLD response to reward anticipation	48 (MZ pairs)	50 (n=24 pairs)	15–17	Correlations between MZ twins in ~½ of striatal regions	.38–.53
Stice, 2012	Multilocus composite	BOLD response to reward anticipation, feedback	160	51	M=15.3 (SD=1.1)	Multilocus composite associated with ↓ striatum during reward receipt	.27–.35
Adult Studies							
Aarts, 2010	DAT1, COMT	BOLD response to reward anticipation, feedback	20	50	18–27	DAT1 9-repeat associated with ↑ striatum; no effect of COMT on striatum	—
Baker, 2016	DRD4, COMT	RewP to reward feedback	195	75	18–51	No effect DRD4 or COMT on striatum	—
Boecker-Schlier, 2016	DAT, DRD4, COMT	BOLD/EEG response to reward anticipation, feedback	168	58	M=24.5 (SD=0.6)	COMT Met homozygotes showed ↑ VS with early adversity; no effect DAT, DRD4 on VS	—
Bogdan, 2009	Heritability (twin study)	Probabilistic reward learning	20 MZ, 15 DZ twin pairs	MZ: 90; DZ: 87	MZ: M=29.0 (SD=10.9); DZ: 33.7 (SD=13.5)	MZ twin correlations higher than corresponding DZ correlations	MZ: .59; DZ: -.05
Camara, 2010	DRD4, COMT	BOLD response to reward feedback	36	67	18–34	COMT ValVal associated with ↑ VS to large unexpected wins; no effect of DRD4 on striatum	—
Cohen, 2005	DRD2	BOLD response to reward anticipation, feedback	16	44	20–27	DRD2 A1 predicted ↓ VS to reward feedback	—
Dillon, 2010	DAT1, COMT	BOLD response to reward anticipation, feedback	32	50	M=21.7 (SD=3.4)	No main effect DAT1 or COMT on striatum; positive association for DAT1 9-repeat/COMT Met combined	—
Dreher, 2009	DAT1, COMT	BOLD response to reward anticipation	27	41	M=27.3 (SD=5.7)	COMT Met and DAT1 9-repeat associated with ↑ striatum; COMT x DAT1 for striatum	.51–.62
Felsted, 2010	DRD2	BOLD response to food	26	24	—	DRD2 A1 associated with ↓ striatum activation to milkshake	.58
Forbes, 2009	DAT1, DRD2, DRD4, COMT	BOLD response to reward	89	57	M=43.8 (SD=6.5)	DAT1 9-repeat, DRD2, DRD2−141C Del, DRD4 7-repeat associated with ↑ VS; no effect of COMT	.30–.34
Foti, 2012	COMT	ERP to reward anticipation, feedback	83	46	Undergraduates	COMT Met associated with ↑ ERPs to reward anticipation and RewP	.28–.29
Hahn, 2011	DAT	BOLD response to reward anticipation	53	55	18–47	DAT 10-repeat homozygotes x self-reported reward sensitivity scores associated with ↑ VS	—
Heitland, 2012	DAT1, COMT, 5HTTPLR	RewP to reward feedback	60	100	M=20.9 (SD=2.0)	DAT1 and 5HTTPLR associated with enhanced RewP to loss; no effect of COMT	.30–.33
Lancaster, 2017	rs322931 on chromosome 1	BOLD response to pleasant images, reward feedback	S1: 81; S2: 82	S1: 60; S2: 60	19–47	rs322931 associated with ↓ VS to pleasant images and ↑ VS to reward	—
Marco-Pallarés, 2009	DRD4, COMT	RewP to reward feedback	40	75	18–35	COMT ValVal homozygotes associated with ↑ RewP; no effect of DRD4	—
Mueller, 2014a	COMT (moderated by D2 antagonist sulpiride)	BOLD response to reward feedback	86	100	18–31	COMT x D2 antagonist sulpiride on RewP	—
Nikolova, 2011	Multilocus composite	BOLD response to reward feedback	69	54	M=44.5 (SD=6.7)	Multilocus composite predicted ↑ VS	.33
Peciña, 2013	DRD2	BOLD response to reward anticipation	86	50	19–54	No effect DRD2 on striatum	—
Richter, 2017	DRD2	BOLD response during encoding of reward cues	62	—	M=24.6 (SD=2.8)	DRD2 C957T modulates striatum response during reward encoding	—
Schmack, 2008	COMT	BOLD response to reward anticipation, feedback	44	20	M=38.7 (SD=10.0)	COMT Met associated with ↑ VS during anticipation of loss	.35
Weinberg, 2015a	Heritability (sibling study)	RewP to reward feedback	140 (70 sibling pairs)	59	18–30	Positive correlation between siblings on RewP to reward	.31
Weinberg, 2015b	Heritability (twin study)	LPP to pleasant images	479	49	22–38	Stronger positive correlation of LPP to pleasant images for MZ compared to DZ twins	MZ: .28–.55; DZ: .11–.15
Wingo, 2017	rs322931 on chromosome 1	RewP to reward feedback	45	100	Adults (≥ 18)	rs322931 associated with ↑ VS	.36

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Table 1 (*continued*)

First Author, Year	Predictor	PVS Measure	Sample Size (N)	Sample Gender (% Female)	Age Range or M (Years)	PVS Finding	Estimated Effect Size (r)
		BOLD response to pleasant images					
Wittmann, 2013	DAT1	BOLD response to reward anticipation, feedback, memory	24	67	M = 25.3 (SD = 3.9)	DAT1 10-repeat homozygotes associated with ↑ striatum during reward anticipation/memory	.64–.70
Yacubian, 2007	DAT, COMT	BOLD response to reward anticipation, feedback	98	0	18–46	COMT Met homozygotes associated with ↑ striatum; COMT x DAT for VS	.28–.34

Note: BOLD = blood oxygen level dependent; COMT = catechol-O-methyltransferase; DA = dopamine; DAT = dopamine transporter; DRD2 = dopamine receptor D2; DRD4 = dopamine receptor D4; DZ = dizygotic; ERP = event-related potentials; LPP = late positive potential; MZ = monozygotic; RewP = reward positivity; VPS4A = vacuolar protein sorting-associated protein 4A; VS = ventral striatum.

Table 2
Temperament: Overview of studies of temperament and PVS function.

First Author, Year	Predictor	PVS Measure	Sample Size (N)	Sample Gender (% Female)	Age Range or M (Years)	PVS Finding	Estimated Effect Size (r)
Child and Adolescent Studies							
Kessel, 2017	Observed PE at age 6	LPP to pleasant images at age 9	340	46	M= 6.6 (SD = 0.5); follow up: M = 9.1, (SD = 0.4)	Early PE predicted enhanced LPP	.11
Kujawa, 2015a	Observed PE at age 3, self-reported PE at age 9	RewP to reward feedback	381	45	M = 3.6 (SD = 0.3); M = 9.2 (SD = 0.4)	Both measures of PE positively predicted RewP	.11–.12
Speed, 2015	Self-reported E, PE, N	LPP to pleasant images	523	100	13.5–15.5	E and PE positively associated with LPP	.10–.14
Speed, 2018	Self-reported PE, N	RewP to reward feedback	508	100	13.5–15.5	PE positively associated with ΔRewP at low and average N	.09–.12
Adult Studies							
Beaver, 2006	Self-reported BAS	BOLD response to images of appetizing food	12	58	M = 22.0 (SD = 2.4)	BAS positively correlated with VS	.80
Canli, 2001	Self-reported E	BOLD response to pleasant images	14	100	19–42	E positively correlated with striatum	.82–.86
Cohen, 2005	Self-reported E	BOLD response to reward anticipation, feedback	S1: 17; S2: 16	S2: 44	S2: 20–27	E positively correlated with VS to reward feedback	.56–.61
Cooper, 2014	Self-reported E	ERP response to reward feedback	38	47	19–42	E positively correlated with RewP	.36
Geaney, 2015	Self-reported BAS	Effort expended for rewards	97	59	18–44	BAS positively correlated with effort to obtain reward	—
Haas, 2006	Self-reported E, facets	BOLD response to positive words	26	54	18–28	E associated with ACC, but not with striatum	—
Hahn, 2011	Self-reported reward sensitivity	BOLD response to reward anticipation	53	55	18–47	Reward sensitivity positively correlated with VS	.39
Hutcherson, 2008	Self-reported E	BOLD response to amusing films	28	100	18–21	E negatively correlated with VS	.67–.71
Kehoe, 2012	Self-reported E, N	BOLD response to pleasant images	23	100	19–29	No effect on striatum	—

Table 2 (*continued*)

First Author, Year	Predictor	PVS Measure	Sample Size (N)	Sample Gender (% Female)	Age Range or M (Years)	PVS Finding	Estimated Effect Size (r)
Lange, 2012	Self-reported BAS	RewP to feedback in an extinction learning task	85	41	20–29	BAS correlated with RewP following change from reward to non-reward	.22–.26
Mobbs, 2005	Self-reported E, N	BOLD response to positive cartoons	17	47	M = 22.8 (SD = 1.9)	No effect of E, N on striatum	—
Mueller, 2014b	Self-reported E	RewP response to positive feedback (placebo vs. sulpiride)	86	100	18–31	E marginally predicted RewP in placebo group	.30
Rapp, 2008	Self-reported trait cheerfulness	BOLD response to positive cartoons	10	100	—	No effect of cheerfulness on VS	—
Schaefer, 2011	Self-reported E, N	BOLD response to chocolate brand images	12	42	21–31	E associated with ↓ VS; N associated with ↑ VS	.54–.71
Schweckendiek, 2016	Self-reported E, N	BOLD response during reward learning	20	50	19–33	No effect of E on striatum	—
Simon, 2010	Self-reported BAS	BOLD response to reward anticipation, feedback	24	54	M = 24.8 (SD = 3.2)	BAS positively correlated with VS to reward feedback	.44
Smillie, 2011	Self-reported E	RewP to reward feedback	30	47	M = 23.4 (SD = 5.1)	E positively correlated with RewP	.46
Suslow, 2010	Self-reported E, implicit IAT E	BOLD response to happy faces	30	100	M = 23.0 (SD = 1.8)	No effect of E on striatum	—
Weinberg, 2018	Self-reported PE	LPP to rewarding images	205	77	M = 19.3 (SD = 1.8)	Low PE predicted ↓ LPP	.17
Wu, 2014	Self-reported trait positive/ negative arousal	BOLD response to reward anticipation	52	56	21–75	PE positively correlated with VS	.31

Note: ACC = anterior cingulate cortex; BAS = behavioral activation system; BOLD = blood oxygen level dependent; E = extraversion; ERP = event-related potentials; IAT = Implicit Association Test; LPP = late positive potential; mPFC = medial prefrontal cortex; N = neuroticism; PE = positive emotionality; RewP = reward positivity; VS = ventral striatum; Δ = change.

et al., 2019b, 2016; Sandre et al., 2019).

Despite the promise of these findings, progress in translating affective neuroscience to prevention is hindered by limited understanding of how reduced activation of PVS emerges across development, and whether and when trajectories can be altered to promote healthy development. Moving beyond studies of the emergence of psychiatric symptoms, it is critical to chart developmental trajectories of more narrowly-defined dimensions of emotion and behavior, and examine how biological and environmental factors influence their development. Animal research on PVS function implicates complex interactions between genetic predispositions, specific types of stressors, and developmental windows (Cabib and Puglisi-Allegra, 1996; Novick et al., 2018), but we lack an integrated model of individual differences in the development of PVS function in humans.

In this review, we evaluate the evidence for commonly implicated genetic and environmental influences on PVS activation. Conceptualizing alterations in PVS function as a potential vulnerability for psychopathology, we focus on neural (i.e., ERP and functional magnetic resonance imaging [fMRI]) studies of responses to appetitive stimuli and rewards in community samples to avoid confounds with clinical disorders. We limit our review to experimental designs examining responses to positive stimuli, rather than studies of resting brain activity in which PVS function is inferred but not directly manipulated. We include studies of brain activation to pleasant stimuli as well as guessing, monetary incentive delay, and reward learning tasks. We report details of designs and samples in Tables 1–5, organized by child/adolescent and adult studies. Effect size estimates for the main effect of a predictor on a PVS measure are included in the tables when available. In cases of interactions, effect sizes are presented within subgroups and described accordingly. Importantly, *enhanced* activation of PVS function in certain domains is also associated with psychopathology risk, including bipolar disorder, substance use, and impulsivity (Nusslock and Alloy, 2017; Plichta and Scheres, 2014; Stice et al., 2013), but we focus the current review on developmental precursors to low PVS activation, specifically, given growing and consistent evidence of links with depression risk (Keren et al., 2018; Kujawa et al., 2019b; Nelson et al., 2016; Stringaris et al., 2015) and limited research on PVS as a depression prevention target. Finally, we integrate these findings and propose possible models of PVS development in which single variables have relatively modest effects, but combinations of genetic and environmental factors converge early in life to lead to sustained alterations in PVS function and/or sensitivity of PVS to proximal stress.

2. Neural circuitry of PVS

Processing of appetitive stimuli and rewards activates a distributed network of cortical and subcortical regions. Bilateral ventral striatum (VS; including nucleus accumbens) and dorsal striatum (DS; including the caudate nucleus and putamen [see Fig. 1]) are most commonly examined in fMRI research as indicators of individual differences in PVS function, given key roles in dopaminergic pathways, reward processing, and motivated behavior (Davey et al., 2008; Nusslock and Alloy, 2017). In addition to VS and DS, positive stimuli also activate bilateral amygdala and cortical regions, including orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), medial prefrontal cortex (mPFC), and anterior insula (Berridge and Kringelbach, 2015; Liu et al., 2011; Sescousse et al., 2013). Although much of the literature on developmental contributors to these networks describes regions broadly (e.g., mPFC), ventromedial prefrontal cortex (vmPFC) has stronger connections with VS and activation during reward tasks compared to dorsomedial prefrontal cortex (see Fig. 1), which is associated with social cognition and complex cognitive processes (Bzdok et al., 2013; Eickhoff et al., 2016). Ventral portions of ACC (i.e., subgenual and pregenual) and vmPFC are thought to play a key role in regulating reward responses, decision-making, and coordinating action (Davey et al., 2008; Etkin et al., 2011; Forbes and Dahl, 2012) and are commonly examined in PVS research.

Table 3
Parenting: Overview of studies of parenting and PVS function.

First Author, Year	Predictor	PVS Measure	Sample Size (N)	Sample Gender (% Female)	Age Range or M (Years)	PVS Finding	Estimated Effect Size (r)
Child and Adolescent Studies							
Casement, 2014	Parental warmth (parent report) in childhood	BOLD response to reward anticipation	120	100	8–9 for parenting; 16 for fMRI	Low parental warmth predicted ↑ VS during reward anticipation	.33
Kawamoto, 2018	Parental presence with encouragement	RewP to reward feedback	21	52	M = 5.3 (SD = 0.2)	Parent encouragement relative to alone condition ↑ RewP to positive feedback	.24
Kessel, 2019	Exposure to natural disaster and promotion-focused parenting style	RewP to reward feedback assessed before and after natural disaster	74	51	Before disaster: 8.8–10.7; after disaster: 9.6–12.4	Lower promotion-focused parenting predicted ↓ RewP among those exposed to high disaster-related stress	.43 in youth exposed to high stress
Kopala-Sibley, 2018	Observed maternal hostility in childhood	BOLD response to reward feedback	63	81	M = 10.3 (SD = 0.9)	Greater maternal hostility predicted ↓ striatum connectivity with ventrolateral PFC	.37
Kujawa, 2015a	Observed maternal parenting, maternal/paternal report	RewP to reward feedback	344	44	M = 3.6 (SD = 0.3); M = 9.2 (SD = 0.4)	Lower maternal positive parenting and authoritative parenting predicted ↓ RewP in offspring of mothers or fathers with depression	.20–.32 in offspring with parental depression
Morgan, 2014	Observed maternal warmth	BOLD response to reward anticipation, feedback	120	0	18/24 months and 10/11 years for parenting; 20 for fMRI	Lower warmth in childhood x maternal depression predicted ↑ striatum; greater warmth in adolescence x maternal depression predicted ↓ striatum	—
Morgan, 2017	Self-reported maternal rumination, observed maternal disengagement	BOLD response to reward anticipation, feedback	122	0	6 and 10–12 parenting; 20 for fMRI	No effects on striatum	—
Quevedo, 2017	Observed parent-child attachment	BOLD response to reward anticipation, feedback	171	0	6 and 17 months for attachment; 20 for fMRI	Insecure attachment predicted ↑ striatum in reward anticipation	.30
Schneider, 2012	Maternal interpersonal affiliation (parent report)	BOLD response to reward anticipation, feedback	63	41	M = 14.2 (SD = 0.3)	Low maternal affiliation associated with ↓ striatum to reward feedback in girls	.63–.71
Tan, 2014	Observed maternal negative affect	BOLD response to peer acceptance	40	63	11–17	Greater maternal negative affect predicted ↓ VS	.44
Adult Studies							
Acevedo, 2017	Quality of childhood parenting (self-report)	BOLD response to pleasant images	14	100	18–25	Interaction between sensory processing and parenting predicted striatum	—
Holz, 2018	Observed maternal stimulation in childhood	BOLD response to reward anticipation, feedback	172	58	25	High familial risk for a psychiatric disorder: Higher maternal stimulation predicted ↑ striatum during anticipation and ↓ striatum during reward feedback	.22–.27 in high familial risk group

Note: BOLD = blood oxygen level dependent; fMRI = functional magnetic resonance imaging; PFC = prefrontal cortex; RewP = reward positivity; VS = ventral striatum.

Table 4
Early Life Stress: Overview of studies of early life stress and PVS function.

First Author, Year	Predictor	PVS Measure	Sample Size (N)	Sample Gender (% Female)	Age Range or M (Years)	PVS Finding	Estimated Effect Size (r)
Child and Adolescent Studies							
Birn, 2017	Childhood stress (child interview)	BOLD response to reward anticipation, feedback	42	46	M = 10.2 for stress; M = 20.6 for fMRI	No effect of stress on striatum	—
Casement, 2014	Peer victimization (self-report) at age 11 and 12	BOLD response to reward anticipation	120	100	16 for fMRI	No effect of earlier peer victimization on striatum	—
Casement, 2015	Adolescent stressful life events (self-report)	BOLD response to reward anticipation, feedback	157	0	15–18 for stress; 20 for fMRI	No effect of adolescent stress on striatum	—
Dennison, 2016	Childhood maltreatment (self-report and interview)	BOLD response to positive images	59	61	13–20	Maltreatment predicted ↑ striatum	—
Dennison, 2017	Emotional and material deprivation	Observed response to reward anticipation, feedback	94	49	6–19	Food insecurity predicted ↓ reward performance	.33
Goff, 2013	Early deprivation in institutions	BOLD response to happy and fearful faces	Deprivation: 38; Control: 31	Deprivation: 63; Control: 39	Children 5–10 and adolescents 11–15	↓ VS for deprivation group compared to controls in adolescents across face types	.40 in adolescents
Hanson, 2015	Emotional neglect (self-report)	BOLD response to positive feedback	106	48	11.9–15.5 for fMRI 1; 13.8–18.3 for fMRI 2	Emotional neglect associated with ↓ developmental change in VS	.21
Hanson, van den Bos, 2017	Early adversity (self-report)	Probabilistic reward learning	81	51	12–17	Early adversity predicted ↓ reward learning	.44
Kamkar, 2017	Early life events (parent report)	Probabilistic reward learning; BOLD during reward learning task	40 for learning task; 26 for fMRI	60 for learning task; 46 for fMRI	9–12	Early life events predicted ↑ reward learning and ↑ VS	.47–.54
Kessel, 2019	Exposure to natural disaster	RewP to reward feedback assessed before and after natural disaster	74	51	Before disaster: 8.8–10.7; after disaster: 9.6–12.4	No main effect of stress; parenting x stress interaction on RewP	—
Mehta, 2010	Early deprivation in institutions	BOLD response to reward anticipation, feedback	Deprivation: 12; Control: 11	45	Deprivation: M = 16.1 (SD = 0.8); Control: M = 16.0 (SD = 0.9)	Early deprivation predicted ↓ VS to reward feedback	—
Romens, 2015	Childhood public assistance (maternal report)	BOLD response to reward anticipation, feedback	123	100	16	No effect of public assistance on striatum	—
Adult Studies							
Boecker-Schlier, 2016	Early adversity (parent interview across childhood)	BOLD, ERP response to reward feedback	168	58	M = 24.5 (SD = 0.6)	Early adversity predicted ↓ striatum and ↓ ERP during reward anticipation; no effect on RewP	.26–-.29
Dillon, 2009	Childhood maltreatment (multimodal assessment)	BOLD response to reward anticipation, feedback	Maltreated: 13; Control: 29	Maltreated: 69; Control: 45	Maltreated: M = 24.6 (SD = 0.9), Control: M = 37.1 (SD = 13.8)	Childhood maltreatment predicted ↓ striatum	.23
Gonzales, 2016	Neighborhood quality and socioeconomic status	BOLD response to reward anticipation, feedback	83	49	M = 24.4 (SD = 1.1) for fMRI	Lower neighborhood quality predicted ↑ VS	—
Hanson, 2016	Stressful life events (parent report; K through 12)	BOLD response to reward anticipation, feedback	72	0	M = 26.3 (SD = 1.1)	Cumulative life stress during childhood predicted ↓ VS	.33
Hanson, Knodt, 2017	Childhood maltreatment; recent life events (self-report)	BOLD response to positive feedback	926	—	18–22	Childhood maltreatment and recent events interacted to predict VS-mPFC connectivity	.20 (recent life stress)

Note: BOLD = blood oxygen level dependent; ERP = event-related potential; fMRI = functional magnetic resonance imaging; K = kindergarten; mPFC = medial prefrontal cortex; RewP = reward positivity; VS = ventral striatum.

Table 5
Proximal Stress: Overview of studies of proximal stress and PVS function.

First Author, Year	Predictor	PVS Measure	Sample Size (N)	Sample Gender (% Female)	Age Range or M (Years)	PVS Finding	Estimated Effect Size (r)
Child and Adolescent Studies							
Gaffrey, 2018	Frustration task	BOLD response to reward feedback	52	54	M = 6.0 (SD = 0.7)	Trend for association between cortisol response to stress and striatum activation	—
Lincoln, 2019	Social rejection and performance feedback	BOLD response to reward feedback	40	75	12−14	Post-stress ↓ striatum to wins compared to pre-stress	—
Adult Studies							
Admon, 2013	Military service	BOLD response to reward anticipation, feedback	24	50	18	Post-military service VS to reward feedback predicted PTSD symptoms	—
Banis, 2012	Noise stressor	RewP to reward feedback	32	0	18−28	Stress ↓ RewP	—
Banis, 2014	Noise stressor	RewP to reward feedback	61	61	18−40	Stress ↓ RewP	—
Banis, 2017	Aversive movie clips	ERP response to reward	18	100	19−26	Stressor ↓ anticipatory ERP	—
Berghorst, 2013	Threat of shock	Probabilistic reward learning	100	100	18−25	Stress ↓ reward learning	—
Bogdan, 2006	Threat of electric shock, performance feedback	Probability reward learning	80	100	18−25	Stress ↓ reward learning	.24
Bogdan, 2010	Threat of electric shock, iso/val polymorphisms of MR gene (NR3C2)	Probabilistic reward learning	53	100	18−25	Stress ↓ reward learning; strongest effect for val carriers	—
Bogdan, 2011	Threat of electric shock, CRHR1 gene	RewP during probabilistic reward learning	75	100	18−25	Stress ↓ RewP and reward learning; interacted with CRHR1	—
Born, 2010	Impossible cognitive challenge	BOLD response to palatable food	9	100	18−28	Stress ↓ striatum in satiated vs. fasted condition	—
Cavanagh, 2011	Social evaluation	Probabilistic reward learning	50	52	18−25	Stress ↓ reward learning for high BIS participants	—
Ethridge, 2018	Past-year peer victimization	RewP to reward feedback	61	89	18−25	Recent peer victimization associated with ↓ RewP	.26
Ethridge, 2020	Montreal Imaging Stress Task	Delta and theta frequency bands to reward feedback underlying RewP	100	0	18−34	Stress ↓ delta magnitude but not theta magnitude	.60
Glienke, 2015	Socially-evaluated cold pressor task	RewP in reward learning task	40	0	Stress: M = 23.1 (SD = 2.7); Control: M = 25.8 (SD = 3.4)	Stress ↑ RewP; no difference in reward learning	—
Kruse, 2018	Trier Social Stress Task	BOLD response in appetitive conditioning task	56	0	Stress: M = 23.5 (SD = 3.3); Control: M = 23.8 (SD = 2.8)	Stress ↓ striatum to cues paired with reward vs. cues paired without reward	.44
Kumar, 2014	Negative performance feedback	BOLD response to reward anticipation, feedback	18	61	18−25	Stress ↓ striatum to reward feedback and ↑ striatum during reward anticipation	.56−.72
Lighthall, 2013	Cold pressor task	Probabilistic reward learning	96	50	18−85	Stress ↑ reward learning	—
Morris, 2015	Social evaluation during impossible cognitive task	Reward learning	75	100	18−47	Stress ↓ reward learning	.29
Nikolova, 2012	Final exam stress, 5-HTTLPR/rs25531 genotype	Probabilistic reward learning	70	45	M = 18.5 (SD = 0.5)	Stress ↓ reward learning only in participants with one S allele	—
Ossewaarde, 2011	Aversive movie clips	BOLD response to reward anticipation, feedback	27	100	18−25	No effect of stress on striatum	—
Pizzagalli, 2007	Self-reported perceived stress (S1); threat of shock or negative performance (S2)	Reward learning	S1: 88, S2: 80	S1: 55, S2: 100	S1: M = 22.2 (SD = 4.4); S2: M = 21.6 (SD = 2.3)	Stress ↓ reward learning	.26−.27
Porcelli, 2012	Cold pressor task	BOLD response to reward feedback	32	50	18−27	Stress ↓ striatum	.56−.62
			88	100	—		—

Table 5 (*continued*)

First Author, Year	Predictor	PVS Measure	Sample Size (N)	Sample Gender (% Female)	Age Range or M (Years)	PVS Finding	Estimated Effect Size (r)
Treadway, 2017	Cognitive challenge and social evaluation	BOLD response, reward prediction error				No main effect of stress on VS; increase in IL-6 following stress predicted ↓ VS	
van Leeuwen, 2019	Trier Social Stress Task	BOLD response to reward anticipation, feedback	74 (36 siblings of schizophrenia patients)	0	Group means from 32.6−35.4	Stress ↑ striatum to reward feedback in healthy controls only	—
Wei, 2013	Exposure to earthquake	BOLD response in monetary donation task	30	47	19−25	Exposure to earthquake ↓ VS	.37−43

Note: BOLD = blood oxygen level dependent; CRHR1 = corticotropin releasing hormone receptor 1; ERP = event-related potential; IL-6 = Interleukin 6; PTSD = post-traumatic stress disorder; RewP = reward positivity; S1 = Study 1; S2 = Study 2; VS = ventral striatum.

In addition to fMRI, individual differences in PVS activation are reliably measured at the neurophysiological level using ERPs derived from the electroencephalogram (EEG), which are characterized by high temporal but limited spatial resolution and easily assessed across development. Particularly relevant ERP components for assessing PVS function include the reward positivity (RewP) and late positive potential (LPP; see Fig. 1). Longitudinal evidence indicates that both RewP and LPP are reliably elicited in response to reward feedback and pleasant stimuli, respectively (Kujawa et al., 2018; Pegg et al., 2019). RewP, which is thought to reflect reinforcement learning processes (Holroyd and Coles, 2002), appears as a relative positivity in the ERP wave approximately 300 ms after reward or positive feedback compared to loss or neutral feedback. RewP is also referred to as a feedback negativity, which presents as a more negative deflection the ERP wave for loss feedback compared to win feedback. In monetary reward tasks, this component appears to be more accurately described as a positivity for wins and is consistently identified across development (Kujawa et al.,

2018). Combined ERP-fMRI studies have linked RewP to activation in reward-related brain regions including VS, vmPFC, midcingulate and ACC (Becker et al., 2014; Carlson et al., 2011). LPP is a later, more sustained positivity in the ERP wave beginning around 400 ms after stimulus onset that indexes motivated attention towards salient information and is enhanced in response to *both* pleasant and unpleasant stimuli (Cuthbert et al., 2000). LPP is thought to primarily be generated by activation of visual processing regions, but has also been linked to a broad neural network including subcortical regions like the amygdala, and cortical regions, such as OFC, insula, and mPFC (Liu et al., 2012; Sabatinelli et al., 2013).

Reduced activation of VS/DS and reduced RewP and LPP in response to reward and positively-valenced stimuli have consistently been linked to depressive symptoms in both youth and adults. Critically, there is also evidence that these neural indicators of PVS function are associated with *risk* for depression (for reviews, Keren et al., 2018; Kujawa and Burkhouse, 2017; Proudfit et al., 2015). Reduced activation of VS and
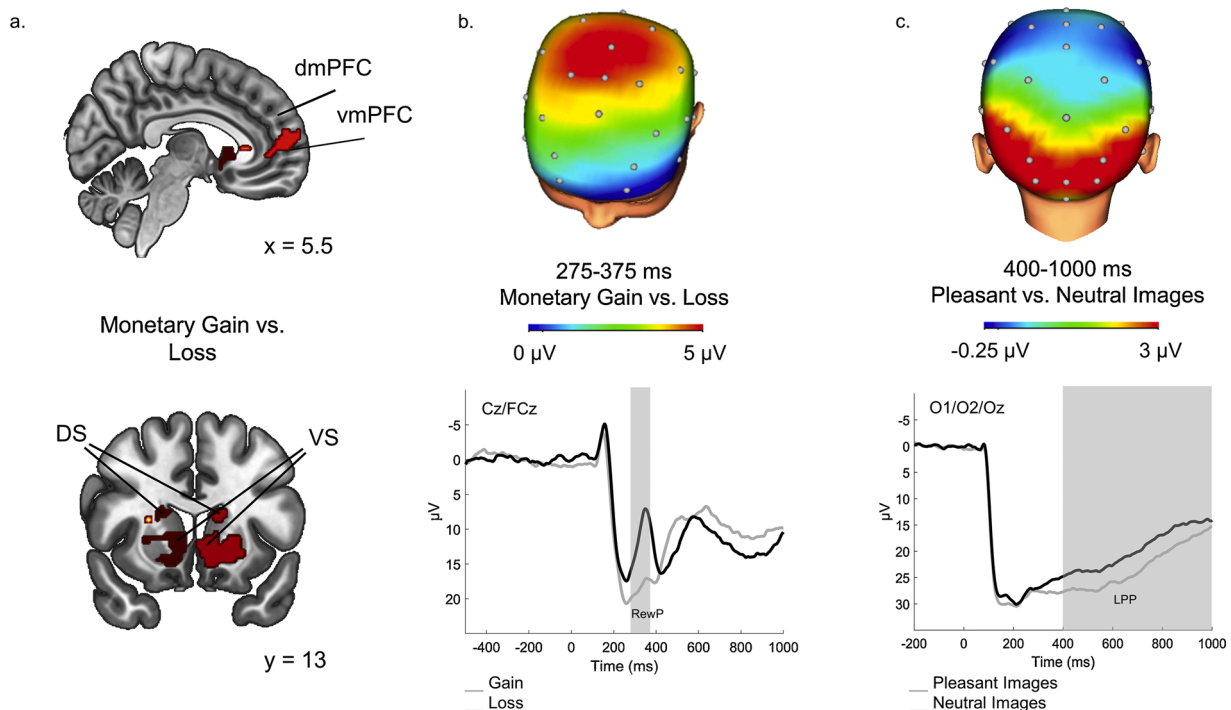

Fig. 1. Examples of neural measures of PVS: a. Activation of ventromedial prefrontal cortex (vmPFC), ventral striatum (VS), and dorsal striatum (DS) in response to monetary gain vs. loss feedback during an fMRI monetary incentive delay task. Dorsomedial prefrontal cortex (dmPFC) is marked for comparison, but is less robustly activated to monetary reward feedback. b. Scalp distribution and event-related potential (ERP; negative up) depicting the reward positivity (RewP) in response to monetary gain vs. loss feedback in a guessing task. c. Scalp distribution and ERP depicting the late positive potential (LPP) in response to pleasant images (e.g., cute animals, children having fun) vs. neutral images.

blunted RewP and LPP have been observed in youth at high risk for depression due to parental history prior to the emergence of symptoms (Kujawa et al., 2014a, 2012; Nelson et al., 2015; Olino et al., 2014; Sharp et al., 2014). Moreover, each of these indicators of PVS function have been shown to prospectively predict the development of psychiatric symptoms in children and adolescents, particularly in combination with stress (Bress et al., 2013; Kujawa et al., 2019b, 2016; Morgan et al., 2013; Nelson et al., 2016; Stringaris et al., 2015).

The current review focuses on how reduced PVS activation—primarily as assessed by these measures—emerges across development. It is important to note that in contrast to VS/DS and RewP/LPP, *increased* activation of vmPFC and ACC to rewards has been associated with depression, although less consistently (for reviews, Forbes and Dahl, 2012; Zhang et al., 2013). Interactions between the VS/DS and vmPFC/ACC are likely key to shaping PVS function and depression risk (Forbes and Dahl, 2012), but the role of broader PVS-related networks in depression risk is less clearly defined than that of the striatum. For this reason, our summaries of neuroimaging results in Tables 1–5 focus primarily on results of studies testing activation of VS/DS, although we also address evidence of associations between our predictors of interest and other PVS-related regions, as well as behavioral measures.

3. Typical development of PVS across levels of analysis

Brain circuits underlying PVS undergo dramatic developmental changes from childhood through adolescence and into adulthood, although specific developmental trajectories differ across levels of analysis and networks. Adolescence is characterized by increased activation of subcortical regions involved in emotional processing and motivation, including the amygdala and striatum (Casey et al., 2016; Galván, 2013; Shulman et al., 2016), and strengthening of connections between subcortical regions, which is thought to facilitate later refinement of connections with cortical regions (Casey et al., 2016). Cortical networks, including both medial and lateral prefrontal cortex (PFC), are involved in cognitive control and self-regulation and continue to develop and mature into adulthood (Shulman et al., 2016), with the strengthening of connections between subcortical and cortical regions facilitating top-down regulation of affective responses (Casey et al., 2016). Consistent with this, there is evidence from animal and human studies of peaks in dopamine function in adolescence compared to childhood and adulthood (Telzer, 2016; Wahlstrom et al., 2010), and meta-analytic evidence supports heightened activation of VS, DS, and amygdala to rewards in adolescence compared to adulthood (Silverman et al., 2015). This adolescent-specific peak in VS activation is also observed when considering developmental changes from childhood to adolescence and is most apparent in response to reward feedback (Braams et al., 2015; Cohen et al., 2010; Ernst et al., 2005; Galván et al., 2006; Somerville et al., 2011; Van Leijenhorst et al., 2009). Although some studies have also found support for increased activation of the striatum during reward *anticipation* in middle or late adolescence (Geier et al., 2010; Lamm et al., 2014; Van Leijenhorst et al., 2009), these findings are more mixed than evidence for developmental change during the feedback stage of processing (Shulman et al., 2016), and others have found reduced striatal activation in adolescents compared to adults during reward anticipation (Bjork, 2004; Bjork et al., 2010). Discrepancies in the literature may be due in part to methodological differences, a lack of specificity in describing regions and defining adolescence, and a focus on chronological age rather than pubertal development (Galván, 2010; Pfeifer and Allen, 2016). Critically, heightened reward responsiveness in adolescence is thought to underlie risky decision-making and impulsivity, particularly in social and emotional contexts (Blakemore and Robbins, 2012), but also play a key role in healthy social development and adjustment, facilitating prosocial behavior and pursuit of goals and activities (Crone and Dahl, 2012; Pfeifer and Allen, 2012; Telzer, 2016).

There is more limited evidence for a comparable developmental trajectory of RewP and LPP. Several studies have failed to find significant developmental changes in RewP from childhood to adolescence or adolescence to adulthood (Kujawa et al., 2018; Lukie et al., 2014; Santesso et al., 2011), although others have found evidence of a relatively enhanced (Hämmerer et al., 2011) or reduced (Kujawa et al., 2019a; Zottoli and Grose-Fifer, 2012) RewP in adolescents compared to younger children or adults. Variability in timing of assessments and cross-sectional vs. longitudinal designs may contribute to mixed results. Consistent with this possibility, a recent longitudinal study of a large sample of 8- to 14-year-olds found an increase in RewP magnitude from late childhood to early adolescence but not into later adolescence (Burani et al., 2019). Several studies have examined developmental change in LPP to happy faces or pleasant images. There is some evidence that the magnitude of LPP decreased from childhood to adolescence, although similar patterns have been observed across emotional and neutral stimuli (Kujawa et al., 2013; MacNamara et al., 2016). Developmental changes in LPP may be best characterized by shifts in the scalp topography of responses from more occipital distributions in childhood to centroparietal into adolescence and adulthood, rather than increasing or decreasing activation of PVS specifically (Pegg et al., 2019). RewP and LPP reflect electrophysiological responses in broad neural networks (e.g., Becker et al., 2014; Liu et al., 2012), which may include regions with distinct developmental trajectories. Compared to neuroimaging measures with high spatial resolution, ERP measures of PVS function may be less sensitive to distinct developmental trajectories of specific brain regions and potentially more stable across development.

Most studies of the development of PVS function rely on chronological age, with limited precision for defining specific windows. Further, youth of the same age vary considerably in pubertal development, which may partly account for mixed findings concerning developmental change (Pfeifer and Allen, 2016). There is considerable evidence from animal studies that dopamine systems are sensitive to circulating gonadal hormones (Caldú and Dreher, 2007), and a review of the literature on pubertal development and brain function in humans supported the possibility that hormonal changes correspond with increases in activation of PVS-related brain systems, although effects of pubertal stage are less consistent (Vijayakumar et al., 2018). Heightened PVS function in adolescence is likely driven in part by increases in testosterone and estradiol during this period (Braams et al., 2015; Op de Macks et al., 2017; Op De Macks et al., 2011). Although similar patterns have been observed in both boys and girls (Op De Macks et al., 2011), there is also evidence of sex differences in associations between testosterone level and activation of the striatum during reward anticipation. For example, one study found that higher levels of testosterone were related to increased DS response during reward anticipation in boys, with the inverse observed in girls (Forbes et al., 2010).

In considering the development of PVS function, it is crucial to also consider context and variability across reward domains. Heightened reward responsiveness in adolescence is thought to depend heavily on the context and is particularly apparent in social or emotional situations (Blakemore and Robbins, 2012; Crone and Dahl, 2012; Telzer, 2016). Consistent with this, adolescents exhibit more of a preference for immediate rewards when with peers vs. alone (O'Brien et al., 2011) and enhanced activation of VS during a driving simulation task when observed by peers vs. alone (Chein et al., 2011). Reward responsiveness has also been shown to be heightened as a function of perceived control over outcomes (Mühlberger et al., 2017). Further, adolescence is thought to be a time of change in social reward systems, particularly increasing salience and importance of peer relationships (Crone and Dahl, 2012; Davey et al., 2008; Silk et al., 2012). Most research on reward-related brain function across development relies on monetary reward tasks, but predictors and trajectories of PVS function may vary depending on the reward domain. Comparable networks are thought to underlie responses to social rewards, like peer acceptance (e.g., Ethridge et al., 2017; Olino et al., 2015), but little research to date has examined developmental trajectories and predictors of neural responses to social

reward specifically.

4. Developmental contributions to reduced activation of PVS

4.1. Genetics

Although self-report and behavioral measures of constructs related to PVS, such as extraversion and positive emotionality, appear to be at least moderately heritable (Borkenau et al., 2001; Pedersen et al., 1988), only a handful of family and twin studies have examined familial aggregation and genetic influence of neural measures of PVS (Hess et al., 2016). Importantly, the heritability of positive emotionality/extraversion changes over development. There appears to be minimal genetic influence in infancy (Planalp et al., 2017), with increasing heritability through age 30, and then declining effects through the rest of the lifespan (Kandler, 2012). The heritability of neural indicators of PVS function may also change across development, but this possibility has yet to be examined.

Twin and family studies support the possibility that PVS function at the neural level is modestly to moderately heritable. For example, one study of adolescent monozygotic twin pairs found correlations between twins in activation of VS/DS to reward (Silverman et al., 2014), but could not separate genetic from environmental effects. One ERP study indicated that RewP was moderately correlated between pairs of siblings (Weinberg et al., 2015a). A similar pattern was observed with an association between RewP in fathers and RewP in children, although RewP in mothers and children were negatively correlated (Moser et al., 2018). Only one study has compared mono- and dizygotic twins on a neural measure of PVS functioning, reporting that sustained attention to pleasant images, as indicated by LPP, exhibited moderate heritability (Weinberg et al., 2015b). Consistent with evidence at the neural level, one study indicated that performance-based reward learning was moderately heritable in a sample of 35 twin pairs (Bogdan and Pizzagalli, 2009).

A larger literature has examined effects of genes on PVS function (Hess et al., 2016), primarily using candidate gene approaches, which are often underpowered, susceptible to false positive findings, and ignore genes that are not assumed on a priori grounds to be associated with PVS (Flint and Kendler, 2014; Hess et al., 2016). Genome-wide association studies (GWAS) have replaced the candidate gene approach as an unbiased strategy for identifying links between gene variants and phenotypes. GWAS indicate that, for most traits, single genes account for very small amounts of variance. For example, a meta-analysis of GWAS of self-reported extraversion identified just one significant single nucleotide polymorphism with a sample of over 63,000 participants (Van den Berg et al., 2016). We are aware of only one study that has applied GWAS to a neural measure of PVS. A region on the vacuolar protein sorting-associated protein 4A (VPS4A) gene, which encodes an ATPase involved in trafficking of G protein-coupled receptors including dopamine receptors, was associated with decreased VS/DS activation during reward anticipation (Jia et al., 2016). In another GWAS study, an association of self-reported positive emotionality with a single-nucleotide polymorphism at rs322931 on chromosome 1 emerged, and the minor allele was subsequently associated with greater VS activation to positive stimuli (Wingo et al., 2017). However, in a replication and extension, the minor allele was associated with *reduced* VS activation in response to pleasant images, but *increased* VS activation to rewards (Lancaster et al., 2017).

Although a number of candidate genes have been tested for a potential role in PVS, the largest literature is on dopamine-related genes, which we focus on in this review. Although some relatively consistent findings emerge across these studies, most studies are limited to relatively small samples of adults (as small as 16, with only a few studies of 100 or more participants, see Table 1 for details). Several studies of the dopamine transporter (DAT) gene DAT1 have reported links with neural measures of PVS (Aarts et al., 2010; Dreher et al., 2009; Forbes et al.,

2009; Heitland et al., 2012; Wittmann et al., 2013). Most of these studies have found evidence of *enhanced* PVS function in those with the 9-repeat allele (Aarts et al., 2010; Dreher et al., 2009; Forbes et al., 2009; Heitland et al., 2012), although one study found enhanced VS activation in 10-repeat homozygotes (Wittmann et al., 2013). Some studies have failed to find main effects of DAT1 on VS/DS activation or RewP (Boecker-Schlier et al., 2016; Dillon et al., 2010), and others found effects only in interaction with other genes (Yacubian et al., 2007) or self-reported reward sensitivity (Hahn et al., 2011).

There is also relatively consistent evidence of associations between the dopamine receptor DRD2 gene and activation of VS/DS to rewards (Cohen et al., 2005; Felsted et al., 2010; Forbes et al., 2009; Richter et al., 2017), with some exceptions (Peciña et al., 2013). Two studies indicated that the presence of the Taq1A A1 allele is associated with reduced PVS function, including reduced VS activation (Cohen et al., 2005; Felsted et al., 2010). Another study found links between the Taq1A A1 allele and better performance on a recognition memory following an incentive task, but only the c957 T polymorphism was related to activation of reward-related brain regions during the reward task (Richter et al., 2017). Finally one DRD2 study focused on the -141C deletion polymorphism, showing greater VS activation to rewards in adults with this polymorphism (Forbes et al., 2009). Thus, despite relatively consistent links between variants of the DRD2 gene and indicators of PVS, associations with specific alleles or polymorphisms vary across studies.

Several studies have also found associations between the catechol-O-methyltransferase (COMT) polymorphism and indicators of PVS, including activation of VS or DS and RewP (Camara et al., 2010; Dreher et al., 2009; Foti and Hajcak, 2012; Marco-Pallarés et al., 2009; Schmack et al., 2008; Yacubian et al., 2007), and another study found an interaction between COMT and the substance sulpiride, which increases dopamine release (Mueller et al., 2014a). Most studies support associations between the Met allele and greater PVS function (Dreher et al., 2009; Foti and Hajcak, 2012; Schmack et al., 2008; Yacubian et al., 2007). Two studies found evidence of enhanced PVS function in those with the Val allele, but these effects were primarily driven by responses to loss (Camara et al., 2010; Marco-Pallarés et al., 2009). Others have failed to find significant effects of COMT on VS/DS activation or RewP (Aarts et al., 2010; Baker et al., 2016; Dillon et al., 2010; Forbes et al., 2009; Heitland et al., 2012). One study simultaneously recorded fMRI and ERP, and did not find main effects of COMT on VS activation or the RewP (Boecker-Schlier et al., 2016). Interestingly, however, a history of early family adversity was positively associated with VS activation to reward feedback in those with the Met/Met genotype (Boecker-Schlier et al., 2016).

Finally, although one study observed an association between the 7-repeat allele of DRD4 and greater VS activation (Forbes et al., 2009), most have not documented links between DRD4 and VS activation or ERP measures of PVS function (Baker et al., 2016; Boecker-Schlier et al., 2016; Camara et al., 2010; Marco-Pallarés et al., 2009).

Although dopamine genes appear to be most consistently related to activation of the striatum in reward tasks, some fMRI studies have found associations between candidate genes involved in dopamine function and activation of other PVS-related brain regions. For example, the DRD2 A1 allele has been associated with reduced activation of OFC and amygdala to rewards (Cohen et al., 2005; Felsted et al., 2010). Further, the Val allele of COMT has been linked to greater relative activation of mPFC to large unexpected rewards compared to losses (Camara et al., 2010).

Although effects of single genes on PVS function are likely to be small in magnitude, associations may increase in magnitude as a function of the number of susceptibility genes an individual possesses. One study found a positive association between a composite score of the DAT1 9-repeat allele and COMT Met allele and DS activation to rewards (Dillon et al., 2010). Two studies examined multilocus composites of DA-related SNPs and associations with PVS function (Nikolova et al.,

2011; Stice et al., 2012). Results were relatively consistent across these two studies, although there were some notable distinctions in coding of composite scores. Specifically, Nikolova et al. (2011) tested a composite linked to high dopamine signaling (DAT 9-repeat, DRD4 7-repeat, DRD2-141C deletion, Taq1A A2, and COMT Met), while Stice et al. (2012) tested a composite linked to low dopamine signaling (TaqIA A1, DRD2 -141C Ins/Ins, DRD4 7-repeat or longer, DAT 10-repeat, and COMT Met), with DRD4 and COMT coded in opposite directions across these two studies. Although effects for individual single-nucleotide polymorphisms were generally non-significant in both studies, composite genetic measures were significantly associated with VS or DS response to reward, such that composites associated with low dopamine signaling were related to lower PVS activation (Nikolova et al., 2011; Stice et al., 2012).

Overall, it appears that neural measures of PVS function are moderately heritable, but few genetically-informative behavior genetic studies and GWAS are available. Although there is evidence of associations between several candidate genes, particularly DAT, DRD2, and COMT, as well as multilocus composites for dopamine signaling, it is critical that future work goes beyond candidate gene studies by conducting well-designed twin and adoption studies and adequately powered GWAS analyses of neural measures of PVS function across development.

4.2. Temperament

Temperament refers to early-emerging dispositions that are partly biogenetic in origin and become more complex and elaborated with development, influencing cognitive and interpersonal styles (Caspi and Shiner, 2006). We conceptualize temperament traits as influencing subsequent neural reactivity to reward and appetitive stimuli, which then in turn shape vulnerability for psychopathology. However, the association between temperament and PVS function at the neural level is likely complex and bidirectional. To some degree, traits manifested through behavior and self-report may reflect similar phenomena to neural measures, viewed at different levels of analysis.

All prominent models of temperament and personality include a PVS-relevant trait dimension such as extraversion or positive emotionality (Caspi and Shiner, 2006) that exhibits impressive continuity from early childhood through adulthood (Roberts and DelVecchio, 2000) and is particularly relevant when considering developmental trajectories of PVS function and is the focus of our review. Although a few studies have examined behavioral observations of temperament in early childhood, most studies testing associations between extraversion or positive emotionality and neural measures of PVS rely on self-report measures in adults. We focus on the traits of extraversion and positive emotionality, which have a very high degree of conceptual and empirical overlap (Watson et al., 2006), but also review studies of self-reported behavioral activation system (BAS) and reward sensitivity, which are conceptually and empirically related to extraversion and positive emotionality (Carver and White, 1994; Torrubia et al., 2001). However, unlike a previous review (Hess et al., 2016), we do not include Cloninger's temperament model, as his reward dependence and novelty seeking scales do not show good convergent and discriminant associations with other widely-used measures of extraversion and positive emotionality (De Fruyt et al., 2000).

Our review of the literature identified five fMRI studies that examined the relationship of positive emotionality-related constructs with VS activation to reward in adults. As predicted, lower self-reported extraversion/positive emotionality and reward sensitivity is associated with reduced VS activity during reward anticipation (Hahn et al., 2011; Wu et al., 2014) and to reward feedback (Cohen et al., 2005; Simon et al., 2010). One fMRI study examined conditioning to images reinforced by monetary reward, and did not find an association between extraversion and appetitive conditioning in VS but did observe positive associations with activation of hippocampus and thalamus (Schweckendiek et al.,

2016). Extending beyond the striatum, there is also evidence of positive associations between extraversion and medial OFC and amygdala activation to reward feedback (Cohen et al., 2005; Simon et al., 2010).

With regard to ERPs to reward, lower self-reported extraversion and BAS has been associated with reduced RewP in adult samples (Cooper et al., 2014; Lange et al., 2012; Mueller et al., 2014b; Smillie et al., 2011). In the longitudinal Stony Brook Temperament Study, we observed that lower positive emotionality in preschoolers modestly predicted reduced RewP in later childhood (Kujawa et al., 2015a). Interestingly, self-reported positive emotionality was also cross-sectionally associated with RewP in later childhood, although observed and self-reported positive emotionality were not associated with each other. Finally, a study of adolescent girls reported that neuroticism moderated the association between positive emotionality and RewP, with positive emotionality positively associated with RewP, but only at lower levels of neuroticism (Speed et al., 2018). Findings of links between individual differences in positive emotionality, extraversion, and BAS with PVS function at the neural level are complemented by a behavioral study indicating that lower BAS scores predicted lower effort to obtain reward in a low probability of reward condition (Geaney et al., 2015).

Despite relatively consistent evidence of effects of positive emotionality-related constructs on brain function in the context of monetary reward, studies of VS activation to other appetitive stimuli provide less compelling support. Two small fMRI studies reported that extraversion or BAS were positively associated with VS activation to appetitive stimuli (Beaver, 2006; Canli et al., 2001), but others failed to find significant associations between positive emotionality-related constructs and VS activity to pleasant stimuli (Kehoe et al., 2012; Mobbs et al., 2005; Rapp et al., 2008; Suslow et al., 2010), and two studies reported inverse associations between extraversion and VS reactivity to amusing films (Hutcherson et al., 2008) and chocolate (Schaefer et al., 2011). Several studies have reported correlations between extraversion and activation of other PVS-related brain regions, including increased activation in ventral portions of ACC to positive words (Haas et al., 2006) and increased ventrolateral PFC and right OFC activation to humorous cartoons (Mobbs et al., 2005).

At the neurophysiological level, fairly consistent evidence emerges for associations between positive emotionality and related constructs and processing of appetitive images. In a sample of young adults, positive emotionality was correlated with LPP to rewarding images (Weinberg and Sandre, 2018). Across development, extraversion in adolescent girls was positively correlated with LPP to both positive and negative images (Speed et al., 2015), and laboratory observations of positive emotionality in 6-year-olds predicted an enhanced LPP to pleasant images in later childhood (Kessel et al., 2017).

Although positive emotionality-related traits have the strongest conceptual links to neural indicators of PVS function, there is a smaller literature linking negative emotionality and behavioral inhibition, characterized by wariness in novel situations, to *heightened* PVS function (Bar-Haim et al., 2009; Guyer et al., 2006; Lahat et al., 2018; Schaefer et al., 2011), with one study indicating that individual differences in neuroticism moderated the effects of positive emotionality on RewP to rewards in adolescent girl (Speed et al., 2018).

In summary, there is consistent evidence of an association between positive emotionality-related traits and sensitivity to reward feedback in fMRI and ERP, although results for reward anticipation are less consistent. Findings of ERP studies indexing attention to appetitive stimuli also support a relationship with extraversion/positive emotionality, but studies using fMRI are predominantly negative. Positive emotionality-related traits may be specifically associated with reward responsiveness subconstructs of PVS. This literature is limited primarily to cross-sectional studies of adults, making it difficult to draw conclusions about the direction and development of these associations. Two studies using observational measures of temperament in young children have shown that positive emotionality predicts neural measures of PVS

function later in development (Kessel et al., 2017; Kujawa et al., 2015a), which provide the strongest support for the role of temperamental emotionality in shaping neural indicators of PVS function across development.

4.3. Parenting

In addition to genetics and temperament, developmental trajectories of PVS function are likely influenced by early experiences, including variability in parenting style and behaviors. Low positive parenting, including lack of support, warmth, and structure, is one possible factor contributing to low PVS function. Consistent with this, in one fMRI study, adolescent girls of mothers low in affiliation showed *decreased* VS activation to reward feedback (Schneider et al., 2012). However, two other fMRI studies found *increased* activation of VS or DS during reward anticipation in adolescents and adults experiencing insecure attachment styles and low parental warmth in early life (Casement et al., 2014; Quevedo et al., 2017).

Although more limited, there is some evidence that negative parenting behaviors may also shape PVS function. One study found that greater maternal negative affect during a mother-adolescent interaction task predicted reduced VS activation to social reward in adolescents (Tan et al., 2014). On the other hand, at the neurophysiological level, there is evidence of specificity for low positive parenting rather than negative parenting in predicting RewP (Kujawa et al., 2015b), which is consistent with experimental work indicating that parental presence and encouragement enhances RewP in young children compared to completing a task alone (Kawamoto and Hiraki, 2018).

Other fMRI studies have identified effects of parenting on VS connectivity and broader reward-related brain networks, rather than activation of VS or DS. For example, in a longitudinal study, observed maternal hostility in early childhood predicted decreased VS connectivity with ventrolateral PFC during a reward task later in childhood (Kopala-Sibley et al., 2020). A second study indicated that maternal rumination and disengagement interacted to predict decreased ventral ACC response, but not VS activation, to reward feedback in adult offspring (Morgan et al., 2017).

Specific effects of parenting on PVS activation likely depend on interactions with genetic predispositions (e.g., Richards et al., 2016), other risk factors, and developmental timing. Consistent with this, effects of parenting style on PVS measures have been shown to be moderated by offspring temperament (Acevedo et al., 2017) and exposure to proximal and acute stressors (Kessel et al., 2019). In addition, effects may be strongest among those at high risk for depression or other forms of psychopathology. A longitudinal study showed that low positive parenting in early childhood predicted a blunted RewP in later childhood only among offspring of parents with a history of depression (Kujawa et al., 2015b). Similar patterns were observed in a longitudinal fMRI study of young men from low-income families, though effects depended on developmental timing. Lower maternal warmth experienced in very early childhood predicted *greater* VS response during reward processing in offspring of depressed mothers, but lower maternal warmth in early adolescence predicted *reduced* VS activation (Morgan et al., 2014). Effects may also differ depending on stage of reward processing. For example, a recent study of young adults followed since birth indicated that among those with a family history of psychiatric disorders, greater maternal stimulation of offspring in infancy predicted *increased* DS activation during reward anticipation but *decreased* activation during reward feedback (Holz et al., 2018).

Taken together, there is evidence to support associations between parenting and PVS activation. However, directions of results vary across studies and depend on interactions with other risk factors, particularly family history of psychopathology, and developmental timing. Given evidence of genetic contributions to PVS function, associations between parenting and PVS function may reflect both heritable affective styles and aspects of the home environment.

4.4. Stress

Stress is often considered to be a key contributor to reduced PVS function, with effects thought to be mediated by elevated inflammatory cytokines and alterations in dopamine function (for reviews, Nusslock and Miller, 2015; Pizzagalli, 2014). Indeed, a relatively large literature has examined associations between naturalistic and lab-induced stress on PVS. Although it is clear that stress can have profound effects on PVS function, the direction of effects varies across studies, likely as a function of the type of stressor, developmental timing, and measure of PVS. Here, we evaluate the evidence for effects of stress on PVS function, reviewing research on effects of stress early in life, as well as associations between proximal stressors and PVS activation in adolescents and adults.

4.4.1. Early life stress

We found five studies that presented evidence that childhood adversity and stress predict *reduced* activation of VS or DS to reward anticipation or feedback in adolescence or adulthood (Boecker-Schlier et al., 2016; Dillon et al., 2009; Hanson et al., 2016, 2015; Mehta et al., 2010), and one study with evidence of a general reduction in VS activation to faces among adolescents exposed to early adversity (Goff et al., 2013). At the same time, others have indicated that early life stress predicts *increased* activation of the striatum to rewards (Dennison et al., 2016; Gonzalez et al., 2016; Kamkar et al., 2017). Patterns of blunted VS/DS activation more consistently emerge among adolescents and adults exposed to severe and prolonged childhood adversity, including institutionalization, emotional neglect, and maltreatment (Dillon et al., 2009; Hanson et al., 2015; Mehta et al., 2010), although there are exceptions (e.g., Dennison et al., 2016). Similar patterns also emerge for cumulative life stress (e.g., moves, deaths, parental divorce) assessed repeatedly across development (Boecker-Schlier et al., 2016; Hanson et al., 2016), which may more sensitively detect level of total stress exposure. On the other hand, less direct or severe stress, including lower neighborhood quality and normative experiences assessed at a single time point, have been shown to predict *increased* activation of VS during reward anticipation in adulthood (Gonzalez et al., 2016; Kamkar et al., 2017).

We also found five studies that failed to find effects of cumulative life events, peer victimization earlier in life, and poverty in childhood or adolescence on later VS/DS activation to reward, but observed associations with other PVS-related brain regions (Birn et al., 2017; Casement et al., 2015, 2014; Romens et al., 2015). Several studies have implicated alterations in mPFC activation to reward anticipation following stress exposure (Casement et al., 2014, 2015; Romens et al., 2015), although the direction of associations between stress and mPFC activation varies, potentially due to the specific type of stress. Beyond activation, functional connectivity studies have the potential to inform understanding of the effects of stress on reward networks. In a large sample of young adults, the combination of self-reported childhood maltreatment and recent life stress predicted increased VS-mPFC connectivity to reward (Hanson et al., 2017a).

At the neurophysiological level, one study indicated that family adversity in childhood predicted a blunted ERP in anticipation of reward (i.e., contingent negative variation [CNV]) in adulthood but had no significant effect on RewP (Boecker-Schlier et al., 2016). At the behavioral level, childhood exposure to maltreatment or food insecurity has been shown to predict poorer performance on reward tasks (Dennison et al., 2017; Hanson et al., 2017b), but one study indicated that greater normative childhood stress predicted *enhanced* reward learning in late childhood (Kamkar et al., 2017). Deficits in reward learning may emerge only in response to more severe, prolonged, or uncontrollable early life experiences. Further, specific manifestations of stress exposure may depend both on the developmental timing of exposure and the window in which PVS is assessed.

Taken together, evidence across levels of analysis indicates that exposure to stress in early childhood alters development of PVS

function, but the directions of effects are inconsistent and likely depend on the type of exposure. Consistent with this, animal models indicate that prolonged and uncontrollable stress inhibits dopamine function and reward behavior (Cabib and Puglisi-Allegra, 1996; Pizzagalli, 2014). In addition to chronicity and type of stress, there is support for effects of developmental timing of stress, with some evidence that exposure to stress in childhood may have stronger effects on PVS function than stress experienced later in life (Boecker-Schlier et al., 2016; Hanson et al., 2015). It is possible that stress exposure in adolescence may have stronger effects on cortical regions involved in regulation of emotional responses, as well as connectivity between cortical and subcortical regions, due to the timing of development of these networks (Casey et al., 2016; Spear, 2000).

4.4.2. Proximal stress

In contrast to the mixed findings for early life stress, a relatively robust and consistent literature has demonstrated a blunting effect of laboratory-induced stress (typically uncontrollable) on PVS function in adults. Laboratory studies have employed psychosocial stressors including social evaluation (Cavanagh et al., 2011; Kumar et al., 2014; Morris and Rottenberg, 2015; Treadway et al., 2017), noxious stimuli (Banis et al., 2014; Banis and Lorist, 2017, 2012; Ossewaarde et al., 2011), and painful stimuli (Berghorst et al., 2013; Bogdan et al., 2010; Bogdan and Pizzagalli, 2006; Lighthall et al., 2013; Porcelli et al., 2012). Neuroimaging studies largely find decreased VS or DS activation to reward feedback after acute stress (Born et al., 2010; Kumar et al., 2014; Lincoln et al., 2019; Porcelli et al., 2012; but also see van Leeuwen et al., 2019), but results may differ at other stages of processing. For example, one study of 18 adults showed that stress led to deactivation of the DS to reward feedback, but *enhanced* activation during reward anticipation (Kumar et al., 2014). In another study of adult men using a reward conditioning paradigm, laboratory stress was associated with less of a difference in activation of the DS to reward compared to no-reward cues (Kruse et al., 2018). In addition to effects on VS/DS activation, acute stress has been associated with differential activation of the OFC to rewards, with two studies finding *reduced* activation of OFC to rewards following stress (Born et al., 2010; Porcelli et al., 2012) and one study finding *increased* activation of OFC to rewards after stress (van Leeuwen et al., 2019).

The majority of laboratory stress studies examining ERP and behavioral measures find evidence of a blunted RewP following stress (Banis et al., 2014; Banis and Lorist, 2012; Bogdan et al., 2011; Ethridge et al., 2020), as well as a reduced CNV ERP during reward anticipation (Banis and Lorist, 2017) and reward learning impairments (Berghorst et al., 2013; Bogdan et al., 2011, 2010; Bogdan and Pizzagalli, 2006; Cavanagh et al., 2011; Morris and Rottenberg, 2015), although two studies found opposite effects (Glienke et al., 2015; Lighthall et al., 2013).

Patterns of reduced PVS function have also been observed following proximal naturalistic stressors, rather than laboratory stressors. For example, recent perceived stress, military service, peer victimization, and academic stress have been associated with decreased activation of VS or RewP to rewards (Admon et al., 2013; Ethridge et al., 2018; Wei et al., 2013) and impaired reward learning (Nikolova et al., 2012; Pizzagalli et al., 2007).

Critically, there are individual differences in the extent to which PVS function fluctuates in response to proximal stress. For example, a recent study of 4- to 6-year-old children indicated that greater cortisol responses to a laboratory stressor predicted reduced amygdala reactivity during a reward task administered at a separate assessment, although associations with activation of VS/DS did not reach significance (Gaffrey et al., 2018). Similarly, reduced reward sensitivity at the behavioral level was observed only among adults exposed to stress who were also considered high stress responders due to the magnitude of their cortisol responses (Berghorst et al., 2013).

The extant literature examining the effects of acute laboratory and proximal naturalistic stress supports the idea that proximal stress can induce at least a temporary reduction in PVS functioning in adolescents and adults. Importantly, despite the relative consistency of these effects, there is also evidence of moderators of the effects of proximal stress on PVS activation, including sex differences, genes, personality, and early life experiences (Bogdan et al., 2011, 2010; Cavanagh et al., 2011; Kessel et al., 2019; Nikolova et al., 2012), suggesting that there are individual differences in the extent to which PVS activation fluctuates as a result of proximal stress. It remains largely unexamined the extent to which reductions in PVS function following an acute stressor persist across time or whether individual differences in degree of change in PVS function in response to stress predicts psychiatric symptoms over and above a single PVS assessment.

5. Developmental trajectories of PVS function

Integrating developmental, genetic, temperament, parenting, and stress research, it becomes clear that developmental trajectories to reduced activation of PVS are complex, dynamic, and shaped by a number of biological and environmental factors. Although the literature reviewed suggests a range of contributors to PVS development, a close examination of the evidence reveals that effect sizes of single variables tend to be modest to moderate in magnitude and directions of associations can be inconsistent (see Tables 1–5). While large effect sizes are observed at times, these may be overestimates due to small samples (Button et al., 2013) or biases in selections of voxels and thresholding procedures (Vul et al., 2009). Indeed, as outlined in detail in Tables 1–5, sample sizes varied considerably across studies, with many studies relying on fewer than 20 participants. Importantly, sample sizes tend to be larger for more recent studies, indicating that more accurate estimates of effect size will continue to emerge.

Early-emerging genetic factors and biobehavioral temperament traits have the potential to exert both main and interactive effects in combination with environmental experiences to shape styles of processing and responding to positive reinforcers, alterations in which then increase vulnerability for depression and other disorders (Admon et al., 2013; Corral-Frías et al., 2015; Keren et al., 2018; Kujawa and Burkhouse, 2017). The evidence to date indicates there is not one single mechanistic pathway to reduced PVS activation. Instead, inconsistent findings emerge for even the most commonly implicated factors, like early life stress, highlighting the need for a more nuanced perspective of the emergence of PVS function considering multiple potential developmental trajectories.

Although research in young children is limited, tendencies toward reduced activation of PVS may emerge early in life as a function of genetic predispositions and biobehavioral traits such as low extraversion/positive emotionality. Indeed, there is fairly consistent evidence linking genetics and temperament to neural measures of PVS, although candidate gene approaches have limitations and must be interpreted cautiously. These early-emerging tendencies may be further exacerbated among children exposed to stress, particularly severe, prolonged, or uncontrollable stress (Boecker-Schlier et al., 2016; Dillon et al., 2009; Hanson et al., 2016, 2015; Mehta et al., 2010), and to parenting styles that are low in warmth and structure (Acevedo et al., 2017; Kujawa et al., 2015b; Morgan et al., 2014; Schneider et al., 2012). However, the specific effects of parenting style and stress on PVS function vary dramatically depending on types of experiences, developmental timing, and interactions with other variables.

It is clear from the current review that a range of factors have the potential to alter PVS function. It is less clear exactly how genes, temperament, parenting and stress affect the *course* of PVS development, and we propose three possibilities for atypical developmental trajectories of PVS function. That is, it is possible that individuals who exhibit relatively reduced PVS function at a single time point may actually be characterized by distinct developmental trajectories, which could partly account for inconsistencies in the literature reviewed above.

5.1. Chronically low PVS function

First, early-emerging factors may trigger a chronically low trajectory of PVS function (Fig. 2). Youth on this trajectory may experience a developmental increase in PVS function in adolescence (at least at some levels of analysis), but continue to show relatively reduced PVS functioning compared to those with typical developmental trajectories of PVS. A chronically low trajectory may be most apparent among those with early-emerging risk factors, including genetic predispositions, temperamental low positive emotionality, and low positive parenting in early childhood (Kessel et al., 2017; Kujawa et al., 2015b, 2015a). Few studies have examined neural indicators of PVS function in early childhood, but there is some evidence that reduced activation can emerge prior to adolescence (Belden et al., 2016; Kujawa et al., 2015b; Luking et al., 2016), raising the possibility that at least for a subset of people, chronically low PVS function may appear early in childhood and persist across development.
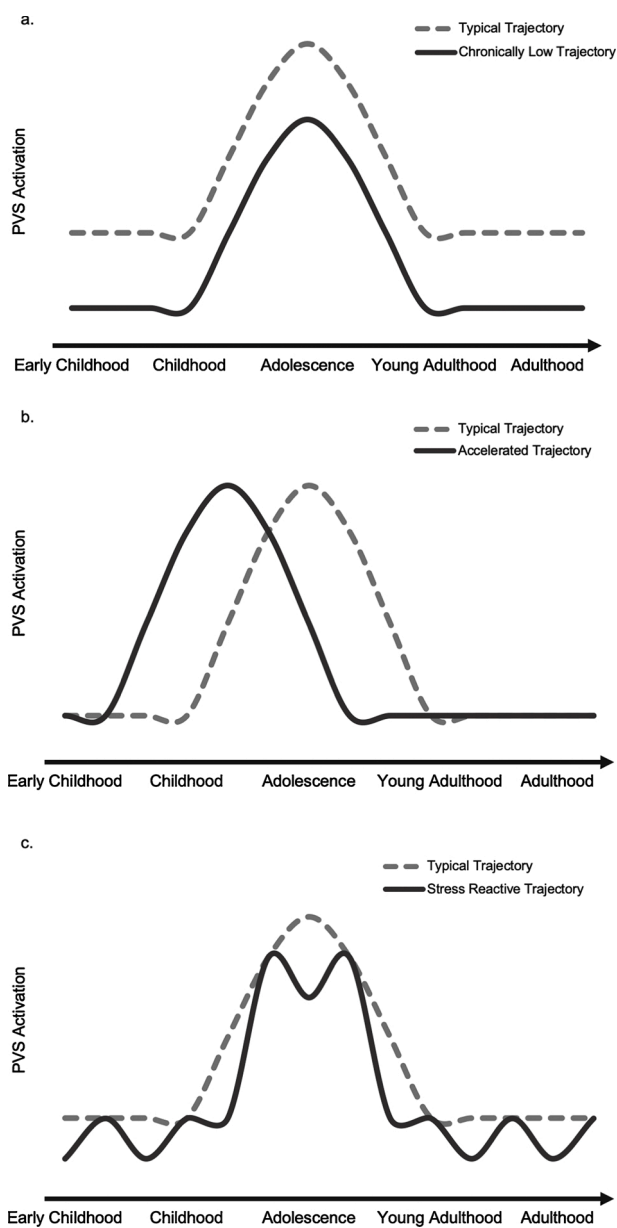
Fig. 2. Models depicting three potential developmental trajectories leading to relatively reduced PVS activation: a. chronically low, b. accelerated, and c. stress reactive.

5.2. Accelerated development of PVS function

For others, adverse experiences early in life may alter the trajectory of PVS function (Fig. 2). Accelerated development theories posit that maturation of the brain may be evolutionarily adaptive for youth in particularly adverse environments, allowing them to reach adult-like brain function at an earlier age (Callaghan and Tottenham, 2016; Tyborowska et al., 2018). As such, youth exposed to particularly severe, chronic, and/or uncontrollable stress may show a relative increase in PVS function in childhood, similar to the developmental increase commonly observed in adolescence, but shift to relatively reduced activation in adolescence and adulthood. Consistent with this trajectory, *increased* VS activation and reward learning was observed in children exposed to stress (Kamkar et al., 2017), suggesting the possibility that they exhibit more adolescent-like patterns of brain activation. In addition, young adolescents exposed to emotional neglect showed *less* of a developmental increase in VS activation across two years (Hanson et al., 2015), possibly because the experience of emotional neglect led to an earlier peak in PVS function.

5.3. Stress reactive PVS function

Finally, rather than the sustained trajectory of PVS function, genetic factors, temperament, and early experiences may shape the extent to which PVS activation fluctuates as a result of proximal stress (Fig. 2). Among the most consistent evidence for contributors to low PVS function is the effect of laboratory stress on reductions in PVS function. That is, in addition to sustained developmental change, PVS appears sensitive to stress and daily life experiences throughout adulthood, but stress may not always have large or lasting effects on PVS function. Instead, there are likely individual differences in how reactive PVS function is to stress. Studies in both young children and adults have indicated that those who showed greater cortisol responses to stress showed reduced reward reactivity at the neural and behavioral level (Berghorst et al., 2013; Gaffrey et al., 2018), and there is evidence that sex, genes, personality, and early life experiences moderate the magnitude of the effects of proximal stress on PVS function (Bogdan et al., 2011, 2010; Cavanagh et al., 2011; Kessel et al., 2019; Nikolova et al., 2012).

5.4. Overlapping trajectories

It should also be noted that these trajectories may not be fully independent. For example, people with a chronically low or accelerated trajectory may subsequently show a more stress reactive pattern of PVS function. Alternatively, certain risk factors may predispose to alterations in both the sustained trajectory and extent to which PVS function is reactive to stress. This idea is consistent with double hit models of psychopathology risk, in that early experience may shape vulnerabilities that then enhance stress reactivity later in life, with the combination of both "hits" leading to psychopathology (Koss and Gunnar, 2018). Further, there is evidence that individuals with relatively enhanced PVS function show reduced physiological and subjective stress reactivity (Ethridge et al., 2020; Heller et al., 2013; Vidal-Ribas et al., 2019). Although it is unclear whether fluctuations in PVS function underlie these physiological and subjective responses, these data support the possibility that multiple trajectories may combine in such a way that sustained trajectories of low PVS function can also predispose to increased stress reactivity.

In conclusion, PVS function at a given time point may reflect relatively stable individual differences shaped by early genetic, temperamental, and environmental factors, but also acute fluctuations in response to stress and changes in mood. Biobehavioral predispositions likely shape an individual's typical level of PVS activation, as well as developmental trajectories and reactivity to stress. Early life stress and certain parenting styles potentially have persistent effects on PVS activation, but PVS activation also shows continued fluctuations as a

function of exposure to stress across adolescence and adulthood. In this way, it may be the combination of both early and later stress that leads to the most disruption in PVS activation (Hanson et al., 2017a; Kessel et al., 2019), and greatest risk for psychopathology, particularly depression.

6. Future research priorities

Since the introduction of the RDoC initiative (Sanislow et al., 2010), considerable progress has been made in understanding the development of PVS. Yet further work is needed to clarify developmental trajectories and translational implications. First, it should be noted that we collapse across broad neuroanatomical structures and regions (e.g., VS and DS, subregions of mPFC) in our review. Yet, subregions of the striatum and mPFC have distinct functions and patterns of connectivity (e.g., Bzdok et al., 2013). More precise labeling of regions in charting developmental trajectories and comparisons across studies should be a priority for future work (Pfeifer and Allen, 2016). In addition, longitudinal studies examining PVS function beginning early in life and with repeated assessments across time are critically needed to chart developmental course. Obtaining valid and developmentally-appropriate neural and performance measures of PVS in young children is a challenge, although recent work (Belden et al., 2016; Gaffrey et al., 2018) suggests that it is possible and should be a priority. Multi-method longitudinal studies are needed to chart trajectories of PVS function across levels of analysis and examine effects of age and hormonal changes associated with puberty. Such designs should account for both individual differences at rest, as well as the magnitude and persistence of change in PVS function in response to stress. Further, there is a need to extend research on reward responsiveness to the social domain, given the salience of social relationships in adolescence and the role of interpersonal stress in the development of depression. Several tasks have been developed for measuring responses to social feedback (e.g., Jarcho et al., 2016; Kujawa et al., 2014b, 2017; Olino et al., 2015), but little work has examined developmental trajectories of neural responses to social reward.

At the genetic level, research must extend to well-designed twin and adoption studies and adequately powered GWAS. Given evidence of developmental changes in the heritability of extraversion/positive emotionality (Kandler, 2012), it is important to use longitudinal designs to study multiple points in development. For temperament, research is needed to evaluate temporal dynamics of these associations, beginning in early childhood with repeated assessments of both temperament and neural measures to compare concurrent and longitudinal relationships. EEG/ERP research provides a useful complement to fMRI research in this regard, in that these methods are relatively economically and easily applied in large samples across development and guidelines for ERP research with young children are emerging (Brooker et al., 2019).

For parenting, studies comparing specific parenting behaviors are needed to identify key factors in shaping PVS development. This is crucial as parenting may be a prime target for prevention, yet it remains unclear what types of parenting are likely to be effective in promoting healthy PVS development. For example, there is some evidence of specific links between positive parenting, rather than negative parenting, and RewP in children (Kujawa et al., 2015b), but determining whether this is driven by parental warmth or affection, structure and consistency, and/or displays of positive affect is needed for prevention. For stress, longitudinal research is needed to compare chronically low and accelerated development models of stress exposure, as well as factors that predict specific developmental trajectories. Improved understanding of how patterns observed in response to laboratory-induced stressors translate to naturalistic stress is needed. To our knowledge, no studies have examined whether effects of laboratory stressors on PVS are evident beyond the session. This makes it difficult to evaluate the extent to which such experiences lead to persistent change in PVS, as well as individual differences in the amount of time required for PVS function to return to baseline. As naturalistic stressors can be episodic or chronic,

and co-occur with other stressors, careful assessment and designs will be needed to disentangle the effects of specific stressors from other stressors occurring concurrently as well as in prior and subsequent developmental periods.

7. Clinical implications

Understanding trajectories of PVS function is essential for translating findings from clinical and affective neuroscience to prevention. There is growing evidence to suggest that low PVS function may be modifiable target for prevention. For example, treatment studies indicate that that neural indicators of PVS function may be sensitive to intervention, at least for some people (Barch et al., 2019; Burkhouse et al., 2018; Dichter et al., 2009). Further, we recently demonstrated that a brief motivational manipulation was effective in enhancing neural and behavioral indicators of PVS in a nonclinical sample of emerging adults, supporting the feasibility of targeting PVS function earlier in development to reduce risk for later psychiatric disorders (Pegg and Kujawa, 2020).

A few directions for future research will advance the translational implications of research on PVS function. First, determining the earliest point at which alterations in PVS trajectories can be reliably identified across development could allow for very early identification of youth in need of prevention. Moreover, the trajectory of PVS activity may have greater predictive value than the level of PVS activation at a single time point (e.g., Hanson et al., 2015), and, as such, the identification of both typical and atypical trajectories of change is essential. Second, identifying early factors—and combinations of factors—that shape PVS function will provide specific processes to target to promote healthy PVS development (e.g., increasing supportive parenting in families with a history of depression vs. reducing harsh parenting). Finally, examining trajectories of both baseline PVS function and stress reactivity is essential for developing interventions for youth exhibiting signs of low PVS. If chronically low PVS function is the target, interventions to increase positive affect may be most relevant (e.g., Craske et al., 2019). If sensitivity of PVS function to stress is the target, interventions to enhance ability to cope with stress may be most effective (e.g., Compas et al., 2015).

8. Conclusions

Charting typical and atypical developmental trajectories of core dimensions of emotion and behavior prior to the emergence of psychopathology is needed to understand the pathophysiology of psychiatric conditions, as well as when and how to intervene. Although progress has been made in identifying biological and environmental contributors to reduced activation of PVS, the effects of single factors appear to be relatively small and inconsistent. Greater consideration of developmental processes and interactions amongst variables is needed to advance understanding of how these patterns emerge and predispose to risk for psychopathology. Continuing to test and refine an integrated model of developmental trajectories to reduced activation for PVS will move us towards translational work to reduce the burden of these conditions.

9. Financial disclosures

Declaration of Competing Interest

None.

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