

Sample description by age group

Table S1: Participant characteristics by sample and age group.

Children			
	ASD	TD	group comparison
Total N	38	40	
Demographics			
Sex (m/f)	29/9	24/16	$\chi^2(1)=2.382, p=.123$
Age (years)	10.06 ± 1.35 (7.56 - 11.97)	10.26 ± 1.26 (7.57 - 11.98)	$t(76)=-.675, p=.502$
IQ (full IQ)	112.55 ± 13.16 (86.36 - 148.00)	110.42 ± 12.10 (76.00 - 133.00)	$t(75)=.740, p=.462$
Handedness (right/left/ambidextrous/unknown)	31/4/0/3	28/3/1/8	$\chi^2(3)=3.519, p=.318$
Medication use (no/yes/unknown)	12/14/12	20/3/17	$\chi^2(2)=9.935, p=.007$
fMRI quality control			
SID Mean framewise displacement (FD; in mm)	.18 ± .08 (.05 - .36)	.14 ± .07 (.06 - .34)	$t(76)=2.579, p=.012$
SID Volumes with FD >0.5 mm (in %)	5.49 ± 6.08 (0 - 18.24)	2.52 ± 4.10 (0 - 14.86)	$t(64.51)=2.523, p=.014$
SID Signal-to-noise ratio	11.18 ± .62 (9.96 - 12.51)	11.20 ± .84 (8.28 - 13.10)	$t(76)=-.130, p=.897$
MID Mean framewise displacement (FD; in mm)	.18 ± .07 (.05 - .29)	.16 ± .08 (.05 - .41)	$t(76)=1.563, p=.122$
MID Volumes with FD >0.5 mm (in %)	4.83 ± 4.59 (0 - 12.84)	3.53 ± 4.58 (0 - 16.89)	$t(76)=1.252, p=.215$
MID Signal-to-noise ratio	11.35 ± .78 (10.05 - 13.43)	11.60 ± 1.11 (8.22 - 14.08)	$t(76)=-1.118, p=.267$
Clinical characteristics			
ADI-R			
Social interaction	14.81 ± 6.77 (1 - 25)		
Communication	12.62 ± 5.93 (3 - 24)		
RRB	3.97 ± 3.04 (0 - 12)		
ADOS			
Social affect	5.33 ± 2.32 (1 - 9)		
RRB	4.06 ± 2.87 (1 - 9)		
Total	4.58 ± 2.42 (1 - 9)		
SRS-2			
rawscore	89.91 ± 31.27 (32 - 163)	19.68 ± 14.29 (2 - 74)	$t(47.14)=11.813, p<.000$
t-score	72.88 ± 11.98 (49 - 90)	45.12 ± 5.61 (37 - 66)	$t(47.76)=12.129, p<.000$
ADHD reserach diagnosis*(ADHD/noADHD/missing)	18/16/4	2/30/8	$\chi^2(1)=17.016, p<.001$
DAWBA comorbidities			
ADHD symptoms	2.30 ± 1.51 (0 - 5)	.39 ± 1.03 (0 - 4)	$t(50.45)=5.792, p<.000$
Anxiety symptoms	2.77 ± 1.55 (0 - 5)	1.21 ± .70 (0 - 4)	$t(44.16)=5.464, p<.000$
Depression symptoms	.97 ± 1.33 (0-5)	.12 ± .33 (0-1)	$t(34.94)=3.461, p=.001$
Adolescents			
	ASD	TD	group comparison
Total N	88	61	
Demographics			
Sex (m/f)	68/20	39/22	$\chi^2(1)=3.166, p=.075$
Age (years)	15.09 ± 1.75 (12.07 - 17.90)	15.61 ± 1.59 (12.44 - 17.99)	$t(147)=-1.841, p=.068$
IQ (full IQ)	102.63 ± 15.19 (75.00 - 143.00)	104.01 ± 12.46 (76.82 - 123.00)	$t(147)=-.584, p=.560$
Handedness (right/left/ambidextrous/unknown)	60/10/4/14	45/5/0/11	$\chi^2(3)=3.388, p=.336$
Medication use (no/yes/unknown)	22/37/29	25/6/30	$\chi^2(2)=18.264, p<.001$
fMRI quality control			
SID Mean framewise displacement (FD; in mm)	.13 ± .07 (.03 - .41)	.11 ± .06 (.05 - .29)	$t(147)=2.298, p=.023$
SID Volumes with FD >0.5 mm (in %)	2.36 ± 3.47 (0 - 13.51)	1.67 ± 3.16 (0 - 14.19)	$t(147)=1.228, p=.221$
SID Signal-to-noise ratio	9.51 ± 1.23 (6.28 - 12.21)	9.63 ± 1.11 (6.49 - 12.38)	$t(147)=-.596, p=.552$
MID Mean framewise displacement (FD; in mm)	.15 ± .08 (.03 - .36)	.13 ± .07 (.05 - .35)	$t(147)=2.106, p=.037$
MID Volumes with FD >0.5 mm (in %)	3.62 ± 5.03 (0 - 19.59)	2.23 ± 3.75 (0 - 16.22)	$t(147)=-.671, p=.503$
MID Signal-to-noise ratio	9.55 ± 1.33 (6.08 - 12.80)	9.69 ± 1.23 (6.40 - 12.03)	$t(146.16)=1.942, p=.054$
Clinical characteristics			
ADI-R			
Social interaction	16.85 ± 6.43 (2 - 29)		
Communication	13.21 ± 5.68 (1 - 26)		

RRB	3.99 ± 2.67 (0 - 12)		
ADOS			
Social affect	6.18 ± 2.72 (1 - 10)		
RRB	4.28 ± 2.32 (1 - 9)		
Total	5.36 ± 2.81 (1 - 10)		
SRS-2			
rawscore	91.34 ± 29.37 (22 - 151)	22.40 ± 14.22 (1 - 67)	<i>t</i> (115.98)=17.946, <i>p</i> <.000
t-score	73.03 ± 11.51 (45- 90)	45.72 ± 5.35 (38 - 63)	<i>t</i> (113.72)=18.318, <i>p</i> <.000
ADHD reserach diagnosis*(ADHD/noADHD/missing)	29/43/16	5/45/11	$\chi^2(1)=13.457$, <i>p</i> <.001
DAWBA comorbidities			
ADHD symptoms	2.03 ± 1.58 (0 - 5)	.18 ± .59 (0 - 3)	<i>t</i> (87.43)=8.472, <i>p</i> <.000
Anxiety symptoms	2.52 ± 1.32 (0 - 5)	.73 ± .60 (0 - 2)	<i>t</i> (119.87)=8.251, <i>p</i> <.000
Depression symptoms	.80 ± 1.09 (0-4)	.35 ± .62 (0-2)	<i>t</i> (118.00)=2.516, <i>p</i> =.013

Adults

	ASD	TD	group comparison
Total N	86	80	
Demographics			
Sex (m/f)	60/26	52/28	$\chi^2(1)=.429$, <i>p</i> =.512
Age (years)	22.50 ± 3.49 (18.02 - 30.60)	23.00 ± 3.16 (18.07 - 30.78)	<i>t</i> (164)=-.966, <i>p</i> =.336
IQ (full IQ)	105.94 ± 14.36 (75.56 - 148.00)	108.40 ± 12.30 (75.56 - 141.00)	<i>t</i> (164)=-1.179, <i>p</i> =.240
Handedness (right/left/ambidextrous/unknown)	58/12/4/12	49/7/3/21	$\chi^2(3)=4.459$, <i>p</i> =.216
Medication use (no/yes/unknown)	30/31/25	27/3/50	$\chi^2(2)=31.374$, <i>p</i> <.001
fMRI quality control			
SID Mean framewise displacement (FD; in mm)	.09 ± .05 (.03 - .27)	.09 ± .06 (.03 - .34)	<i>t</i> (164)=-.115, <i>p</i> =.908
SID Volumes with FD >0.5 mm (in %)	.95 ± 2.00 (0 - 11.49)	1.23 ± 3.00 (0 - 16.22)	<i>t</i> (164)=-.717, <i>p</i> =.474
SID Signal-to-noise ratio	9.40 ± 1.03 (7.10 - 11.97)	9.45 ± 1.02 (6.93 - 12.13)	<i>t</i> (164)=-.328, <i>p</i> =.744
MID Mean framewise displacement (FD; in mm)	.10 ± .06 (.03 - .31)	.10 ± .06 (.03 - .36)	<i>t</i> (164)=-.195, <i>p</i> =.845
MID Volumes with FD >0.5 mm (in %)	1.13 ± 2.65 (0 - 15.54)	1.21 ± 2.46 (0 - 15.54)	<i>t</i> (164)=-.192, <i>p</i> =.848
MID Signal-to-noise ratio	9.45 ± 1.17 (7.05 - 13.62)	9.44 ± 1.06 (6.82 - 11.77)	<i>t</i> (164)=.015, <i>p</i> =.988
Clinical characteristics			
ADI-R			
Social interaction	14.27 ± 6.48 (0 - 28)		
Communication	11.63 ± 5.54 (0 - 24)		
RRB	3.84 ± 2.52 (0 - 12)		
ADOS			
Social affect	5.46 ± 2.40 (1 - 10)		
RRB	4.39 ± 2.38 (1 - 10)		
Total	4.51 ± 2.42 (1 - 10)		
SRS-2			
rawscore	76.06 ± 29.34 (20 - 143)	29.27 ± 15.07 (4 - 87)	<i>t</i> (123.83)=12.270, <i>p</i> <.000
t-score	62.56 ± 10.33 (43- 86)	46.15 ± 5.33 (37 - 66)	<i>t</i> (124.09)=12.215, <i>p</i> <.000
ADHD reserach diagnosis*(ADHD/noADHD/missing)	69/118/25	11/130/40	$\chi^2(1)=9.376$, <i>p</i> =.002
DAWBA comorbidities			
ADHD symptoms	1.20 ± 1.41 (0 - 4)		
Anxiety symptoms	2.59 ± 1.14 (0 - 4)		
Depression symptoms	.82 ± 1.29 (0-5)		

Participant characteristics, split by age group. ADI-R: Autism Diagnostic Interview-Revised. Scores were computed for reciprocal interaction (social interaction), communication, and restrictive, repetitive stereotyped behaviors and interests (RRB). ADOS-2: Autism Diagnostic Observation Schedule 2. Calibrated severity scores were computed for social affect, restricted and repetitive behaviors (RRB) and the overall total score. SRS-2: Social Responsiveness Scale-2. Total raw and total T scores (sex+age normalized) are reported. The raw SRS-2 scores were used in our analyses. ADHD research diagnosis was based on applying DSM-V criteria to symptom scores in the parent- and self-rated ADHD rating scale. Self-rated scores were used when parent-rated scores were not available. Comorbid symptoms of ADHD, depression and anxiety were assessed with the Development and Well Being Assessment (DAWBA), generating six levels (ordinal scores 0 to 5) of prediction of the probability of a disorder (~0.1%, ~0.5%, ~3%, ~15%, ~50%, >70%). SID social incentive delay task, MID monetary incentive delay task.

Standard operation procedures and quality control

Standard operation procedures were implemented to harmonize data acquisition between sites and across time (1). This included hands-on and face-to-face training according to detailed protocols before study rollout and regular exchange between sites during data acquisition. Hard- and software for data acquisition was aligned as closely as possible between sites. Procedures were undertaken to optimize the MRI sequences for the best scanner-specific options while harmonizing across sites, and phantoms and travelling heads were employed to assure standardization and quality assurance of the multi-site image-acquisition. Test-retest reliability of the fMRI task battery was ensured (2-4).

Of the total sample, n=285 ASD participants and n=217 typically developing (TD) individuals had an IQ above 75 and data for both the MID and SID available. Several quality assessment (QA) metrics were calculated (<http://preprocessed-connectomes-project.org/quality-assessment-protocol/>) for these participant's datasets. Head motion was quantified as frame-wise displacement (FD, (5)), with two separate scores extracted for each dataset: mean FD and percent of volumes exceeding 0.5 mm FD. Additional temporal QA metrics comprised the temporal signal-to-noise ratio and the average change of a volume's mean intensity across time points (DVARs (6)). We additionally calculated the signal-to-noise ratio (SNR) based on the mean volume of the realigned time-series. In the present study, bad functional data quality was defined as excessive head movement with more than 20 percent of frames with a frame-wise displacement (FD) > 0.5mm (5) and/or signal loss with less than 80% overlap of the individual brain mask with the template mask in MNI space.

Participants were excluded based on anatomical brain abnormality (n=16), incomplete fMRI scan (n=12), technical problems (n=77), incorrect task performance (n=21), bad functional data quality (N=111) and data corruption (n=4) or a combination of these reasons. A comparison of the main QA metrics between ASD and TD in the final sample is presented in table 1 in the main text.

Experimental paradigm

Participants were asked to give a speeded response (button press) to a visual target screenflash. A cue arrow pointing upwards indicated the possibility to obtain a reward if responses were given within a predefined response time window (win trial). No reward option was given in trials preceded by a horizontal cue arrow (neutral trial). The response time window was continuously adapted to ensure a comparable number of reward events across subjects and groups (~60 %). Sufficiently fast responses on win trials were followed by the presentation of a 2€/2£ coin in the MID and a smiling female face in the SID as feedback. Blurred control stimuli were presented in neutral trials and as feedback following slow responses in win trials. In total, 30 win trials and 30 neutral trials were presented in a pseudorandomized order during each task. SID and MID were collected as separate paradigms with SID always presented first, followed by MID. Task order was not randomized across participants in order to avoid loss of motivation after performing the monetary task. Note that the feedback presentation was temporally decoupled from the target presentation but not from the button press. For a visualization of the task design and stimuli, please refer to figure 1 in the main text.

First level fMRI data analysis

SID and MID tasks were combined as two sessions in a general linear model (GLM) on the single subject level. Each session was modeled using the six task regressors (cue win, cue neutral, target, feedback win, feedback lost and feedback neutral) and six realignment parameters were included for both tasks. All regressors were modeled as stick functions, convolved with a canonical hemodynamic response function (HRF). At the model estimation stage, a high-pass filter with a cutoff of 128 seconds and an autoregressive model of the first order were applied. Contrast images (cue win > cue neutral; feedback won > feedback neutral) were created for each task separately and for both tasks combined. Additionally, a contrast image for the interaction between condition (win, neutral) and task (SID, MID) was calculated.

Behavioural data

Behaviourally, individuals with autism have been reported to show decreased accuracy compared to typically developing (TD) individuals in these tasks in some cases (7, 8) but not others (9-11). Typically, reaction times (RT) are faster when a subsequent reward is possible for all participants and do not differ between groups (7, 9, 10, 12, 13), but see (11, 14).

Reaction times (RT) and accuracy (percentage of successful trials) were analyzed using SPSS Software package (Version 25, IBM Corp., Armonk, NY, USA). A repeated measures ANOVAs with the within subject factors condition (win, neutral) and task (MID, SID) and between subject factor of diagnosis (TD, ASD) and covariates age (mean-centered) sex, and study sites (dummy coded) were used to assess effects of task, condition and diagnosis.

There were no significant effects of diagnosis, task or condition on RT. Accuracy was higher for win ($M=70.5$) compared to neutral trials ($M=49.9$, $F_{(1,380)}=25.989$, $p<.001$, $\eta^2=.064$). A significant task*condition interaction ($F_{(1,380)}=3.896$, $p=.049$, $\eta^2=.010$) revealed higher accuracy during win trials in the MID ($M=73.2$) compared to the SID ($M=67.8$, $p<.001$) while during neutral trials accuracy was higher in the SID ($M=51.6$) compared to the MID ($M=48.2$, $p<.001$). There was no significant effect of diagnosis on accuracy.

Whole brain activation analyses for tasks separately

Although there was a significant interaction between task and condition during both reward anticipation and delivery, revealing stronger differential activation in the MID compared to the SID (see fig 2 and table 2 in main text), this interaction was not different between individuals with ASD and TD and not associated with SRS-2 scores. However, here we still assess differences between ASD and TD in the SID and MID separately (see figures S1 and S2, and tables S2 and S3).

The separate analysis of the MID revealed similar results to the combined analysis, with the effect of diagnostic group remaining significant at the whole brain level in the right VS ($F_{(1,384)}=22.53$, $p_{FWE}=.022$, $k=9$). In contrast, separate analysis of the SID revealed no significant effect of diagnosis at the whole brain level. For details, see figure S1 and tables S2 and S3. Explorative ROI analyses in the SID and MID separately revealed that group differences remained

significant in the MID (left VS: $F_{(1,384)}=15.201$, $p<.001$, partial $\eta^2=.038$, right VS $F_{(1,384)}=19.462$, $p<.001$, partial $\eta^2=.048$) and in the right VS for the SID ($F_{(1,384)}=6.732$, $p=.010$, partial $\eta^2=.017$). Effects in the left VS for the SID did not survive correction for multiple testing ($F_{(1,384)}=4.876$, $p=.028$, partial $\eta^2=.013$). For details, see figure S1 and supplementary table S3.

Separate analyses of the MID and SID also yielded no significant effect of diagnosis in either task during reward delivery. For details, see figure S2 and supplementary tables S2 and S3). Explorative ROI analysis in the MID revealed that the effect of diagnosis reached significance in the right VS: ($F_{(1,370)}=5.557$, $p=.019$, partial $\eta^2=.015$) while it remained below threshold for the left VS ($F_{(1,370)}=4.804$, $p=.029$, partial $\eta^2=.013$). There was no significant effect of diagnosis in the SID (left VS: $F_{(1,370)}=1.383$, $p=.240$, partial $\eta^2=.004$, right VS $F_{(1,370)}=.802$, $p=.371$, partial $\eta^2=.002$). For details, see figure S2 and table S3.

Across both tasks, there was also no effect of continuous SRS-2 scores on the whole brain or ROI level.

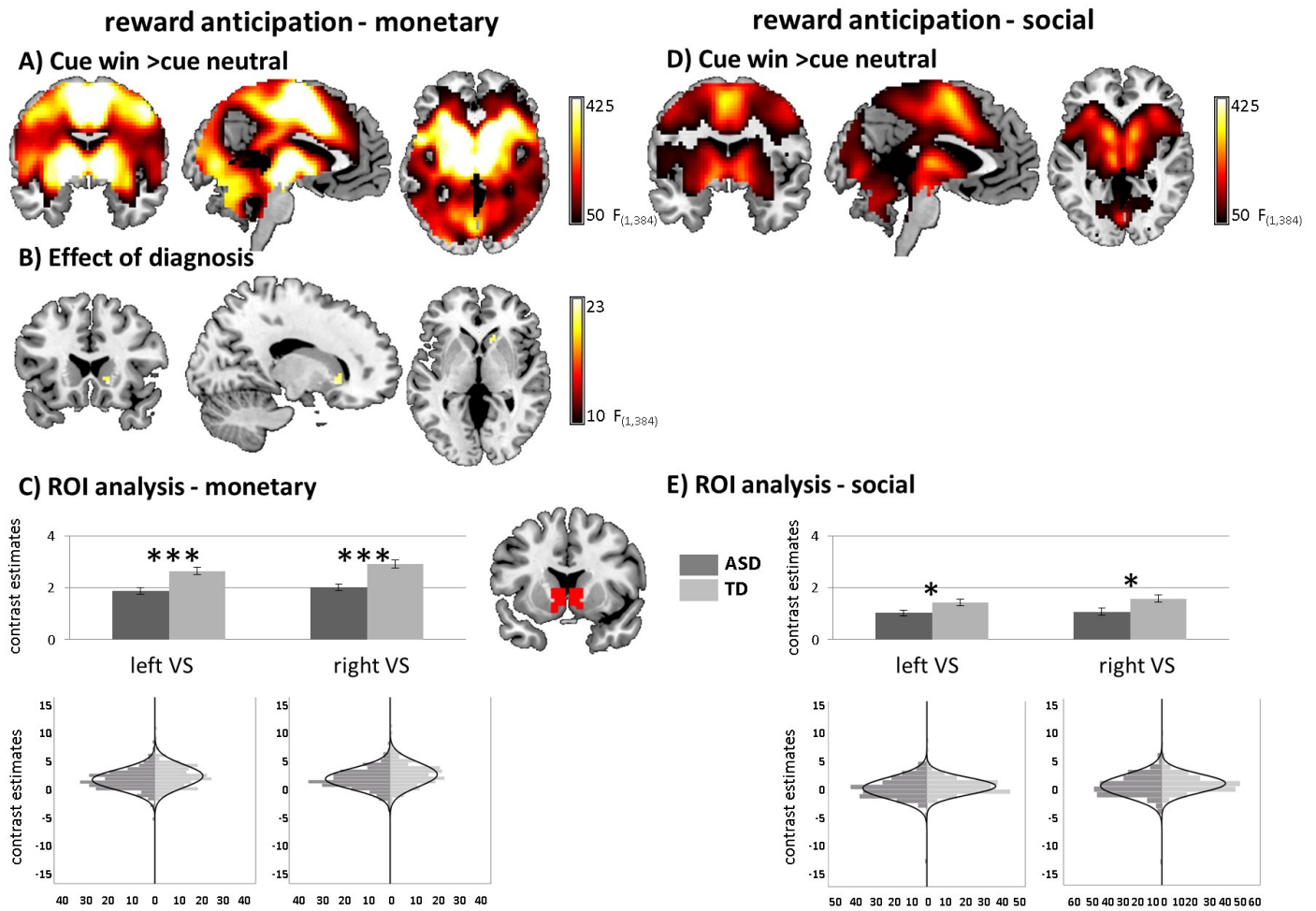


Figure S1: Whole-brain familywise error corrected brain activation to win compared to neutral cues for the MID and SID separately. A) activation across both ASD and TD individuals in the MID. B) Effect of diagnosis in right ventral striatum in the MID. C) Effect of diagnosis in the region of interest (ROI) analysis of the left and right ventral striatum with corresponding distribution plots for the MID. D) activation across both ASD and TD individuals in the SID. E) Effect of diagnosis in the region of interest (ROI) analysis of the left and right ventral striatum with corresponding distribution plots for the SID. *** $p < .001$, * $p < .05$.

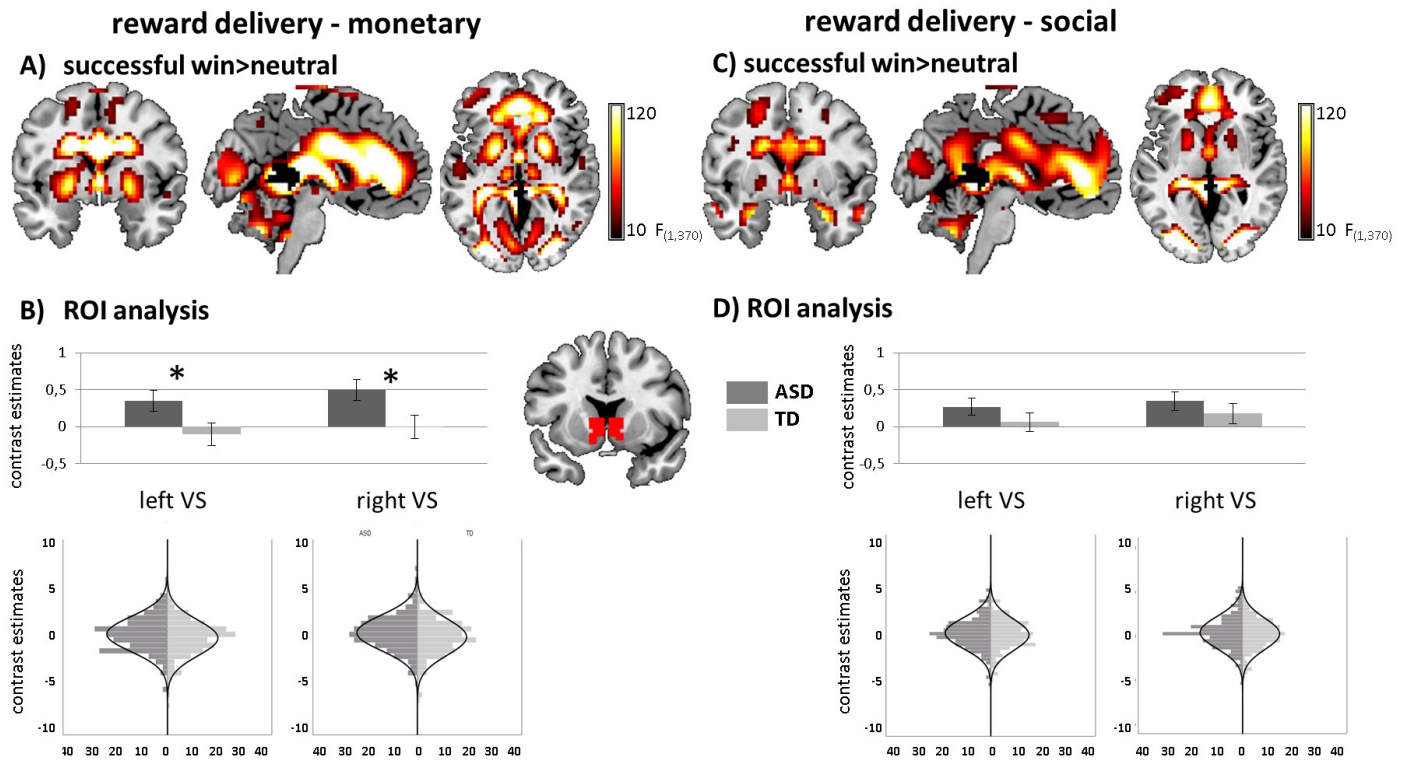


Figure S2: Whole-brain familywise error corrected brain activation to successful win compared to neutral trials for the MID and SID separately. A) activation across both ASD and TD individuals in the MID. B) Effect of diagnosis in the region of interest (ROI) analysis of the left and right ventral striatum with corresponding distribution plots for the MID. C) activation across both ASD and TD individuals in the SID. D) Effect of diagnosis in the region of interest (ROI) analysis of the left and right ventral striatum with corresponding distribution plots for the SID. * $p < .05$.

Table S2: Whole-brain effects of brain activation for SID and MID separately.

Region	Hemisphere	Direction	k	x	y	z	F	$p_{(FWE-corr)}$
ANTICIPATION MID								
EFFECT OF TASK								
supplementary motor area	r	win>neutral	42326	3	2	53	870.267	0.000
nucleus accumbens	r	win>neutral		12	8	-4	815.828	0.000
supplementary motor area	l	win>neutral		-6	8	44	797.693	0.000
pallidum	l	win>neutral		-12	8	-4	793.943	0.000
middle cingulate gyrus	r	win>neutral		9	11	41	781.462	0.000
thalamus, intralaminar	l	win>neutral		-9	-19	-1	776.746	0.000
thalamus, mediodorsal lateral parvocellular	r	win>neutral		9	-16	-1	707.410	0.000
precentral gyrus	l	win>neutral		-36	-16	50	655.909	0.000
precentral gyrus	l	win>neutral		-36	-10	53	655.081	0.000
precentral gyrus	l	win>neutral		-27	-28	65	626.463	0.000
middle cingulate gyrus	l	win>neutral		-9	-25	44	563.826	0.000
insula	r	win>neutral		30	26	2	533.024	0.000
insula	l	win>neutral		-27	23	5	530.611	0.000
precentral gyrus	l	win>neutral		-24	-10	65	517.364	0.000
middle cingulate gyrus	r	win>neutral		9	-28	47	512.844	0.000

middle frontal gyrus	r	win>neutral		39	-7	53	482.072	0.000
EFFECT OF DIAGNOSIS								
caudate	r	TD>ASD	9	12	17	-1	22.533	0.022
DELIVERY MID								
EFFECT OF TASK								
middle occipital gyrus	r	s. win>neutral	9359	30	-88	-1	744.741	0.000
middle occipital gyrus	l	s. win>neutral		-24	-94	-1	734.134	0.000
inferior occipita gyrus	l	s. win>neutral		-27	-88	-7	637.429	0.000
pallidum	r	neutral>s. win	420	21	5	-1	137.279	0.000
insula	r	neutral>s. win		36	-7	8	33.433	0.000
insula	l	s. win>neutral	165	-33	14	-16	132.034	0.000
pallidum	l	neutral>s. win	469	-21	2	-1	123.290	0.000
insula	l			-36	-7	5	36.622	0.000
superior temporal gyrus	l			-39	-13	-7	21.866	0.032
angular gyrus	r	neutral>s. win	694	60	-55	32	98.575	0.000
middle temporal gyrus	r	neutral>s. win		54	-64	11	93.408	0.000
supramarginal gyrus	r	neutral>s. win		66	-19	32	79.452	0.000
lingual gyrus	l	neutral>s. win	2393	-6	-79	2	90.596	0.000
calcarine	r	neutral>s. win		6	-79	5	87.877	0.000
angular gyrus	l			-48	-64	47	75.276	0.000
middle temporal gyrus	l	neutral>s. win	183	-45	-70	8	77.507	0.000
vermis		neutral>s. win	34	0	-37	-40	75.909	0.000
middle frontal gyrus	l	neutral>s. win	967	-39	20	47	74.903	0.000
middle frontal gyrus	l	neutral>s. win		-36	26	41	66.322	0.000
middle frontal gyrus	l	neutral>s. win		-36	38	26	57.545	0.000
supplementary motor area	l	s. win>neutral	70	0	-13	74	74.385	0.000
supplementary motor area	l	s. win>neutral		0	2	71	50.649	0.000
paracentral lobule	r	s. win>neutral		0	-28	74	49.733	0.000
superior frontal gyrus	l	neutral>s. win	145	-21	-7	59	59.315	0.000
superior frontal gyrus	r	neutral>s. win	300	27	50	20	44.435	0.000
middle frontal gyrus	r	neutral>s. win		30	44	26	40.945	0.000
middle frontal gyrus	r	neutral>s. win		36	23	38	37.056	0.000
supplementary motor area	r	neutral>s. win	126	12	5	47	38.244	0.000
superior frontal gyrus	r	neutral>s. win		18	-4	65	33.302	0.000
paracentral lobule	r			9	-28	62	26.585	0.004
rolandic operculum	r	neutral>s. win	49	60	8	14	36.222	0.000
precentral gyrus	l	s. win>neutral	12	-24	-22	74	35.440	0.000
precentral gyrus	r	neutral>s. win	33	30	-7	50	33.639	0.000
Inferior frontal gyrus, opercular part	l	neutral>s. win	31	-54	5	11	31.498	0.001
superior temporal gyrus	r	neutral>s. win	50	51	2	-13	28.538	0.002
superior temporal gyrus	r	neutral>s. win		54	-7	-1	28.514	0.002

Heschl's gyrus	r	neutral>s. win	8	39	-28	14	25.125	0.008
Inferior frontal gyrus, triangular part	r	neutral>s. win	5	57	26	14	22.722	0.022

ANTICIPATION SID

EFFECT OF TASK

putamen	l	win>neutral	35668	-15	5	-7	366.221	0.000
nucleus accumbens	r	win>neutral		12	8	-7	347.367	0.000
supplementary motor area	l	win>neutral		-3	-1	59	338.159	0.000
precentral gyrus	l	win>neutral		-42	-13	53	329.747	0.000
thalamus, mediodorsal medial magnocellular	l	win>neutral		-6	-19	8	320.730	0.000
supplementary motor area	r	win>neutral		6	5	50	301.722	0.000
thalamus, mediodorsal medial magnocellular	r	win>neutral		6	-13	5	297.261	0.000
middle cingulate gyrus	l	win>neutral		-6	11	38	287.338	0.000
red nucleus	l	win>neutral		-9	-19	-7	274.031	0.000
red nucleus	l	win>neutral		-6	-22	-10	271.665	0.000
precentral gyrus	l	win>neutral		-27	-28	59	262.482	0.000
red nucleus	r	win>neutral		9	-19	-10	259.582	0.000
middle frontal gyrus	r	win>neutral		42	-7	56	231.990	0.000
insula	l	win>neutral		-30	26	2	214.125	0.000
lingual gyrus	l	win>neutral		0	-76	2	207.940	0.000
insula	r	win>neutral		33	26	-4	202.890	0.000

DELIVERY SID

EFFECT OF TASK

Calcarine fissure and surrounding cortex	r	s. win>neutral	8891	24	-91	-1	907.615	0.000
inferior occipita gyrus	l	s. win>neutral		-24	-91	-4	695.419	0.000
middle occipital gyrus	l	s. win>neutral		-21	-97	5	645.510	0.000
middle frontal gyrus	l	neutral>s. win	1877	-39	35	29	89.398	0.000
middle frontal gyrus	l	neutral>s. win		-33	44	14	63.507	0.000
superior frontal gyrus	l	neutral>s. win		-21	2	53	59.495	0.000
inferior parietal gyrus	l	neutral>s. win	1153	-36	-43	44	79.507	0.000
inferior parietal gyrus	l	neutral>s. win		-51	-28	38	72.565	0.000
precuneus	l	neutral>s. win		-12	-67	53	69.840	0.000
supramarginal gyrus	r	neutral>s. win	303	63	-22	35	63.666	0.000
supramarginal gyrus	r	neutral>s. win		36	-37	44	44.921	0.000
supramarginal gyrus	r			57	-31	47	32.139	0.000
cuneus	r	neutral>s. win	438	6	-85	17	57.019	0.000
Inferior frontal gyrus, opercular part	r	neutral>s. win	174	57	11	11	56.174	0.000
insula	r	neutral>s. win		39	20	5	30.799	0.001
superior parietal gyrus	r		109	18	-67	53	54.793	0.000
middle frontal gyrus	r	neutral>s. win	311	36	32	35	54.568	0.000
superior frontal gyrus	r	neutral>s. win		27	50	14	33.088	0.000
supplementary motor area	r	s. win>neutral	34	3	-13	74	51.326	0.000

pallidum	r	neutral>s. win	71	18	8	-1	35.084	0.000
superior frontal gyrus	r		28	27	-7	53	27.116	0.004
angular gyrus	l	s. win>neutral	20	-45	-64	23	26.356	0.005
middle temporal gyrus		s. win>neutral	8	-51	-40	2	23.989	0.015

Table provides test statistic of significant peak voxel(s) for whole-brain analysis. Voxel-level statistics were family-wise error (FWE) corrected for the number of voxels across the whole brain for each test. Significance was defined as $p_{FWE} < .05$ with a cluster threshold of $k \geq 5$. Significant whole-brain results are localized in MNI space and labeled according to the automated anatomical labeling atlas 3 (aal3). SID social incentive delay task, MID monetary incentive delay task, ASD autism spectrum disorder, TD typically developing, s. win successful win.

Table S3: Overview over effects of diagnosis (categorical) and autism traits (dimensional) on functional brain activation separately in the MID and SID task.

	TD vs. ASD			effect of SRS-2		
	whole-brain	left VS	right VS	whole-brain	left VS	right VS
original results (reported in main text)						
anticipation	<i>right VS</i> $F_{(1,384)}=22.84$, $p_{FWE}=.017$	$F_{(1,384)}=14.163$, $p<.001$	$F_{(1,384)}=18.693$, $p<.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,370)}=4.829$, $p=.029$	$F_{(1,370)}=4.719$, $p=.030$	no effect	no effect	no effect
MID						
anticipation	<i>right VS:</i> $F_{(1,384)}=22.53$, $p_{FWE}=.022$	$F_{(1,384)}=15.20$, $p<.001$	$F_{(1,384)}=19.46$, $p<.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,370)}=4.80$, $p=.029$	$F_{(1,370)}=5.56$, $p=.019$	no effect	no effect	no effect
SID						
anticipation	no effect	$F_{(1,384)}=4.88$, $p=.028$	$F_{(1,384)}=6.73$, $p=.010$	no effect	no effect	no effect
delivery	no effect	no effect	no effect	no effect	no effect	no effect

Table provides test statistic of region of interest (ROI) analysis and of significant peak voxel(s) for whole-brain analysis. Voxel-level statistics were family-wise error (FWE) corrected for the number of voxels across the whole brain for each test. Significance was defined as $p_{FWE} < .05$ with a cluster threshold of $k \geq 5$. To correct for investigating left and right VS activity separately in the ROI analysis, critical alpha was adjusted to $p < .025$ based on the Bonferroni procedure. Deviations from results reported in the main text are highlighted in bold. Significant whole-brain results are localized in MNI space and labeled according to the automated anatomical labeling atlas (aal). Abbreviations: TD typically developing, ASD autism spectrum disorder, VS ventral striatum, SID social incentive delay task, MID monetary incentive delay task, IA interaction.

Control analyses

Please note that we did not correct for the number of tests performed in the control analyses to maximize sensitivity.

Age group

Previous research in TD individuals has demonstrated several, but inconsistent age effects (for reviews, see 15, 16). Regarding reward anticipation, studies have reported hypoactivation in adolescents compared to adults and children (17-19). Regarding reward consumption or responses to entire reward processing trials, studies reported either no age-related effects (18, 20) or increased activity in adolescence in the VS (21-24) or dorsal caudate (25). Other studies show a linear increase of VS activation with age during reward anticipation (26), a linear decrease during reward delivery (26) or a linear increase of VS activity assessing the entire trial (27). Further, the pattern of age-related differences in reward processing in ASD remains elusive. We hypothesize a similar quadratic effect of age on VS activity in both TD and ASD individuals. We assessed linear and quadratic effects of age as well as interactions between age and diagnosis dimensionally across the whole sample in separate models where age and age² were added as additional covariates of interest.

During reward anticipation, no linear or non-linear effects of age were observed.

During reward delivery, a linear increase of activation with increasing age was observed in the right superior medial frontal gyrus ($F_{(1,370)}=23.58$, $p_{FWE}=.016$, $k=8$, figure S3 A). There was no linear association of age and VS. Quadratic age yielded an effect on the whole-brain level in a cluster comprising the left amygdala and pallidum ($F_{(1,369)}=27.10$, $p_{FWE}=.004$, $k=18$, figure S3 B). A trend for a quadratic effect of age was observed in the right VS ($F_{(1,367)}=3.609$, $p=.058$) and is illustrated in figure S3 C. In the left VS a trend for an interaction between age² and reward type was observed ($F_{(1,367)}=3.626$, $p=.058$) and is illustrated in figure S3 C. Follow up analysis revealed a significant effect of age² in the MID ($F_{(1,368)}=5.242$, $p=.023$), but not in the SID ($F_{(1,368)}=.010$, $p=.921$). No interactions between age or age² and diagnosis were observed.

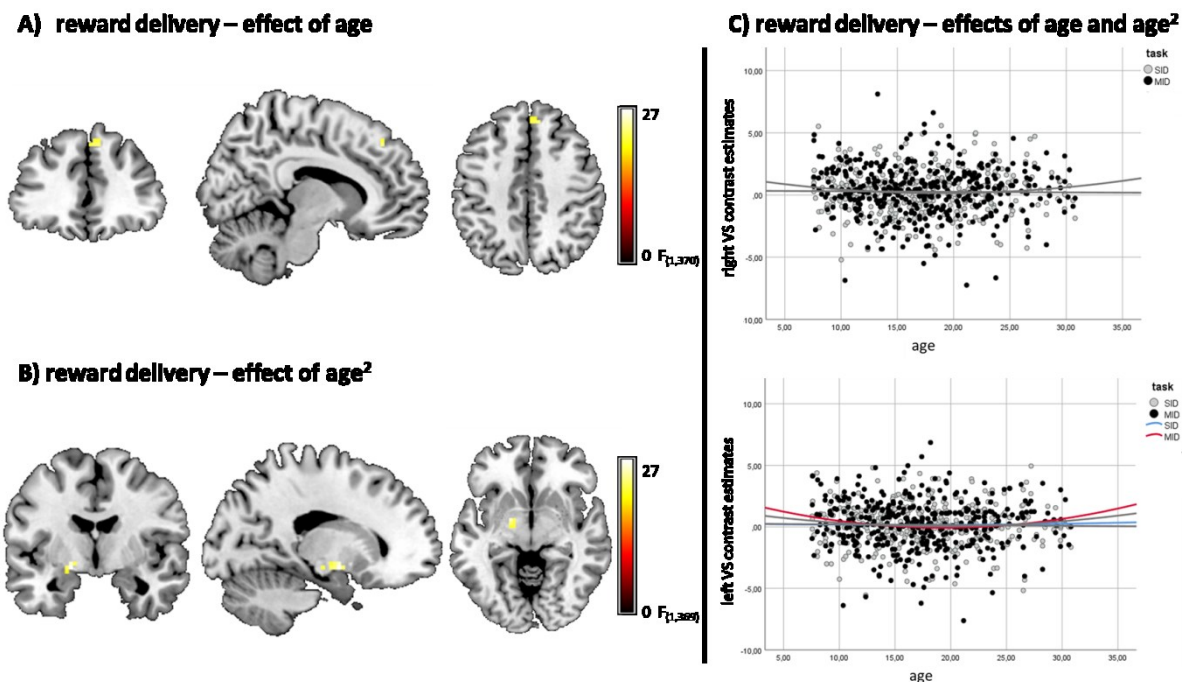


Figure S3: Linear and non-linear effects of age. A) Whole-brain familywise error corrected brain activation associated with age for the contrast of won compared to neutral trials. B) Whole-brain familywise error corrected brain activation associated with age² for the contrast of won compared to neutral trials. C) Scatterplot with linear and quadratic model fits for the association between age and contrast estimates of right and left ventral striatal activation across social and monetary reward for the contrast of won compared to neutral trials. Plot for left VS includes quadratic model fits for both tasks separately (MID in red, SID in blue).

Investigating effects of age yielded a linear increase of activity within the right superior medial frontal gyrus during reward delivery. This region in the medial prefrontal cortex (mPFC) has been shown to encode task value and expectation of monetary reward (28) and more frontal parts of this region have been associated with valuation of social interactions (29, 30). Further, our results are in line with previous studies reporting increased activation in the mPFC in older compared to younger participants during reward delivery (27, 31, 32). The trend-level significant effects for age² and the interaction between task and age² in the right VS and left VS, respectively, revealed lowest VS activity in adolescence and early adulthood with higher activation in childhood and towards the upper end of the age range. Importantly, our results are in contrast to findings of Schreuders et al. (24) showing a quadratic association of VS activity during delivery and age, with peak VS activity levels in adolescence. While this study was similar in sample size and age-range, the diverging results may be explained by the different tasks applied and, more importantly, by the fact that Schreuders et al. employed a longitudinal design across three assessment timepoints. The longitudinal approach of the LEAP study will allow us to test the replicability of our finding in future investigations. Further, as none of the

age-related effects we observed differed significantly between individuals with ASD and TD, a within-subject design should complement the current cross-sectional analyses with higher sensitivity for differences in developmental changes of motivational processes due to a better control of between- and within-subject confounds.

To allow comparability to previous studies explore effects within narrower age-ranges, we repeated these main analyses within age-specific subgroups: children (7.0-11.9 years), adolescents (12.0-17.9 years) and adults (18.0-30.9 years).

A main effect of diagnosis was found only in the adult subsample (see table S4 and figure S4 A). However, in children a significant interaction between task and diagnosis emerged in the right VS during reward anticipation with increased VS activation in TD compared to ASD only during the MID ($p=.003$) but not during the SID task ($p=.896$). During reward delivery, the interaction between task and diagnosis in the right VS was significant at trend-level in the childhood subsample with increased right VS activation to monetary compared to social rewards in ASD ($p=.022$), but not in TD children ($p=.562$). For details see table S4 and figure S4 B.

Table S4: Overview over effects of diagnosis and age on functional brain activation for adults, adolescents and children separately.

	TD vs. ASD			effect of age			quadratic effect of age			effect of SRS-2		
	whole-brain	left VS	right VS	whole-brain	left VS	right VS	whole-brain	left VS	right VS	whole-brain	left VS	right VS
full sample (group comparison as reported in main text)												
anticipation	<i>right VS</i> $F_{(1,384)}=22.84$, $p_{FWE}=.017$	$F_{(1,384)}=14.16$ 3, $p<.001$	$F_{(1,384)}=18.69$ 3, $p<.001$	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect
delivery	no effect	$F_{(1,370)}=4.829$, $p=.029$	$F_{(1,370)}=4.719$, $p=.030$	<i>right superior medial frontal gyrus</i> $F_{(1,370)}=23.58$, $p_{FWE}=.016$	no effect	no effect	<i>left amygdala/pallidum:</i> $F_{(1,367)}=27.10$, $p_{FWE}=.004$; <i>left hippocampus:</i> $F_{(1,367)}=22.18$, $p_{FWE}=.029$	no effect <i>IA task*age_quad</i> $F_{(1,367)}=3.626$, $p=.058$	$(F_{(1,367)}=3.609$, $p=.058$	no effect	no effect	no effect
adults (n=166)												
anticipation	Superior parietal lobule: $F_{(1,384)}=23.08$, $p_{FWE}=.028$	$F_{(1,157)}=10.56$ 1, $p=.001$	$F_{(1,157)}=11.50$ 4, $p=.001$	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect
delivery	no effect	$F_{(1,152)}=11.18$ 6, $p=.001$	$F_{(1,152)}=10.63$ 1, $p=.001$	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect
adolescents (n=149)												
anticipation	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect
delivery	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect	no effect
children (n=78)												
anticipation	no effect	no effect	no effect <i>IA task*diagnosis</i> $F_{(1,70)}=5.451$, $p=.022$	no effect	no effect	no effect	no effect	no effect	no effect <i>IA task*age_quad</i> $F_{(1,67)}=3.869$, $p=.053$	no effect	no effect	no effect
delivery	no effect	no effect	no effect <i>IA task*diagnosis</i> $F_{(1,68)}=3.924$, $p=.052$	right middle frontal gyrus: $F_{(1,64)}=30.89$, $p_{FWE}=.011$ left middle frontal gyrus:	no effect	$F_{(1,68)}=4.70$, $p=.034$	no effect	no effect	no effect	no effect	no effect	no effect

				$F_{(1,64)}=28.91,$ $p_{FWE}=.019$								
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Table provides test statistic of region of interest (ROI) analysis and of significant peak voxel(s) for whole-brain analysis. Voxel-level statistics were family-wise error (FWE) corrected for the number of voxels across the whole brain for each test. Significance was defined as $p_{FWE} < .05$ with a cluster threshold of $k \geq 5$. To correct for investigating left and right VS activity separately in the ROI analysis, critical alpha was adjusted to $p < .025$ based on the Bonferroni procedure. Deviations from results reported in the main text are highlighted in bold. Significant whole-brain results are localized in MNI space and labeled according to the automated anatomical labeling atlas (aal). Abbreviations: TD typically developing, ASD autism spectrum disorder, VS ventral striatum, SID social incentive delay task, MID monetary incentive delay task.

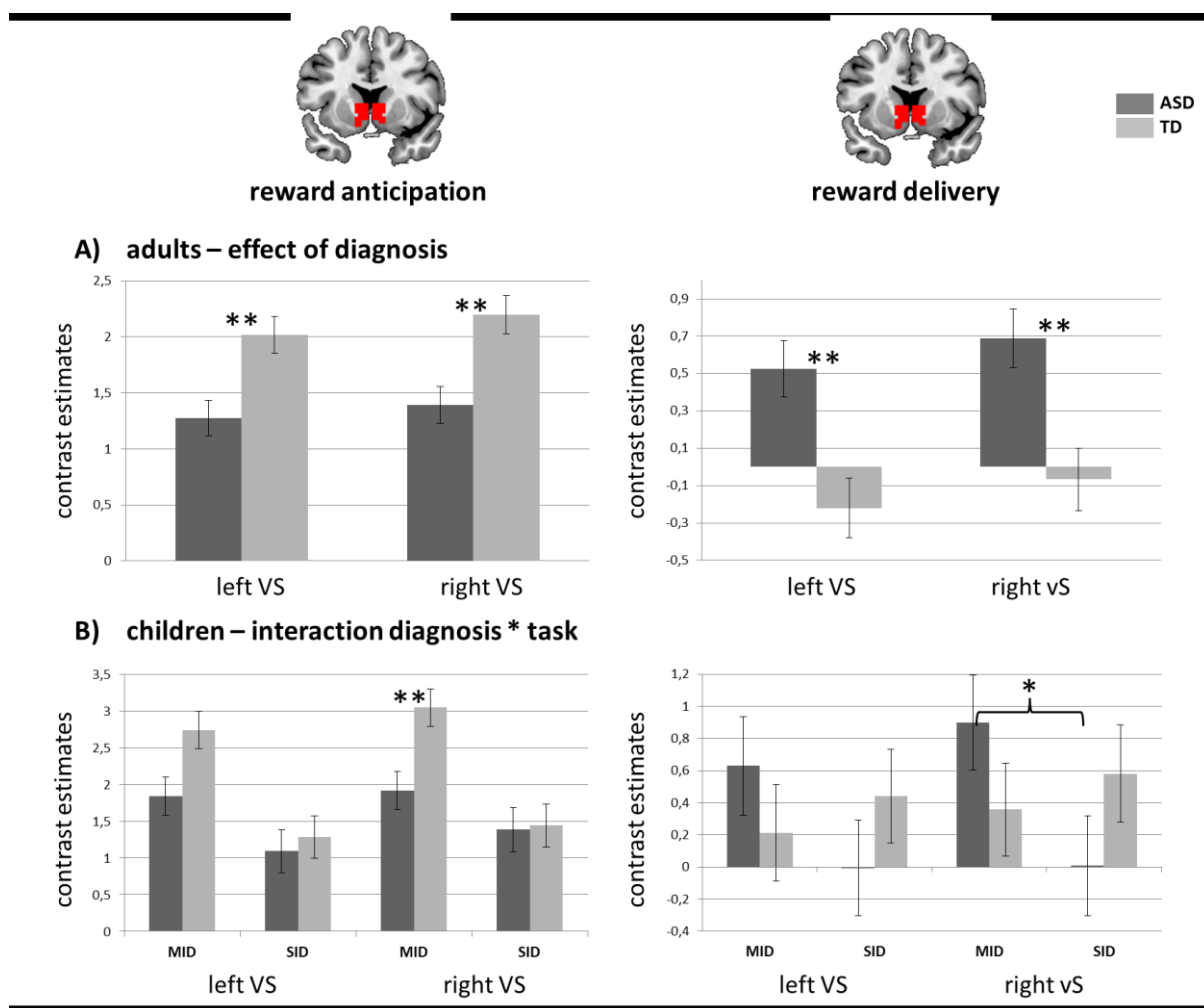


Figure S4: Contrast estimates for ventral striatal activation during reward anticipation and delivery for A) adults and B) children. ** $p < .01$, * $p < .05$.

ADHD comorbidity

Given the significant comorbidity between ASD and ADHD, and based on the extensive literature on ventral striatal abnormality in ADHD, we investigated the effect of diagnosis while controlling for ADHD comorbidity. Note that information on the presence of a confirmed diagnosis of ADHD was not available in our sample. As a proxy, we calculated an artificial diagnosis applying DSM-V criteria based on symptom scores in the parent- and self-rated ADHD rating scale (33). The pattern of results in the left and right VS was similar when controlling for ADHD comorbidity. The whole-brain level finding of reduced right VS activity in ASD was not replicated when controlling for ADHD, which might, however, also be due to the reduced sample size of individuals with completed ADHD rating-scale.

Table S5: Overview over effects of diagnosis and ADHD comorbidity on functional brain activation.

	TD vs. ASD		
	whole-brain	left VS	right VS
original results (reported in main text)			
anticipation	<i>Right VS</i> $F_{(1,384)}=22.84$, $p_{FWE}=.017$	$F_{(1,384)}=14.163$, $p<.001$	$F_{(1,384)}=18.693$, $p<.001$
delivery	no effect	$F_{(1,370)}=4.829$, $p=.029$	$F_{(1,370)}=4.719$, $p=.030$
results controlling for ADHD comorbidity			
anticipation	no effect	$F_{(1,317)}=9.895$ $p=.002$	$F_{(1,317)}=13.187$, $p<=.001$
delivery	no effect	$F_{(1,307)}=7.598$ $p=.006$	$F_{(1,307)}=5.074$ $p=.025$

Table provides test statistic of region of interest (ROI) analysis and of significant peak voxel(s) for whole-brain analysis. Voxel-level statistics were family-wise error (FWE) corrected for the number of voxels across the whole brain for each test. Significance was defined as $p_{FWE}<.05$ with a cluster threshold of $k\geq 5$. To correct for investigating left and right VS activity separately in the ROI analysis, critical alpha was adjusted to $p<.025$ based on the Bonferroni procedure. Deviations from results reported in the main text are highlighted in bold. Significant whole-brain results are localized in MNI space and labeled according to the automated anatomical labeling atlas (aal). Abbreviations: TD typically developing, ASD autism spectrum disorder, ADHD attention-deficit/hyperactivity disorder, VS ventral striatum.

Site

All analyses were controlled for site. However, to further examine systematic effects while preserving statistical power we employed a leave-one-out approach, recalculating the statistical maps while holding out one site at a time. The pattern of results remained largely the same with vanished whole-brain level effect in the right VS, possibly due to decreased statistical power, when dropping all sites except UCAM. For ROI analysis, the pattern remained largely stable when dropping all sites except when dropping RUN and KCL for reward delivery. As these two sites contributed the largest groups of participants, this is likely due to decreased statistical power.

Table S6: Overview over effects of diagnosis and age on functional brain activation after controlling for site using the leave-one-out approach.

	TD vs. ASD			effect of SRS-2		
	whole-brain	left VS	right VS	whole-brain	left VS	right VS
original results (reported in main text)						
anticipation	<i>Right VS</i> $F_{(1,384)}=22.84$, $p_{FWE}=.017$	$F_{(1,384)}=14.163$, $p<.001$	$F_{(1,384)}=18.693$, $p<.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,370)}=4.829$, $p=.029$	$F_{(1,370)}=4.719$, $p=.030$	no effect	no effect	no effect
drop site RUNMC						
anticipation	no effect	$F_{(1,270)}=11.561$, $p=.001$	$F_{(1,270)}=16.557$, $p_{F<.001}$	no effect	no effect	no effect
delivery	no effect	no effect	$F_{(1,257)}=3.561$, $p=.060$	no effect	no effect	no effect
Drop site UMCU						
anticipation	no effect	$F_{(1,315)}=9.458$, $p=.002$	$F_{(1,315)}=13.065$, $p<.001$	no effect	no effect	no effect

delivery	no effect	$F_{(1,313)}=4.777,$ $p=.030$	$F_{(1,313)}=3.929,$ $p=.048$	no effect	no effect	no effect
Drop site KCL						
anticipation	no effect	$F_{(1,302)}=11.094,$ $p=.001$	$F_{(1,302)}=13.334,$ $p<.001$	no effect	no effect	no effect
delivery	no effect	no effect	no effect	no effect	no effect	no effect
Drop site UCAM						
anticipation	left caudate/thalamus $F_{(1,333)}=22.24,$ $p_{FWE}=.025$ right VS $F_{(1,333)}=21.97,$ $p_{FWE}=.028$	$F_{(1,332)}=14.269,$ $p<.001$	$F_{(1,332)}=18.623,$ $p<.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,318)}=4.602,$ $p=.033$	$F_{(1,318)}=4.478,$ $p=.035$	no effect	no effect	no effect
Drop site CIMH						
anticipation	no effect	$F_{(1,340)}=12.636,$ $p<.001$	$F_{(1,340)}=16.557,$ $p<.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,326)}=5.724,$ $p=.017$	$F_{(1,326)}=5.167,$ $p=.024$	no effect	no effect	no effect
Drop site UCBM						
anticipation	no effect	$F_{(1,358)}=12.326,$ $p=.001$	$F_{(1,358)}=16.151,$ $p<.001$	no effect	no effect	no effect
delivery		$F_{(1,345)}=4.049,$ $p=.045$	$F_{(1,345)}=3.860,$ $p=.050$		no effect	no effect

Table provides test statistic of region of interest (ROI) analysis and of significant peak voxel(s) for whole-brain analysis. Voxel-level statistics were family-wise error (FWE) corrected for the number of voxels across the whole brain for each test. Significance was defined as $p_{FWE}<.05$ with a cluster threshold of $k \geq 5$. To correct for investigating left and right VS activity separately in the ROI analysis, critical alpha was adjusted to $p<.025$ based on the Bonferroni procedure. Deviations from results reported in the main text are highlighted in bold. Significant whole-brain results are localized in MNI space and labeled according to the automated anatomical labeling atlas (aal). Abbreviations: TD typically developing, ASD autism spectrum disorder, VS ventral striatum, KCL Kings College London, UCBM University Campus Bio-Medico of Rome, UMCU University Medical Centre Utrecht, RUNMC Radboud University Nijmegen Medical Centre, CIMH Central Institute of Mental Health in Mannheim, UCAM University of Cambridge .

IQ

Level of intellectual abilities was assessed using the Wechsler Abbreviated Scales of Intelligence – Second Edition, WASI-II (Wechsler, 2011) or – in countries where the WASI is not translated (i.e. The Netherlands, Germany and Italy) – the four-subtest short-forms of the German, Dutch or Italian WISC-III/IV (Wechsler, 1991; 2005 for children) or WAIS-III/IV (Wechsler, 1997; Wechsler, 2008 for adults). The shortened versions were used for feasibility reasons to not further prolong the testing sessions for participants. All versions included two verbal subscales (Vocabulary, Similarities) and two non-verbal subscales (Block Design, Matrix Reasoning). To standardise data across sites, IQ was pro-rated from two verbal subtests (vocabulary and similarities) and two performance subtests (matrix reasoning and

block design) using an algorithm developed by Sattler (1992) that produces an estimated IQ score that is highly correlated ($r = .93$) with a Full-Scale IQ obtained by administering the complete test. Age-appropriate national population norms were available for each participating site and these were used to derive standardised estimates of an individual's intellectual functioning. Where recent IQ scores from previous assessments were available (less than 12 months in children; less than 18 months in adolescents and adults) IQ tests were not repeated.

To assess the influence of intellectual ability, full IQ scores were added as additional covariate of no interest to the second level models assessing brain activation differences between individuals with ASD and TD and autism trait scores.

Results were not significantly impacted by controlling for IQ.

Table S7: Overview over effects of diagnosis on functional brain activation after controlling for intellectual ability.

	TD vs. ASD			Effect of SRS-2		
	whole-brain	left VS	right VS	whole-brain	left VS	right VS
original results (reported in main text)						
anticipation	<i>right VS</i> $F_{(1,384)}=22.84$, $p_{FWE}=.017$	$F_{(1,384)}=14.163$, $p<.001$	$F_{(1,384)}=18.693$, $p<.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,370)}=4.829$, $p=.029$	$F_{(1,370)}=4.719$, $p=.030$	no effect	no effect	no effect
results controlling for IQ						
anticipation	<i>Right VS</i> $F_{(1,381)}=22.14$, $p_{FWE}=.023$	$F_{(1,383)}=14.346$, $p<.001$	$F_{(1,383)}=18.848$, $p<.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,369)}=4.774$, $p=.030$	$F_{(1,369)}=4.684$, $p=.031$	no effect	no effect	no effect

Table provides test statistic of region of interest (ROI) analysis and of significant peak voxel(s) for whole-brain analysis. Voxel-level statistics were family-wise error (FWE) corrected for the number of voxels across the whole brain for each test. Significance was defined as $p_{FWE}<.05$ with a cluster threshold of $k \geq 5$. To correct for investigating left and right VS activity separately in the ROI analysis, critical alpha was adjusted to $p<.025$ based on the Bonferroni procedure. Deviations from results reported in the main text are highlighted in bold. Significant whole-brain results are localized in MNI space and labeled according to the automated anatomical labeling atlas (aal). Abbreviations: TD typically developing, ASD autism spectrum disorder, VS ventral striatum.

Medication

Medication use was confirmed in 82 individuals with ASD and 12 control participants. Most frequently used were psychostimulants and other drugs used to treat ADHD (44.6%), hypnotics/sedatives (31.9%), and antidepressants (24.5%).

Adding a dichotomous covariate indicating medication use (yes/no) to the second level models assessing brain activation did not change the pattern of diagnosis effects on the whole-brain level or within the VS. However, assessing differences between ASD and TD in unmedicated and medicated participants separately yielded no significant whole-brain level and ROI effects, besides a trend for a diagnosis effect in the left VS during reward delivery. This possibly reflects decreased statistical power due to the significantly reduced sample sizes.

Table S8: Overview over effects of diagnosis and age on functional brain activation after controlling for site using the leave-one-out approach.

	TD vs. ASD in unmedicated participants only (n=136)			TD vs. ASD in medicated participants only (n=94)			TD vs. ASD controlled for effect of medication (whole sample)		
	whole-brain	left VS	right VS	whole-brain	left VS	right VS	whole-brain	left VS	right VS
anticipation	no effect	no effect	no effect	no effect	no effect	no effect	<i>Right VS</i> $F_{(1,381)}=22.14$, $p_{FWE}=.023$	$F_{(1,383)}=14.302$, $p<.001$	$F_{(1,383)}=18.525$, $p<.001$
delivery	no effect	$F_{(1,123)}=3.564$, $p=.061$	no effect	no effect	no effect	no effect	no effect	$F_{(1,369)}=4.596$, $p=.033$	$F_{(1,369)}=4.433$, $p=.036$

Table provides test statistic of region of interest (ROI) analysis and of significant peak voxel(s) for whole-brain analysis. Voxel-level statistics were family-wise error (FWE) corrected for the number of voxels across the whole brain for each test. Significance was defined as $p_{FWE}<.05$ with a cluster threshold of $k \geq 5$. To correct for investigating left and right VS activity separately in the ROI analysis, critical alpha was adjusted to $p<.025$ based on the Bonferroni procedure. Deviations from results reported in the main text are highlighted in bold. Significant whole-brain results are localized in MNI space and labeled according to the automated anatomical labeling atlas (aal). Abbreviations: TD typically developing, ASD autism spectrum disorder, VS ventral striatum.

Motion

In order to control for potential effects of head motion on our results, we included mean FD as covariate of no interest in the second level models assessing brain activation. Results were not significantly impacted by controlling for head motion. This suggests that the effect of diagnosis is not driven by the fact that individuals with ASD show partly more movement than TD individuals, for example in the SID (see table 1, main text).

Table S9: Overview over effects of diagnosis on functional brain activation after controlling for head motion

	TD vs. ASD			effect of SRS-2		
	whole-brain	left VS	right VS	whole-brain	left VS	right VS
original results (reported in main text)						

anticipation	<i>right VS</i> $F_{(1,384)}=22.84$, $p_{FWE}=.017$	$F_{(1,384)}=14.163$, $p<.001$	$F_{(1,384)}=18.693$, $p<.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,370)}=4.829$, $p=.029$	$F_{(1,370)}=4.719$, $p=.030$	no effect	no effect	no effect
Controlling for head motion						
anticipation	<i>Right VS:</i> $F_{(1,383)}=21.75$, $p_{FWE}=.027$	$F_{(1,382)}=13.630$, $p<.001$	$F_{(1,382)}=18.066$, $p<.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,368)}=6.762$, $p=.010$	$F_{(1,368)}=6.221$, $p=.013$	no effect	no effect	no effect

Table provides test statistic of region of interest (ROI) analysis and of significant peak voxel(s) for whole-brain analysis. Voxel-level statistics were family-wise error (FWE) corrected for the number of voxels across the whole brain for each test. Significance was defined as $p_{FWE}<.05$ with a cluster threshold of $k\geq 5$. To correct for investigating left and right VS activity separately in the ROI analysis, critical alpha was adjusted to $p<.025$ based on the Bonferroni procedure. Deviations from results reported in the main text are highlighted in bold. Significant whole-brain results are localized in MNI space and labeled according to the automated anatomical labeling atlas (aal). Abbreviations: TD typically developing, ASD autism spectrum disorder, VS ventral striatum.

Sex

All analyses were controlled for sex. However, we additionally explored (1) whether ASD diagnosis effects interacted with sex and (2) if we could replicate our findings in males and females separately.

We found no significant interaction between sex and diagnosis at the whole-brain level or for the right VS. In the left VS, a trend-level significant interaction between diagnosis and sex emerged for reward delivery. Post hoc Bonferroni corrected pairwise comparisons yielded increased left VS activity during reward delivery only in males with ASD compared to male TD ($p=.004$) while in females there was no significant difference between ASD and TD ($p=.709$). Similarly, we could only replicate the effect of diagnosis during reward delivery in male participants ($n=272$) but not female participants ($n=121$). This finding likely suggests a lack of statistical power in the female sample, but warrants further inspection in future studies.

Table S10: Overview over effects of diagnosis and age on functional brain activation in male and female participants separately.

	TD vs. ASD			effect of SRS-2		
	whole-brain	left VS	right VS	whole-brain	left VS	right VS
sex*diagnosis interaction						
anticipation	no effect	no effect	no effect		no effect	no effect
delivery	no effect	$F_{(1,369)}=3.885$, $p=.049$	no effect		no effect	no effect
original results (reported in main text)						
anticipation	<i>right VS</i> $F_{(1,384)}=22.84$,	$F_{(1,384)}=14.163$, $p<.001$	$F_{(1,384)}=18.693$, $p<.001$	no effect	no effect	no effect

	$p_{FWE}=.017$					
delivery	no effect	$F_{(1,370)}=4.829,$ $p=.029$	$F_{(1,370)}=4.719,$ $p=.030$	no effect	no effect	no effect
only male participants (n=272)						
anticipation	no effect	$F_{(1,264)}=9.017,$ $p=.003$	$F_{(1,264)}=13.509,$ $p<.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,252)}=8.632,$ $p=.004$	$F_{(1,252)}=7.854,$ $p=.005$	no effect	no effect	no effect
only female participants (n=121)						
anticipation	no effect	$F_{(1,113)}=6.352,$ $p=.013$	$F_{(1,113)}=6.589,$ $p=.012$	no effect	no effect	no effect
delivery	no effect	no effect	no effect	no effect	no effect	no effect

Table provides test statistic of region of interest (ROI) analysis and of significant peak voxel(s) for whole-brain analysis. Voxel-level statistics were family-wise error (FWE) corrected for the number of voxels across the whole brain for each test. Significance was defined as $p_{FWE}<.05$ with a cluster threshold of $k \geq 5$. To correct for investigating left and right VS activity separately in the ROI analysis, critical alpha was adjusted to $p < .025$ based on the Bonferroni procedure. Deviations from results reported in the main text are highlighted in bold. Significant whole-brain results are localized in MNI space and labeled according to the automated anatomical labeling atlas (aal). Abbreviations: TD typically developing, ASD autism spectrum disorder, VS ventral striatum.

Handedness

The majority (68.9%, $n=271$) of participants were right handed. However, to rule out any impact of handedness on brain activation we repeated the main analyses in righthanded participants only, replicating group differences during reward anticipation and delivery.

Table S11: Overview over effects of diagnosis and age on functional brain activation in righthanders ($n=271$).

	TD vs. ASD			effect of SRS-2		
	whole-brain	left VS	right VS	whole-brain	left VS	right VS
anticipation	no effect	$F_{(1,263)}=10.161,$ $p=.002$	$F_{(1,263)}=12.159,$ $p=.001$	no effect	no effect	no effect
delivery	no effect	$F_{(1,254)}=4.529,$ $p=.034$	$F_{(1,254)}=5.511,$ $p=.020$	no effect	no effect	no effect

Table provides test statistic of region of interest (ROI) analysis and of significant peak voxel(s) for whole-brain analysis. Voxel-level statistics were family-wise error (FWE) corrected for the number of voxels across the whole brain for each test. Significance was defined as $p_{FWE}<.05$ with a cluster threshold of $k \geq 5$. To correct for investigating left and right VS activity separately in the ROI analysis, critical alpha was adjusted to $p < .025$ based on the Bonferroni procedure. Deviations from results reported in the main text are highlighted in bold. Significant whole-brain results are localized in MNI space and labeled according to the automated anatomical labeling atlas (aal). Abbreviations: TD typically developing, ASD autism spectrum disorder, VS ventral striatum.

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