showed 51% of functional activity altered (61% was hypoactivity). The ventrolateral prefrontal cortex (VLPFC) presented 28% of aberrant fMRI (75% was hyperactivity). Thalamus presented alteration activity with 20% (71% was hypoactivity). The Cingulate was also altered during activation with 20% of patients (85% was hypoactivity). Finally, functional alterations of the Amygdala were present in 14% of the selected patients (80% was hypoactivation).

Discussion: This larger review suggests that there are several possible areas apart from fronto temporal pathways (Mwansisya et al., 2017), that have to be taken into account at the early course of psychosis, such as lymbic system, thalamo-cortical networks and cingulate. These functional activation abnormalities seem to be different to the reported in the previous review. The different results seem to be clearly influenced by the kind of paradigm. Moreover, our finding is not in concordance with the suggestion that thalamic alterations became only prominent at the chronic phase of psychosis (Li et al., 2017).

References:

1. Moher, D et al., Preferred reporting items for systematic reviews and meta-analyses: The Prisma statement. PLoS Med, 2009, 6, 1000097.
2. Mwansisya, T et al., Task and resting-state fMRi studies in first-episode schizophrenia: A systematic review. Schizophrenia Research article in press, 2017.
3. Li, T et al., Brain-wide analysis of functional connectivity in first episode and chronic stages of schizophrenia: Schizophrenia Bulletin 2016, 43, 436–448.

S154. THE ROLE OF DOPAMINE IN PROCESSING THE MEANINGFUL INFORMATION OF OBSERVATIONS, AND IMPLICATIONS FOR THE ABERRANT SALIENCE HYPOTHESIS OF SCHIZOPHRENIA

Matthew Nour*[,1], Tarik Dahoun[2], Philipp Schwartenbeck[3], Rick Adams[4], Thomas FitzGerald[5], Christopher Coello[6], Matthew Wall[7], Raymond Dolan[3], Oliver Howes[8]
[1]King's College London, Institute of Psychiatry; [2]Imperial College London, Institute of Clinical Sciences; [3]University College London, Wellcome Trust Centre for Neuroimaging and Max Planck UCL Centre for Computational Psychiatry and Ageing Research; [4]University College London, Institute of Cognitive Neuroscience and Division of Psychiatry; [5]University of East Anglia; [6]University of Oslo, Institute of Basic Medical Sciences; [7]Imanova Centre for Imaging Sciences, Hammersmith Hospital; [8]King's College London, Institute of Psychiatry, Imperial College London, Institute of Clinical Sciences, MRC London Institute of Medical Sciences

Background: The aberrant salience hypothesis of schizophrenia proposes that symptoms such as paranoia arise when behavioural salience is attributed to neutral stimuli. Mesolimbic dopamine dysfunction is thought to be central to this mechanism; building on findings that activity in this pathway conveys a (signed) reward prediction error signal. Given that many psychotic symptoms are not explicitly related to reward learning, it is relevant that recent studies in rodents have demonstrated a role for midbrain dopamine neurons in value-neutral associative learning. Direct evidence for this role in humans, however, is lacking.

In this study we asked whether the mesolimbic dopamine circuit is involved in encoding the value-neutral meaningful information of observations, using a model-based functional magnetic resonance imaging (fMRI) task and dopamine positron emission tomography (PET). We define 'meaningful information' as the degree to which an observation results in a belief-update to an agent's internal model of the environment (Kullback-Leibler divergence from prior to posterior beliefs; 'Bayesian surprise').

Methods: Participants were tasked to infer the current (hidden) state of the environment, using partially-informative observations at each trial, and then report their belief at the end of each trial. Participant beliefs were modelled using a Hidden Markov Model of the task and iterative application of Bayes' rule, allowing us to quantify the Bayesian surprise (meaningful information content) associated with a trial observation. Crucially, our task de-correlated Bayesian surprise from both the pure sensory unexpectedness of an observation (unexpected but meaningless information) and its signed reward prediction error. 39 healthy participants (22M, mean age 26y) performed 180 task trials within an fMRI scanner. 36 participants also had a [11C]-(+)-4-propyl-9-hydroxy-naphthoxazine (PHNO) PET scan to quantify dopamine-2/3 receptor (D2/3R) availability. 17 participants additionally had a second PET scan 3hrs post 0.5mg/kg oral dexamphetamine, to quantify striatal dopamine release capacity. Neuroimaging analyses were restricted to the bilateral substantia nigra/ventral tegmental area (SN/VTA) and ventral striatum (VS).

Results: Our computational model closely predicted participant behaviour (R2= .67), and there was a negative correlation between subclinical paranoia and the degree to which participant behaviour approximated normative Bayesian performance (rho = -.60, P<0.001). Neuronal activation encoding the meaningful information content of an observation (Bayesian surprise) was present in SN/VTA and VS (both P(peak)<0.05, SVC), whereas no such encoding was present for sensory unexpectedness or reward-prediction error. Crucially, activation encoding Bayesian surprise was inversely correlated with D2/3R availability in the SN/VTA (rho = -.43, P=0.009), consistent with a tonic inhibitory role for midbrain D2/3Rs. Moreover, activation encoding Bayesian surprise was inversely related to dopamine release capacity in the VS (rho = -.66, P=0.005), indicating that subjects with high dopamine release capacity showed blunted striatal activation in response to belief-changing information, as is also found in schizophrenia.

Discussion: We provide direct evidence in humans that a mesolimbic dopamine circuit is involved in encoding the meaningful information content of observations, distinct from its involvement in processing signed reward prediction error. These results implicate dopamine in a wider range of function than reward learning, including updating a predictive associative model of the world, and are therefore relevant for the aberrant salience hypothesis of schizophrenia.

S155. SENSORY ATTENUATION DURING AUDITORY PROCESSING IN PARTICIPANTS AT CLINICAL-HIGH RISK FOR PSYCHOSIS: EVIDENCE FROM MAGNETOENCEPHALOGRAPHY

Lingling Hua*[,1], Marc Recasens[2], Tineke Grent-'t-Jong[3], Emmi Mikanmaa[2], Hanna Thuné[2], Andrew Gumley[2], Matthis Schwannauer[4], Stephen Lawire[4], Ruchika Gajwan Gajwan[2], Joachim Gross[2], Peter Uhlhaas[2]
[1]School of Psychology; [2]University of Glasgow; [3]University of Glasgow, Institute of Neuroscience and Psychology, Centre for Cognitive NeuroImaging, School of Psychology; [4]University of Edinburgh

Background: The ability to predict the sensory feedback of self-generated stimuli against incoming sensory information is of importance to distinguish internal from external stimuli and is associated with sensory attenuation. Furthermore, it has been proposed that deficits in sensory attenuation could contribute to clinical symptoms of schizophrenia, including hallucinations and delusions, involving potential deficits in corollary discharge. The current study examined the hypothesis whether sensory attenuation is present in participants at clinical high–risk (CHR) for psychosis.

Methods: Sixty-four CHR-participants and 32 healthy controls were presented with auditory stimuli during two experimental conditions: 1) In a