dependence of auditory evoked potential (LDAEP) is a suitable biomarker of inhibitory action in signal processing. Variations in response inhibition can have great impact on different aspects of life. Individuals with reduced capability of inhibitory control have a tendency to impulsive behavior. Studies showed that they have stronger LDAEP values. Patients with schizophrenia may exhibit alterations in the responsiveness to sensory stimuli. Thus, a reduced LDAEP was found in these patients. However, these deviances differed in clinical features of the disorder. Therefore, we would like to further elucidate the relationship between multimodal neuroimaging methods and dimensions of symptoms, observable behavior, personality traits and general psychopathological dysfunction.

Methods A sample of 20 healthy controls and 20 patients with manifest schizophrenia will be examined with the LDAEP paradigm in a trimodal approach with customary imaging tools. PET measurements with the radiotracer [11C]-flumazenil will be used to assess the binding potentials of GABA-A receptors. MRS will provide data about GABA concentrations. Simultaneously recorded EEG-fMRI data will permit new insight in the relationship between LDAEP and impulsivity.

Discussion The project will use alternative approaches to psychiatric classification. Response inhibition in sensory processing will be investigated from different angles (biochemical, neurophysiological, and neuroanatomical) and combined with psychological characteristic values.

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Obsessive-compulsive disorder

FC48

Actions speak louder than words: Enhanced action tendencies in obsessive-compulsive disorder: An ERP study

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Obsessive-compulsive disorder (OCD) is characterized by repeated thoughts and behaviors. Several studies have detected deficient response inhibition ability in individuals with OCD, leading researchers to suggest this deficit as an endophenotype of OCD. However, other researchers maintain that the effect size of this deficit is modest and that it lacks clinical significance. The current investigation examines a potential alternative explanation for difficulties in response inhibition, namely enhanced action tendencies in response to stimuli. Therefore, early processes of motor response preparation preceding action performance (or inhibition) were studied with the event-related potential (ERP) component of readiness potential (RP). RP measures brain reactions related to motor activity in response to external stimuli. ERPs were recorded while 15 participants with OCD and 16 healthy controls performed a variation of a go/no-go task and a stop-signal task using schematic faces (angry and neutral). The OCD group presented with a greater RP slope gradient and amplitude over bilateral parietal areas corresponding to the motor cortex. The amplitude effect was further enhanced under negative valence, compared with the neutral condition. Differences in RP between the OCD and control groups remained significant when controlling for levels of trait anxiety. Results support the hypothesis that a stronger readiness for action might characterize OCD, especially in the presence of threatening stimuli. This finding, specific to OCD and not to anxiety symptoms, may underlie habitual tendencies in OCD. This study suggests that early-stages of motor preparation might be important to the etiology and maintenance of OCD.

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Pain and treatment options

FC49

The net suppression effect of pain catastrophic cognition on anxiety sensitivity

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Introduction The existing literature on chronic pain points to the effects anxiety sensitivity, pain hypervigilance, and pain catastrophizing on pain-related fear; however, the nature of the relationships remains unclear. The three dispositional factors may affect one another in the prediction of pain adjustment outcomes. The addition of one disposition may increase the association between another disposition and outcomes, a consequence known as suppressor effects in statistical terms.

Objective This study examined the possible statistical suppressor effects of anxiety sensitivity, pain hypervigilance and pain catastrophizing in predicting pain-related fear and adjustment outcomes (disability and depression).

Methods Chinese patients with chronic musculoskeletal pain ($n = 401$) completed a battery of assessments on pain intensity, depression, anxiety sensitivity, pain vigilance, pain catastrophizing, and pain-related fear. Multiple regression analyses assessed the mediating/moderating role of pain hypervigilance. Structural equation modeling (SEM) was used to evaluate suppression effects.

Results Our results evidenced pain hypervigilance mediated the effects of anxiety sensitivity (Model 1: Sobel $z = 4.86$) and pain catastrophizing (Model 3: Sobel $z = 5.08$) on pain-related fear. Net suppression effect of pain catastrophizing on anxiety sensitivity was found in SEM where both anxiety sensitivity and pain catastrophizing were included in the same full model to predict disability (Model 9: CFI = 0.95) and depression (Model 10: CFI = 0.93) (all $P < 0.001$) (see Tables 1 and 2, Figs. 1 and 2).

Conclusions Our findings evidenced that pain hypervigilance mediated the relationship of two dispositional factors, pain catastrophic cognition and anxiety sensitivity, with pain-related fear. The net suppression effects of pain catastrophizing suggest that anxiety sensitivity enhanced the effect of pain catastrophic cognition on pain hypervigilance.

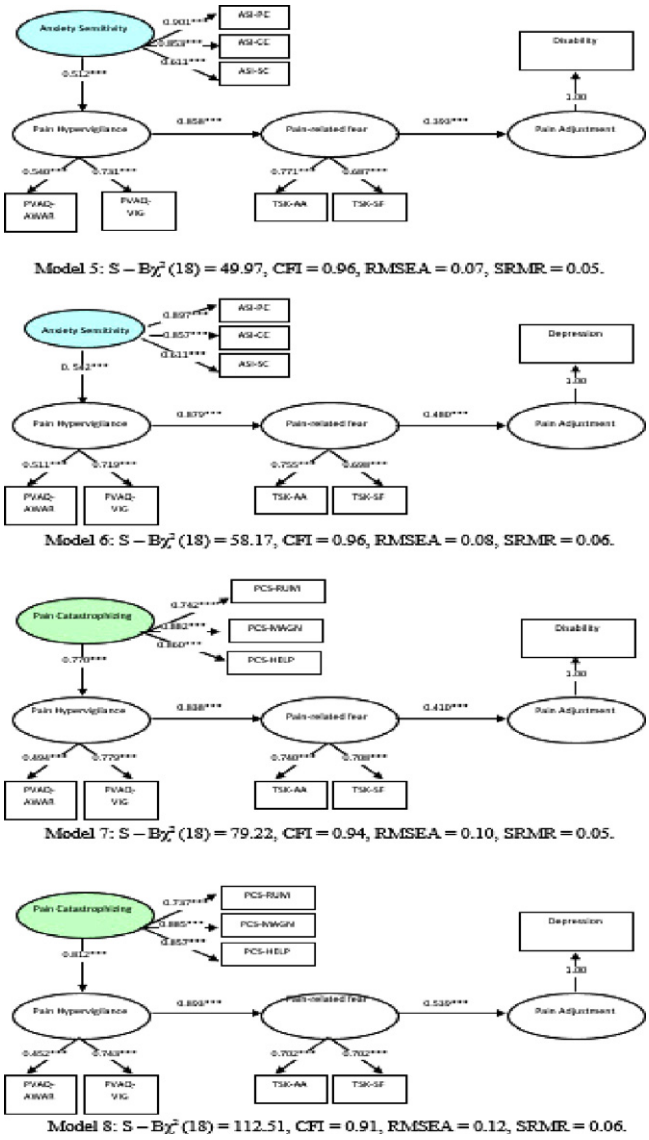

Fig. 1 Simple models testing the mediating role of pain hypervigilance in the link between anxiety sensitivity and pain-related fear (Models 7 and 8) predicting disability and depression. Anxiety Sensitivity was indexed by the Anxiety Sensitivity Index (ASI). PC: ASI Physiological Concerns subscale; CC: ASI Physiological Concerns subscale; SC: ASI Social Concerns subscale. Pain catastrophizing was indexed by the Pain Catastrophizing scale (PCS). RUM: PCS Rumination subscale; MAGN: PCS Magnification subscale; HELP: PCS Helplessness subscale. Pain hypervigilance was indexed by the Pain Vigilance and Awareness Questionnaire (PVAQ). AWAR: PVAQ Passive Awareness subscale; VIG: PVAQ Active Vigilance subscale. Pain-related fear was indexed by the Tampa Scale for Kinesiophobia (TSK). AA: TSK Activity Avoidance subscale; SF: TSK Somatic Focus. Disability was indexed by the Chronic Pain Grade Disability score. Depression was indexed by the Depression subscale of the Hospital Anxiety and Depression scale. S–$B_X{}^2$: Satorra and Bentler scaled Chi^2 statistic; CFI: comparative fit index; RMSEA: root mean square error of approximation; SRMR: standardized root mean square residual. $^{***}P < 0.001$.

Table 1 Multivariate regression analyses of the relationships between pain hypervigilance, anxiety sensitivity, pain-related fear.

Model	β	SE	95% CI	P value
Model 1ª: Anxiety sensitivity → Pain hypervigilance → Pain-related fear				
Anxiety sensitivity (Predictor) → Pain hypervigilance (Mediator)	0.25	0.04	0.17, 0.34	<0.001
Pain hypervigilance (Mediator) → Pain-related fear (Outcome)	0.19	0.02	0.15, 0.23	<0.001
Anxiety sensitivity (Predictor) → Pain-related fear (Outcome)	0.15	0.02	0.12, 0.18	<0.001
Anxiety sensitivity (Predictor) → Pain-related fear (Outcome) | Pain hypervigilance (Mediator)ᵇ	0.11	0.02	0.08, 0.14	<0.001
Sobel test	$Z = 4.86$		$P < 0.001$	
Model 2ª: (Anxiety sensitivity × Pain hypervigilance) → Pain-related fear				
Anxiety sensitivity (Predictor)	0.12	0.02	0.09, 0.15	<0.001
Pain hypervigilance (Moderator)	0.15	0.02	0.11, 0.18	<0.001
Anxiety sensitivity (Predictor) × Pain hypervigilance (Moderator)	-0.00	0.00	-0.00, 0.00	0.186
Model 3ª: Pain catastrophizing → Pain hypervigilance → Pain-related fear				
Pain catastrophizing (Predictor) → Pain hypervigilance (Mediator)	0.47	0.05	0.38, 0.57	<0.001
Pain hypervigilance (Mediator) → Pain-related fear (Outcome)	0.18	0.02	0.15, 0.22	<0.001
Pain catastrophizing (Predictor) → Pain-related fear (Outcome)	0.21	0.02	0.17, 0.24	<0.001
Pain catastrophizing (Predictor) → Pain-related fear (Outcome) | Pain hypervigilance (Mediator)ᵇ	0.15	0.02	0.11, 0.19	<0.001
Sobel test	$Z = 5.08$		$P < 0.001$	
Model 4ª: (Pain catstrophizing × Pain hypervigilance) → Pain-related fear				
Pain catastrophizing (Predictor)	0.15	0.02	0.11, 0.19	<0.001
Pain hypervigilance (Moderator)	0.11	0.02	0.07, 0.15	<0.001
Pain catastrophizing (Predictor) × Pain hypervigilance (Moderator)	-0.00	0.00	-0.01, 0.00	0.068

Note. β: Unstandardized beta coefficient; SE: standard error; CI: confidence interval; NS: non-significant P value at 0.05 level. All regression equations were controlled for age, sex, number of pain site, and pain duration. The total scores of the measurement scale of anxiety sensitivity, pain hypervigilance and pain-related fear were used in all regression models.
ªFour separate regression models were generated to test the mediation pathway of pain hypervigilance on the link between anxiety sensitivity and pain-related fear (Model 1) and the link between pain catastrophizing and pain-related fear (Model 3).
ᵇPain hypervigilance, as mediator, was controlled in the regression equation.
ᶜUsing pain-related fear as dependent variable, one regression model was generated to test the moderation pathway of pain hypervigilance on the link between anxiety sensitivity and pain-related fear (Model 2) and the link between pain catastrophizing and pain-related fear (Model 4).

Table 2 Results of SEM testing the relationships between anxiety sensitivity, pain catastrophizing, and pain hypervigilance for two pain adjustment outcomes.

Model	S–$B\chi^2$	*df*	CFI	NNFI	RMSEA	90% CI	SRMR
Simple model: Anxiety sensitivity → Pain hypervigilance → Pain-related fear → Adjustment outcome							
Model 5: Disability	49.97	18	0.96	0.94	0.07	0.05, 0.10	0.05
Model 6: Depression	58.17	18	0.96	0.93	0.08	0.06, 0.10	0.06
Simple model: Pain catastrophizing → Pain hypervigilance → Pain-related fear → Adjustment outcome							
Model 7: Disability	79.22	18	0.94	0.91	0.10	0.08, 0.12	0.05
Model 8: Depression	112.51	18	0.91	0.96	0.12	0.10, 0.15	0.06
Full model							
Model 9: Disability	122.24	40	0.95	0.93	0.08	0.06, 0.09	0.05
Model 10: Depression	154.49	40	0.93	0.91	0.09	0.08, 0.11	0.06

Note: The full models include both anxiety sensitivity and pain catastrophizing, and specify that pain hypervigilance mediates the link of both anxiety sensitivity and catastrophizing with pain-related fear, which in turn predicts adjustment outcomes. Disability was indexed by the Chronic Pain Grade Disability score; Depression was indexed by the Depression subscale of the Hospital Anxiety and Depression Scale; S–$B\chi^2$ = Satorra & Bentler scaled chi-square statistics; *df* = degrees of freedom; CFI = comparative fit index; NNFI = non-normed fit index; RMSEA = root mean square error of approximation; CI = confidence interval; SRMR = standardized root mean square residual.

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Personality and personality disorders

FC50

Prevalence, mortality and healthcare utilization of cluster B personality disorders in Quebec: A province cohort study, 2001–2012

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Background Cluster B personality disorder (PD) is a highly prevalent mental health condition in general population (1 to 6% depending on the subtype and study). Patients affected are known to be heavier users of both mental and medical healthcare than other clinical conditions such as depression. Few studies have highlighted their elevated mortality rate compared to general population.