

Cognitive ability and motivation in a new effort-based decision-making task testing  
healthy participants showing subclinical symptoms of schizophrenia

Thea Simensen, NTNU- Norges teknisk-naturvitenskapelige universitet  
Master thesis in psychology. Learning- brain, behaviour, environment

Supervisor: Gerit Pfuhl

May 2019



## Table of contents

Preface .....	v
Abstract.....	vii
Abstrakt.....	ix
Introduction.....	1
Motivation as effort-cost computations .....	1
Mental illnesses as a continuum.....	3
Psychotic symptoms as a continuum and the transition to disorder .....	4
Psychotic disorders: schizophrenia and bipolar disorder.....	4
Positive symptoms .....	6
Cognitive symptoms.....	6
Affective symptoms .....	7
Motivational deficits and affective symptoms. ....	7
Negative symptoms.....	7
Motivational deficits in negative symptoms. ....	8
Effort-based decision making .....	8
New method measuring effort-based decision-making.....	10
The current study.....	12
Methods .....	13
Participants .....	13
Materials .....	14
Neurocognitive tests: Trail Making Test A and B, and Digit Symbol Substitution Test.....	14
Precision and Motivation Task. ....	14
CAPE-42.....	16
Procedures .....	17
Ethics.....	18
Statistical analysis .....	19
Results.....	19
Participants .....	19
CAPE-42.....	20

Neurocognitive tests.....	20
TMT A and B.....	21
DSST.....	21
Precision and motivation task.....	21
Visual short-term memory and metacognitive precision.....	21
Activational motivation.....	22
Directional motivation.....	23
Subjective cost.....	24
Discussion.....	25
Precision and Motivation Task.....	26
Activational and directional motivation.....	26
Visual short-term memory and metacognitive precision.....	27
Subjective cost.....	28
Neurocognitive tests.....	30
The CAPE.....	31
Limitations.....	33
Conclusion.....	34
References.....	35
Appendix A. Demographics form.....	45
Appendix B. Procedures precision and motivation task.....	46
Appendix C. Approval from REK.....	54
Appendix D. Consent form.....	61

## Preface

As motivation is a part of all human behaviour it seemed exciting to me to be able to learn about this topic and perhaps to be able to contribute to this field. Further, after learning that motivational deficits in schizophrenia currently is hard to treat it made the project even more meaningful to take on. My supervisor Gerit Pfuhl already had some ideas about the project and had a research question in place. In her lab research are being done to try to identify what contributes to deliberate reasoning and rational behaviour by exploring cognitive effort, motivation and attributing uncertainties. A study similar to mine, but with schizophrenic patients was performed by a former student in 2017.

During this project I have learned a great deal. Firstly, I got introduced to preregistration, an aspect of scientific research that was new to me. By writing the preregistration in close guidance by Gerit, I got a better overview of what we were going to do and why we were doing it, by reading about motivation, schizophrenia, effort-based decision-making and mental illnesses as a continuum. Gerit helped with the specifics such as design and analysis plan. Secondly, I got to learn about recruiting and data collection and the challenges associated with this process. To recruit participants, I travelled to Tromsø, Trondheim, Oslo, Østfold and Buskerud and I contacted several institutions to ask for help spreading information about the project. I also administered the tests and made the consent form.

I want to thank my supervisor Gerit for guiding me through this process, for helping me a great deal with the data analysis and for giving me advice and support when I needed it. I also want to thank everyone who participated in the study and to those who helped me recruit participants by sharing information about my project.



### Abstract

Effort-based decision-making have been shown to be closely linked to the motivational deficits seen in negative symptoms in schizophrenia and other psychotic disorders, and several paradigms have been developed to be able to more objectively measure motivation and effort. These tasks measure how much effort one is willing to exert across different reward and probability conditions. However, there is substantial research suggesting that cognitive deficits affect these processes, and current paradigms do not consider how cognitive functioning may affect effort-based decision-making. In addition, previous research supports that individuals with schizophrenia show deficits in effort-cost computations, but less is known about individuals with subclinical symptoms of schizophrenia. By studying healthy individuals, one might get insight into behavioural, clinical and environmental risk and protective factors associated with the development of the disorder and to be able to limit the progression of illness. The aim of this study was to identify which of the several symptoms in psychotic disorders or schizophrenia contribute the most to the lack of motivation and goal-directed behaviour in a non-clinical sample. A recently developed effort-based decision-making paradigm was used, controlling for neurocognitive ability, metacognitive precision, probability and subjective cost. Fifty-three healthy individuals from the general population participated in the study. To measure subclinical symptoms, the participants completed the Community Assessment of Psychic Experiences. Our results showed that individuals scoring highly on one symptom dimension, scored highly on the other symptom dimensions as well, making it difficult to test our hypothesis properly. Nevertheless, elevated degrees of negative symptoms were associated with less effort. Interestingly, it was positive symptoms and not negative symptoms that was associated with neurocognitive deficits. Thus, in our study, deficits in neurocognition did not account for the reduced effort. Further, the cost of performing the task was the same for all participants, regardless of symptom severity. Lastly, all participants were rational in their decision making, i.e. they reduced their effort as the probability decreased. Due to the overlap of symptoms, more research is needed to see which of several symptoms in schizophrenia contribute the most to the lack of motivation and goal-directed behaviour.

**Keywords:** Schizophrenia, subclinical symptoms, neurocognition, effort-cost computations, motivation, mental illness as a continuum.





### Abstrakt

Det har blitt vist at anstrengelsesbasert beslutningstaking er tett assosiert med motivasjonsproblemer sett i negative symptomer hos personer med schizofreni og andre psykoselidelser. På bakgrunn av dette har det blitt utviklet flere paradigmer designet for å måle motivasjon og anstrengelse mer objektivt. Disse paradigmene måler hvor mye innsats en person er villig til å gi på bakgrunn av belønning og sannsynlighet for å få belønning. Det er imidlertid mye forskning som viser at problemer med kognisjon ofte assosiert med negative symptomer påvirker anstrengelsesbasert beslutningstaking, og paradigmer som blir brukt i dag tar ikke kognitiv fungering i betraktning. Mye forskning viser at personer med schizofreni har problemer i anstrengelsesbasert beslutningstaking, men det er mindre forskning som viser om denne effekten også finnes hos personer som viser subkliniske symptomer i schizofreni. Ved å studere friske individer som viser symptomer kan man få innsikt i atferdsmessige, kliniske og miljømessige beskyttende- og risikofaktorer assosiert med utviklingen av lidelsen, og muligens å kunne hindre utviklingen. Målet med denne studien var å identifisere hvilke av symptomene i schizofreni eller psykoselidelser som bidrar mest til manglende motivasjon og målrettet atferd i et ikke-klinisk utvalg. Et nylig utviklet paradigme som kontrollerer for kognitiv evne, metakognisjon, sannsynlighet for belønning og subjektiv kostnad ble brukt for å måle anstrengelsesbasert beslutningstaking og motivasjon i 53 friske deltakere. For å måle subkliniske symptomer ble «the Community Assessment of Psychic Experiences» brukt. Resultatene viste at personer som skåret høyt på ett symptom også skåret høyt på de andre symptomene, noe som gjorde det vanskelig å teste hypotesene våre ordentlig. Likevel viste resultatene at mengden negative symptomer var assosiert med mindre anstrengelse. Interessant nok fant vi at det var graden av positive symptomer og ikke negative symptomer som viste en sammenheng med dårligere testskårer på kognitive tester. Med andre ord var det ikke forstyrrelser i kognitiv funksjon som stod for den lavere anstrengelsen sett hos de med negative symptomer. Videre fant vi at den subjektive kostnaden for å utføre oppgaven var lik for alle deltakere, uavhengig av symptomgrad. Til sist fant vi at alle deltakere var rasjonelle i beslutningstakingen, sett ved at de reduserte anstrengelsen når sannsynligheten for belønning ble lavere. På grunn av overlappet mellom symptomene trengs det mer forskning for å identifisere hvilke symptomer i schizofreni eller psykoselidelser som bidrar mest til manglende motivasjon og målrettet atferd.

Nøkkelord: Schizofreni, subkliniske symptomer, kognisjon, anstrengelsesbasert beslutningstaking, motivasjon, mentale lidelser som et kontinuum



What makes people motivated? There are several theories and definitions of motivation as well as factors affecting it. Motivation can be defined as the energizing of behaviour in pursuit of goals and is a fundamental property of all behaviours (Simpson & Balsam, 2015). It starts with a goal and one needs a cognitive representation of the end-state of that goal, of what is to be acquired, achieved or conducted (Austin & Vancouver, 1996). One may or may not be aware of the goal, it may be implicit or explicit but without motivation all behaviour would be random. Motivation may also be defined as the process of overcoming the costs of effortful actions to achieve desired outcomes (Chong, Bonnelle, & Husain, 2016). This is a complex phenomenon influenced by cultural, developmental and environmental factors and is further challenging to study due to interindividual variability, ranging from highly motivated healthy individuals to patients with disorders of motivation such as apathy (Chong et al., 2016).

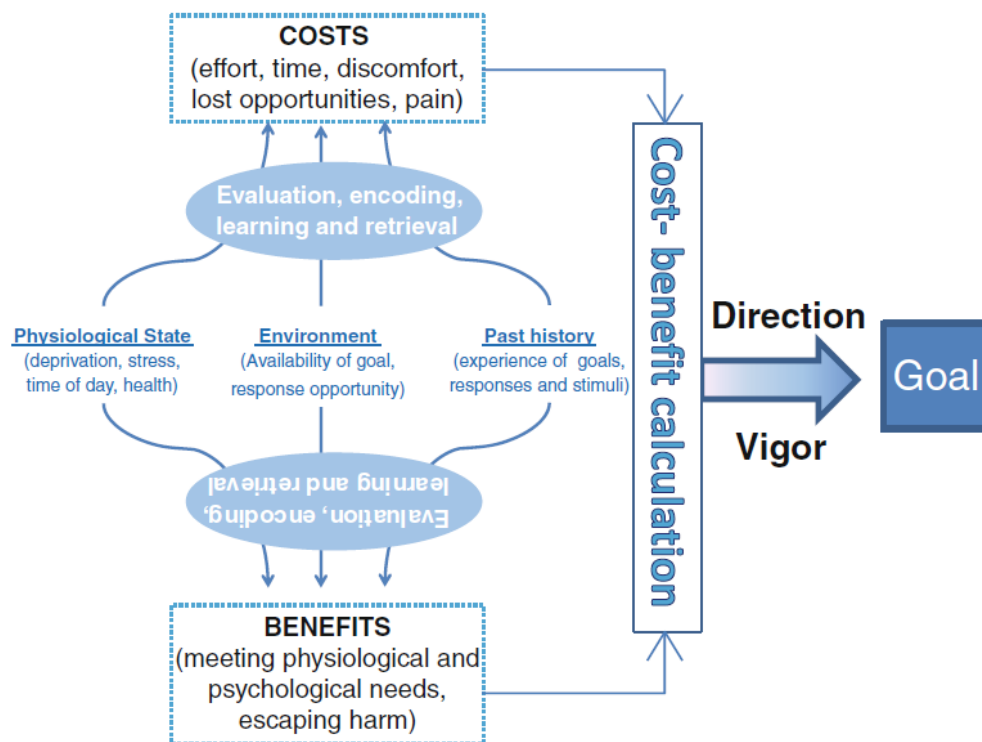
Studies on motivational deficits indicate that there is disruption in mechanisms underlying how rewards are processed to motivate behaviour (Barch, Pagliaccio, & Luking, 2016). To measure motivation in illness and health self-report measures and clinical interviews have traditionally been used, but these methods have several shortcomings such as the lack of ability to provide mechanistic explanations of the underlying processes of motivation (Chong et al., 2016). To address these shortcomings new paradigms have been developed such as effort-based decision-making tasks, measuring how much effort one is willing to exert for rewards (Green, Horan, Barch, & Gold, 2015). The aim of this thesis was to assess motivation seen as effort-cost computations in relation to motivational deficits in mental illnesses, more specifically negative symptoms in schizophrenia and other psychotic disorders. In the next section, literature on motivation as effort-cost computations will be reviewed, before a review on symptoms seen in schizophrenia and other psychotic disorders, especially how symptoms may be seen along a continuum. Further, we will see how different symptoms in schizophrenia relate to motivational deficits. Lastly, there will be a presentation of effort-based decision-making tasks before the current study is presented.

### **Motivation as effort-cost computations**

Motivation is conceptualized to consist of an arousal, activational component and a goal directed, directional component (Duffy, 1957; Hebb, 1955). Directional motivation is referring to that motivated behaviour is usually directed towards certain kinds of stimuli (e.g. pleasurable activities or food) and away from other types of stimuli (e.g. dangerous activities

or stress). Activational motivation refers to that motivated behaviour is characterized by behavioural activation, manifested by speed, vigour or persistence observed in initiation and maintenance of the behaviour (Salamone & Correa, 2012). Motivation can either be intrinsic, driven by an interest in and enjoyment of goals and activities for their own sake in absence of external rewards, or extrinsic, driven by attaining external rewards (Ryan & Deci, 2000).

Several factors influence motivation, such as the individuals internal physiological state, past history, experiences and the current environmental condition (Simpson & Balsam, 2015). Information about these factors must be processed in different ways to affect motivation; it must be encoded and evaluated, something that is influenced by retrieval processes and learning (see figure 1). The costs associated with behaviour might include mental or physical effort, discomfort, time, danger and loss of potential opportunities. The benefits associated with behaviour may include fulfilling psychological and physiological needs, obtaining reinforcement related to those needs, avoidance of costs or escaping from harm. The value of these costs and benefits must be encoded and calculated. After encoding



*Figure 1.* A simplified figure of processes that are involved in and factors influencing motivation by Simpson and Balsam (2015), in which cost-benefit analyses are central. The individual's past history, physiological state and the environment influence motivation, and these factors undergo several processes (represented in the blue circle). Information will undergo retrieval and learning processes, resulting in a cost-benefit calculation that will affect direction and vigour of action towards a goal.

the benefits and costs, a cost-benefit computation determines the appropriate vigour and direction of behaviour. For example, Students must decide how much to study for an exam based on the subjective importance of doing well (intrinsic motivation), and employees decide how much effort to put into their jobs based on their wage (extrinsic motivation).

Several disorders are associated with amotivation such as schizophrenia and major depressive disorder (Pelizza & Ferrari, 2009) and is often referred to in the context of anhedonia (Thomsen, Whybrow, & Kringelbach, 2015) or negative symptoms (Blanchard & Cohen, 2005). However, motivational problems are not only seen in patients, it is likely to be on a continuum (Chong & Husain, 2016). One might find healthy individuals struggling with some aspects of motivation, i.e. an individual might have high social motivation, but struggle with procrastinating school or work. The alterations in motivation play a major role in clinical outcome and functioning (Barch, Treadway, & Schoen, 2014; Strauss, Horan, et al., 2013). In addition to being closely linked to functional outcome, motivational deficits may be seen early in the progression of psychotic disorders, making it crucial to be able to treat. However, the underpinnings of motivational deficits remain unclear.

### **Mental illnesses as a continuum**

Mental illnesses have recently gained more attention due to the burden they have on the individual and society. Poor mental health does not only affect patients diagnosed with a mental illness. People in the general population also express symptoms of mental illness, but below the threshold of clinical significance (Caspi et al., 2014). Thus, as with motivation, mental illnesses may be seen along a continuum. Subthreshold symptoms do not always develop to disorder, but there is well-replicated evidence that they might; for instance, bipolar disorder can be traced to subthreshold mania (Tijssen et al., 2010), major depression may be traced to subthreshold depressive states (Judd, Schettler, & Akiskal, 2002), and psychotic disorders to subthreshold psychotic experiences (Linscott & Van Os, 2013). This indicates that early symptoms may reflect vulnerability for later illness, but most symptoms are shown to be transient (Dominguez, Wichers, Lieb, Wittchen, & van Os, 2009; Zammit et al., 2013).

Early symptoms may also be transdiagnostic to other mental disorders. It has been suggested that early symptoms are non-specific and that motivational alterations, affective dysregulation, anxiety, depression and other early symptoms affect each other in a way that later develop to specific syndromes (Dominguez, Saka, Lieb, Wittchen, & van Os, 2010; Fusar-Poli, Nelson, Valmaggia, Yung, & McGuire, 2012; van Rossum, Dominguez, Lieb,

Wittchen, & van Os, 2009; Wigman et al., 2013). Thus, focusing on symptoms such as motivational alterations instead of disorders may be a more productive approach.

### **Psychotic symptoms as a continuum and the transition to disorder**

As mentioned above, psychotic disorders are examples of illnesses that may be seen along a continuum with non-specific early symptoms affecting the progression of disorder. There is now a general agreement that the expression and experience of psychotic symptoms may be seen at a subclinical level in the general population (Calkins et al., 2017). The transition to disorder depends on several symptom factors such as frequency and intrusiveness of symptoms, and on cultural and personal factors such as illness behaviour, coping mechanisms and societal tolerance (Johns & Van Os, 2001). These findings have led researchers to develop the proneness-persistence-impairment model of psychosis (Van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009), stating that genetic vulnerability (both subclinical and clinical psychotic disorders tend to show familial clustering) interact with environmental risk factors to influence the early expressions of psychotic-like experiences to be persistent and later become clinically impairing. Exposure to alcohol, cannabis and other drugs, stressful and traumatic experiences, and urbanicity are shown to be non-genetic risk factors for both psychosis and subclinical psychosis (Van Os et al., 2009).

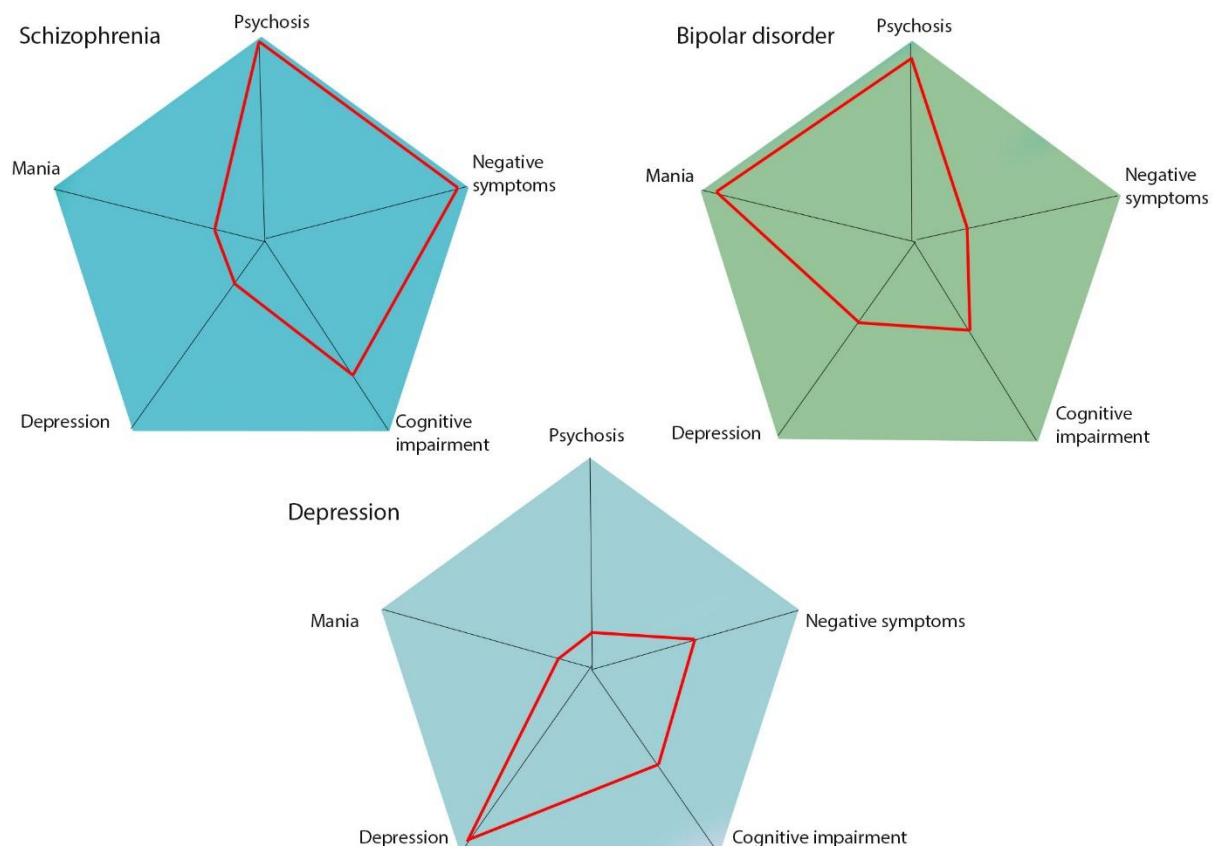
Different degrees of the subthreshold symptoms of psychoses may differently predict help-seeking behaviour and functional outcome (Dominguez et al., 2010). Even though the prevalence of clinical disorder may be relatively low, the prevalence of symptoms seems to be a great deal higher. To explore early symptoms, such as motivational deficits, may be a way to limit progression of disorders by gaining insight into risk and protective factors. Further, as early symptoms have a higher prevalence, it makes studying them easier.

### **Psychotic disorders: schizophrenia and bipolar disorder**

Symptoms of schizophrenia and psychotic disorders may be grouped into five main categories including (1) psychosis (delusions and hallucinations- positive symptom dimension), (2) changes in volition and drive (deficiencies in motivation, social withdrawal and diminished spontaneous speech- negative symptom dimension), (3) alterations in neurocognition (issues in attention, memory and executive functioning- cognitive symptom dimension), and (4 and 5) difficulties in regulating affect, causing manic and depressive (bipolar) symptoms (Van Os & Kapur, 2009). These symptoms are apparent both in individuals with a clinical diagnosis, and in individuals experiencing subthreshold symptoms

(Barragan, Laurens, Navarro, & Obiols, 2011; Calkins et al., 2017), indicating that the degree of symptoms and which symptoms one may have vary.

Criteria distinguishing between the different categories of psychotic disorder are based on duration, bizarreness of delusions, substance use, and presence of mania or depression (Van Os & Kapur, 2009). For example, a patient with few negative symptoms, but with positive symptoms and high levels of affective symptoms, may be diagnosed with bipolar disorder (figure 2). The progression of illness is usually gradual, beginning with the depressive and negative symptoms, followed by social and cognitive impairment. The positive symptoms often emerge later (Hafner, Löffler, Maurer, & Hambrecht, 1999; Häfner et al., 2003). The negative symptom dimension is associated with impairments in neurocognition, but the affective symptom dimension and the positive symptom dimension are not (Dominguez, Viechtbauer, Simons, van Os, & Krabbendam, 2009). Negative symptoms and the associated problems such as motivational and cognitive deficits have a special need for treatment as they are central aspects of the illness affecting daily functioning and do not



*Figure 2.* Three hypothetical typical patients diagnosed with schizophrenia (blue), bipolar disorder (green) and depression (grey). The red lines show scores on the five dimensions of psychopathology

currently respond well enough to existing antipsychotic treatment (Green et al., 2015; Kirkpatrick, Fenton, Carpenter, & Marder, 2006).

**Positive symptoms** Positive symptoms or psychotic symptoms entail loss of contact with reality in the form of perceptual experiences not shared with others (known as hallucinations), false beliefs (known as delusions), and bizarre behaviour (Mueser & McGurk, 2004). Psychotic experiences, as with the other symptoms, are commonly seen in the general population (Linscott & Van Os, 2013), and the presence, duration and severity of these symptoms tend to vary (Heilbronner, Samara, Leucht, Falkai, & Schulze, 2016), as well as the presence of distress related to the symptoms. For example, Lim, Gleeson, and Jackson (2012) found that highly religious people scoring high on psychotic symptoms showed less distress in relation to their symptoms compared to individuals with psychotic disorders.

Positive symptoms are not associated with neurocognitive deficits such as alterations in memory accuracy, but they are associated with deficits in metacognition. It has been shown that clinical samples, individuals with subclinical delusions and people at high risk of psychosis are overconfident in incorrect or implausible judgments (Broome et al., 2007; Zawadzki et al., 2012), as seen in a jumping to conclusion bias (JTC; to base strong judgements on little evidence) or liberal acceptance bias (LA), and in a bias against disconfirmatory evidence (BADE; A tendency to not reduce the plausibility of an original explanation in the light of disconfirmatory evidence) (Moritz, Woodward, Jelinek, & Klinge, 2008). For example, Zawadzki et al. (2012) found that subclinical delusional ideation was associated with LA, BADE and JTC, but in a milder fashion.

### **Cognitive symptoms**

Research has shown that neurocognitive deficits associated with psychotic disorders are detectable at the subclinical level in the general population as well, but to a lesser degree. In their meta-analysis Van Os et al. (2009) found that individuals at a genetically risk of developing psychosis and individuals with subclinical symptoms showed impaired cognition, although not as generalized as seen in patients with psychotic disorders. Sitskoorn, Aleman, Ebisch, Appels, and Kahn (2004) performed a meta-analysis on cognitive performance in healthy first-degree family members of people with schizophrenia, indicating that family members expressed less cognitive efficiency than controls on various measures of cognition such as the Trail Making Test (TMT). Relatives showed deficiencies especially in executive functioning, verbal memory and attention. The severity of negative and cognitive symptoms are shown to correlate (Harvey, Koren, Reichenberg, & Bowie, 2005), especially the motivational component of negative symptoms (Foussias et al., 2015), indicating that



cognitive deficits could partly be a factor affecting motivational deficits seen in negative symptoms.

### **Affective symptoms**

Affective symptoms entail self-depreciation, hopelessness, feelings of guilt, suicidal ideation, rumination and active social avoidance (Dominguez, Viechtbauer, et al., 2009). Anxiety and depressive symptoms have been suggested to mark the onset of the initial prodrome to psychosis with large studies suggesting comorbidity of 69 and 78 percent, respectively (Salokangas et al., 2012; Woods et al., 2009). Further, depressive symptoms and negative symptoms are shown to be closely associated with each other in clinical and nonclinical samples (Stefanis et al., 2002), especially social withdrawal and avolition (Barragan et al., 2011; Brébion et al., 2000).

As with positive symptoms, depressive symptoms are associated with disturbances in aspects of metacognition, only these symptoms are associated with underconfidence in decisions related to elevated apathy (Rouault, Seow, Gillan, & Fleming, 2018), consistent with the notion that depression or dysphoria is characterized by pervasive negative self-evaluation and the tendency to overattribute negative outcomes (Garrett et al., 2014).

**Motivational deficits and affective symptoms.** Little research has been conducted on the relation between motivation and specifically affective symptoms of schizophrenia or psychotic disorders. However, regarding motivation individuals with dysphoria, or individuals with depression show reduced hedonic responses to enjoyable stimuli and monetary rewards, and this relates to elevated levels of anhedonia (Barch et al., 2016; Gotlib et al., 2010). Such deficits might contribute to impairments in other areas related to hedonic responses, such as learning, anticipation, action selection and effort. Dysphoric or depressed individuals show impairments in reward anticipation, prediction errors, reinforcement learning, value representation and effort allocation (Barch et al., 2016). Furthermore, Yang et al. (2014) showed that patients with major depressive disorder, individuals with subsyndromal depression and first episode patients with depression had reduced motivation in an effort-based decision-making task. They concluded that motivational deficits were correlated with stage of illness and depression severity, with greater deficits during depressive episodes and before onset (Barch et al., 2014). Thus, it seems likely that affective symptoms may play a part in motivational deficits in schizophrenia.

### **Negative symptoms**

Negative symptoms refer to, in broad terms, the impairment or absence of normal functions in sociality, emotion, communication and productive goal-directed behaviour

(Reddy, Horan, & Green, 2016). They may be separated into two factors with five core symptoms: (1) A factor of diminished expressivity, including alogia (reduced verbal spontaneous speech and verbal production) and restricted affect (reduced emotional expressions, including gestures, speech and facial expressions) and (2) a motivational factor consisting of anhedonia (diminished intensity and range of pleasant emotions), avolition (diminished engagement in occupational, intellectual and social pursuits) and asociality (social avoidance or withdrawal and lack of interpersonal relationships) (Blanchard & Cohen, 2005; Kirkpatrick et al., 2006; Strauss, Horan, et al., 2013). Negative symptoms have traditionally been described in relation to schizophrenia, but they also occur in other psychiatric disorders such as bipolar disorder, major depressive disorder and the general population though not explicitly termed negative symptoms (Strauss & Cohen, 2017).

The motivational component of negative symptoms have long been understood to be one of the core components of the illness with a great need for treatment as it seems more closely linked to core aspects of the disorder such as subjective quality of life, functional outcome and recovery (Kirkpatrick et al., 2006; Strauss, Horan, et al., 2013). Although negative symptoms have an extensive impact on patients functioning, current available treatment options are scarce, and their underlying cause remains unclear.

**Motivational deficits in negative symptoms.** The reduced or absent ability for hedonic experiences, anhedonia, was long assumed to be the cause of negative symptoms and thus the lack of motivation and goal-directed behaviour. However, recent studies have revealed a different picture, showing that individuals with schizophrenia have intact in-the-moment hedonic experiences (Llerena, Strauss, & Cohen, 2012). These findings have led researchers to explore other possibilities, now suggesting that individuals with negative symptoms have deficits in a range of reward-related processes, making it difficult to translate reward information into motivated behaviour (Whitton, Treadway, & Pizzagalli, 2015). Abnormalities in cortical-striatal interactions may be associated with these behaviours, including value representation, reinforcement learning, effort-based reward related decision making, effort-cost computations and uncertainty-driven exploration (Strauss, Waltz, & Gold, 2013; Whitton et al., 2015).

### **Effort-based decision making**

Recently there has been a growing interest in exploring effort-cost computations, i.e. how much effort an individual is willing to exert for a given level of reward. These computations involve valuation and effort (Shenhav, Botvinick, & Cohen, 2013) in that one

first has to make a representation of the value of a potential reward and how much that reward is worth relative to another reward, something that depends on the amount (e.g., monetary reward) and the probability of receiving that reward. The valuation of a reward is also affected by several factors in the individual such as the mood, the perceived cost, and the working memory capacity (figure 3). Further, one must create a representation of the perceived cost of the effort, and to be willing to change one's effort as a function of changes in reward value.

There is not much research on effort-based decision-making tasks and subclinical symptoms of schizophrenia, but several studies have consistently shown a pattern that individuals with psychotic disorders or schizophrenia have effort-related impairments (Whitton et al., 2015) and that there is an association between degree of negative symptoms and these impairments (Bergé et al., 2018). However, one study performed by Terenzi, Mainetto, Barbato, Rumiati, and Aiello (2019) showed that healthy individuals scoring high on negative symptoms as measured by the CAPE were less willing to exert effort for higher rewards. Green et al. (2015) reviewed results from studies on negative symptoms in schizophrenia using varying methods. It was most common to use physical effort tasks with either grip effort tasks or button-pressing tasks such as the Effort Expenditure for Rewards Task (EEfRT) and the Balloon Effort Task. The most consistent results were found in the button-pressing physical effort tasks showing impairments in individuals with negative

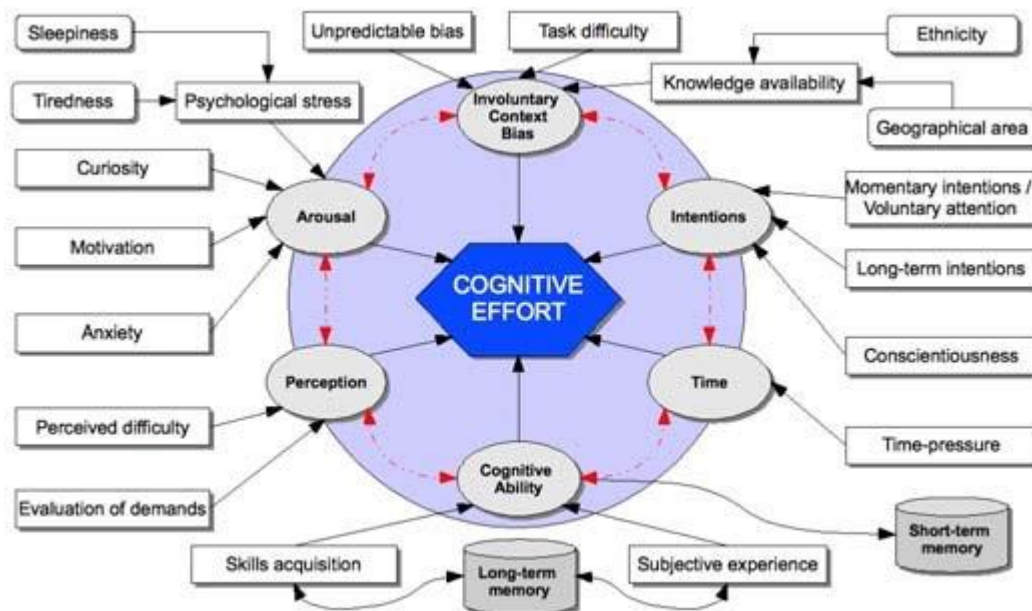


Figure 3. Overview of cognitive effort and influencing factors by Longo and Barrett (2010, p. 67). The focus of this thesis is on motivation, cognitive ability and perception.

symptoms. More precisely, individuals with negative symptoms chose easy tasks more often than controls at higher probability levels and monetary reward levels.

There is conflicting evidence when it comes to affective symptoms and effort-based decision-tasks. In the review of Green et al. (2015), four out of five of the studies reporting on affective symptoms did not find a significant relationship. However, when studying individuals with symptoms of dysphoria and anhedonia also found in schizophrenia, there seems to be a different picture. Yang et al. (2014) found that individuals with subsyndromal depression showed reduced motivation on effort-based decision making using the EEfRT, and that this effect was correlated with symptom severity and anhedonia. Further, Barch et al. (2014) tested healthy controls varying on symptoms of depression and anhedonia in a study using the EEfRT task. Anhedonia and level of depressive symptoms was associated with fewer hard task choices in high reward and high probability conditions.

We have seen that symptoms seen in schizophrenia may be seen along a continuum and that early symptoms might develop to clinical disorders. To address early symptoms is important to be able to prevent the development to disorder and by studying healthy individuals with different degrees of symptoms this provides a window into the interaction between affective, negative and cognitive factors of motivation. In addition, by studying healthy individuals, one is able to avoid the possible confounding effect of psychotropic drugs. Further, negative, affective and cognitive symptoms affect motivation in complex ways, and it is unclear exactly how they affect each other. As previously stated, there is an overlap between negative and affective symptoms, making it difficult to disentangle the effect of these symptoms on cognitive symptoms and motivation. Several studies have reported significant relationships between negative, depressive and cognitive symptoms, but there is disagreement in which ways (Bowie & Harvey, 2005; Brébion, David, Jones, & Pilowsky, 2009; Foussias et al., 2015). Nevertheless, it seems clear that effort-based decision-making and motivation is affected by cognitive ability and symptoms seen in schizophrenia, and more research is needed to see which of several symptoms that affect motivation the most, taking cognitive ability into account. As current methods measuring effort-cost decision-making do not control for cognitive ability, new methods have been developed.

### **New method measuring effort-based decision-making**

A new method measuring effort-based decision-making has recently been developed, the Precision and Motivation Task (PMT). This computer-based task was developed by Gerit Pfuhl and is based on a mathematical model (Pfuhl, Tjelmeland, Molden, & Biegler, 2009) trying to quantify the question of how much effort one should invest in an activity and when

to abandon it in terms of how much reward one gets compared to the cost of doing it. As with the other methods measuring effort-cost computations, value and probability will affect the equation. However, as seen in figure 3, several factors are at play to affect our effort-cost computations. The mathematical model assumes that people are rational beings not affected by emotions, which they are not (De Martino, Kumaran, Seymour, & Dolan, 2006). To see how motivation and neurocognition affects our decision making is therefore an important aspect to take into consideration.

The task consists of encoding, recall and search (effort), where the participants are instructed to remember a shape, and then finding it again among similar shapes through performing a search. The subjective understanding of probability of finding the shape and a known uncertainty that the shape is there sets the base for effort the participant puts into the task. Therefore, the effort will have to be adjusted according to the probability for reward. Neurocognitive ability and metacognitive precision may affect the process in that lower cognitive ability could influence the representation of value, and the cost of performing the task might be greater due to these deficits (see materials for more information about PMT).

To illustrate, imagine the activity of looking for a receipt for an important purchase in your house. The amount of time you spend (effort) looking for the receipt is dependent on your memory. If you know that your memory is precise and you know that it was in the kitchen drawer, you are going to (and should) presume that your roommate threw it away and stop searching right away. If you know your memory is very poor, you should not start the search at all, because the cost (in terms of physical cost or time spent) will be greater than the reward of finding it, in that you might have to search the whole house. Further, your metamemory will affect this equation, as will the reward of finding it. If you wanted to find the receipt to get a refund of a pair of socks, the reward will probably be lower than the cost of finding it. Finally, the search will depend on the probability of the receipt being in your house at all. Someone might have thrown it away, or maybe you did not bring it from the store. If the probability is low, the effort you put in your search should be lower than if the probability of it being there is high. In between the extremes of knowing for sure, and having no idea, there is an optimal limit to investment in the search. As you forget (memory of the location of the receipt becomes less precise), the optimal search limit first rises, but then it declines steeply. If the probability of the receipt being in your house is high, the poorer memory you can have before you rationally should stop searching (see also Fig 2 in Pfuhl et al. 2009).

### **The current study**

The aim of this study was to identify which of the several symptoms in psychotic disorders contribute the most to the lack of motivation and goal-directed behaviour. The PMT assesses effort-based decision-making as well as motivation, controlling for neurocognitive ability (visual short-term memory), metacognitive precision (precision of visual short-term memory) and effort. In addition, PMT controls for the relative cost of performing the task. Lastly, PMT measures directional and activational motivation (Pfuhl et al., 2009). By using PMT, one may see differences in effort-cost computations between individuals with varying degrees of symptoms due to several non-exhaustive factors that interact with each other. Participants may exert the same effort (time spent searching) but due to a possible difference in cognitive ability the cost of doing so may differ. Conversely, there is a possibility for the same costs, but different effort. As an analogy, think of a seven-year-old boy and his father both reading a newspaper. They spend the same time on the task, but it is more demanding for the boy who has not yet mastered reading at the level of his father. When reading the headlines for example, the cost might be similar, but the father is faster, so this would be measured as less time spent/ effort. This example illustrates that one needs to measure both effort and costs to assess efficacy.

We have deviated from the preregistration (Pfuhl & Simensen, 2019) in several ways to answer this question. In the initial preregistration we wanted to compare participants with familial psychosis and people scoring high on dysphoria in motivation. This was based on the assumption that individuals with familial psychosis tend to show mild cognitive symptoms in the same way patients do. Further, individuals with dysphoria are assumed to have the affective or perhaps the negative symptoms seen in psychotic disorders. The reason we deviated from the preregistration is twofold. Firstly, during the course of the project we got to know the recent literature better and we realized that recent research focus more on the continuum aspect of mental illnesses in contrast to the earlier dichotomous classification system, arguing that there is high comorbidity in mental illnesses and that one should focus on the common underlying symptoms rather than distinct disorders (Craddock & Owen, 2007; Van Os, 2013; Van Os et al., 2009). Secondly, recruiting participants with familial psychosis in a short timeframe is challenging, something we recognized early on in the data collection procedures. This insight led to the decision to compare individuals with varying degrees of psychotic symptoms (some scoring high on only negative, affective or positive symptoms, but not the other symptoms) on the PMT.

Our initial hypotheses were based on the symptoms relating to dysphoria and familial psychoses. After changing our approach, we translated our hypothesis to fit the symptom clusters. Hypothesis 1 predicted more neurocognitive deficits in the participants scoring high on negative symptoms. In hypothesis 2 we expected meta-cognition to be lower in the individuals scoring high on depressive and positive symptoms compared to healthy controls with no symptoms (i.e. dysphoric participants would be underconfident and participants scoring high on positive symptoms would be overconfident). Hypothesis 3 predicted that activation motivation in the PMT to be lower in the participants with negative symptoms and dysphoria than in the ones scoring high on positive symptoms and control group with no symptoms. Lastly, in hypothesis 4 we expected that participants with negative symptoms and dysphoria would search less (lower directional motivation), whereas the positive symptom group would search similarly to their capture area.

## Methods

### Participants

To recruit participants, flyers were distributed around campus at UiT and NTNU. Further, we used social media to spread information and encourage people to contact us. More specifically we posted information in Facebook support groups for people with mental illness or their relatives or web-pages with information about mental health. We specified that we were looking for individuals in Tromsø, Trondheim, Oslo or Østfold who either had a first-degree relative with a psychotic disorder or people feeling dysphoric or sad and had been doing so for quite some time. By recruiting dysphoric participants and people with first-degree relatives with psychotic disorders we hoped to sample across the symptom continuum. More specifically, as previously mentioned, dysphoric people have anhedonia (as in affective and negative symptoms) but should have less of the other symptoms. Further, we expected that relatives would have a higher probability of having higher degrees of cognitive or positive symptoms but not the other symptoms. Local institutions specialising in mental health were contacted to help spread information about the project and we were able to put flyers in waiting rooms. Lastly, we used snowballing (i.e. recruit through friends or family members). Participation was rewarded with a gift certificate worth 150kr.

Fifty-three individuals participated in the study with age ranging from 18 to 48, with mean age of 26 ( $SD = 7,10$ ). Out of these 37 were females and 16 males. Participants were excluded if they presented a substance use disorder (except nicotine), or if they had

neurological disorders. One participant was excluded due to abandoning testing after two tasks, leaving fifty-two participants.

### **Materials**

**Neurocognitive tests: Trail Making Test A and B, and Digit Symbol Substitution Test.** Participants started by completing the Trail Making Test (TMT) A and B (Reitan & Wolfson, 1985) providing information on visual search, cognitive flexibility, scanning, speed of processing and executive functions (Tombaugh, 2004). TMT-A requires a participant to draw lines connecting 25 numbers semi randomly distributed on a sheet of paper. TMT-B resembles TMT A, except the individual must alternate between connecting numbers and letters (e.g. 1, A, 2, B, 3, C, etc.). The score on each task comprises the amount of time it takes the participant to complete the task. An example was provided to each task beforehand to make sure the task was understood. The task was administered and interpreted as described in Bowie and Harvey (2006).

The Digit Symbol Substitution Task (DSST) is a subtest from WAIS (Wechsler, 1955) measuring visual scanning, attention and psychomotor speed. It consists of a template connecting specific numbers with hieroglyphic-like symbols, and rows of boxes with the number on the top and empty spaces at the bottom of each box. The score is computed by counting the number of correct pairings completed in 90 seconds. The higher the score the better performance. The participants were first shown an example and got to practice a sample before the timed task.

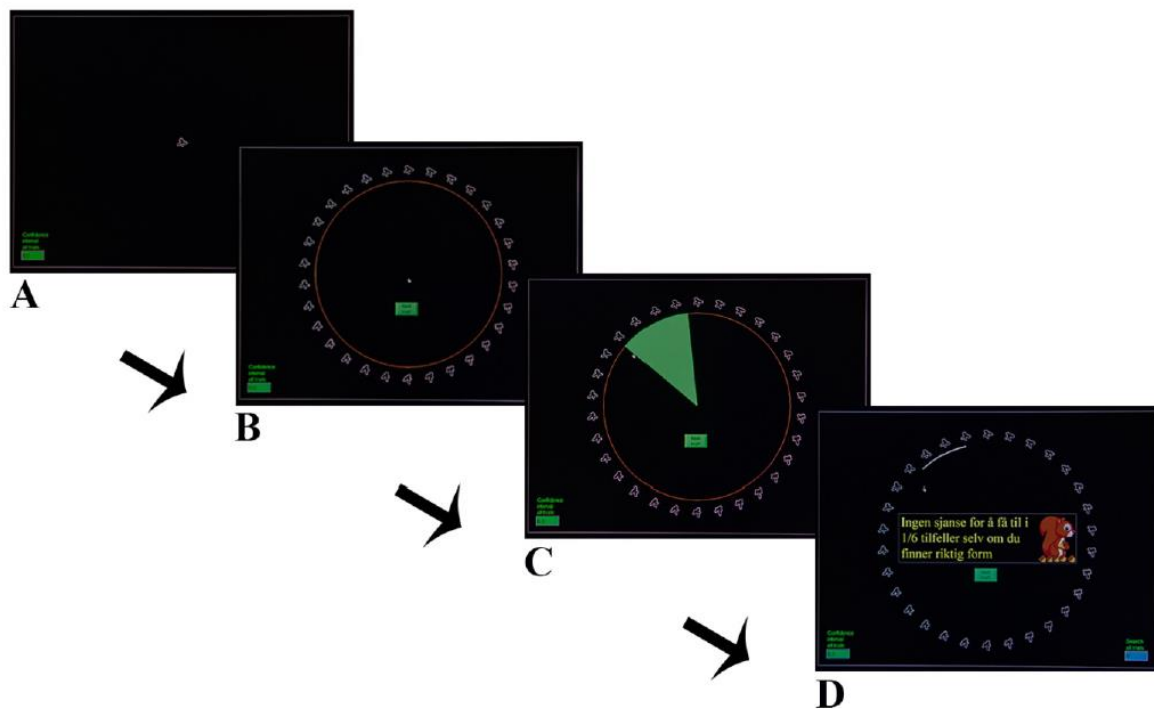
**Precision and Motivation Task.** This computer-based task is presented as a game, where the participants are presented with a background story saying that they must compete with two hungry squirrels to find hidden nuts. The game has four stages. An abstract shape said to be a nut is shown for two seconds in the first stage (Figure 4A). The participant is instructed to remember the shape as she will have to find it among 30 similar shapes arranged in a circle (Figure 4B). The shapes look a lot like each other, but they are a little different. The participant is told that one or more of the shapes are very similar to the shape she was asked to remember.

In the second stage the participant is instructed to indicate where among the 30 shapes the remembered shape is located. If she does not remember, she is allowed to move on by clicking the “next trial” button. The participant moves to the next phase when she has indicated the location of the shape. This stage is where neurocognition is measured, by calculating the deviation between indicated location of the shape and correct location. In the third phase the indicated location of the shape is used as a starting point for making the



capture area (Figure 4C). The Capture area represents perceived precision and the participant is instructed to make the area large enough so that she is sure the shape is located somewhere inside the capture area (Graf, Warren, & Maloney, 2005). Points are received depending on the accuracy of the capture area in relation to real precision (Figure 5). The maximum score is ten points in each trial, with points subtracted if the capture area is made too large, but no points earned if the capture area is made too small. The points are presented after each trial.

The fourth phase is where the participant searches for the shape after being presented with information about the probability of finding it, represented by a mean or a kinder squirrel (Figure 6). The mean squirrel is hungry and steals the nut  $\frac{2}{6}$  times. The kind squirrel is less hungry and steals the nut  $\frac{1}{6}$  times (representing 67% and 83% probability of finding the nut). The participant is informed that the nut is hidden and that she must search for it by clicking the left mouse key repeatedly. The search will start where she first indicated the nut to be located and expand to each side (Figure 4D), and end when she passes the area the nut is located. Ten points will be received if the nut is found and zero points will be received if it is

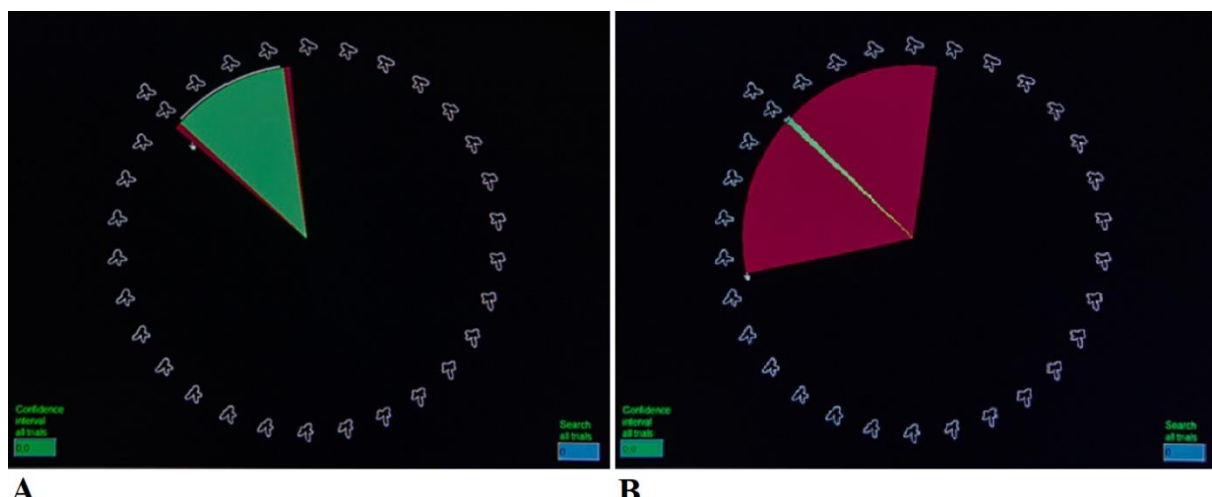


*Figure 4.* Precision and motivation task. A: Sample phase- the squiggly shape is shown for two seconds. B: Retrieval phase- 30 similar shapes are organized in a circle. Using the sample phase as a reference, the participant indicates the location of the shape looking most alike. C: Confidence phase- the participant makes a capture area large enough that he/she is certain that the shape from the sample phase is included in the area. D: search phase- the participant searches for the “nut”, starting at the indicated location from the retrieval phase. A white line stretches in both directions for each click, indicating the search. The probability of finding the “nut” is indicated by the squirrel, either  $\frac{5}{6}$  or  $\frac{4}{6}$  chance.

not. The search phase represents motivation and is seen in relation to the capture area. Keep in mind that the capture area is made large enough to be sure that the shape, aka nut, is located inside of it. Accordingly, searching less than the indicated area means that one is less motivated to find the nut. This can only be assessed in the few ( $n=10$ , 5 per probability condition) trials where the nut cannot be found, i.e. the participant must give up and move on by clicking the “next trial” button. The PMT has 45 trials, in which there is 30 trials with  $1/6$  probability of the nut being stolen, and 15 trials have  $2/6$  probability of the nut being stolen. It is in the trials where the nut is stolen that one is able to see the maximal effort a participant is willing to exert for a reward.

The software is programmed to measure latency to start a trial, latency to making the capture area, latency to start searching, and vigour (activational, speed at which the search clicks are made), deviation between indicated stimulus positioning and correct positioning (visual short-term memory), size of capture area, number of clicks, number of clicks (effort) invested in “no chance”- trials where the individual gave up and moved on, and if the stimulus was included in the capture area (hits, metacognitive index).

**CAPE-42.** The participants filled out a Norwegian version of the CAPE-42 (Community Assessment of Psychic Experiences) (Stefanis et al., 2002), developed to measure the lifetime prevalence of psychotic experiences in the general population. It was computer based, implemented in Qualtrics. It is a self-report questionnaire containing a negative, positive, and a depressive dimension score, as well as a distress score. There are 14 negative symptom items, 20 positive symptom items and 8 depressive symptom items. It uses



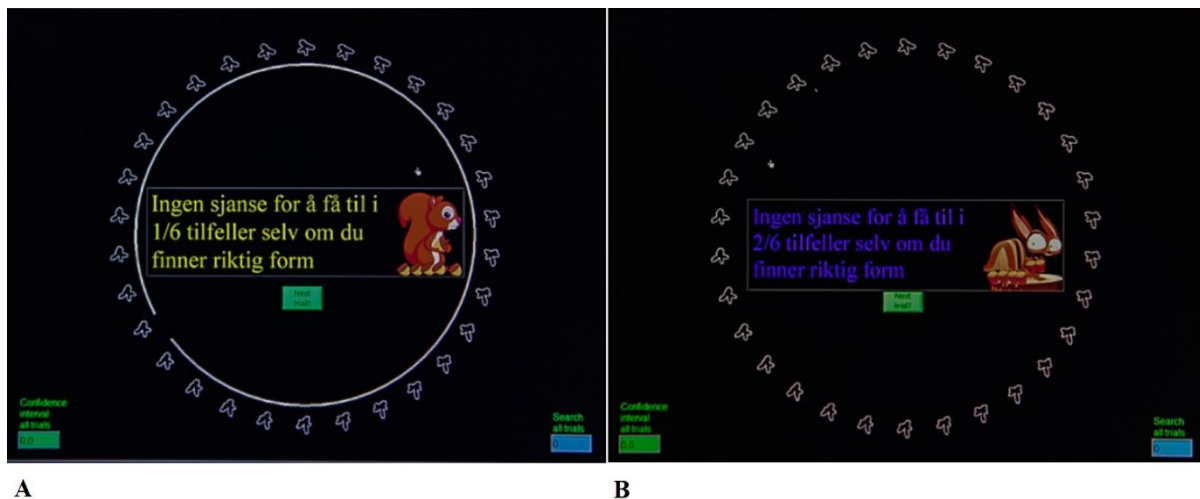
**A** **R**  
 Figure 5. A search where the “nut” has been found and may be seen outside the circle. The participant was rewarded with ten points for finding the nut. The capture area was precise (little red marking) and gave close to ten points. Figure 4B: The search gave ten points for finding the “nut”. However, the capture area is disproportionately large (the capture area is mostly red), giving less points.

a four-point scale ranging from 1 (never) to 4 (nearly always). It provides an overall score per dimension and a total score by adding up the number of positive responses to the frequency question. The total score of the CAPE ranges from 42-168. Scores on the positive dimension range from 20-80 points, the depressive dimension score ranges from 8-32 points and the negative dimension ranges from 14 to 56 points. It also provides a distress score by adding up the scores on the distress questions.

### Procedures

The data collection was mainly performed in a computer lab, but some performed the study in a quiet room with no distractions, as all software needed was available on a laptop and we were flexible to meet participants where it was convenient for them. Initially the participants read and signed the consent form and completed a survey containing questions about demographics and background information such as education and employment, alcohol habits, medication and substance use habits, mental health and neurological disorders (see appendix A).

In the next step the participants performed TMT A and B. They received the instructions and performed a test trial prior to starting the timed task in both subtests. DSST was presented in the same manner, and both tests were performed with pen and paper. We recorded the time in seconds it took to complete the TMT and used a stop watch of 90 seconds on the DSST. TMT A and B and DSST took approximately 11 minutes to complete.



*Figure 6.* Figure A shows the visual representation of the kind squirrel (83% probability of finding the nut). The search line is stretching around the whole circle, indicating a “no-chance” trial where the participant has invested maximum effort. Figure B shows the visual representation of the mean squirrel (63% probability of finding the nut).

Before performing the Precision and Motivation Task the participants went through a demonstration with the administrator. The demonstration had seven trials where all the components were present (high and low probability), where all principles of the task was gone through step by step (see appendix B for script and procedures). In the first three trials the basic idea of the experiment was presented. This included the shape and that it represented a nut, that she was supposed to remember it and to indicate its location, the capture area and the search. The principles of the points and probability (the squirrels) were presented in the fourth trial. This is also where they were presented with the squirrel analogy. The participant was encouraged to participate and ask questions during the demonstration to make sure they understood the task. They had all the information they needed by trial five, where they were instructed to try for themselves while the administrator observed. We did not move on to the main task until the participant completely understood the task. The task took approximately 22 minutes to complete.

Lastly, participants answered the CAPE-42, directly after completing the PMT. As it was computer-based we had to exit and open the survey in a new tab. The front page provided information about the aim of the survey, that there were no right or wrong answers and that they should not spend too much time on each question. One question appeared on the screen at the time and one was not able to move on until one had answered the question.

### **Ethics**

When conducting research there are several ethical considerations. It is important to protect personal integrity, to respect privacy and to provide sufficient information to the participants. Prior to data collection the project was approved by REK Vest (2011/1198/REK vest; see appendix C) and an informed consent form was formulated by using a template by the regional committees for medical and health research ethics (see appendix D). The participation was voluntary, and the participant could withdraw their consent at any time. No participants were contacted by us, they had to contact us after seeing flyers about the project. The flyers did not contain in depth information, thus more information was provided to those who were interested prior to participation. The consent form was signed prior to any data collection. All information about the participants was treated confidential. They received an ID number to ensure that no information could be traced back to them. The consent forms containing the participants names were stored separately in a locked cabinet. After completing the test, participants were debriefed and were encouraged to ask questions.

### **Statistical analysis**

All analyses were conducted using the software package JASP version 0.9.2. Demographic variables were summarised using descriptive statistics. First, in order to investigate the reliability of the variables representing the negative, positive and depressive domains, the Cronbach alpha coefficient was used as a measure of internal consistency for each symptom cluster and for the CAPE total score. Alpha values of 0.7 are regarded as satisfactory. Second, associations between the depressive, positive and negative symptoms were explored using Pearson correlation. Following Cohen's criteria, the effect size of correlational coefficients is defined as small when  $r = 0.10$ , medium when  $r = 0.30$  and large when  $r = 0.50$  (Cohen, 1992).

Third, multiple linear regressions were conducted to examine the relationship between symptoms and task-related variables (TMT A and B, DSST, latencies, vigour, real precision, perceived precision, meta-cognition, trial-by-trial awareness and subjective cost). Positive, negative and depressive symptoms were predictors and the task-related variables outcome variables. Assumptions of normality, linearity and homoscedasticity were met before interpretation of the results.

## **Results**

### **Participants**

16 participants reported having family members with psychotic disorders (at-risk participants) whereas 13 reported feeling depressed or dysphoric. In the at-risk group, 10 participants had family members with bipolar disorder, 2 participants had family members with schizophrenia or psychosis, and 4 participants had family members with both bipolar disorder and schizophrenia. On average participants had 5.72 years of education ( $SD = 2.07$ ) after junior high school in which 2 (3.8%) had completed junior high school, 32 (60.4%) had completed upper secondary school, 17 (32.1%) had completed a bachelor's degree and 2 (3.8%) had completed a master's degree. Table 1 presents the age of the participants, the mean scores on the subscales of the CAPE, the mean time spent to complete TMT A and B and the mean pairings completed in the DSST task.

**Table 1**

Descriptive Statistics for CAPE-42 and neurocognitive tasks

	Age	CAPE_P	CAPE_ND	TMT-A	TMT-B	DSST
<b>Mean</b>	26.77	25.56	42.63	24.74	59.16	57.48
<b>Std. Deviation</b>	7.579	4.840	11.05	5.870	16.55	7.925
<b>Minimum</b>	18.00	20.00	26.00	14.37	30.49	37.00
<b>Maximum</b>	48.00	41.00	69.00	38.18	100.8	73.00

*Note:* TMT A and TMT B is measured in seconds, DSST is measured as number of correct symbols. CAPE\_P = positive symptom scale. CAPE\_ND = negative/depressive symptom scale.

**CAPE-42.** CAPE total scores ranged from 49 to 145 ( $M = 68.19$ ,  $SD = 14.67$ ), negative subscale scores ranged from 17 to 45 ( $M = 26.88$ ,  $SD = 7.17$ ), positive subscale scores ranged from 20 to 41 ( $M = 25.56$ ,  $SD = 4.84$ ) and depressive subscale scores ranged from 9 to 26 ( $M = 15.75$ ,  $SD = 4.38$ ). Satisfactory internal consistency was found for the CAPE-42 total score ( $\alpha = .94$ ), as well as for the positive symptom subscale ( $\alpha = .84$ ), the negative symptom subscale ( $\alpha = .90$ ) and the depressive symptom subscale ( $\alpha = .87$ ). In our sample, higher scores on one symptom was associated with higher scores on the other symptoms in that all the symptoms were highly correlated (see table 2). The high correlation between negative and depressive symptom subscales may indicate that they are measuring the same construct, thus the two subscales were combined.

**Table 2**

Correlations between the three subscales of the CAPE-42

	Pearson's r	p	Lower 95% CI	Upper 95% CI
CAPE_P - CAPE_N	0.597	< .001	0.387	0.748
CAPE_P - CAPE_D	0.668	< .001	0.483	0.796
CAPE_N - CAPE_D	0.823	< .001	0.709	0.895

*Note:* CAPE\_P = Cape positive scale; CAPE\_N = Cape negative scale; CAPE\_D: Cape depressive scale

### Neurocognitive tests

Hypothesis 1 predicted that neurocognition would be reduced in participants with higher levels of negative symptoms as measured by the TMT A and B, the DSST and memory accuracy / real precision. Hypothesis 2 predicted that meta-cognition to be lower in the individuals scoring high on depressive and positive symptoms compared to healthy controls with no symptoms (i.e. dysphoric participants would be underconfident and participants scoring high on positive symptoms would be overconfident) as measured by whether the

target shape was included within the capture area or not. Hypothesis 3 predicted that activation motivation would be lower in individuals scoring high on negative or depressive symptoms as measured by latencies and vigour in the PMT. Lastly, hypothesis 4 predicted that directional motivation would be poorer in people scoring high on negative or depressive symptoms, i.e. that they would search less than indicated by capture area whereas the positive symptom group would search similarly to their capture area.

**TMT A and B.** There were no significant relationship between TMT A and the degree of positive ( $\beta = 0.18, t = 1.06, p = .29$ ) or negative/depressive symptoms ( $\beta = -0.07, t = -0.42, p = 0.68$ ), but there was a significant relationship of age and TMT A ( $\beta = 0.50, t = 3.83, p < .001$ ). There was no significant relationship of TMT B and the degree of positive symptoms ( $\beta = 0.37, t = 2.00, p = 0.051$ ), and no significant relationship of TMT B and negative or depressive symptoms ( $\beta = -0.22, t = -1.19, p = .24$ ), or age ( $\beta = 0.12, t = 0.87, p = .39$ ).

**DSST.** There was a significant negative relationship between the degree of positive symptoms and completed pairings in DSST ( $\beta = -0.52, t = -3.19, p = .003$ ) and between age and DSST ( $\beta = -0.38, t = -3.04, p = .004$ ), showing that a higher degree of positive symptoms and higher age was associated with fewer pairings, but positive symptoms were a stronger predictor than age. There was no significant relationship between DSST and negative/depressive symptoms ( $\beta = 0.08, t = 0.49, p = 0.63$ ).

### **Precision and motivation task**

**Visual short-term memory and metacognitive precision.** Mean real precision was 18.75 degrees (SD = 7.84), ranging from 7.23 to 40.47. Visual short-term memory (real precision) measured as deviation between indicated location of the shape and correct location was significantly positively associated with the degree of positive symptoms ( $\beta = 0.63, t = 3.91, p < .001$ ), meaning that the more positive symptoms the poorer visual short-term memory. There was no significant relationship between negative/depressive symptoms and visual short-term memory ( $\beta = -0.20, t = -1.23, p = .22$ ) (Fig 7).

The mean hit rate was 0.65 (SD = 0.11), ranging from 0.42 to 0.96. There was no significant relationship between metacognitive precision and positive symptoms ( $\beta = -0.32, t = -1.77, p = .08$ ) or negative/depressive symptoms ( $\beta = 0.20, t = 1.07, p = .29$ ) as measured by whether the target shape was included within the capture area or not (Fig 8). However, as previously stated, the capture area is in a way a measure of how well they think they remember, because they made the capture area large enough that they were sure the shape would be located inside the area. A large capture area would indicate that they thought they

had poorer memory, and a smaller capture area would indicate that they thought they had better memory. Mean capture area was 18.85 degrees (SD = 4.97) ranging from 10.49 to 36.48. Multiple linear regression analysis showed that the positive symptom dimension was significantly positively associated with the size of the capture area ( $\beta = 0.36$ ,  $t = 2.05$ ,  $p = .05$ ), but the negative/depressive symptom dimension was not ( $\beta = 0.03$ ,  $t = 0.15$ ,  $p = .89$ ).

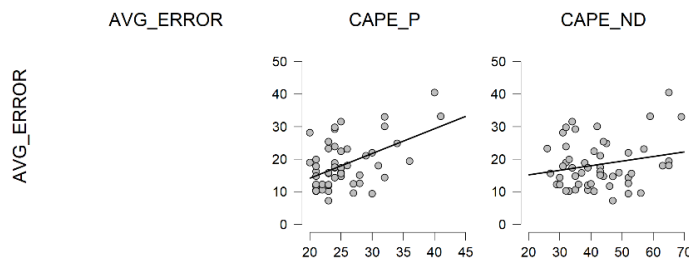


Figure 7. Correlations between visual short-term memory and the two symptom dimensions.

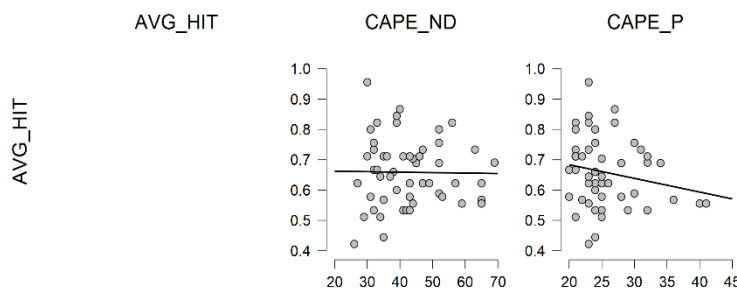


Figure 8. Correlations between metacognitive precision and the two symptom dimensions.

**Activational motivation.** The average latency to start trial was 2.75 seconds (SD = 1.14), ranging from 1.18 to 6.26 and the average latency to make capture area was 8.92 seconds (SD = 3.09), ranging from 4.04 to 17.22. The average latency to start searching in the high probability condition was 14.17 seconds (SD = 4.45) ranging from 6.72 to 25.76 and in the low probability condition the mean was 15.39 (SD = 4.89), ranging from 6.78 to 31.37. Lastly, the average vigour in low probability trials was 15.39 (SD = 4.89) ranging from 6.78 to 31.37 and 14.33 (SD = 4.44) in the high probability trials, ranging from 6.70 to 25.48. There was no significant relationship between positive symptoms and average latencies to start a trial ( $\beta = -0.16$ ,  $t = -0.86$ ,  $p = .40$ ), make a capture area ( $\beta = 0.03$ ,  $t = 0.14$ ,  $p = .89$ ) or initiate search in either low or high probability trials (high probability:  $\beta = -0.06$ ,  $t = -0.31$ ,  $p = .76$ ; low probability:  $\beta = -0.05$ ,  $t = -0.28$ ,  $p = .78$ ), nor a relationship between positive symptoms and vigour in low ( $\beta = -0.05$ ,  $t = -0.28$ ,  $p = .78$ ) or high ( $\beta = -0.06$ ,  $t = -0.30$ ,  $p = .76$ ) probability trials. Further, there was no significant relationship between



negative/depressive symptoms and average latencies to start a trial ( $\beta = 0.03, t = 0.17, p = .86$ ), make a capture area ( $\beta = -0.02, t = -0.14, p = .89$ ) or initiate search in either low or high probability trials (high probability:  $\beta = -0.11, t = -0.60, p = .55$ ; low probability:  $\beta = -0.09, t = -0.50, p = .62$ ), nor a relationship between negative/depressive symptoms and vigour in low ( $\beta = -0.04, t = -0.50, p = .62$ ) or high ( $\beta = -0.09, t = -0.59, p = 0.56$ ) probability trials.

**Directional motivation.** Next, we investigated the relationship between the degree of symptoms and invested effort in high and low probability trials in the search phase of the PMT. Remember, only the ten trials with no chance of finding the shape nut assess maximum effort the participant was willing to exert. One could say that the higher search radius, the more motivated the participants were to find the hidden nut, but one also must take the capture area into account. If the participant was completely rational, she would search as far as the size of the capture area giving a log ratio of 1. If she would search less than the indicated capture area, it could imply that she is less motivated, giving a log ratio smaller than 1. To search longer than the capture area is not necessarily rational because she does not think it is there, and it would therefore be counterproductive to search longer, i.e. the probability of the nut being past the capture area is low according to her memory. To search longer than the capture area would give a log ratio larger than 1.

A stepwise multiple regression was performed for the five “no chance” trials in the low probability trials. The mean search radius in the low probability condition was 0.60 (SD = 0.49) ranging from -0.82 to 1.65. The first predictor entered was the negative/depressive symptom dimension score. This model was statistically significant [ $F(1, 50) = 4.86, p = .03$ ] and explained 30% of the variance in the search radius in the low probability trials (Fig 9). After entry of the positive symptom dimension at step 2 the total variance explained by the model as a whole did not change and remained at 30% [ $F(2, 49) = 2.43, p = .10$ ]. The introduction of the positive symptom dimension explained 0% of the variance in search radius, after controlling for the negative/depressive dimension [ $R^2$  change = .00;  $F(1, 49) = 0.10; p = .76$ ]. In the final adjusted model, there were no statistically significant variables either for the negative/depressive symptom dimension ( $\beta = -0.26, t = -1.45, p = .15$ ) or the positive symptom dimension ( $\beta = -0.06, t = -0.31, p = .76$ ).

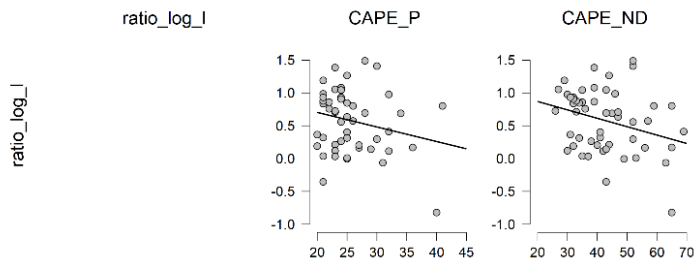


Figure 9. Correlations between effort invested in the search phase in low probability trials and the two symptom dimensions.

A second analysis was performed for the five “no chance” trials in the high probability condition. The mean search radius in the high probability condition was 0.71 (SD = 0.48), ranging from -0.97 to 1.79. The first predictor entered was the negative/depressive symptom dimension score. This model was not statistically significant [ $F(1, 50) = 3.39, p = .07$ ] and explained 25% of the variance in the search radius in the high probability condition (Fig 10). After entry of the positive symptom dimension at step 2 the total variance explained by the model as a whole only changed to 27% [ $F(2, 49) = 1.89, p = .16$ ]. The introduction of the positive symptom dimension explained 1% of the variance in search radius, after controlling for the negative/depressive dimension [ $R^2$  change = .01;  $F(1, 49) = 0.43, p = .52$ ]. In the final adjusted model, there were no statistically significant variables either for the negative/depressive symptom dimension ( $\beta = -0.18, t = -0.96, p = .34$ ) or the positive symptom dimension ( $\beta = -0.12, t = -0.65, p = .52$ ).

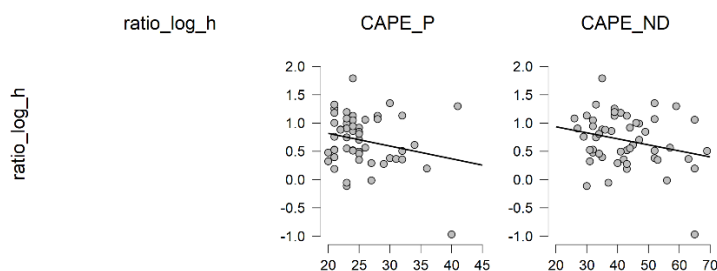


Figure 10. Correlations between effort invested in the search phase in high probability trials and the two symptom dimensions.

**Subjective cost.** According to the mathematical model used, the subjective cost of performing the search affects the search for the hidden nut. When calculating cost, memory is accounted for due to the assumption that poorer memory leads to an increased cost of performing the search. Only the 10 trials where the nut could not be found were used for calculating the costs (the formula can be found in Pfuhl et al., 2009). There was no significant relationship between the positive symptom dimension and the subjective cost of performing

the search in the high ( $\beta = -0.14$ ,  $t = -0.77$ ,  $p = .45$ ) or low ( $\beta = -0.07$ ,  $t = -0.39$ ,  $p = .70$ ) probability conditions; nor a significant relationship between the negative/depressive symptom dimension and the subjective cost of performing the search in the high ( $\beta = 0.22$ ,  $t = 1.19$ ,  $p = .24$ ) or low ( $\beta = 0.15$ ,  $t = 0.80$ ,  $p = .43$ ) probability condition.

### Discussion

The aim of this study was to identify which of the several symptoms in psychotic disorders or schizophrenia contribute the most to the lack of motivation and goal-directed behaviour by using a new paradigm measuring effort-based decision-making in a non-clinical sample. The method used was the PMT, based on a mathematical model quantifying how much effort one rationally should invest in a given activity relative to the subjective cost of doing it, taking neurocognitive ability and metacognitive precision into account. The task also measured activation and directional motivation as humans tend not to be rational (De Martino et al., 2006) and are likely to be affected by their motivation when performing a task. By testing participants with different degrees of symptoms, some with only negative, depressive or positive symptoms we hoped to get insight into the underpinnings of motivation in mental illnesses.

In this study, with this specific sample it seems like the degrees of negative/depressive symptoms are associated with lower directional motivation. Further, it does not seem like reduced neurocognition mediates this relationship, as it was positive and not negative or depressive symptoms that was associated with poorer neurocognition. In addition, the perceived cost of performing the task did not increase with symptom severity, thus the reduced effort cannot be attributed to increased subjective cost. Lastly, participants were rational in their effort-cost computations, as they lowered their effort as a function of lowered probability for reward. The results support the view that processes underlying schizophrenia and other psychotic disorders are expressed along a continuum as symptoms may be seen at a subclinical level.

To test the hypotheses in this study there was an assumption that participants would score differently on the symptom dimensions in CAPE. However, due to the distribution of symptoms in our sample it was difficult to test our hypothesis. Participants who scored highly on one symptom dimension scored highly on the other symptom dimensions as well, and those who had low symptom scores on one dimension also had low scores on the other symptom dimensions. These findings make it difficult to draw conclusions about which

symptom contribute the most to the lack of goal directed behaviour and motivation. We cannot rule out that the interplay between symptoms affected the results.

### **Precision and Motivation Task**

The PMT is based on a mathematical model trying to quantify the question of how much effort one should invest in an activity and when to abandon it in terms of how much reward one gets compared to the cost of doing it, taking motivation and neurocognition into account to see how it affects our decision making. It consists of encoding, recall and search (effort). The subjective understanding of probability of finding the nut and a known uncertainty that the nut is there sets the base for effort the participant puts into the task; thus, the effort will have to be adjusted according to probability for reward. Neurocognitive ability and metacognitive precision were assumed to affect the process in that lower cognitive ability could influence the representation of value, and the cost of performing the task could be greater due to these deficits.

**Activational and directional motivation.** There were no significant differences in activational motivation between individuals with more negative/depressive or positive symptoms and those with fewer symptoms on either average latencies to start trial, make capture area or initiate search in either low or high probability trials, nor a relationship between symptoms and vigour in low or high probability trials. Further, we found that there was no relationship of symptoms on the probability judgement in our sample. All participants were able to discriminate between high and low probability trials and adjusted their effort accordingly, i.e. they searched less when the probability of finding the nut was lower. Accordingly, individuals with symptoms show the same rational cost-benefit evaluations as individuals without symptoms in this regard. These findings are in contrast to previous studies finding that individuals in the general population scoring high on anhedonia and patients with schizophrenia have a reduced ability to adequately estimate and adjust effort in relation to reward and reward probability, more specifically that they allocate less effort in high reward and probability conditions (Gold et al., 2013; Treadway, Buckholz, Schwartzman, Lambert, & Zald, 2009). However, when Gold et al. (2013) compared patients with high versus low degrees of negative symptoms they found that individuals with low degrees of negative symptoms did not show impaired effort-cost computations regarding probability. They argued that these deficits were associated with symptom severity, something that is in line with our results testing healthy individuals with symptoms beneath clinical significance. It is worth noting that the PMT is not purely a physical task such as other paradigms used to measure effort-cost computations, as the PMT has a memory component. The results may also be

explained by the integration of memory together with reward probability and physical effort (vigour), and future research should explore this possibility in subclinical samples.

There was a significant association between individuals scoring high on negative/depressive symptoms and directional motivation. This was shown in that people with negative/depressive symptoms searched shorter than indicated by their capture area in trials where there was no chance of finding the nut. Participants were instructed to make the capture area large enough that they were sure that the figure would be located inside the area. If they assumed that the figure was inside this area and they searched shorter, it indicates that they were not motivated to find it to gain a reward. There were no such effects regarding positive symptoms. These findings are in accordance with the literature indicating that individuals with negative and depressive symptoms show less effort than controls in effort-based decision-making tasks (Barch et al., 2014; Green et al., 2015). It is important to note that there might be individual differences in how participants interpret the instruction that they are to “be sure” that the nut is located inside the capture area. We did not control for how sure they were, if they were a hundred percent sure or if they were satisfied with being somewhere between fifty and ninety-nine percent sure, for example. This issue might have affected the result regarding our measure of motivation, as it is possible that participants knew that they were not at all very sure and therefore searched less than they indicated. The results should therefore be interpreted with caution, and future research should find a way to control for this.

**Visual short-term memory and metacognitive precision.** Our results indicated that there was an association between positive symptoms and poorer visual short-term memory measured by calculating the deviation between correct stimulus positioning and indicated stimulus positioning. This is not in accordance with previous research as discussed below, but we see that in our sample positive symptoms are associated with several cognitive alterations. As previously mentioned, several studies suggest that cognitive deficits are partly responsible for reduced effort in decision making tasks. However, Bergé et al. (2018) found that participants with schizophrenia showed reduced effort regardless of cognitive deficits.

Metacognitive precision was measured by seeing whether the nut was included in the capture area or not. Here, we did not find any association with the degree of symptoms. It should be noted, however, that this measure of metacognition is unprecise, and it is therefore challenging to be able to draw conclusions from this measure. It would have been possible to calculate metacognition in another way by calculating real precision (deviation between indicated location of the nut and correct location) / perceived precision (the size of the capture area made by participants measured from the centre to the edge) per participant. We chose not

to use this method as it has other shortcomings. Assessing online metacognition may require other tools, including physiological measures, and future research should identify reliable measures of metamemory for continuous variables.

The results showed that there was an association between scoring higher on positive symptoms and making a larger capture area. This indicates that people scoring high on positive symptoms were aware that their memory was not perfect, and compensated by making a larger capture area. This stands in contrast to other studies such as Laws and Bhatt (2005) who found that people scoring high on delusional thinking in the general population had poorer recall memory and were more confident in erroneous responses than those with low levels of delusional thinking. They found that individuals with low levels of delusional thinking did not show this bias, thus it is possible that this effect is related to symptom severity. Further, they only investigated the relationship between delusional thinking and not other aspects of positive symptoms, possibly explaining the different results. More research is needed to investigate other aspects of positive symptoms in subclinical samples. Participants scoring highly on negative/depressive symptoms did not show underconfidence i.e. made the capture area larger than necessary as we expected. As previously mentioned, one would expect dysphoric or depressed individuals to have negative self-evaluations and to overattribute negative outcomes, thus to be underconfident in their judgements (Garrett et al., 2014). In a study by, Fu, Koutstaal, Poon, and Cleare (2012), they found that depressed patients were underconfident in their judgements of their own performance in a recognition task, but that dysphoric participants were relatively accurate in their judgements, compared to healthy controls that were overconfident. It is possible that dysphoric individuals do not have the same negative self-evaluations as more severely depressed individuals. Taken together, the relationship between real and perceived precision was the same for all the participants, as the individuals with positive symptoms compensated by making the capture area larger when their neurocognition was lower.

**Subjective cost.** Initially we hypothesized that individuals with negative symptoms would experience a higher subjective cost of performing the task due to poorer performance on neurocognitive tests as stated by the mathematical model used. However, contrary to our assumption, individuals with positive symptoms had poorer neurocognitive functioning and this hypothesis became obsolete. Note, the cost of performing the search did not differ with degrees of symptoms in either high or low probability trials, meaning that poorer effort could not be explained by an increased subjective cost. These findings stand in contrast to other studies. For example, Culbreth, Westbrook, and Barch (2016) examined cognitive effort and

subjective cost. They found that both healthy controls and patients with schizophrenia find cognitive effort costly, but to a larger degree in individuals with more negative symptoms and avolition. The same has been found regarding physical effort (Barch et al., 2014). It should be noted, however, that these studies did not control for neurocognition as the PMT does.

The PMT differs from other effort-based decision-making task such as the EEfRT in several ways possibly explaining some of the differing results. Green et al. (2015) highlighted several factors that might influence the differences seen in performance in these tasks such as reward sensitivity and temporal delay discounting (i.e. deciding between small sooner rewards versus larger, later rewards). Firstly, in other tasks, participants must choose if they want to work harder for different levels of monetary rewards, but the subjective value of money may vary between individuals. A person valuing money more might be more willing to work harder for monetary rewards. In the PMT this possible problem is avoided as it measures intrinsic motivation and there are not different reward levels. All participants received 150 NOK regardless of how well they performed on the task. They only received points with no reference to how well others had performed, thus they were competing with themselves to perform well on the task. Secondly, in some paradigms it takes longer to complete harder tasks than easy tasks, thus temporal delay discounting may be at play affecting the results. This is not a possible confounding variable in the PMT as the time spent searching do not differ between the conditions as search should be guided by one's memory accuracy, not by reward probability.

Our findings are in part in line with previous research showing that negative/depressive symptoms are associated with lower effort, but not in accordance with the mathematical model in which the PMT is based, as individuals scoring poorer on visual short term memory did not differ in their cost, and the model states that effort should be more costly due to poorer memory accuracy. It is possible that all participants had a fixed cost (cognitive effort is costly for everyone and we want to avoid it if we can). Therefore, when participants indicate a large capture area (indicating how far they should search) they do not search as far as they thought they should, indicating that they do not act on their belief. In addition, our results stand in contrast to several studies suggesting that poorer cognitive functioning affects the lowered effort in negative symptoms (Foussias et al., 2015). However, it is difficult to draw any firm conclusions given the high overlap of positive and negative/depressive symptom dimensions.

### Neurocognitive tests

There was no significant association between the degree of symptoms and TMT A or B, but there was a significant association between the number of completed pairings in DSST measuring visual scanning, attention and psychomotor speed and positive symptoms showing that individuals with positive symptoms performed poorer. There was also a significant association between DSST and age, but positive symptoms were a stronger predictor than age. Even though there are somewhat conflicting results in other studies, these findings contrast with most studies examining the association between symptoms in both subclinical and clinical schizophrenia and neurocognition, suggesting that negative or depressive symptoms are associated with neurocognitive deficits.

There is less literature on the association between specific symptom dimensions and neurocognitive deficits in the general population compared to studies using clinical samples. Many of these studies seem to focus on whether there are cognitive deficits at all in either family members of people with schizophrenia or individuals at high risk of developing psychosis, without including specific symptoms. If they do, depressive symptoms are not included. Here too, there are contrasting results in this literature, some studies showing that there is a relationship between negative symptoms and neurocognitive deficits (Barrantes-Vidal et al., 2003; Cochrane, Petch, & Pickering, 2012; Delawalla et al., 2006), one study finding no relationship between neurocognitive deficits and either negative or positive symptoms (Niendam et al., 2006), and one study finding that positive subclinical psychotic symptoms measured by the CAPE was associated with better performance on a measure developed to specifically assess cognitive domains known to be impaired in psychotic disorders and that negative symptoms associated with better performance on a measure of estimated IQ (Korponay, Nitzburg, Malhotra, & DeRosse, 2014).

There is less conflicting evidence in clinical samples where there is a large body of literature suggesting that there is an association between negative symptoms and neurocognitive deficits in patients with schizophrenia (Bora, Akdede, & Alptekin, 2017; Dominguez, Viechtbauer, et al., 2009). However, one study suggested that previous studies have failed to include depressive symptoms in the equation (Brébion, Bressan, Pilowsky, & David, 2009) and found that depressive symptoms and not negative symptoms are associated with neurocognitive deficits (Brébion et al., 2000; Brébion, David, et al., 2009). In addition, disorganization, one of the subdimensions of positive symptoms have shown to be associated with poorer performance on neurocognitive tests, even when controlling for affective and negative symptoms (Najas-Garcia, Carmona, & Gómez-Benito, 2018).



It seems that cognitive deficits in subclinical symptoms need further investigation but based on our results it seems that there is a difference between subclinical and clinical samples regarding cognitive functioning. It also seems like there is a gap in the literature on this topic as researches do not always agree on how to subdivide symptoms. In addition, there are several studies examining the association between neurocognitive deficits and psychotic-like experiences in the general population showing that individuals with psychotic-like experiences have greater neurocognitive deficits than individuals who do not have these experiences (Bora et al., 2014). As psychotic-like experiences are considered positive symptoms (as it includes hallucinations and delusions) one may say that our results are in accordance with this literature. However, these studies tend not to control for negative and affective symptoms, making it difficult to conclude that positive symptoms or psychotic-like experiences are the sole reason for this observed effect.

It is important to keep in mind that participants scoring high on positive symptoms also scored high on negative symptoms in our sample. It is possible that in subclinical samples where the degree of symptoms is expected to be lower than in clinical samples, the presence of both negative and positive symptoms is needed to see an effect on cognition, as the total amount of symptoms are higher and therefore could be more distressing and troublesome. It is also possible that the differing results of the association between neurocognition and symptom dimensions is due to different measures of cognitive ability, symptoms and the use of different samples. It is important that future research is able to better separate the symptom dimensions to be able to see which of the symptom dimension contribute the most to the lack of motivation and goal directed behaviour.

### **The CAPE**

The correlation between CAPE depressive and negative symptoms in our sample was very large resulting in that the two subscales were combined. Most studies reviewing the psychometric properties of the CAPE have shown that the CAPE is reliable with good factorial validity. For example, Mark and Touloupoulou (2015) found support for a three-factor model consisting of negative, depressive and positive symptoms in their meta-analysis. However, they noted that one item (lacking energy) loading on the negative dimension of CAPE in the initial study by Stefanis et al. (2002) was now found to load on depressive dimension of CAPE, highlighting the difficulties in separating the symptoms. Further, Hanssen et al. (2003) performed a study testing participants with a diagnosis of schizophrenia and other psychotic disorders, individuals with anxiety disorders, affective disorders and healthy controls on the CAPE. Firstly, their study showed similar high correlations between

negative and depressive symptom dimensions as in our study. Secondly, their results indicated that individuals with depression scored equally high on negative symptoms and depressive symptoms. They raised the question that the CAPE might not provide sufficient discriminatory power between depressive and negative dimensions in patients with depression. It is possible that this could apply to individuals feeling dysphoric and might therefore apply to our study as several individuals reported feeling dysphoric. In addition, Stefanis et al. (2002) noted that correlations between symptom dimensions are a great deal higher when testing the general population compared to testing patients, arguing that symptoms in the general population are more attenuated, making it more difficult to discriminate between them and to measure them sensitively. Taking this together, the reason we were not able to discriminate between negative and depressive symptoms might be that we tested both healthy participants and individuals reporting feeling dysphoric. Another possible reason for not managing to discriminate between negative and positive symptoms as other studies have, is that we used a Norwegian version of the CAPE. It is possible that the CAPE is not sensitive across cultures, possibly due to a different understanding of items.

Recent studies have found that the positive and negative symptom dimensions of the CAPE are comprised of three subcomponents (Mark & Touloupoulou, 2015). In the positive symptom dimension three factors corresponding to delusional ideations, bizarre experiences and perceptual anomalies are found, while three factors corresponding to affective flattening, social withdrawal and avolition are found in the negative symptom dimension. To further divide symptoms into subcomponents may be fruitful, as one might get better insight into the underpinnings of motivation and cognition. For example, several studies have found that specifically avolition in negative symptoms is associated with poorer neurocognitive deficits (Brébion et al., 2000). Further, it is common to subdivide positive symptoms into disorganization and reality distortion, and it has been found that disorganization is associated with impaired neurocognition in several domains (Najas-Garcia et al., 2018), but the CAPE does not account for disorganization.

It should be noted that even though the CAPE has several shortcomings, it can be argued that it is better than several other scales. For example, the Multidimensional schizotypy Scale- brief do not take affective symptoms into account (Gross, Kwapil, Raulin, Silvia, & Barrantes-Vidal, 2018). Other scales such as the Peters Delusion Inventory (Peters, Joseph, & Garety, 1999) only addresses one aspect of schizotypy and one would therefore need several, more time-consuming scales to capture all the symptom dimensions.

Nevertheless, as the CAPE had several shortcomings, future studies could possibly benefit from trying other scales.

### **Limitations**

There are several limitations in the current study. Firstly, even though we mainly recruited healthy participants, a few of them ( $n=3$ ) reported that they previously had been diagnosed with anxiety or depression, whereas some of those were on antidepressants ( $n=2$ ). The use of antidepressants as well as the use of other types of drugs like marijuana the past three months was not but should have been controlled for in the study. We did not have sufficient information about dosage, making it difficult to control for this. This decision not to exclude them was driven by the aim to have enough participants. It is not clear how these factors could affect the results as these medications may have side effects such as sleepiness and dizziness (NHS, 2018), and future research should have stricter exclusion criteria. However, several studies have not found the use of medication to affect effort-based tasks (Hartmann et al., 2014; Horan et al., 2015). Further, we did not exclude one participant reporting having ADHD. A study performed by Egeland, Nordby, and Ueland (2010) revealed that children with ADHD have impairments in effortful learning and motivation. It cannot be ruled out that the diagnosis of ADHD could be a confounding variable, and these individuals should be excluded in future research on this topic.

Secondly, future research would benefit from having a greater sample size than we did. When testing symptoms in the general population the symptoms are likely to be attenuated compared to clinical samples possibly influencing the high correlations between the three symptom dimensions. With a larger sample size, one might be able to measure symptoms more sensitively and be able to discriminate between them.

Thirdly, assessment of symptoms was based on self-reports. To use structured interviews aimed to measure symptoms or at-risk states in the general population such as the Structured Interview for Psychosis Risk Syndromes (SIPS) or the Comprehensive Assessment of At-Risk Mental States (CAARMS) would possibly give a more accurate measure of symptoms. However, these interviews are time-consuming and might not be suitable to test individuals who are not help-seekers in the general population and would need a trained interviewer. Nevertheless, there is extensive literature of the validity of self-reported positive, negative and depressive symptoms (Konings, Bak, Hanssen, Van Os, & Krabbendam, 2006; Mark & Toulopoulou, 2015).

### **Conclusion**

In this study we aimed to shed light on which of several symptom dimensions commonly seen in subclinical and clinical schizophrenia and other psychotic disorders contribute the most to lack of motivation and goal directed behaviour. A new effort-based decision-making paradigm based on a mathematical model was used to see how subjective understanding of probability and reward influence effort, taking cognitive functioning and the subjective cost of performing the task into account.

We did not get to test the hypotheses properly and our results must be interpreted with caution due to the symptom distribution in our sample, indicating that individuals scoring high on one symptom dimension scored highly on the other symptom dimensions in the CAPE as well. Results indicated that negative/depressive symptoms were associated with poorer effort, pointing to lower directional motivation. Surprisingly the results showed that positive symptoms were associated with poorer scores on neurocognitive test, contrary to other studies in the field. Accordingly, it seems that poorer effort was not mediated by cognitive ability in our study. Further, poorer effort was not associated with a higher subjective cost of performing the task. Lastly, the participants were rational in their decision-making as seen when the participants lowered their effort in low probability trials. Future research is needed to determine which symptoms relate the most to the lack of goal-directed behaviour. These studies should have larger sample sizes to be able to discriminate between symptoms and could possibly benefit from trying a different approach when recruiting participants to get a better symptom distribution.

### References

- Barch, D. M., Pagliaccio, D., & Luking, K. (2016). Mechanisms Underlying Motivational Deficits in Psychopathology: Similarities and Differences in Depression and Schizophrenia. In E. H. Simpson & P. D. Balsam (Eds.), *Behavioral Neuroscience of Motivation* (pp. 411-449). Cham: Springer International Publishing.
- Barch, D. M., Treadway, M. T., & Schoen, N. (2014). Effort, anhedonia, and function in schizophrenia: Reduced effort allocation predicts amotivation and functional impairment. *Journal of Abnormal Psychology, 123*(2), 387-397.  
doi:10.1037/a0036299
- Barragan, M., Laurens, K., Navarro, J., & Obiols, J. (2011). Psychotic-like experiences and depressive symptoms in a community sample of adolescents. *European Psychiatry, 26*(6), 396-401. doi:10.1016/j.eurpsy.2010.12.007
- Barrantes-Vidal, N., Fañanás, L., Rosa, A., Caparrós, B., Riba, M. D., & Obiols, J. E. (2003). Neurocognitive, behavioural and neurodevelopmental correlates of schizotypy clusters in adolescents from the general population. *Schizophrenia Research, 61*(2-3), 293-302. doi:10.1016/S0920-9964(02)00321-3
- Bergé, D., Pretus, C., Guell, X., Pous, A., Arcos, A., Perez, V., & Vilarroya, O. (2018). Reduced willingness to invest effort in schizophrenia with high negative symptoms regardless of reward stimulus presentation and reward value. *Comprehensive Psychiatry, 87*, 153-160. doi:10.1016/j.comppsy.2018.10.010
- Blanchard, J. J., & Cohen, A. S. (2005). The structure of negative symptoms within schizophrenia: implications for assessment. *Schizophrenia Bulletin, 32*(2), 238-245. doi:10.1093/schbul/sbj013
- Bora, E., Akdede, B. B., & Alptekin, K. (2017). Neurocognitive impairment in deficit and non-deficit schizophrenia: a meta-analysis. *Psychological Medicine, 47*(14), 2401-2413. doi:10.1017/S0033291717000952
- Bora, E., Lin, A., Wood, S., Yung, A., McGorry, P., & Pantelis, C. (2014). Cognitive deficits in youth with familial and clinical high risk to psychosis: a systematic review and meta-analysis. *Acta Psychiatrica Scandinavica, 130*(1), 1-15. doi:10.1111/acps.12261
- Bowie, C. R., & Harvey, P. D. (2005). Cognition in schizophrenia: impairments, determinants, and functional importance. *Psychiatric Clinics, 28*(3), 613-633. doi:10.1016/j.psc.2005.05.004
- Bowie, C. R., & Harvey, P. D. (2006). Administration and interpretation of the Trail Making Test. *Nature Protocols, 1*(5), 2277-2281.

- Brébion, G., Amador, X., Smith, M., Malaspina, D., Sharif, Z., Gorman, J. M., & Brébion, G. (2000). Depression, psychomotor retardation, negative symptoms, and memory in schizophrenia. *Neuropsychiatry, Neuropsychology, & Behavioral Neurology*, *13*(3), 177-183.
- Brébion, G., Bressan, R. A., Pilowsky, L. S., & David, A. S. (2009). Depression, avolition, and attention disorders in patients with schizophrenia: associations with verbal memory efficiency. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *21*(2), 206-215. doi:10.1176/jnp.2009.21.2.206
- Brébion, G., David, A. S., Jones, H. M., & Pilowsky, L. S. (2009). Working memory span and motor and cognitive speed in schizophrenia. *Cognitive and Behavioral Neurology*, *22*(2), 101-108. doi:10.1097/WNN.0b013e3181a722a0
- Broome, M., Johns, L., Valli, I., Woolley, J., Tabraham, P., Brett, C., . . . McGuire, P. (2007). Delusion formation and reasoning biases in those at clinical high risk for psychosis. *The British Journal of Psychiatry*, *191*(S51), 38-42. doi:10.1192/bjp.191.51.s38
- Calkins, M. E., Moore, T. M., Satterthwaite, T. D., Wolf, D. H., Turetsky, B. I., Roalf, D. R., . . . Gur, R. C. (2017). Persistence of psychosis spectrum symptoms in the Philadelphia Neurodevelopmental Cohort: a prospective two-year follow-up. *World Psychiatry*, *16*(1), 62-76. doi:10.1002/wps.20386
- Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., . . . Poulton, R. (2014). The p factor: one general psychopathology factor in the structure of psychiatric disorders? *Clinical Psychological Science*, *2*(2), 119-137. doi:10.1177/2167702613497473
- Chong, T.-J., Bonnelle, V., & Husain, M. (2016). Quantifying motivation with effort-based decision-making paradigms in health and disease. *Progress in Brain Research* (Vol. 229, pp. 71-100): Elsevier.
- Chong, T.-J., & Husain, M. (2016). The role of dopamine in the pathophysiology and treatment of apathy. *Progress in Brain Research* (Vol. 229, pp. 389-426): Elsevier.
- Cochrane, M., Petch, I., & Pickering, A. D. (2012). Aspects of cognitive functioning in schizotypy and schizophrenia: evidence for a continuum model. *Psychiatry Research*, *196*(2-3), 230-234. doi:10.1016/j.psychres.2012.02.010
- Cohen, J. (1992). A power primer. *Psychological Bulletin*, *112*(1), 155-159. doi:10.1037/0033-2909.112.1.155

- Craddock, N., & Owen, M. J. (2007). Rethinking psychosis: the disadvantages of a dichotomous classification now outweigh the advantages. *World Psychiatry, 6*(2), 84-91.
- Culbreth, A., Westbrook, A., & Barch, D. (2016). Negative symptoms are associated with an increased subjective cost of cognitive effort. *Journal of Abnormal Psychology, 125*(4), 528-536. doi:10.1037/abn0000153
- De Martino, B., Kumaran, D., Seymour, B., & Dolan, R. J. (2006). Frames, Biases, and Rational Decision-Making in the Human Brain. *Science, 313*(5787), 684-687. doi:10.1126/science.1128356
- Delawalla, Z., Barch, D. M., Fisher Eastep, J. L., Thomason, E. S., Hanewinkel, M. J., Thompson, P. A., & Csernansky, J. G. (2006). Factors mediating cognitive deficits and psychopathology among siblings of individuals with schizophrenia. *Schizophrenia Bulletin, 32*(3), 525-537. doi:10.1093/schbul/sbj082
- Dominguez, M.-d.-G., Saka, M. C., Lieb, R., Wittchen, H.-U., & van Os, J. (2010). Early expression of negative/disorganized symptoms predicting psychotic experiences and subsequent clinical psychosis: a 10-year study. *American Journal of Psychiatry, 167*(9), 1075-1082. doi:10.1176/appi.ajp.2010.09060883
- Dominguez, M.-d.-G., Viechtbauer, W., Simons, C. J., van Os, J., & Krabbendam, L. (2009). Are psychotic psychopathology and neurocognition orthogonal? A systematic review of their associations. *Psychological Bulletin, 135*(1), 157-171. doi:10.1037/a0014415
- Dominguez, M.-d.-G., Wichers, M., Lieb, R., Wittchen, H.-U., & van Os, J. (2009). Evidence that onset of clinical psychosis is an outcome of progressively more persistent subclinical psychotic experiences: an 8-year cohort study. *Schizophrenia Bulletin, 37*(1), 84-93. doi:10.1093/schbul/sbp022
- Duffy, E. (1957). The psychological significance of the concept of "arousal" or "activation". *Psychological Review, 64*(5), 265-275. doi:10.1037/h0048837
- Egeland, J., Nordby, S. J., & Ueland, T. (2010). Do Low-Effort Learning Strategies Mediate Impaired Memory in ADHD? *Journal of Learning Disabilities, 43*(5), 430-440. doi:10.1177/0022219409355473
- Foussias, G., Siddiqui, I., Fervaha, G., Mann, S., McDonald, K., Agid, O., . . . Remington, G. (2015). Motivated to do well: an examination of the relationships between motivation, effort, and cognitive performance in schizophrenia. *Schizophrenia Research, 166*(1-3), 276-282. doi:10.1016/j.schres.2015.05.019

- Fu, T. S.-T., Koutstaal, W., Poon, L., & Cleare, A. J. (2012). Confidence judgment in depression and dysphoria: The depressive realism vs. negativity hypotheses. *Journal of behavior therapy and experimental psychiatry*, *43*(2), 699-704.  
doi:10.1016/j.jbtep.2011.09.014
- Fusar-Poli, P., Nelson, B., Valmaggia, L., Yung, A. R., & McGuire, P. K. (2012). Comorbid depressive and anxiety disorders in 509 individuals with an at-risk mental state: impact on psychopathology and transition to psychosis. *Schizophrenia Bulletin*, *40*(1), 120-131. doi:10.1093/schbul/sbs136
- Garrett, N., Sharot, T., Faulkner, P., Korn, C. W., Roiser, J. P., & Dolan, R. J. (2014). Losing the rose tinted glasses: neural substrates of unbiased belief updating in depression. *Frontiers in Human Neuroscience*, *8*(1), 639. doi:10.3389/fnhum.2014.00639
- Gold, J. M., Strauss, G. P., Waltz, J. A., Robinson, B. M., Brown, J. K., & Frank, M. J. (2013). Negative symptoms of schizophrenia are associated with abnormal effort-cost computations. *Biological Psychiatry*, *74*(2), 130-136.  
doi:10.1016/j.biopsych.2012.12.022
- Gotlib, I. H., Hamilton, J. P., Cooney, R. E., Singh, M. K., Henry, M. L., & Joormann, J. (2010). Neural processing of reward and loss in girls at risk for major depression. *Archives of General Psychiatry*, *67*(4), 380-387.  
doi:10.1001/archgenpsychiatry.2010.13
- Graf, E. W., Warren, P. A., & Maloney, L. T. (2005). Explicit estimation of visual uncertainty in human motion processing. *Vision Research*, *45*(24), 3050-3059.  
doi:10.1016/j.visres.2005.08.007
- Green, M. F., Horan, W. P., Barch, D. M., & Gold, J. M. (2015). Effort-based decision making: a novel approach for assessing motivation in schizophrenia. *Schizophrenia Bulletin*, *41*(5), 1035-1044. doi:10.1093/schbul/sbv071
- Gross, G. M., Kwapil, T. R., Raulin, M. L., Silvia, P. J., & Barrantes-Vidal, N. (2018). The multidimensional schizotypy scale-brief: Scale development and psychometric properties. *Psychiatry Research*, *261*(1), 7-13.  
doi:https://doi.org/10.1016/j.psychres.2017.12.033
- Hafner, H., Löffler, W., Maurer, K., & Hambrecht, M. (1999). Depression, negative symptoms, social stagnation and social decline in the early course of schizophrenia. *Acta Psychiatrica Scandinavica*, *100*(2), 105-118.



- Hanssen, M., Peeters, F., Krabbendam, L., Radstake, S., Verdoux, H., & van Os, J. (2003). How psychotic are individuals with non-psychotic disorders? *Social Psychiatry and Psychiatric Epidemiology*, *38*(3), 149-154. doi:10.1007/s00127-003-0622-7
- Hartmann, M. N., Hager, O. M., Reimann, A. V., Chumbley, J. R., Kirschner, M., Seifritz, E., . . . Kaiser, S. (2014). Apathy but not diminished expression in schizophrenia is associated with discounting of monetary rewards by physical effort. *Schizophrenia Bulletin*, *41*(2), 503-512. doi:10.1093/schbul/sbu102
- Harvey, P. D., Koren, D., Reichenberg, A., & Bowie, C. R. (2005). Negative symptoms and cognitive deficits: what is the nature of their relationship? *Schizophrenia Bulletin*, *32*(2), 250-258. doi:10.1093/schbul/sbj011
- Hebb, D. O. (1955). Drives and the CNS (conceptual nervous system). *Psychological Review*, *62*(4), 243-254. doi:10.1037/h0041823
- Heilbronner, U., Samara, M., Leucht, S., Falkai, P., & Schulze, T. G. (2016). The longitudinal course of schizophrenia across the lifespan: clinical, cognitive, and neurobiological aspects. *Harvard Review of Psychiatry*, *24*(2), 118-128. doi:10.1097/HRP.0000000000000092
- Horan, W. P., Reddy, L. F., Barch, D. M., Buchanan, R. W., Dunayevich, E., Gold, J. M., . . . Green, M. F. (2015). Effort-based decision-making paradigms for clinical trials in schizophrenia: part 2—external validity and correlates. *Schizophrenia Bulletin*, *41*(5), 1055-1065. doi:10.1093/schbul/sbv090
- Häfner, H., Maurer, K., Löffler, W., Der Heiden, W. A., Hambrecht, M., & Schultze-Lutter, F. (2003). Modeling the early course of schizophrenia. *Schizophrenia Bulletin*, *29*(2), 325-340. doi:10.1093/oxfordjournals.schbul.a007008
- Johns, L. C., & Van Os, J. (2001). The continuity of psychotic experiences in the general population. *Clinical Psychology Review*, *21*(8), 1125-1141. doi:10.1016/S0272-7358(01)00103-9
- Judd, L. L., Schettler, P. J., & Akiskal, H. S. (2002). The prevalence, clinical relevance, and public health significance of subthreshold depressions. *Psychiatric Clinics*, *25*(4), 685-698. doi:10.1016/S0193-953X(02)00026-6
- Kirkpatrick, B., Fenton, W. S., Carpenter, W. T., & Marder, S. R. (2006). The NIMH-MATRICES consensus statement on negative symptoms. *Schizophrenia Bulletin*, *32*(2), 214-219. doi:10.1093/schbul/sbj053
- Konings, M., Bak, M., Hanssen, M., Van Os, J., & Krabbendam, L. (2006). Validity and reliability of the CAPE: a self-report instrument for the measurement of psychotic

- experiences in the general population. *Acta Psychiatrica Scandinavica*, 114(1), 55-61.  
doi:10.1111/j.1600-0447.2005.00741.x
- Korponay, C., Nitzburg, G. C., Malhotra, A. K., & DeRosse, P. (2014). Positive and negative subclinical symptoms and MCCB performance in non-psychiatric controls. *Schizophrenia Research: Cognition*, 1(4), 175-179.
- Laws, K. R., & Bhatt, R. (2005). False memories and delusional ideation in normal healthy subjects. *Personality and Individual Differences*, 39(4), 775-781.  
doi:10.1016/j.paid.2005.03.005
- Lim, M. H., Gleeson, J. F., & Jackson, H. J. (2012). The jumping-to-conclusions bias in new religious movements. *The Journal of Nervous and Mental Disease*, 200(10), 868-875.  
doi:10.1097/NMD.0b013e31826b6eb4
- Linscott, R., & Van Os, J. (2013). An updated and conservative systematic review and meta-analysis of epidemiological evidence on psychotic experiences in children and adults: on the pathway from proneness to persistence to dimensional expression across mental disorders. *Psychological Medicine*, 43(6), 1133-1149.  
doi:10.1017/S0033291712001626
- Llerena, K., Strauss, G. P., & Cohen, A. S. (2012). Looking at the other side of the coin: a meta-analysis of self-reported emotional arousal in people with schizophrenia. *Schizophrenia Research*, 142(1-3), 65-70. doi:10.1016/j.schres.2012.09.005
- Longo, L., & Barrett, S. (2010). A Computational Analysis of Cognitive Effort. In *Intelligent Information and Database Systems* (pp. 65-74). Berlin: Springer-Verlag Berlin Heidelberg.
- Mark, W., & Touloupoulou, T. (2015). Psychometric properties of “community assessment of psychic experiences”: review and meta-analyses. *Schizophrenia Bulletin*, 42(1), 34-44.  
doi:10.1093/schbul/sbv088
- Moritz, S., Woodward, T., Jelinek, L., & Klinge, R. (2008). Memory and metamemory in schizophrenia: a liberal acceptance account of psychosis. *Psychological Medicine*, 38(6), 825-832. doi:10.1017/S0033291707002553
- Mueser, K. T., & McGurk, S. R. (2004). Schizophrenia. *The Lancet*, 363(9426), 2063-2072.
- Najas-Garcia, A., Carmona, V. R., & Gómez-Benito, J. (2018). Trends in the Study of Motivation in Schizophrenia: A Bibliometric Analysis of Six Decades of Research (1956–2017). *Frontiers in Psychology*, 9(63). doi:10.3389/fpsyg.2018.00063
- NHS. (2018). Side effects - Antidepressants. Retrieved from  
<https://www.nhs.uk/conditions/antidepressants/side-effects/>

- Niendam, T. A., Bearden, C. E., Johnson, J. K., McKinley, M., Loewy, R., O'Brien, M., . . . Cannon, T. D. (2006). Neurocognitive performance and functional disability in the psychosis prodrome. *Schizophrenia Research, 84*(1), 100-111.  
doi:0.1016/j.schres.2006.02.005
- Pelizza, L., & Ferrari, A. (2009). Anhedonia in schizophrenia and major depression: state or trait? *Annals of General Psychiatry, 8*(1), 22-31. doi:10.1186/1744-859X-8-22
- Peters, E. R., Joseph, S. A., & Garety, P. A. (1999). Measurement of delusional ideation in the normal population: introducing the PDI (Peters et al. Delusions Inventory). *Schizophrenia Bulletin, 25*(3), 553-576. doi:10.1093/oxfordjournals.schbul.a033401
- Pfuhl, G., & Simensen, T. (2019). Motivation, precision task, negative symptoms. Retrieved from osf.io/bzepx
- Pfuhl, G., Tjelmeland, H., Molden, S., & Biegler, R. (2009). Optimal cache search depends on precision of spatial memory and pilfering, but what if that knowledge is not perfect? *Animal Behaviour, 78*(4), 819-828.
- Reddy, L. F., Horan, W. P., & Green, M. F. (2016). Motivational Deficits and Negative Symptoms in Schizophrenia: Concepts and Assessments. In E. H. Simpson & P. D. Balsam (Eds.), *Behavioral Neuroscience of Motivation* (pp. 357-373). Cham: Springer International Publishing.
- Reitan, R. M., & Wolfson, D. (1985). *The Halstead-Reitan neuropsychological test battery: Theory and clinical interpretation* (Vol. 4): Reitan Neuropsychology.
- Rouault, M., Seow, T., Gillan, C. M., & Fleming, S. M. (2018). Psychiatric symptom dimensions are associated with dissociable shifts in metacognition but not task performance. *Biological Psychiatry, 84*(6), 443-451.  
doi:10.1016/j.biopsych.2017.12.017
- Ryan, R. M., & Deci, E. L. (2000). Self-determination theory and the facilitation of intrinsic motivation, social development, and well-being. *American Psychologist, 55*(1), 68-78.  
doi:10.1037/0003-066X.55.1.68
- Salamone, J. D., & Correa, M. (2012). The mysterious motivational functions of mesolimbic dopamine. *Neuron, 76*(3), 470-485. doi:10.1016/j.neuron.2012.10.021
- Salokangas, R. K., Ruhrmann, S., von Reventlow, H. G., Heinimaa, M., Svirskis, T., From, T., . . . Dingemans, P. (2012). Axis I diagnoses and transition to psychosis in clinical high-risk patients EPOS project: prospective follow-up of 245 clinical high-risk outpatients in four countries. *Schizophrenia Research, 138*(2-3), 192-197.  
doi:10.1016/j.schres.2012.03.008

- Shenhav, A., Botvinick, M. M., & Cohen, J. D. (2013). The expected value of control: an integrative theory of anterior cingulate cortex function. *Neuron*, *79*(2), 217-240. doi:10.1016/j.neuron.2013.07.007
- Simpson, E. H., & Balsam, P. D. (2015). The behavioral neuroscience of motivation: an overview of concepts, measures, and translational applications. In *Behavioral Neuroscience of Motivation* (pp. 1-12): Springer.
- Sitskoorn, M. M., Aleman, A., Ebisch, S. J., Appels, M. C., & Kahn, R. S. (2004). Cognitive deficits in relatives of patients with schizophrenia: a meta-analysis. *Schizophrenia Research*, *71*(2-3), 285-295. doi:10.1016/j.schres.2004.03.007
- Stefanis, N., Hanssen, M., Smirnis, N., Avramopoulos, D., Evdokimidis, I., Stefanis, C., . . . Van Os, J. (2002). Evidence that three dimensions of psychosis have a distribution in the general population. *Psychological Medicine*, *32*(2), 347-358. doi:10.1017/S0033291701005141
- Strauss, G. P., & Cohen, A. S. (2017). A transdiagnostic review of negative symptom phenomenology and etiology. *Schizophrenia Bulletin*, *43*(4), 712-719. doi:10.1093/schbul/sbx066
- Strauss, G. P., Horan, W. P., Kirkpatrick, B., Fischer, B. A., Keller, W. R., Miski, P., . . . Carpenter Jr, W. T. (2013). Deconstructing negative symptoms of schizophrenia: avolition–apathy and diminished expression clusters predict clinical presentation and functional outcome. *Journal of Psychiatric Research*, *47*(6), 783-790. doi:10.1016/j.jpsychires.2013.01.015
- Strauss, G. P., Waltz, J. A., & Gold, J. M. (2013). A review of reward processing and motivational impairment in schizophrenia. *Schizophrenia Bulletin*, *40*(Suppl\_2), 107-116. doi:10.1093/schbul/sbt197
- Terenzi, D., Mainetto, E., Barbato, M., Rumiati, R. I., & Aiello, M. (2019). Temporal and Effort cost Decision-making in Healthy Individuals with Subclinical Psychotic Symptoms. *Scientific Reports*, *9*(1), 2151. doi:10.1038/s41598-018-38284-x
- Thomsen, K. R., Whybrow, P. C., & Kringelbach, M. L. (2015). Reconceptualizing anhedonia: novel perspectives on balancing the pleasure networks in the human brain. *Frontiers in Behavioral Neuroscience*, *9*(49), 1-23. doi:10.3389/fnbeh.2015.00049
- Tijssen, M. J., Van Os, J., Wittchen, H.-U., Lieb, R., Beesdo, K., Mengelers, R., . . . Wichers, M. (2010). Evidence that bipolar disorder is the poor outcome fraction of a common developmental phenotype: an 8-year cohort study in young people. *Psychological Medicine*, *40*(2), 289-299. doi:10.1017/S0033291709006138

- Tombaugh, T. N. (2004). Trail Making Test A and B: normative data stratified by age and education. *Archives of Clinical Neuropsychology*, *19*(2), 203-214. doi:10.1016/S0887-6177(03)00039-8
- Treadway, M. T., Buckholtz, J. W., Schwartzman, A. N., Lambert, W. E., & Zald, D. H. (2009). Worth the 'EEfRT'? The effort expenditure for rewards task as an objective measure of motivation and anhedonia. *PloS One*, *4*(8), e6598. doi:10.1371/journal.pone.0006598
- Van Os, J. (2013). The Dynamics of Subthreshold Psychopathology: Implications for Diagnosis and Treatment. *American Journal of Psychiatry*, *170*(7), 695-698. doi:10.1176/appi.ajp.2013.13040474
- Van Os, J., & Kapur, S. (2009). Schizophrenia. *The Lancet*, *374*, 635-645.
- Van Os, J., Linscott, R. J., Myin-Germeys, I., Delespaul, P., & Krabbendam, L. (2009). A systematic review and meta-analysis of the psychosis continuum: evidence for a psychosis proneness–persistence–impairment model of psychotic disorder. *Psychological Medicine*, *39*(2), 179-195. doi:10.1017/S0033291708003814
- van Rossum, I., Dominguez, M.-d.-G., Lieb, R., Wittchen, H.-U., & van Os, J. (2009). Affective dysregulation and reality distortion: a 10-year prospective study of their association and clinical relevance. *Schizophrenia Bulletin*, *37*(3), 561-571. doi:0.1093/schbul/sbp101
- Wechsler, D. (1955). *Manual for the Wechsler adult intelligence scale*. New York: Psychological Corporation.
- Whitton, A. E., Treadway, M. T., & Pizzagalli, D. A. (2015). Reward processing dysfunction in major depression, bipolar disorder and schizophrenia. *Current Opinion in Psychiatry*, *28*(1), 7-12. doi:10.1097/YCO.0000000000000122
- Wigman, J. T., van Os, J., Thiery, E., Derom, C., Collip, D., Jacobs, N., & Wichers, M. (2013). Psychiatric diagnosis revisited: towards a system of staging and profiling combining nomothetic and idiographic parameters of momentary mental states. *PloS One*, *8*(3), 247-257. doi:10.1371/journal.pone.0059559
- Woods, S. W., Addington, J., Cadenhead, K. S., Cannon, T. D., Cornblatt, B. A., Heinssen, R., . . . Walker, E. F. (2009). Validity of the prodromal risk syndrome for first psychosis: findings from the North American Prodrome Longitudinal Study. *Schizophrenia Bulletin*, *35*(5), 894-908. doi:10.1093/schbul/sbp027
- Yang, X.-h., Huang, J., Zhu, C.-y., Wang, Y.-f., Cheung, E. F., Chan, R. C., & Xie, G.-r. (2014). Motivational deficits in effort-based decision making in individuals with

subsyndromal depression, first-episode and remitted depression patients. *Psychiatry Research*, 220(3), 874-882. doi:10.1016/j.psychres.2014.08.056

Zammit, S., Kounali, D., Cannon, M., David, A. S., Gunnell, D., Heron, J., . . . Wolke, D. (2013). Psychotic experiences and psychotic disorders at age 18 in relation to psychotic experiences at age 12 in a longitudinal population-based cohort study. *American Journal of Psychiatry*, 170(7), 742-750. doi:10.1176/appi.ajp.2013.12060768

Zawadzki, J. A., Woodward, T. S., Sokolowski, H. M., Boon, H. S., Wong, A. H. C., & Menon, M. (2012). Cognitive factors associated with subclinical delusional ideation in the general population. *Psychiatry Research*, 197(3), 345-349. doi:10.1016/j.psychres.2012.01.004

**Appendix A. Demographics form**

ID-nummer: \_\_\_\_\_

**Bakgrunnsopplysninger/demografi**Alder: \_\_\_\_\_ Kjønn: \_\_\_\_\_ Fargeblind: **J N** Hendtset: **H V**

Bosituasjon: \_\_\_\_\_

Sivilstand: \_\_\_\_\_

Egne barn: **JA NEI** Antall: \_\_\_\_\_

Høyeste fullført skolegang: \_\_\_\_\_

Antall år e. u.skole: \_\_\_\_\_

Tid som arbeidstaker (m ca. stillingsbrøk): \_\_\_\_\_

År verken skole eller arbeid: \_\_\_\_\_

Nåværende skole-/jobbsituasjon: \_\_\_\_\_

Alkoholvane: \_(ca gng/mnd og enheter/gng) \_\_\_\_\_

Sist merkbart beruset: \_\_\_\_\_

Narkotika siste 3mnd: \_\_\_ **JA NEI** \_\_\_\_\_

Medisin (psykofarmaka): \_\_\_\_\_

Bivirkninger: \_\_\_\_\_

Tidl. psykisk lidelse: \_\_\_\_\_ Symptomfri siden: \_\_\_\_\_

Kjente nevrologiske skader: \_\_\_\_\_

Kjent psykisk lidelse i familien: \_\_\_\_\_

## Appendix B. Procedures precision and motivation task

The order of which we will demonstrate the trials is as follows:

- Trial 1 – Chance trial:** Learning the principles, what is your task
- Trial 2- Chance trial:** Repetition of trial 1
- Trial 3 - No chance trial:** Learning about the possibility of a no chance in 1/6 accounts
- Trial 4 – No chance trial:** Learning about the possibility of a no chance in 2/6 accounts.
- Trial 5 – Chance trial:** Learning about too large intervals and points
- Trial 6 – Chance trial:** Learning about too small intervals and points

**Note: Bold text is information to the administrator. Normal text is what you say to the participant.**

The administrator and the participant will sit together in front of the screen during the demo. Place the participant approximately 50 cm from the screen. Make sure to flip the switch to “demo”, fill in ID number, age and sex, and chose the preferred language. If the participant uses reading glasses, ask her or him to put them on.

For patients go through the demo twice. The administrator will do it the first time while encouraging the participant to play an active role. The second time the participant will do it. For non-patients it is only necessary to go through the demo once. Here the administrator will do the first 2-3 trials before letting the participant take over.

1. The task is about recognising unknown figures. The figures are quite similar, but if you concentrate you will be able to see them apart. You will see them one at a time, and then you are going to learn them, remember them and find them again.

You will receive points for how well you remember a figure, and points for every time you can find them again. The task is in three part, which we now will demonstrate.

Administrator shows which buttons to use (left mouse button and shift)

Click continue to start demo

**Administrator shows which buttons to use (left mouse button and shift). Click continue to start demo**



2. **Trial 1: Chance trial.** The start of every trial is when you see a white cross on the screen.

3. **Task 1:** A figure will appear for two seconds when you click the cross. Try to remember this figure as well as you can. Click the cross now.

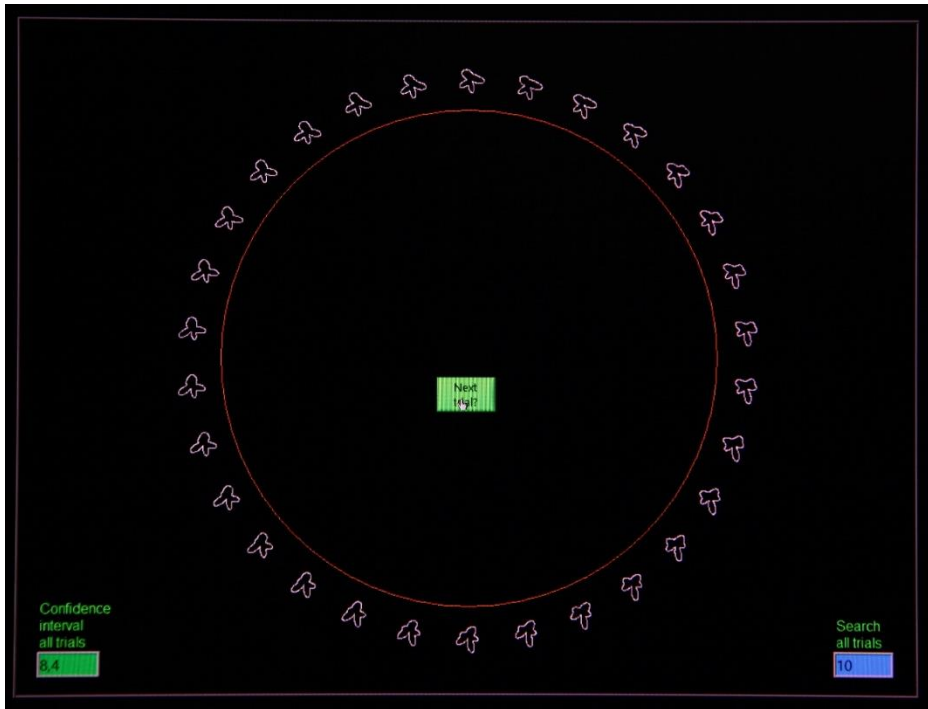


*A cross is the first to appear at every trial.*



*Then an unknown, abstract figure appears for two seconds.*

4. Now a new picture comes up, it contains of a circle with 30 different figures around.



5. **Task 2:** Your figure is hidden somewhere along the circle. Where? It does not have to be exactly like the ones around the circle.

Find the area you think the figure belongs and click with the left mouse button inside the circle. Be sure to be precise when you click.

6. Around the point you click you shall now expand a green interval. Your interval should be large enough for you to be sure that your figure is in this area. Keep the marker inside the circle.

7. When the interval covers the area where your figure is you will receive points for finding the figure.



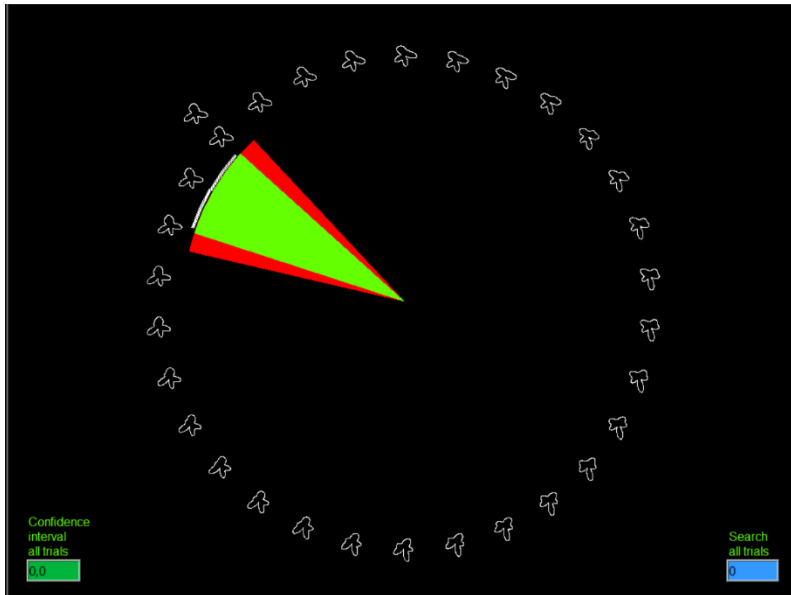
*Mark the area where your figure is hidden with the green interval. Your figure is hidden where the figures around the circle are most similar.*

8. Save the area by pressing SHIFT.

9. **PS:** If you have forgotten how the figure looks like, you can at any time press the green button in the middle of the screen to proceed to the next trial.

10. **Task 3:** You will now search after the figure where you think it is. Click anywhere in the circle to start the search. You can see that every click expands a white line moving from the place you originally clicked in the previous task. When the white line arrives at the place where the figure hides it will automatically pop up outside the circle.

For the moment pay no attention to the squirrel, we will explain that later.



*Here you can see both the white search line and the correct figure outside the circle.*

11. **Trial 2:** Chance trial. Repeating the basic principles.

12. **Trial 3:** No chance trial.

**Begin by clicking your way to where you see the squirrel.**

13. Imagine that the figures are in fact acorns. In this game there are two hungry squirrels. They keep themselves hidden from you, but are after acorns, which they sometimes steal. That means sometimes a squirrel will take your acorn before you can find it and before you know it (You will not be able to stop it by being faster, sometimes it just happens).

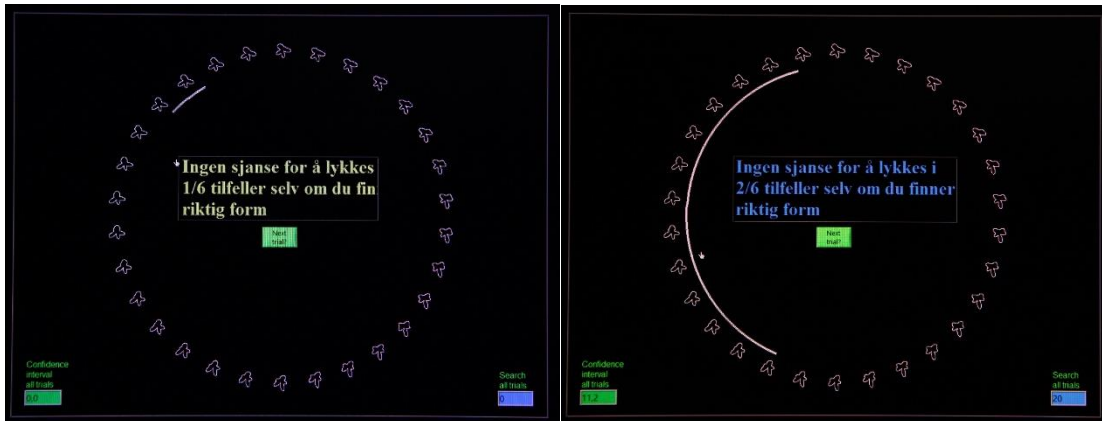
Squirrel 1 is the least hungry. In an equivalent to 1 out of 6 this squirrel has taken your acorn before you can find it. You will therefore click past where your acorn should have been on the circle. Since it is gone it will not pop up.

At some point you just have to give up and move along to the next trial. Press “next trial” to move on. When you decide to do this is all up to you.

14. **Trial 4: No chance trial.**

**Repeat the previous steps until you see the squirrel.** As mentioned earlier there are two squirrels in this game. Squirrel two is twice as hungry, he will take your acorn 2 out of 6 times. Which squirrel you are facing can be seen in the middle of the screen.

15. Mark: In most trials you will find your acorn and receive 10 points. If you do not find your acorn you will receive 0 point.



### 16. Trial 5: Chance trial.

#### Before you click on the cross:

You receive point for two different things. 1: How well you remember and are able to localize each acorn hidden in the circle. 2: For every acorn you find.

The green box is points for how precisely you indicate your interval. The blue box is points for each acorn found. The maximum points in a trial is 10 for each thing.

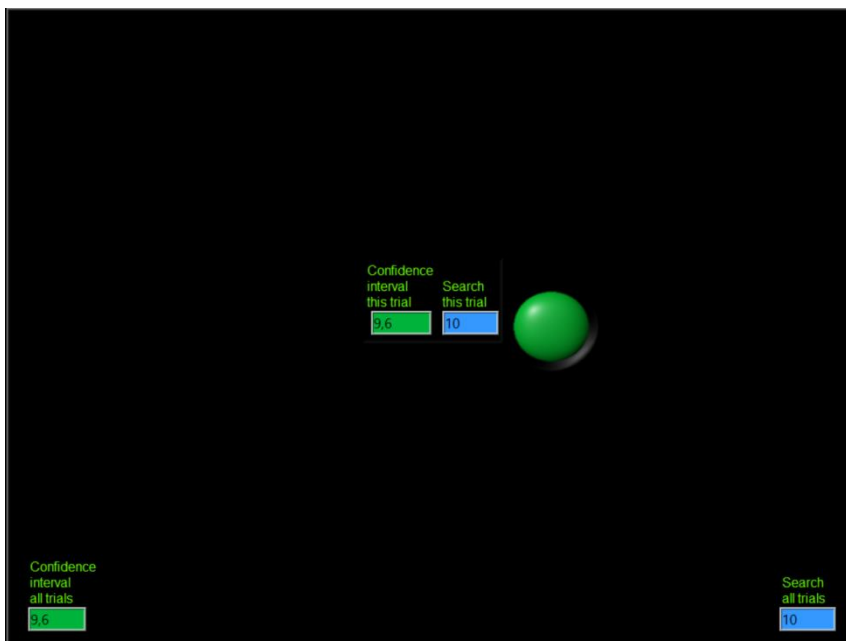
#### 17. Repeat the steps.

The points you receive is shown at the end of each trial and at the bottom of the screen your total.

Click until you find the figure.

Here you received x out of 10 points for your interval.

The red part shows you how much bigger than necessary your interval was. This time the red part is small because we made a precise interval



*An example of 9,6/10 for the interval and 10 point for finding the acorn.*

### 18. Trial 6: Chance trial.

**Before you click on the cross:**

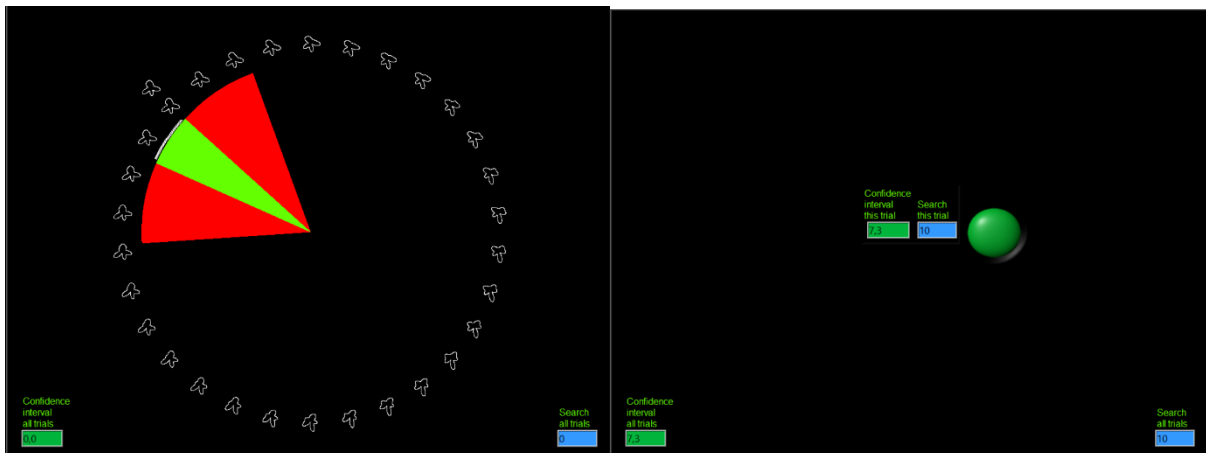
Think about your interval.

The green score is dependent on that you make a reasonably large interval. Make it too big and you will receive a low score, make it fairly accurate and you will receive a high score.

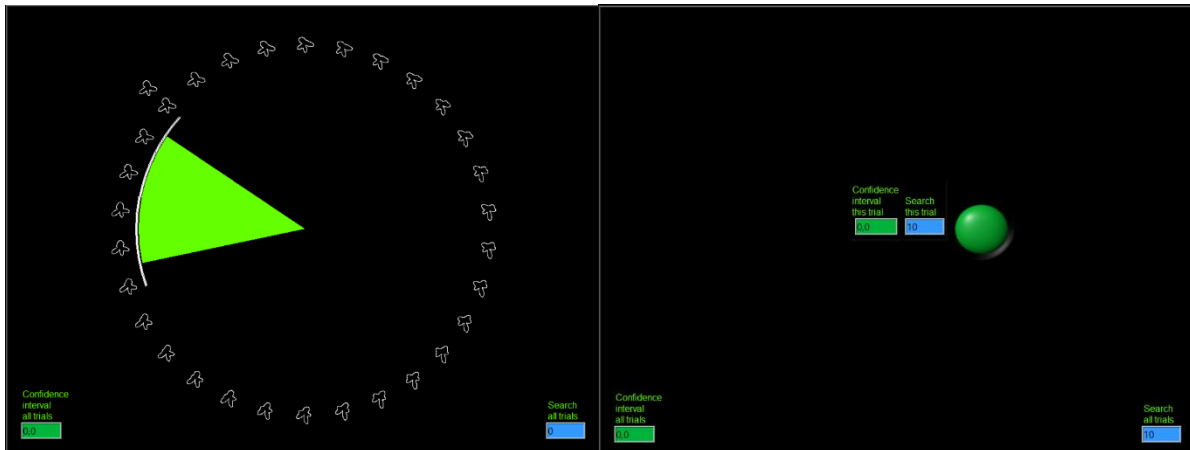
And if your interval is too small and not covering where your acorn is you will receive zero points. This apply even if a squirrel has taken the acorn you are looking for. In short you have to make an interval large enough for you to be sure that it covers where your acorn is, but not so big that you lose too many points.



*This is a good interval, and we received 9,6 out of a possible 10 points.*



*This interval is a bit too big, and we therefore only received 7,6 out of 10 points.*



*Here we made an interval that was too small, and therefore did not hit the figure we were looking for. We then receive 0 out of 10 points.*

In the blue box you will receive 10 points every time you find the acorn. When you don't find the acorn you will still receive point for how precise your interval is.

**Remember to switch to “experiment”. Make sure the participant understood the task and ask if they have any questions before moving on.**

**The task has 45 trials. When they finish the task, they will automatically get back to the start page. You do not have to save before exiting.**

## Appendix C. Approval from REK



---

<b>Region:</b>	<b>Saksbehandler:</b>	<b>Telefon:</b>	<b>Vår dato:</b>	<b>Vår referanse:</b>
REK vest	Fredrik Rongved	55978498	12.06.2018	2011/1198/REK vest
			<b>Deres dato:</b>	<b>Deres referanse:</b>
			07.05.2018	

Vår referanse må oppgis ved alle henvendelser

Wenche ten Velden Hegelstad

Psykoseforskning

### 2011/1198 TIPS II

**Forskningsansvarlig:** Helse Stavanger HF - Stavanger universitetssjukehus

**Prosjektleder:** Wenche ten Velden Hegelstad

Vi viser til søknad om forhåndsgodkjenning av ovennevnte forskningsprosjekt. Søknaden ble behandlet av Regional komité for medisinsk og helsefaglig forskningsetikk (REK vest) i møtet 15.08.2018. Vurderingen er gjort med hjemmel i helseforskningsloven (hfl.) § 10.

### Prosjektomtale

*I TIPS III er en studie på første gangs psykoser med vekt på tidlig oppdagelse. Vi har også fokus på om det finnes undergrupper av pasienter med rusmisbruk og psykose, karakterisert ved biologiske, symptomatologiske, kognitive og demografiske kjennetegn, som har en høynet risiko for utvikling av schizofrenispekter lidelse ved rusmisbruk. En ønsker også å undersøke effekten av vedvarende rusmisbruk på schizofrenispekter og andre psykoser*

### Vurdering

#### Ønsket tilbakemelding

REK vest ønsket tilbakemelding om følgende:

- Det må forklares hvordan de nye kontrollpersonene rekrutteres til studien
- Et nytt informasjonsskriv rettet mot de kontrollpersonene må sendes til REK vest

#### Tilbakemelding

Prosjektleder har gitt tilbakemelding på rekruttering av kontrollpersonene og vedlagt informasjonsskriv rettet mot dem.

REK vest ved leder vurderte saken.

### Vurdering



REK vest har ingen innvendinger til rekrutteringsprosedyren, men har noen merknader til informasjonsskrivet:

Honorering: REK vest har ikke vært klar over at kontrollpersonene skulle honoreres 100 kroner for deltakelse. Dette burde vært meldt tydeligere ifra om. REK vest har ikke noen ytterligere merknader til dette og har ingen innvendig mot at deltakerne honoreres 100 kroner.

Begrep: Det står først i informasjonsskrivet at data *anonymiseres* like etter innsamling. Under kapittel B står det at hver deltaker vil tildeles en ID som kobler deres opplysninger, men at ingen personidentifiserbare opplysninger vil beholdes. Dette virker forvirrende. REK vest går ut fra at data i første omgang registreres i aidentifisert form, og at de anonymiseres eller slettes ved prosjektslutt.

Prosjektslutt: Informasjonsskrivet spesifiserer ikke når prosjektet slutter. REK vest ber om at prosjektslutt skrives inn i informasjonsskrivet og at det deretter sendes på epost.

#### **Vilkår:**

REK vest setter følgende vilkår:

- Revidert informasjonsskriv sendes til REK vest på epost. Bruk [post@helseforskning.etikkom.no](mailto:post@helseforskning.etikkom.no). Skriv «REK vest» og «2011/1198» i emnefeltet
- Data anonymiseres eller slettes ved prosjektslutt

#### **Vedtak**

*REK vest godkjenner prosjektendringen på betingelse av at ovennevnte vilkår tas til følge.*

#### **Klageadgang**

Du kan klage på komiteens vedtak, jf. forvaltningsloven § 28 flg. Klagen sendes til REK vest. Klagefristen er tre uker fra du mottar dette brevet. Dersom vedtaket opprettholdes av REK vest, sendes klagen videre til Den nasjonale forskningsetiske komité for medisin og helsefag for endelig vurdering.

Med vennlig hilsen

Marit Grønning

Prof. dr.med. Komiteleder

Fredrik Rongved  
rådgiver

**Kopi til:** forskning@sus.no

**Emne:** Sv: revidert informasjonsskriv  
**Fra:** post@helseforskning.etikkom.no  
**Dato:** 26.06.2018 14:54  
**Kopi:** forskning@sus.no  
**Til:** wenchetenvelden@me.com

**Vår ref. nr.: 2011/1198**

**Prosjekttittel: "TIPS 11"**

**Prosjektleder: Wenche ten Velden Hegelstad**

Til Wenche ten Velden Hegelstad.

Vi viser til revidert informasjonsskriv innsendt 12.06.2018. Komiteen tar dette til orientering og har ingen ytterligere merknader.

Med vennlig hilsen

Jessica Svärd

rådgiver

[post@helseforskning.etikkom.no](mailto:post@helseforskning.etikkom.no)

T: 55978497

**Regional komité for medisinsk og helsefaglig  
forskningsetikk REK vest-Norge (REK vest)**

**<http://helseforskning.etikkom.no>**



**Prosjektendring** Skjema for søknad om godkjenning av prosjektendringer i de regionale komiteer for medisinsk og helsefaglig forskningsetikk (REK)

2011/1198-20

Dokument-id: 1008265 Dokument mottatt 26.03.2018

## TIPS II (2011/1198)

---

### 1. Generelle opplysninger

#### a. Prosjektleder

CRISStin Person ID	51863
Navn:	Wenche ten Velden Hegelstad
Akademisk grad:	PhD
Klinisk kompetanse:	Psykologspesialist
Stilling:	PI TIPS Stavanger
Hovedarbeidsgiver	Stavanger Universitetssykehus
Arbeidsadresse:	Psykoceforskning
Postnummer	4016
Sted	Stavanger
Telefon	98262356
E-post adresse	wenchetenvelden@mac.com

#### b. Prosjekt

Hvilket prosjekt skal endres?

TIPS II (2011/1198)

#### c. Ny Prosjektleder?

Skal prosjektet ha ny prosjektleder?

Nei

<b>d. Forskningsansvarlig(e)</b>
----------------------------------

Forskningsansvarlig(e) som beholdes

<b>Institusjon</b>	<b>Kontaktperson</b>	<b>Stilling</b>	<b>E-post adresse</b>
Helse Stavanger HF	Tor Ketil Larsen	professor/forskningssjef	forskning@sus.no
Helse Stavanger HF Stavanger universitetssjukehus			forskning@sus.no

<b>e. Prosjektmedarbeider(e)</b>
----------------------------------

Prosjektmedarbeider(e) som beholdes

<b>Navn:</b>	<b>Stilling:</b>	<b>Institusjon:</b>	<b>Akademisk Rolle:</b>	<b>Rolle:</b>
Ingvild Aase	PhD kandidat	SUS	cand psychol stipendiat	
Wenche ten Velden Hegelstad	Fagsjef	SUS	PhD	Forsker
Kolbjørn Brønnick	seniorforsker/professor	SUS	PhD	Forsker
Inge Joa	Forsker	SUS	PhD	Forsker
Robert Jørgensen	Psyk sykepleier	SUS	Bachelor	Medarbeider

Ny(e)

prosjektmedarbeider(e)

<b>Navn</b>	<b>Stilling</b>	<b>Institusjon</b>	<b>Akademisk Rolle</b>	<b>Rolle</b>
			<b>rolle</b>	

---

Thea Simensen

Student

UiT

Mastergrad Doktorgradstudent

---

## 2. Endring(er)

### a. Endringen(e) innebærer

Ny(e) prosjektmedarbeider(e) som angitt

---

Annen prosjektendring

---

#### *Redegjør for endringer*

Friske kontrollpersoner i delstudien som omhandler motivasjon (prosjektendring godkjent 21.12.2015; del av TIPS II) har vært intervjuet ved hjelp av PANSS (Positive and Negative Syndromes Scale). En ny gruppe på 10 friske kontrollpersoner vil nå undersøkes ved hjelp av selvrapporteringskjemaet med CAPE 42 (vedlegg) i stedet for PANSS.

---

### b. Begrunnelse for endringen(e)

*Praktisk, faglig og vitenskapelig begrunnelse for endringen(e)*

CAPE 42 er mer hensiktsmessig for å utelukke generell psykopatologi enn PANSS, som er et instrument utarbeidet spesifikt for personer med psykose, eller hvor det er sterk mistanke om psykose. Da et hovedkriterie for inklusjon som matchet kontrollperson i denne delstudien er at kontrollperson ikke oppfyller kriterier på noen akse-I diagnose i DSM IV ønsker iv her å be om godkjenning for å erstatte PANSS med CAPE 42 for disse personene. I tillegg er CAPE 42 mye enklere å administrere og mindre belastende og tidkrevende for kontrollpersonene, enn PANSS. Ellers følges forskningsprotokoll jfr endringer og beskrivelse av delstudie av desember 2015.

---

## 3. Avveining av nytte og risiko ved prosjektendringene

*Hvorfor er det forsvarlig å gjennomføre endringene? Gi en begrunnet avveining av fordelene og ulempene ved prosjektendringene.*

Fordeler:

\*Instrumentet dekker mer allmenn psykotpatologi enn bare psykose.

\*Instrumentet er selvutfyllbart, det er mer privat, mindre invaderende, og mindre tidkrevende

Ulemper:

Ingen kjente. Dersom personer ved CAPE 42 viser seg å ha behandlingstrengende psykiske vansker vil de ikke lenger være aktuelle som kontrollperson og vil hjelpes til adekvat behandling.

---

#### 4. Vedlegg

#	Type	Filnavn	Lagt inn dato
1.	Øvrige vedlegg	Stefanis CAPE 42.pdf	26.03.18
2.	Øvrige vedlegg	CAPE_42 norsk.pdf	26.03.18

#### 5. Ansvarserklæring

Jeg erklærer at prosjektet vil bli gjennomført

---

i henhold til gjeldende lover, forskrifter og retningslinjer

---

i samsvar med opplysninger gitt i denne søknaden

---

i samsvar med eventuelle vilkår for godkjenning gitt av REK

---

## Appendix D. Consent form



### Forespørsel om deltakelse i forskningsprosjektet

#### MOTIVASJON OG MÅLRETTET ATFERD HOS PERSONER MED PSYKISKE LIDELSER - KONTROLLPERSONER

Studien Tidlig Intervensjon ved Psykose (TIPS2) undersøker forløpet til psykoser og effekt av behandling. Det testes også kontrollpersoner som aldri har hatt psykoser, personer som deg. Vi ønsker å se på en spesiell type symptomer litt nøyere: Noen pasienter med psykose opplever at det er tungt å komme i gang med ting og å holde på motivasjon. Disse plagene kalles negative symptomer. Dette er et spørsmål til deg om å delta i studien for å forstå mer av hvordan mental anstrengelse henger sammen med negative symptomer.

I denne studien vil vi undersøke sammenhengen mellom graden av negative symptomer og motivasjon når den måles gjennom anstrengelse. Anstrengelse og motivasjon måles ved hjelp av en datatest på ca. 20 minutter. Vi definerer at økt anstrengelse indikerer sterkere evne til å initiere og igangsette målrettet atferd. Anstrengelse gir således et mål på motivasjon. Vi observerer sammenlagt innsats over flere forsøk med varierende vanskelighetsgrad i dataoppgaven.

#### Hva innebærer prosjektet?

Du vil ta to korte tester, en på papir og en på datamaskin som likner litt på gammeldagse dataspill og undersøker dette med anstrengelse og motivasjon.

#### Mulige fordeler og ulemper

Økt forståelse av negative symptomer hos pasienter vil kunne hjelpe oss mot en bedre og mer effektiv tilnærming og behandlingstilbud. Deltakelse innebærer ikke noen ulemper.

#### Frivillig deltakelse og mulighet for å trekke sitt samtykke

Det er frivillig å delta i studien. Du kan når som helst og uten å oppgi noen grunn trekke ditt samtykke til å delta i studien. Dette vil ikke få konsekvenser for deg. Dersom du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Om du nå sier ja til å delta, kan du senere trekke tilbake ditt samtykke.

Har du spørsmål til studien, ta kontakt med Wenche ten Velden Hegelstad, tlf. 51515877, Thea Simensen på telefon: 41419711 eller på mail [theasim@ntnu.no](mailto:theasim@ntnu.no), eller Gerit Pfuhl: [gerit.pfuhl@uit.no](mailto:gerit.pfuhl@uit.no).

### **Hva skjer med informasjonen om deg?**

#### **Personvern**

Du får en ID og alle resultater lagres med ID, ikke ditt personnavn eller andre identifiserbare informasjon.

#### **Rett til innsyn og sletting av opplysninger om deg**

Hvis du sier ja til å delta i studien, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg. Du har videre rett til å få korrigert eventuelle feil i de opplysningene vi har registrert. Dersom du trekker deg fra studien, kan du kreve å få slettet innsamlede prøver og opplysninger, med mindre opplysningene allerede er inngått i analyser eller brukt i vitenskapelige publikasjoner. Opplysningene vil da ikke brukes videre i studien.

Forskningsdata vil bli anonymisert og lagres bare anonymisert på datamaskin. Data vil bli slettet senest 5 år etter prosjektslutt.

Informasjonen som registreres om deg skal kun brukes slik som beskrevet i hensikten med studien. Alle opplysningene vil bli behandlet uten navn og fødselsnummer eller andre direkte gjenkjennende opplysninger. Det vil ikke være mulig å identifisere deg i resultatene av studien når disse publiseres.

#### **Økonomi**

Du vil få 150 kr som kompensasjon for reise- og oppholdsutgifter i form av et gavekort i forbindelse med testene.

#### **Godkjenning**

Prosjektet er godkjent av Regional komite for medisinsk og helsefaglig forskningsetikk, (2011/1198/REK vest)