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The interactions between prenatal and postnatal maternal depressive symptoms, protective factors, stressful life events and neurophysiology in early infancy

Deutscher Titel der Dissertation:

Die Interaktionen zwischen pränatalen und postnatalen mütterlichen depressiven Symptomen, Schutzfaktoren, belastenden Lebensereignissen und Neurophysiologie im frühen Kindesalter

vorgelegt von:

Polina Plamenova Stoyanova, M. Sc. Psych.

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DISSERTATION

Gutachterinnen:

Prof. Dr. Birgit Mathes, BRISE Universität Bremen

Prof. Dr. Karina Weichold, Institut für Psychologie Arbeitsbereich Jugendforschung Friedrich-Schiller-Universität Jena

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ABSTRACT

Prenatal and postnatal maternal depressive symptoms have been linked to infants' neurophysiological impairments, cognitive deficits, developmental delay, and persisting emotional and behavioral problems. Stressful life events may increase self-perceived depressive symptoms of mothers. Concurrently, social support may reduce prenatal and postnatal maternal depressive symptoms. This dissertation investigated how prenatal and postnatal maternal depressive symptoms relate to infants' neurophysiological outcomes, as well as to protective factors for the mothers and stressful life events. The impact of maternal educational status on these relations was considered. 150 mothers and infants from socially and culturally disadvantaged families participating in a longitudinal study (Bremen Initiative to Foster Early Childhood Development). The participants were interviewed 1) after the 30th week of their pregnancy and till the 10th week after child birth, and 2) between the 2nd and 4th month after child birth, and 3) between the 6nd and 8th month after child birth. Neurophysiological activity was examined in six-to-eight-month-old infants as assessed by spontaneous Electroencephalogram (EEG). Different statistical analyses were conducted in this dissertation to investigate the interactions firstly, between prenatal and postnatal maternal depressive symptoms and infants' neurophysiology and secondly, between prenatal and postnatal maternal depressive symptoms, stressful life events and overall social and partner social support. The results revealed changes of spontaneous brain activity during alert resting states in infants of depressed mothers, which were similarly observed in depressed patients. Low frontal and parietal alpha power and alpha desynchronization were reported for high-risk infants and related to their mother's depressive symptoms and partly to their educational status. Stressful life events were

predictors for the occurrence of maternal depressive symptoms during pregnancy. Overall social and partner social support reduced maternal depressive symptoms and thereby, were significant resources for depressed mothers during both prepartum and postpartum. Neurophysiological markers, such as adverse alpha power and alpha desynchronization, might play an important role for the identification of infants at risk. Understanding the role of maternal depressive symptoms and its interactions with overall social and partner social support, stressful life events and maternal educational status may help developing effective intervention programs for families during the phase of pregnancy and early infancy.

ZUSAMMENFASSUNG

Pränatale und postnatale mütterliche depressive Symptome wurden bislang mit neurophysiologischen Beeinträchtigungen des Säuglings sowie mit kognitiven Defiziten, Entwicklungsverzögerungen und anhaltenden emotionalen und Verhaltensproblemen in Verbindung gebracht. Lebensereignisse, welche durch Stress gekennzeichnet sind, können die selbst wahrgenommenen depressiven Symptome von Müttern verstärken. Gleichzeitig kann die soziale Unterstützung die pränatalen und postnatalen depressiven Symptome der Mütter reduzieren. Diese Dissertation untersuchte, wie pränatale und postnatale mütterliche depressive Symptome mit neurophysiologischen Outcomes des Säuglings sowie mit Ressourcen und belastenden Lebensereignissen von Müttern zusammenhängen. Dabei wurde der Einfluss des mütterlichen Bildungsstatus auf diese Zusammenhänge berücksichtigt. Es nahmen 150 Mütter und Säuglinge aus sozial und kulturell benachteiligten Familien an einer Längsschnittstudie (im Rahmen der Bremer Initiative zur Förderung der frühkindlichen Entwicklung) teil. Die Befragungen fanden 1) nach der 30. Schwangerschaftswoche und bis zur 10. Woche nach der Geburt des Kindes und 2) zwischen dem 2. und 4. Monat nach der Geburt des Kindes sowie 3) zwischen dem 6. und 8. Monat nach der Geburt des Kindes statt. Die neurophysiologische Aktivität wurde bei sechs bis acht Monate alten Säuglingen anhand eines spontanen Elektroenzephalogramms (EEG) untersucht. In dieser Dissertation wurden verschiedene statistische Analysen durchgeführt, um die Interaktionen erstens zwischen pränatalen und postnatalen depressiven Symptomen der Mutter und der Neurophysiologie des Säuglings und zweitens zwischen pränatalen und postnatalen depressiven Symptomen der Mutter, belastenden Lebensereignissen und allgemeine soziale und partnerschaftliche soziale Unterstützung zu untersuchen. Die

ZUSAMMENFASSUNG

Ergebnisse zeigten Veränderungen der spontanen Hirnaktivität während des wachen Ruhezustands bei Säuglingen der depressiven Mütter, welche in ähnlicher Form bei depressiven Patienten beobachtet wurden. Eine niedrige frontale und parietale Alpha-Power und eine Alpha-Desynchronisierung der Hirnaktivität wurden für Hochrisikokinder berichtet und standen in Zusammenhang mit den depressiven Symptomen der Mutter und teilweise mit ihrem Bildungsstatus. Stressige Lebensereignisse waren Prädiktoren für das Auftreten von mütterlichen depressiven Symptomen während der Schwangerschaft. Allgemeine soziale und partnerschaftliche soziale Unterstützung verringerten die depressiven Symptome der Mütter und stellten somit bedeutende Ressourcen für depressive Mütter sowohl während der Zeit vor als auch nach der Geburt des Kindes dar. Neurophysiologische Marker wie beeinträchtigte Alpha-Power und eine Alpha-Desynchronisierung könnten eine wichtige Rolle für die Identifizierung von gefährdeten Säuglingen spielen. Das Verständnis der Rolle mütterlicher depressiver Symptome und ihrer Interaktionen mit allgemeiner sozialer und partnerschaftlicher sozialer Unterstützung, belastenden Lebensereignissen und dem Bildungsstatus der Mutter kann dazu beitragen, wirksame Interventionsprogramme für Familien in der Phase der Schwangerschaft und der frühen Kindheit zu entwickeln.

1 CHAPTER ONE: INTRODUCTION

The story of life begins with a cry, and then humans become a part of an emotionally rich world, surrounded by people, full of different interactions with both positive and negative directions. Thus, during the lifespan development varios interrogations have been faced. Concurrently, periods of developmental transitions in life, such as prenatal development and infancy, have a tremendous meaning for the whole life *continuum* (Van den Bergh et al., 2017). Additionally, life transitions, such as pregnancy and becoming a mother, are associated with lots of changes, causing distress for the woman and the baby and difficulties to adjust (Van Batenburg-Eddes et al., 2013). Moreover, stress-related challenges might be determinant and render some groups at greater risk. Besides, disadvantaged families might be under higher risk for experiencing more stressful life events, such as financial problems, job loss, death or disease in family, and depressive symptoms (Van den Bergh et al., 2017). Furthermore, stressful life events could be a risk factor for developing depressive symptoms during pregnancy.

The occurrence of depressive symptoms during the transition into motherhood is related to infants' protracted neurophysiological outcomes (Van Batenburg-Eddes et al., 2013; Van Den Bergh, Dahnke, & Mennes, 2018). Moreover, previous research shows that prenatal and postnatal maternal depressive symptoms have a negative impact on the neural development and cognitive abilities of children throughout their lifespan (Kinsella & Monk, 2009; O'Connor et al., 2014; O'Donnell et al., 2009; Van Batenburg-Eddes et al., 2013; Van den Bergh et al., 2017; Van den Bergh et al., 2018). Previous electroencephalogram (EEG) research has discovered associations between maternal depressive symptoms and the infants' frontal alpha desynchronization patterns (Van den Bergh et al., 2017; Van den Bergh et al., 2018). Studies with infants from

prenatally and postnatally depressed mothers indicate adverse neurophysiological outcomes, such as right-hemispheric dominance of frontal alpha desynchronization, impairments in cognitive and emotional processing developmental delays, changes resembling self-regulations and behavior problems (O'Connor et al., 2014; O'Donnell et al., 2009; Talge et al., 2007). Even though there are many studies investigating the association between the neurophysiology and depression, there are still some unanswered questions.

Concurrently, maternal depressive symptoms might be positively influenced from maternal resources, such as social support provided from partners, families and friends (Kinsella & Monk, 2009; O'Connor et al., 2014; O'Donnell et al., 2009; Van Batenburg-Eddes et al., 2013; Van den Bergh et al., 2017; Van den Bergh et al., 2018). Previous studies have shown that social support for the mothers help to better cope with stress and reduce depressive symptoms (Elsenbruch et al., 2007; Glazier et al., 2004). Hence, resources for socially and culturally disadvantaged mothers serve to prevent disparities in maternal risk factors such as maternal depressive symptoms. Moreover, social support through partners, families, relatives or friends can be an important protective factor for women during pregnancy and the early infancy period. Furthermore, the investigation of protective factors as preventive tools for mothers and infants at risk might be of crucial importance.

This study will be examined the interactions between prenatal and postnatal depressive maternal symptoms, resources, stressors, educational status and infants' neurophysiology in a sub-clinical sample, representing different cultures and social context. In this dissertation, firstly, prenatal and postnatal maternal depressive symptoms and their impact on infants' neurophysiology will be investigated. Secondly, the predictors of depressive symptoms will be separately presented. Next, the results

will be interpreted and compared with previous research and further aspects will be discussed. Finally, the limitations and the importance of the study sample and future research within groups of socially and/or culturally disadvantaged people will be highlighted.

1.1 The current project: BRISE (The Bremen Initiative to Foster Early Childhood Development)

The data in this dissertation were collected and generated out of the Bremen Initiative to Foster Early Childhood Development (BRISE) Project, 1st wave. The project investigates longitudinally the family environment and early cognitive, social, emotional and neurophysiological development of children from pregnancy through the school entrance. BRISE aims to detect strategies that ameliorate social support structures and to improve educational equity.

The sample in this study includes the first 150 mothers and their infants, from socially and/or culturally disadvantaged families. These are the 150 women from the 1st wave, who attended the project when were pregnant or when their babies were not elder than 10 weeks. The participants have been selected because they lived in specific districts of the German province *Bremen* and had at least one possible risk factor for the infants' development. These risk factors might be socio-economic included low family income, unemployment, low parental educational status and migration background of at least one of the newborn's parents or grandparents. The characterisation "socially and culturally disadvantaged" does not mean that the participants in the BRISE project live in extreme poverty, but rather means that they experience more stressors and disturbances due to the above-mentioned risk factors and hazards than other groups. As

the sample had a large percentage of families with a migration background (46%), the study represents different cultures and contexts. Moreover, BRISE study emphasizes the importance of conducting research with culturally and socially diverse populations, having the differing social settings, reflecting on the social environment and possibly on the transition to motherhood.

1.2 Depression and Depressive symptoms

More than 300 million people over the world suffer from depression or depressive episode and the numbers have increased drastically every year (World Health Organization, 2017). According to the World Health Organization (WHO, 2017), low- and middle-income regions as well as socially and culturally disadvantaged people who lived in well-developed countries are more vulnerable to mental disorders in general and specifically to depression. The socially and culturally disadvantaged people are nearly twice more likely to develop depression (Akhtar-Danesh & Landeen, 2007). A higher socio-economic status (SES) might be protective and preventing depression, reinforce findings from previous studies (Akhtar-Danesh & Landeen, 2007; Freeman et al., 2016; Lorant et al., 2003; Schlax et al., 2019). A meta-analysis on SES inequalities in depression show that for each additional year of education, the odds of being depressed decreased by 3 % (Lorant et al., 2003). A Canadian study found that low educational status and low income were associated with a higher risk of major depressive episodes (Wang et al., 2010). Moreover, the risk for developing the depressive disorder is increased not only by low SES but by inconsistent family situation and other illnesses as well (Akhtar-Danesh & Landeen, 2007).

Women suffer more often from depression than men as well (World Health Organization, 2017). Female have double to triple the prevalence rate for 12 month depression than male (Akhtar-Danesh & Landeen, 2007). In Germany, the probability to develop depression at least once in lifetime is around 17.1%, with a much higher rate for women (23%) than men, whose lifetime prevalence for depression is 11.1% (Jacobi et al., 2014).

Depression leads to tremendous decrease in physiological and psychological health and well-being of the people, affecting the cognitive and socio-emotional processing (Lee et al., 2011). The main depressive symptoms are fatigue, loss of motivation, feeling of sadness and hopelessness, sleep and physical disturbances, anxiety and restlessness (American Psychiatric Association, 2013). Of course, there are variation in the occurrence and severity of the symptoms. Concurrently, depression has comorbidity and some similar characteristics with other affective disorders such as anxiety (Ulrich & Petermann, 2016).

Diagnosis of depression is made by clinical expert based on diagnostic interviews, structured around the updated diagnosis classification systems of Diagnostic and Statistical Manual of Mental Disorders (DSM-V) and previously (DSM-IV) (American Psychiatric Association, 2013), when the duration of symptoms last at least two weeks. The criteria on which a person is diagnosed with depression are based on self-reported symptoms associated with behavioral, cognitive, emotional and somatic disturbances (Lee et al., 2011). Even though the classical approach is to conduct an interview by clinicians reflected by self-reported assessment of the patients (Lee et al., 2011), different biomarkers identifying depression or other mental disorder might be an important tool to support future diagnosis. According to Olbrich & Arns (2013), during a self-reported interview patient and expert biases might be introduced, thereby, new

ways to diagnose depression using EEG to highlight important markers should be implemented. Thus, a biomarker might be able to assess and diagnose objectively a disorder.

1.2.1 Depressive symptoms in prenatal and postnatal periods

As depression impairs the mental health which is considered as a predictor for well-being, lifetime balance and better overall health (World Health Organization, 2017), its investigation is a research topic with a tremendous importance. Especially, during and after pregnancy, when women experience different transformations, like changing hormone levels, physical, and psychological changes which make them much more vulnerable to a depressive episode (Glover, 2014; Kinsella & Monk, 2009; Van Batenburg-Eddes et al., 2013). Moreover, during prepartum and postpartum, new behaviors are lay down, psychosocial trajectories are canalize, and a new epidemiology of diseases might emerge (Glover, 2014; Kinsella & Monk, 2009; Van Batenburg-Eddes et al., 2013). Therefore, the period of pregnancy (prenatal period or prepartum) and after birth (postnatal period or postpartum) are related to the transition to motherhood and might be associated with more challenges for women (Van Batenburg-Eddes et al., 2013) and marked as stressful life events (Geller, 2004). Especially when at this time, some other stressful life events (divorce, job loss, death or disease in family) appear (Van den Bergh et al., 2017). Such stressors might be a risk factor for having emotional disturbances for every woman and more vulnerable might be women during pregnancy, with genetic predispositions and from disadvantaged families (Van den Bergh et al., 2017). Depression that occurs during pregnancy is referred to as a prenatal depression and after birth is referred to as a postnatal depression (Grote et al., 2010; Ulrich &

Petermann, 2016).

Depression seems to be the most common mental disorders during pregnancy because up to 18.4% of pregnant women develop depression or experience depressive symptoms (Grote et al., 2010; Ulrich & Petermann, 2016). Concurrently, the psychosocial risk factors and the occurrence of depressive episode that appear during pregnancy and after birth often remain unclear (Glover, 2014; Ulrich & Petermann, 2016). In utero, high levels of maternal cortisol as a steroid hormone released by the hypothalamic-pituitary-adrenal HPA axis, may cause, by crossing the placenta, a prenatal and postnatal developmental risk and transfer it to the infants (O'Connor et al., 2014; O'Donnell et al., 2009; Talge et al., 2007). Moreover, the disruption of this HPA axis cascade, as the main biological indicators of stress reactivity, has been responsible for the development of the depressive symptoms (Natsuaki et al., 2009). According to Natsuaki and colleagues (2009), the cortisol reactivity increases moderately during motherhood transition and is closely related to age and gender, which together seem to contribute to women's vulnerability to external stressors. Therefore, cortisol reactivity mirrors the stress responses in the transactions between biology and environment.

It seems that prenatal depressive symptoms affected more severely the infants than postnatal depressive symptoms (Kinsella & Monk, 2009). However, both prenatal and postnatal depressive symptoms impair the women's mental health and influence negative the infants (O'Connor et al., 2014; O'Donnell et al., 2009; Van den Bergh et al., 2017) and should be well studied. Many studies focus on the impact of prenatal maternal depressive symptoms and birth parameters of the infants (Grote et al., 2010; Staneva et al., 2015). These studies indicated that prenatal complications like premature delivery and low birth weight of the newborn increase the risk for developmental delays (Grote et al., 2010; Staneva et al., 2015). Both prenatal and postnatal maternal

depressive symptoms may have an early and lasting negative impact on neural development and cognitive abilities on the offspring (Kinsella & Monk, 2009; O'Connor et al., 2014; O'Donnell et al., 2009; Van Batenburg-Eddes et al., 2013; Van den Bergh et al., 2017; Van den Bergh et al., 2018). Infants of mothers who have depressive symptoms during prenatal and postnatal periods often have difficulties adapting to new stimuli, and are more likely to show reduced motor skills deficits and disabilities in regulating their attention, behavior and emotions (Van den Bergh et al., 2017; Van den Bergh et al., 2018). The potential risk transmissions and the vulnerabilities, for instance, psychobiological dysfunction and lack of skills in the cognitive, emotional and social domains, make the offspring of depressed mothers to develop depression more likely as well (Kinsella & Monk, 2009; O'Connor et al., 2014; O'Donnell et al., 2009; Van Batenburg-Eddes et al., 2013; Van den Bergh et al., 2017; Van den Bergh et al., 2018). In the context of prenatal and postnatal depression, the question is how to detect such risk mechanisms as early as possible. The earlier a risk is detected, the sooner preventive measures can be initiated. At this point, some studies have already begun to use neuropsychological measurement methods, to entangle the patterns that are particularly found in people with depression.

1.3 Infancy and developmental risk for infants of depressed mothers

Infancy (0–2 years) is a developmental period characterized by the massive growth and change in comparison to any other developmental period in human's life (Grossmann and Johnson, 2007). One of the hallmark in infancy is the rapid brain development, which is crucial for motor, cognitive, social, emotional and self-regulatory skills

associated with important milestones (Pauen, 2017).

As infancy is described with a tremendous maturation and developmental milestones, some authors marked it as a “sensitive period” and claimed that the brain development starts 2 weeks after conception and lasts approximately 20 years (Tierney & Nelson, 2009, p. 12). Moreover, the brain development starts already in the womb and it is sensitive to sensory interaction during pregnancy (Mcmahon et al., 2012). Furthermore, the neurocognition starts to develop already in utero because neonates are able to recognized and distinguished their mothers’ voice and smell, immediately after birth, and prefer them in comparison to the strangers, which is also evidence for the importance of prenatal experience (Mcmahon et al., 2012). Between the 2nd and 4th month of infants’ life important milestones, such as grasping and distinguishing between different stimuli are taking place (Pauen et al., 2012). Infants are able to hold his head and through this to explore the environment around it and search for stimuli (Heilig & Pauen, 2013). Moreover, between 6th and 8th month of infants’ life, the discrimination process improves and infants are able to distinguish more precisely subjects with a strong preference for human faces (Heilig & Pauen, 2013; Hoehl, 2016).

Often, theories in the developmental psychology, such as Bronfenbrenner's theory (1986), are characterized by approaches that claim that different environmental factors might influence the development. Bronfenbrenner's (1986) socio-ecological model of human development suggests a theoretical approach, involving a combination of person and environment, and it is applied to many areas of life and over the entire lifespan. It describes a complex system consisting of the micro- (parents and family), meso- (peer-groups), exo- (mass media and public agencies) and macro-system (laws and regulations in society) (Bronfenbrenner, 1986; Eriksson et al., 2018). BRISE project assumes a close interaction on microsystem (parents and family) during infancy. The

developmental processes have mostly been studied in cross-sectional research designs related to individual differences and longitudinal investigation in order to identify changes over time (Grossmann & Johnson, 2007).

According to the WHO, the phase of early childhood development is the most important development phase over the entire life. In this phase, social, emotional and cognitive development equally affect well-being, mental and physical health over the entire life course (World Health Organization, 2018) and thus, the exposure of emotional disturbances due to the mother might have serious and long-lasting consequences on early infant development (Pechtel & Pizzagalli, 2011; Peltola et al., 2014).

Maternal depression during pregnancy is associated with preterm birth, dysmaturation and adverse neurophysiological patterns of the infants, secondary related to emotional and cognitive impairments (Hayakawa et al., 1997). During pregnancy, maternal cortisol from the HPA axis as stress response has been shown to predict offspring cortisol reactivity to stressors and should be responsible for the long-term effects of distress on the mothers and on the babies (O'Connor et al., 2014; O'Donnell et al., 2009; Talge et al., 2007). Moreover, newborns of depressed mothers have had higher cortisol level, lower dopamine and serotonin levels (Field, Diego, & Hernandez-Reif, 2004). The salvia and diurnal cortisol increased in the infants of depressed mothers from prenatal to postnatal testing (Field, 2017). Many authors accentuate prenatal maternal depressive symptoms as a risk factor, leading to functional and structural brain changes (Kinsella & Monk, 2009; O'Connor et al., 2014; O'Donnell et al., 2009; Van Batenburg-Eddes et al., 2013; Van den Bergh et al., 2017; Van den Bergh et al., 2018). Hence, the occurrence of maternal depressive symptoms during pregnancy impairs the development of fetal brain structures, important to regulate the infant's stress response and social cognition (Van den Bergh et al., 2017; Van den Bergh et al., 2018). Risk

factors in infancy, such as postnatal maternal depressive symptoms, that often mirror a withdrawal behavior of the mothers toward the infants (Diego et al., 2006), have been linked to infants' negative neurophysiological patterns, emotional disturbances and a delay in developmental stages observed already in infancy as well (Wen et al., 2017). Moreover, as both genetic (prenatal) and environmental (postnatal) circumstances shape infants' neurodevelopment (Newman et al., 2016), postnatal maternal depression is also related to neurophysiological alternations (Wen et al., 2017). Given the close interaction between mother and baby during prepartum and postpartum and thereafter, the probability that neurophysiological risk patterns might be transmitted is significant. Furthermore, both prenatal and postnatal maternal depressive symptoms are associated with neurophysiological brain changes and deficits in attachment, cognitive, behavior disturbances and illness onset in infants (Pérez-Edgar et al., 2006; Van Batenburg-Eddes et al., 2013; Van den Bergh et al., 2017; Van den Bergh et al., 2018; Wen et al., 2017; Wu et al., 2018). Significant influence factors in infants with a maternal depression are course and severity of the maternal illness, occurrences of stressful life events and impaired social interaction and emotional attachment of the parents (Van den Bergh et al., 2017; Van den Bergh et al., 2018). As members of societies differ regarding their income, social prestige, education and neighborhood quality, some studies address the role of low SES of parents as a risk factor, predicting stressful life events and affecting negatively their offspring's language abilities, working memories, executive functions and intelligence skills (Hackman & Farah, 2009; Raizada, 2010).

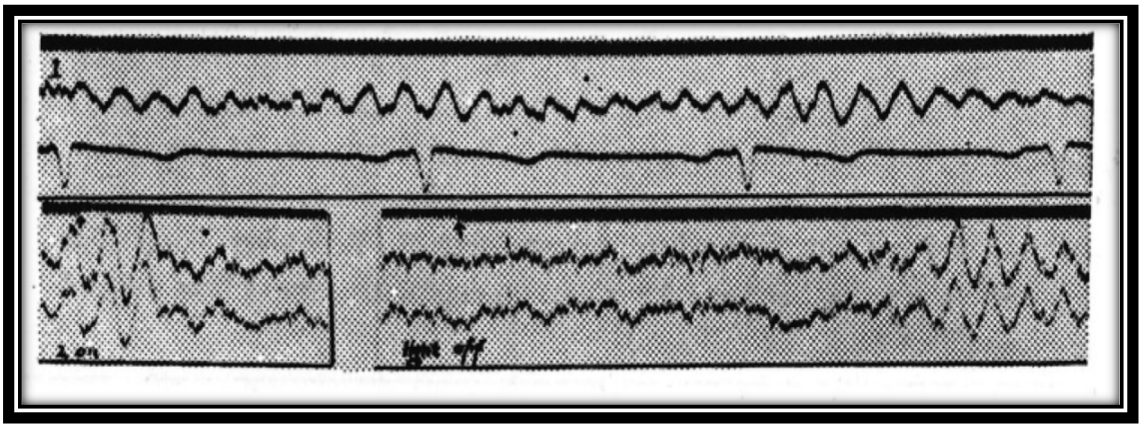
Therefore, the influence of prenatal and postnatal maternal depressive symptoms and possible impactful factors that undergo developmental changes in infants needs to be further examined and, thereby, this dissertation aimed to investigate it.

1.4 Electroencephalogram (EEG)

The electroencephalogram (*EEG*) is an electrophysiological non-invasive method to record electrical activity of the brain, which is low cost and concurrently, it is one of most feasible technique to investigate human brain (La Vaque, 1999). Hans Berger was the pioneer who published the first recordings of the human EEG in 1929 motivating an enormous amount of research. Hans Berger plotted the EEG signal on streams of paper with an oscillograph and galvanometer (Berger, 1929; Ledwidge et al., 2018). Just a few years later, in 1935, the neuroscientists, Herbert Jasper and Hallowell Davis published in the United States, the first human EEG study (Avoli, 2010; Stone & Hughes, 2013). They described *(Figure 1)* „spontaneous fluctuations in magnitude of the alpha waves, the reduction of these waves by visual stimulation, and their consistent frequency in the same individual upon repeated examinations“ (Avoli, 2010, p. 3-4). Since this time, EEG recording method have been used for understanding the relationships between the human brain and behavior (La Vaque, 1999).

Figure 1

The first recording of human EEG published in Science in the USA



Note. On the upper line the ups and downs of the alpha waves are displayed and on the bottom two lines the alpha-blocking by visual stimulus are presented. Figure after Avoli (2010).

An EEG measures brain activity with high temporal precision and detects *when* and *how* neural activity changes during rest or sensory, cognitive and motor tasks (Saby & Marshall, 2012). EEG data is usually recorded and analyzed through event-related potential (ERP) elicited as a brain response to motor or cognitive stimulation or while recording spontaneous EEG and reflect the electrical activity of neurons that underlie cognitive and sensory processing (Csibra et al., 2008). ERPs process rather small amplitudes compared to the random background activity of the spontaneous EEG (Csibra et al., 2008). In this dissertation spontaneous EEG will be conducted. During spontaneous EEG the signal comes from spontaneous activity and it provides information about the brain activity at the frequency domain of the alertness of the person (Csibra et al., 2008).

As EEG provides rich information about neural development, it can be easily applied across the lifespan (Mathes et al., 2016; Peykarjou et al., 2017; Werkle-Bergner et al., 2006; Wienke et al., 2018) and for diverse patient groups (Mathes et al., 2014; Swingler

et al., 2017). The five main EEG frequency bands are defined by frequency range as follow: delta: below 3.5 Hz; theta: 4–7.5 Hz; alpha: 8–13 Hz; beta: 14–40 Hz; and gamma: above 40 Hz (Csibra et al., 2008; Saby & Marshall, 2012).

The signal of spontaneous EEG is decomposed in space and time and the power is distributed and analyzed in frequency domain (Turnbull et al., 2001). The EEG signal is divided and consists of segments (epochs) with variable length (Procházka et al., 2010) and contains unwanted noise and artifacts (Jiang et al., 2019). There are different kinds of artifacts, such as environmental (noise in background), experimental (faulty electrodes, line noise and high electrode impedance) and physiological artifacts (eye movements, cardiac and muscle activity) (Jiang et al., 2019; Procházka et al., 2010). The extrinsic artifacts, such as environmental and experimental, are easier to be avoided than the intrinsic artifacts e.g. psysiological artifacts (Jiang et al., 2019). There are different procedures to remove the artifacts from the EEG signal and which procedure is applied in this dissertation will be discussed in details further in the method's section of the third chapter of the dissertation: *chapter three: study one: methods: EEG pre-processing.*

In the last 30 decades, neurophysiological changes in infants have been well studied with the use of spontaneous EEG (Tierney & Nelson, 2009). The majority of EEG studies with infants have sought for correlations between particular patterns of EEG activity with particular infant or parent behaviors, or disease like depression. By examining the neurophysiology, it is possible to gain a more comprehensive picture of the mechanisms underlying infant development. EEG is suitable for this research project because it is a source to show neurophysiological alternations and since it can be easily applied for infants. In this dissertation brain activity was measured through spontaneous EEG at 8-months of an infants' age.

1.5 Distal factors and proximal factors

Depressive symptoms might be modified through different factors. The research of social determinants of health promotes the use of the terms *proximal* and *distal* (Krieger, 2008; Krishnakumar & Black, 2002, p. 221). The factors that directly or indirectly affect the health are referred to as an either "proximal (downstream)" or "distal (upstream)", respectively (Krieger, 2008, p. 221). For example, the educational status is a distal factor because it doesn't directly affect health, but indirectly. The diathesis-stress model and its gene–environment interaction framework claims that the occurrence of mental diseases reflects the interplay of proximal and distal factors (Arnau-Soler et al., 2019; Colodro-Conde et al., 2018). The proximal–distal framework of the social determinants of health perspective states that pathogenic processes, causing disease, are produced across the life course via intermediary pathways of the proximal and distal factors, such as physical, behavioral, psychosocial, socio-economic and biological exposures (Krieger, 2008; Krishnakumar & Black, 2002). Thereby, the trajectory of depressive symptoms might be influenced through the stress caused by critical life events and different kinds of support which might reduce the depressive symptoms (Da Costa et al., 1999; Speranza et al., 2006; Ursache & Noble, 2016; Van Den Bergh et al., 2018). Therefore, it seems that depressive symptoms might be moderated by different situational circumstances, such as proximal and distal factors. One proximal and two distal indicators are supposed to be potentially relevant to the prediction of depressive symptoms in this dissertation.

1.5.1 Distal protective factors

In general, protective factors refer to distal factors that decrease the negative outcomes and the probability of the appearance of negative outcome (Fiori & Consedine, 2013). More specifically, protective factors are support structures and resources that might reduce the harmful effect of negative life experiences and mood disorder as anxiety and depression (Fiori & Consedine, 2013; Schwarzer & Leppin, 1991). In this dissertation, distal protective factors are considered to have an important buffering role for the depressive symptoms and interpreted as crucial components. Examples of distal protective factors which have been taken into consideration in this study are the overall social support received by family, partner, relatives and friends and partner social support only received by partner. The social support provided by partner, family, relatives and friends as a distal protective factor might play a crucial role in the prevention of depression (Pierce & Quiroz, 2019; Schwarzer & Leppin, 1991). Hence, it is important to entangle the mechanisms behind a resource, such as social support and its impact for the development of depressive symptoms.

Previous research used different ways and terms to measure and describe social support (Fiori & Consedine, 2013; Glazier et al., 2004; Jennings et al., 2020; Pierce & Quiroz, 2019; Schwarzer & Leppin, 1991). Social support is mostly evaluated from self-reported social support questionnaires focusing on the frequency and level of provided social and emotional integration from networks of people, such as close relatives, family and friends. More exactly, it is measured how often the informal social support focusing on the specific behaviors that the participants reported to obtain from their partners, families, friends and relatives were received, because the frequency is crucial to investigate the received support (Fiori & Consedine, 2013; Glazier et al., 2004;

Jennings et al., 2020; Pierce & Quiroz, 2019; Schwarzer & Leppin, 1991). Moreover, social and emotional integration is associated with the a quality and frequency of the relationship to partner, family relatives and friends (Schwarzer & Leppin, 1991). In this dissertation and in other prevous research (Glazier et al., 2004; Jennings et al., 2020), the focus was on informal social support based on participants' perception for receiving support. More specifically, informal social support was examined in accordance with previous studies to answer the question how these social relationships and behavior relate to outcomes, for instance mental or physical health and in this way, individual well-being (Pierce & Quiroz, 2019).

It should be noticed that there is not a consensus in the past history of research investigated the role of social support, some concerned it as a buffer to negative outcome (Pierce & Quiroz, 2019), others emphasize on it as a resource for better interpersonal development (Feeney & Collins, 2015). The last pointed out the role of social support from partner, relatives and friends as a resource not only in time of needs but as a mechanism for thriving and succeed in life.

1.5.2 Proximal risk factor

Proximal risk factors are adverse situations and factors that are associated with negative events and outcomes having a harmful effect on the person health and well-being (Schwarzer & Schulz, 2000). In this study, proximal risk factors are referred to stressful life events which might have critical, short- or long-term negative effects on personal mental health and well-being or cause emotional disturbances, such as job loss, financial problems, partner conflicts, etc. Other examples of critical life events are: bankrupt due to economic conditions, exposure to war or natural disaster, attending a

car accident (Kessler, 1997) or coronavirus crisis (COVID-19 pandemic).

The terms and definition behind the stress research is so complex and there are variety of different kinds of stress, such as stressful life events, chronic stress, traumatic stress, daily stress, etc. (Kendler et al., 1999; Kessler, 1997; Levine et al., 2020; Schwarzer & Schulz, 2000; Van Praag, 2004). Previous studies focused mostly on life events stressors (Kendler et al., 1999; Kessler, 1997; Levine et al., 2020; Schwarzer & Schulz, 2000), others investigated the chronic stress assessed through cortisol level (Van Praag, 2004).

In 1967, Holmes and Rahe, published the first study which linked stressful events to the appearance of mood disorder. In the study, to the participants was submitted a list of 43 items (Social Readjustment Rating Scale (SRRS) describing stressful life events, such as: “marriage”, “troubles with the boss”, “detention in jail or other institution”, “death of spouse”, etc (Holmes & Rahe, 1967, p. 214). The authors pointed out the influence of different stressful life events to the development of a mood disorder and highlighted the importance of examined such relations. Hence, this study motivated several research projects and started a new era in the psychiatric and psychological research.

Moreover, in the last 30 decades, the findings of major amount of studies reveal that stressful life events might predict depressive episode or depressive disorder (Kendler et al., 1999; Kessler, 1997; Levine et al., 2020; Schwarzer & Schulz, 2000). However, it should be cautiously interpreted if a stressful life event is a main and major reason for developing depression or if it is a minor reason for the development the depression. Of course, it must be assumed that different genetic and situational factors and personal traits might be involved (Kessler, 1997) because not every person who experiences stressful life events, develops a depressive episode.

CHAPTER ONE: INTRODUCTION

This dissertation focuses on stressful life events during pregnancy, such as job loss, divorce, death of a spouse, etc. Moreover, some authors manifest the period of pregnancy and the transition to motherhood as stressful life events because of the life changes and challenges for women during this time (Geller, 2004). When some other stressful life events occur during this period, makes it even more challenging for the women. Around 65% to 70% of women have faced at least one stressful life event during pregnancy, and the prevalence seems to vary by race/ethnicity and SES according to the study of Mukherjee et al., 2017. The same study reported that in the USA, the rate was much more higher for non-Hispanic Black women (2.3) and American Indians/Alaska Native women (2.5) than to non-Hispanic White women (1.7). It seems that there is a relationship between low SES and higher levels of different forms of stress during pregnancy, as well. Hence, women of low SES are much more vulnerable to experience a stressful life event during pregnancy. Moving to a new address was reported as the most commonly experienced stressful life event during prepartum (Mukherjee et al., 2017).

There are still research issues, considering the effects and accuracy behind the relationship between depressive symptoms and stressful life events during pregnancy, which highlighted the importance of future research in this area. The modifying factors of different groups in community samples, control groups and individual events might be necessary to unpuzzled the causal relationships behind stressful life events and depressive symptoms.

In the following, a review of the literature of the effects of the distal protective factors and proximal risk factors on depressive symptoms is provided. The distal protective factors and proximal risk factors are separately investigated in this dissertation for better thematic and topic differentiation. In this study, it is assummed that the maternal

educational status is concerned as a distal risk and/or protective factor, which might have an impact of the maternal depressive symptoms. This dissertation suggests that a low educational status is a distal risk factor and a high educational status is a distal protective factor for women.

1.6 Interactions between distal protective factors, distal and proximal risk factors and depressive symptoms

Positive experiences shared with beloved and important people predict the positive affect, supporting the health and well-being, and negative experiences and exchanges predict negative affect harming the health and well-being of the individual (Fiori & Consedine, 2013). In this dissertation is proposed that distal protective factors and proximal risk factors should be separately examined as influencing factors on depressive symptoms, as several previous study suggested (Gómez-López et al., 2019; Levin & Defrank, 1988). The previous research has been shown that stressful life events are proximal risk factors which might be associated with increased depressive symptoms or even developing a depressive disorder (Kendler et al., 1999; Kessler, 1997; Levine et al., 2020; Schwarzer & Schulz, 2000). Moreover, the lack of overall social and partner social support might be also responsible for an increase in depressive symptoms. Several studies revealed that women who received less social support and experienced more partner conflicts (stressors) during pregnancy, perceived less social support and felt more depressed (Glazier et al., 2004; Gómez-López et al., 2019; Levin & Defrank, 1988; Turner et al., 1999). The researchers found that pregnant women who reported receiving social support experienced decreased levels of depression and better

general health (Glazier et al., 2004). Furthermore, provision of support assisted people to better cope with depression (Da Costa et al., 1999; Walen & Lachman, 2000).

Maternal resources, providing support in time of needs, might be protective for the mother. Several studies have proposed that maternal resources and stressful life events might influenced maternal depressive symptoms (Glazier et al., 2004; Gómez-López et al., 2019; Levin & Defrank, 1988; Turner et al., 1999). As pregnancy is a time of significant life change for women and their partners (Elsenbruch et al., 2007), the social context in which stressors and resources function might be crucial for depressive symptoms during pregnancy. Stressful life events during pregnancy are related to feeling dissatisfied and in general unhealthy (Leahy-warren et al., 2011). Previous research has shown that providing support for mothers during prepartum and postpartum while they are caring for their babies might reduce depressive symptoms and enable the transitions to motherhood and the stress related to it (Leahy-warren et al., 2011). Moreover, it seemed that women who received insufficient social support from their partners, and insufficient social support from their families, relatives and friends might be under higher risk to experience emotional distress and later mood disorder. The insufficient social support has been repeatedly linked to affect maternal well-being, increase stress and prenatal and postnatal maternal depressive symptoms (Glazier et al., 2004; Gómez-López et al., 2019; Levin & Defrank, 1988; Turner et al., 1999). Hence, the lack of overall social and social partner support are risk factors for women during and after pregnancy.

Many studies reveal that romantic relationship and in this way, the emotional support provided from partners is an impactful resource for women in time of needs (Gómez-López et al., 2019). The findings of previous research have reported that a lack of partner social support has been accentuated as a risk factor in women during and after

pregnancy (Martin et al., 2013; Nakamura et al., 2020). More specifically, a lack of emotional support from the partner, unsatisfactory couple relationships, non-involvement in infants' care, are related to maternal depressive symptoms during and after pregnancy (Martin et al., 2013; Nakamura et al., 2020). Women who are less supported from their partners and in this way, they did not receive sufficient social support and understanding from the men while caring for babies, develop depressive symptoms during prepartum and postpartum (Nakamura et al., 2020). Hence, the lack of partner social support might have an adverse impact of women especially during pregnancy.

Previous research (Elsenbruch et al., 2007; Glazier et al., 2004) indicated that proximal risk and distal protective factors might influence prenatal and postnatal depressive symptoms but do not completely entangle the mechanisms behind them. Thus, the investigation of these factors might be from crucial importance especially during the phase of pregnancy and early infancy.

It should be noticed that some people and community sample might be more vulnerable to different kinds of stressors. More specifically, socially and culturally disadvantaged families reported more stressful life events due to low SES, migration background and language problems (Levine et al., 2020). Women from disadvantaged families might be even more vulnerable for developing depression when experienced a stressful event during pregnancy and transition to motherhood, thereby, social support provided from partners, families, friends and relatives might be protective for females (Da Costa et al., 1999; Speranza et al., 2006; Ursache & Noble, 2016; Van den Bergh et al., 2018). Hence, the need for research in disadvantaged families who are the ones, who need more support as a buffer to the negative outcome, increases in particular during pregnancy.

Therefore, depressive symptoms might be associated not only with the described above proximal risk and distal protective factors but possibly with socio-economic factors, which might be concerned as distal protective or risk factors. A previous study has provided evidence for the relationship between SES and depressive symptoms during prepartum and postpartum (Goyal et al., 2010). The study suggests that a low SES is a distal risk factor for developing depression and that a high SES is a distal protective factor for women. Women with low SES, such as low monthly income, unemployed, less than a college education and unmarried, have increased depressive symptoms in late pregnancy and at 2 and 3 months after birth in comparison with women with no socio-economic risk factors (Goyal et al., 2010).

Low SES is often associated with the absence of overall social and partner support and more occurrence of stressful life events (Goyal et al., 2010). The interactions between these distal risk factors and their negative contribution to the transition to a motherhood should be better investigated. The low SES might be interpreted as a distal risk factor for developing depression and a high SES might be interpreted as a distal protective factor for women.

Considering socio-economic risk factors might have a tremendous importance for future preventive purposes, especially within group of disadvantaged families. Moreover, people with low educational status are more vulnerable to experience different kinds of stressful life events (e.g. financial problems, job loss, or disease in family) (Van den Bergh et al., 2017) that might increase depressive symptoms. Women with low educational status have difficulties to access health care services and are least likely to report depressive symptoms to midwives, health care professionals and trained support providers (Goyal et al., 2010). There are very few studies investigating how educational status and demographic factors like age, are connected with prenatal and postnatal

depressive symptoms, especially within longitudinal design from the prenatal period through 8 months postpartum. This study would not examine the impact of the different SES components, such as an income and occipation, but it would consider the impact of maternal educational status on prenatal and postnatal maternal depressive symptoms. In the context of this dissertation, the low educational status might be interpreted as a distal risk factor for developing depression and a high educational status might be interpreted as a distal protective factor for women. Of course, how women cope with the stress of motherhood depends on the person's resources to manage stressors and it is very individual as well (Goyal et al., 2010).

There are particular gaps in the research, for instance, in the investigation of the dynamics of depressive symptoms over time, the involvement of different maternal resources for reduction of adverse outcomes and possible confounders as maternal educational status. Attending to these research gaps, the goals of this dissertation are to examine the interactions between prenatal and postnatal maternal depressive symptoms, maternal educational status, stressful life events and overall social and partner support during prepartum and postpartum.

1.7 The Diathesis-stress model and diathesis model by Davidson

"the ones in which genuine meaning attaches to the commonly repeated statement that heredity and environment interact"

(David Rosenthal, 1963, as cited in Colodro-Conde et al, 2018, p. 1590)

The diathesis–stress model is a bio-psycho-social model that represents close interactions between gene and environment which reflects a psychopathology (Colodro-Conde et al., 2018). It has been already 50 years, since the model was first created to spell out the origin behind schizophrenia and in the last 40 decades it was widely used to explain the genesis of depression (Colodro-Conde et al., 2018). The model supposes that stressful events or stress (environment) might be a potential risk factor that makes people more vulnerable to develop a mood disorder (gene). Hence, stress, gene and individual's vulnerability interacts in a complex way (diathesis) to create psychopathology and there is a perplex dependency on the diathesis (Arnau-Soler et al., 2019). In this context, the model provides an overview of the bio-psycho-social factors that are behind the development of depression and the mechanisms of risk transmission that are influenced by vulnerabilities.

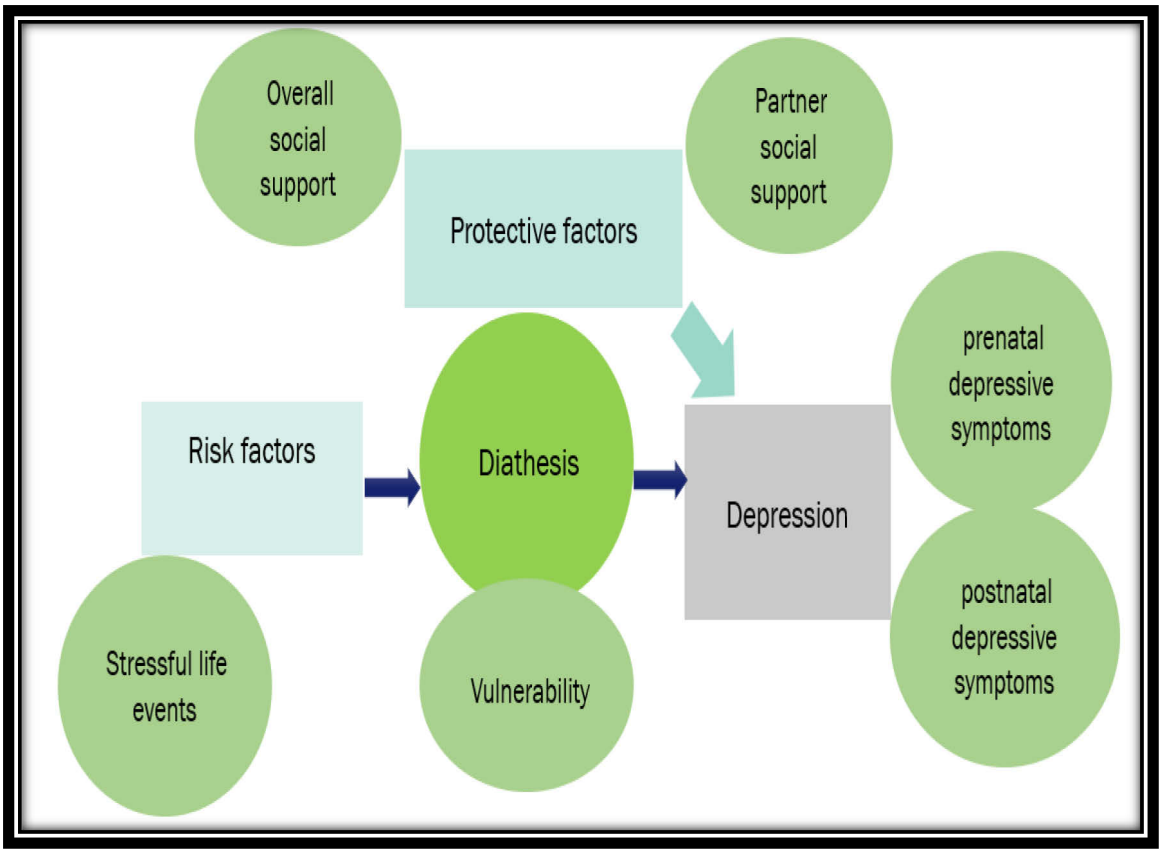
In 5221 depressed participants, the hypothesis behind diathesis-stress model has been significantly confirmed (Colodro-Conde et al., 2018). Moreover, the findings revealed that the occurrence of stressful life events predicted a depression mostly within group of women than men. A new study validates recently the diathesis-stress theory and find significant effects in 4919 participants with stronger effect to females than males (Arnau-Soler et al., 2019). It seems that the hypothesis behind the diathesis-stress is

mostly confirmed within group of female participants and this way, it is suitable for research projects that include only pregnant women and mothers of young children.

Even though the diathesis-stress model represents the complex interactions within the bio-psycho-social multifactorial trajectories, it does not provide a well-comprehensive rationale of the weighting of these factors and weighting the interactions between them (Arnau-Soler et al., 2019; Colodro-Conde et al., 2018). *Figure 2* shows the interactions between risk and protective factors and depression derived from diathesis-stress model and interpreted by this dissertation.

Figure 2

An interpretation of diathesis-stress model supposed by this dissertation



Note. Stressful life events have been risk factors, creating a predispotion do develop depression (prenatal and postnatal depressive symptoms in the context of this research project). Therefore, risk factors and individual's vulnerability interacts in a complex way to reflect psychopathology and there is a dependency on the diathesis with protective factors (overall social support and partner social support in the context of this research project) (Arnau-Soler et al., 2019; Colodro-Conde et al., 2018).

The diathesis model by Davidson

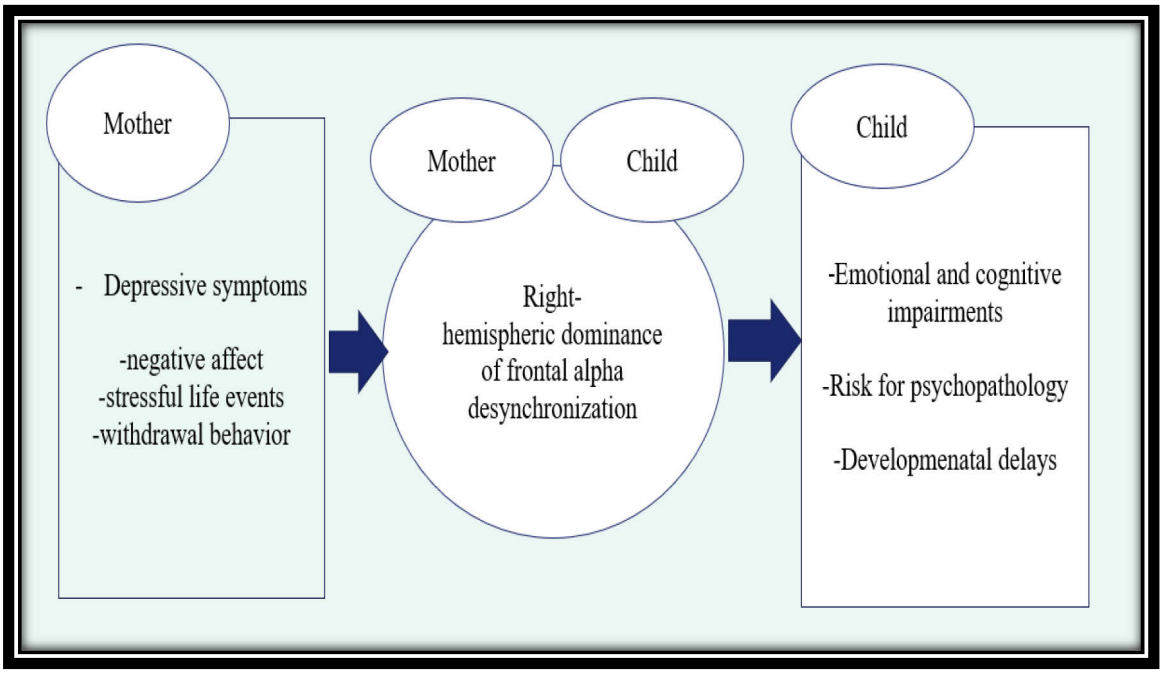
In the last 30 decades with the increased numbers of reported depressive episodes and disorders (World Health Organization, 2017) and the implementation of EEG in the research of depressed individuals, a diathesis model has appeared to explain the relation between depression and EEG brain's asymmetry. Diathesis model by Davidson and Tomarken (1989), also known as the approach-withdrawal model, is developed to describe the above-mentioned relation and it is the most popular model in the EEG context nowadays, motivating several research projects and this work as well. According to the model, there are two dominant motivational systems in response to positive or negative stimuli, associated with approach and withdrawal behavior, respectively (van der Vinne et al., 2017). The balance or disbalance of these systems is reflected in symmetrical or asymmetrical brain activity assessed with EEG (van der Vinne et al., 2017). Davidson and Tomarken claimed that the system which is associated with negative affect is also related to asymmetry or hypo-activity in the left frontal brain area and hyper-activity in the right frontal brain area (Gotlib, 1998).

In 1989, Davidson and his colleagues showed for the first time that there was a relationship between infants' responses to emotional stimuli and individual differences in the EEG hemispheric activity. The authors observed that infants who cried in response to maternal separation while EEG recording exhibited left-hemispheric hypo-activity and right-hemispheric hyper-activity, in comparison to those infants who did not cry. According to Davidson and his colleagues, this hemispheric difference, interpreted in this study context as the right-hemispheric dominance of frontal alpha desynchronization might be a neurophysiological marker of depression reflecting different kinds of impairments in infants, children, adolescents and adults (Davidson & Fox, 1989; Field & Diego, 2008; Tomarken et al., 1990). Moreover, the model proposes

the mechanisms of mother-child risk transmission that are influenced by vulnerabilities *(Figure 3)*. These include the heritability of maternal depression as well as the negative affect and the stressful environment, dysfunctional neuroregulatory and neurophysiological mechanisms of both mother and child, and the child's coping with the mother's withdrawal behaviors *(Figure 3)*.

Figure 3

Overview of the mechanisms of risk transmission in offspring of depressed mothers proposed by the Diathesis model by Davidson (1989)



Note. On the figure is presented how the maternal depressive symptoms, the previous and following but subsequent negative affect, stressful events and withdrawal behavior reflected alpha desynchronization in the right frontal brain area in both mother and child which is associated with emotional and cognitive impairment, developmental problems and risk for developing a mood disorder in child (Gotlib, 1998; Henriques & Davidson, 1991).

Originally, Davidson's model was not aimed to explain psychopathology such as depressive disorders but rather to establish relations between positive or negative affect and EEG patterns (Davidson & Fox, 1989). Later on, when the assumption of Davidson's model was supposed to be related to depression, it motivated several studies with participants with clinical and sub-clinical level within all age groups to investigate the asymmetry patterns of depressed individuals (Davidson et al., 2002; Field & Diego, 2008). Even though the 30 decades history of previous research, there are still methodological questions and inconsistent findings, such as the precise brain location of the asymmetry. Thus, better validation of the results is necessary and this work is focusing on it.

In addition to the findings which claim that asymmetry is a stable trait marker for negative affect and depression (Davidson & Fox, 1989; Davidson et al., 2002; Field & Diego, 2008), there are several studies that suggest that the neurophysiological changes resulted from current emotional state (Alves et al., 2008; Davidson et al., 2002; Field & Diego, 2008). Some authors have found a shift from right-to-left-hemispheric asymmetry associated with positive affect, e.g. when odors were presented (Fernandez et al., 2004), while breastfeeding in comparison with bottle feed (Jones et al., 2004) and in interaction with intrusive (positive) attachment and maternal style in comparison with withdrawn (isolated and unoccupied) maternal style (Diego et al., 2006). However, since Davidson's model considers asymmetry as a stable trait marker, which should also occur in remitted patients without current depressive symptoms (Davidson & Fox, 1989; Davidson et al., 2002; Field & Diego, 2008), a healthy control group and a sub-clinical sample is required for better investigating the model hypothesis.

Furthermore, number of studies who have tested the Davidson model have found

significant differences in parietal brain areas (Bruder et al., 2005, 2007; Marino et al., 2019). Previous EEG studies have emphasized that frontal and limbic brain regions are associated with abnormal function in individuals with depression (Korb et al., 2008). More specifically, the dorsolateral prefrontal cortex (DLPFC), medial prefrontal cortex (mPFC), anterior cingulate cortex (ACC) and orbitofrontal cortex (OFC) have been linked to depression (Korb et al., 2008).

Moreover, another model, the model by Heller and Nitschke (1997), claims that asymmetry at parietal brain areas might also be associated with negative affect and later with depressive symptoms. In general, the model by Heller and Nitschke (1997) has a similar hypothesis such as Davidson's model, but their assumption is transferred on the parietal cortex. Only few studies empirically tested the hypothesis for the parietal region and depression (Bruder et al., 2005, 2007; Marino et al., 2019). The model of Heller and Nitschke (1997) and other empirical studies (Field et al., 2010) point out the importance of comorbid disorders as anxiety for the investigation of the alpha desynchronization patterns. Moreover, when frontal asymmetry is examined in participants without stable asymmetry patterns or without depressive symptoms, its relationship with negative affect is reduced or a shift to the left might be observed (van der Vinne et al., 2017). Concurrently, while the investigation of frontal regions are used to prove the hypothesis behind the Davidson's model, parietal regions and Heller and Nitschke model is typically only used to demonstrate the specificity of EEG asymmetry for frontal regions (Alves et al., 2008; Davidson et al., 2002; Field & Diego, 2008; van der Vinne et al., 2017).

Infants' factors, such as age and gender, have also been shown to moderate the asymmetry (Hernandez-Reif et al., 2006). It seems that mostly females are strongly effected than males, so in this way, girls might be at higher risk (Hernandez-Reif et al.,

2006). Other factors, such as maternal socio-economic factors, might also have an influence on the asymmetry (Feng et al., 2012; Lopez-Duran et al., 2012; Tomarken et al., 2004). More specifically, some studies highlight the moderation role of maternal educational status on asymmetry (Feng et al., 2012; Lopez-Duran et al., 2012). In one of these studies, low maternal educational status was associated with the occurrence of more stressful life events and a tendency towards increased left frontal asymmetry score during all conditions (baseline, happy, sad) (Lopez-Duran et al., 2012). However, there are few studies that investigated the role of maternal educational status on infants' asymmetry and this dissertation aims to fill this gap.

Even though some heterogeneity and inconsistent findings that have been discussed before exist, there is a consensus in recent research regarding certain methodological standards for studies interrogating the Davidson's model. For the duration of the recording, an interval of one minute is recommended in order to achieve acceptable psychometric characteristics of the activation values (Allen et al., 2004; Diego et al., 2010). Studies with more participants both males and females, healthy controls and confounding variables should be conducted. Studies with infants underlying the age-independent significance of Davidson's model are necessary. A particular gap in the research exists in studies investigating the EEG asymmetry in parietal brain regions. The differences in parietal asymmetry between depressed and healthy control subjects remain unclear, so that no completely consistent picture emerges. This could be strongly related to the methodological quality of this work. Although, it cannot be concluded that these conditions offer optimal results, they might be a good example because they ensure a reliable and valid measurement methodology, creating the basis for the interpretability of the results.

In the following, the impact on maternal depressive symptoms and infants' neurophysiological changes related to spontaneous frontal alpha oscillations will be highlighted.

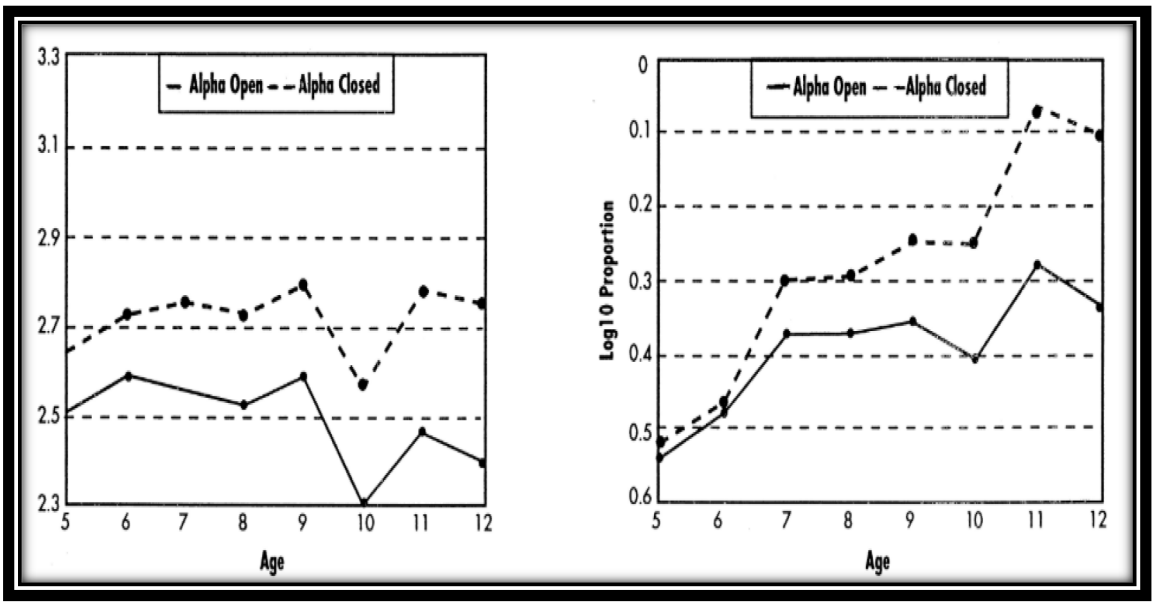
1.8 EEG alpha power and depressive symptoms

Alpha oscillations occur in a frequency range between 8 to 13 Hz in a resting or awake person (Csibra et al., 2008). Alpha becomes more dominant EEG frequency during the development and age maturation *(Figure 4)* (Klimesch, 1999). The absolute alpha power consists of all of the alpha power values and relative alpha power represents a percent of absolute power in each frequency summed over the four frequency bands (Klimesch, 1999).

Figure 4

Overview of alpha frequency changes for absolute and relative alpha power within age

Absolute Power **Relative Power**

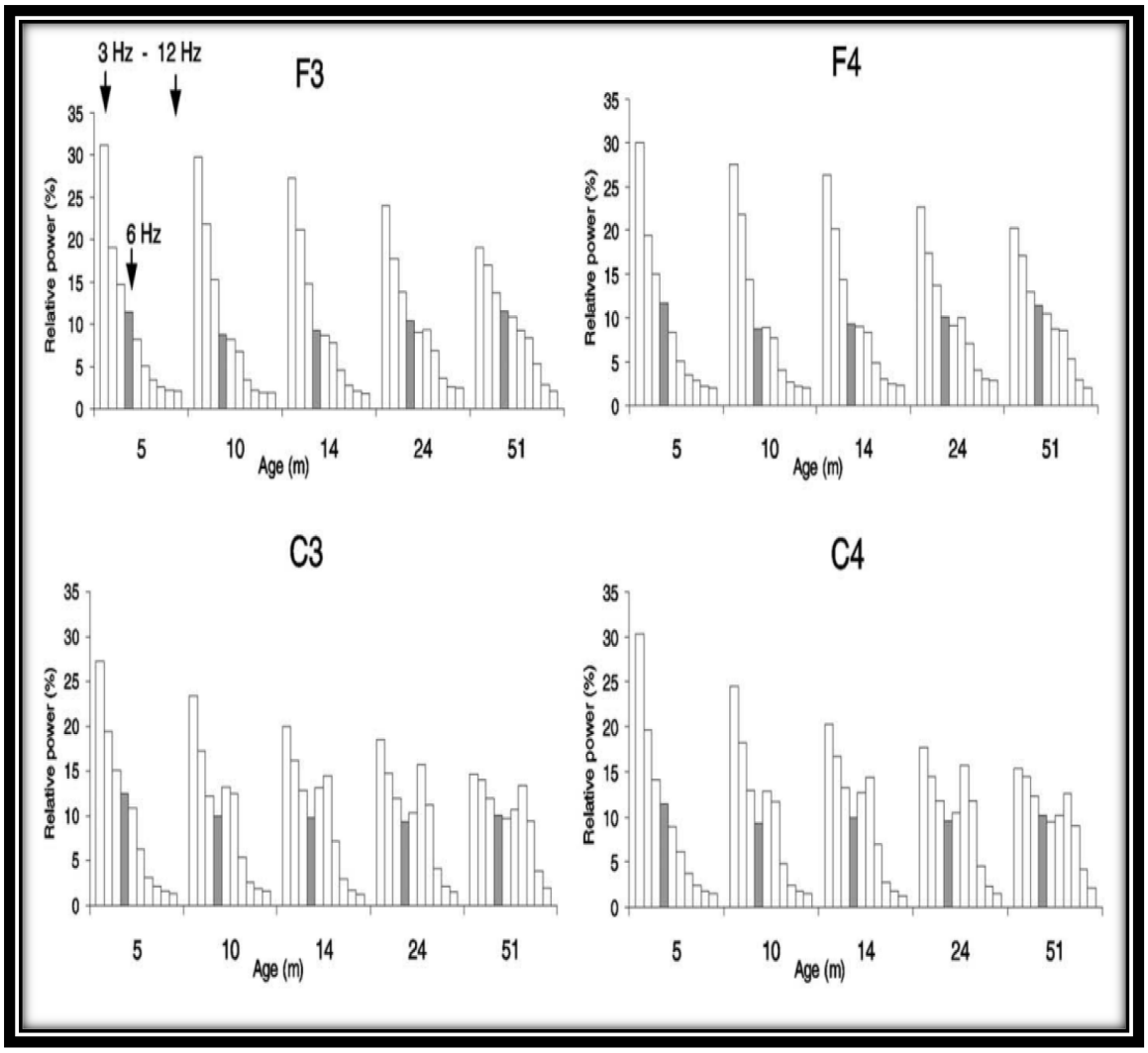


Note. Figure after Klimesch (1999).

In infants, the main alpha frequency is often reduced and it is found in an approximate frequency range between 6 to 8 Hz (Saby & Marshall, 2012). During the development, the alpha frequency tends to enhance and it is observed in the higher frequency (Marshall et al., 2002). *Figure 5* presents how power spectrum (averaged across 29 participants, taking part in the longitudinal study of Marshall et al., 2002) changes across the early infancy till childhood. On the *Figure 5* is displayed a clear developmental pattern for increase in higher frequency power, rising from 7 Hz at 10 months of age to 9 Hz at 4 years of age.

Figure 5

Overview of power spectrum changes for relative power from 5 till 51 months of life



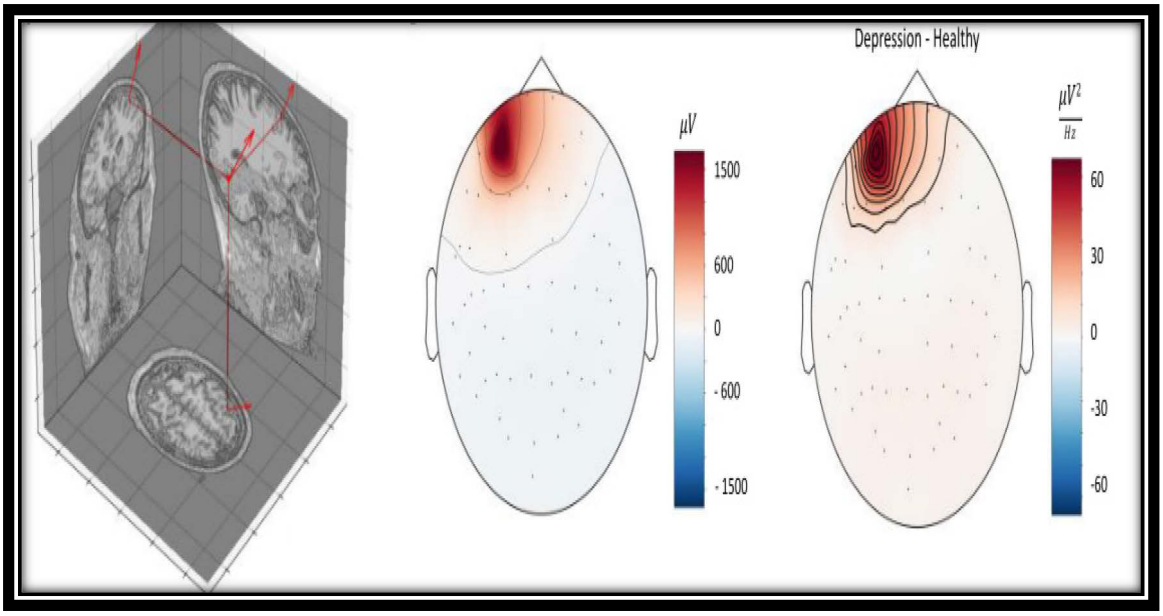
Note. Power spectrum of 29 participants in the 3–12 Hz at the frontal and central electrodes, on the figure the 6 Hz bin is always marked in the gray bar. Figure after Marshall et al. (2002).

Modulations of alpha oscillations are important for sensory processing, attention, memory and inhibitory functions (Klimesch et al., 2012; Basar et al., 2008). Moreover, alpha oscillations inhibit neural activity in synchronizing large-scale networks (Smith et al., 2017). Furthermore, alpha power is strongly related to vigilance which is lower when alpha waves are more synchronized (Başar, 2012; Klimesch, 1999). Hence, alpha

activity enables information flow within cortical networks which are crucial for different kinds of cognitive functions. As alpha is important for cortical maturation, impairments in alpha and theta oscillations might be related to a lack of cortical maturation and an alterned cortical inhibition (Klimesch, 1999). A recent study of Mazza and colleagues (2021) investigates the EEG biomarkers corresponding to microcircuit changes in depression. The findings reveal a reduced cortical inhibition and a significant increase in theta-alpha power in the left orbitofrontal cortex and left dorsolateral prefrontal cortex in relation to depression *(Figure 6)* (Mazza et al., 2021).

Figure 6

Microcircuit EEG signal changes in depression



Note. The spatial distribution of simulated microcircuit EEG signal represents the topography of the grey matter in the left dorsolateral prefrontal cortex and the differences in the distribution over the scalp of the spectral power for theta-alpha band (4 – 12 Hz) between the depressed and non-depressed group, respectively. Figure after Mazza et al. (2021).

Even though alpha oscillations are impaired in many neuropsychiatric illnesses including depression (Başar et al., 2016; Başar, 2012), EEG studies in depressed adults have reported very inconsistent findings of power spectrum differences with increases, decreases to different brain regions or no differences (Korb et al., 2008; Newson & Thiagarajan, 2019; Olbrich & Arns, 2013). Several studies have revealed that an increase of alpha power in posterior and anterior regions have been related to depression in adults and other studies have shown the same relation in parietotemporal regions (Jaworska et al., 2012; Newson & Thiagarajan, 2019; Olbrich & Arns, 2013). In adults with major depressive disorder, increased power in the frontal and temporal regions has been presented (Lee et al., 2011). Moreover, depressed patients showed less frontal alpha activity during emotional task (Mahato & Paul, 2020).

The pre-dominant pattern found in depressed individuals is an increased absolute power value around all frequency bands (Olbrich & Arns, 2013). Despite, these increases are no more presented with regards to relative power across any bands (Korb et al., 2008). Moreover, the enhanced power is observed across lower frequencies (delta and theta) and decreased across higher frequencies (alpha, beta and gamma) (Newson & Thiagarajan, 2019). Other research projects revealed that depression in adults was related to a decrease in alpha power at low frequency and an increase at high frequency above 10 Hz (O'Connor, 1979). Some studies have found decrease alpha waves (Kan & Lee, 2015) and lower parietal-occipital alpha power in depressed individuals (Zoon et al., 2013).

In the above paragraph, studies investigated the relationships between depression and EEG power in adults have been summarized because there are almost no studies with infants of depressed parents and as the outcomes might be secondary related to infants of depressed mothers.

As, there is a great discussion about identifying the alpha frequency in infants and not a completely clear identification of an alpha frequency in infants, some studies have recommended the use of a broad frequency bands or applied the adult frequency bands across infants (Diego et al., 2010; Mizuno et al., 1970). Alpha waves are suitable to show the desynchronization than other frequencies because alpha waves are more rhythmic and occur in states of wakefulness and quiet rest (Jesulola et al., 2015). In the following, the findings that infants of depressed mothers showed right-hemispheric dominance of alpha desynchronization, which might be interpreted as a primary risk factor for depression, will be discussed.

1.9 Right-hemispheric dominance of frontal alpha desynchronization *"asymmetry"*

Depressed adults have been characterized by a brain difference in oscillatory alpha power mostly at frontal electrode sites, more specifically by alpha desynchronization in the left hemisphere versus the right hemisphere (Adolph & Margraf, 2017; Allen et al., 2018; Harrewijn et al., 2016; Smit et al., 2007; Thibodeau et al., 2006). This pattern has often been called frontal alpha asymmetry and indicates alternations of alpha oscillations at the frontal lobe during the resting state, associated with depressive symptoms (Jesulola et al., 2015). The alpha supression over the right and left hemisphere might be interpreted in terms of *alpha desynchronization*, suggests that during "information processing large populations of neurons no longer oscillate in synchrony" (Klimesch et al., 2007, p. 65; Wright et al., 2015). As a relative measure of hemispheric difference, lower/negative frontal alpha desynchronization scores reflect more right than left desynchronization (Coan & Allen, 2004; Smith & Bell, 2010) and it is interpreted in this study in terms of right-hemispheric dominance of frontal alpha

desynchronization. Most studies have calculated hemispheric differences of alpha oscillations by subtracting the natural log-transformed power scores for homologous electrodes sites (right minus the left sites, see e.g. (Lopez-Duran et al., 2012). More specifically, to assess hemispheric-differences, the alpha frequency band (8 and 13 Hz) for adults or for infants (6 and 8 Hz) is extracted from the medial-frontal areas (F3 and F4), from the lateral-frontal areas (F7 and F8) and in few studies from the parietal areas (P3 and P4) and (P7 and P8) (Reznik & Allen, 2018).

The right-hemispheric dominance of frontal alpha desynchronization reflects reduced integration of neural information and selectively increased neural processing in the frontal cortex, and has been related to reduced cognitive functions (Reznik & Allen, 2018; Smith et al., 2017), decreased motivation, increased withdrawal behavior (Jesulola et al., 2015; Nusslock et al., 2015; van der Vinne et al., 2017) and negative emotions (Lusby et al., 2016). Hence, right-hemispheric dominance of frontal alpha desynchronization may be informative regarding the risk for infants of developing emotional and cognitive problems (Field & Diego, 2008; Peltola et al., 2014; Thibodeau et al., 2006), and psychopathology later in life (Meyer, 2017).

As a potential neurophysiological marker predicting illness onset or providing objective information about developmental trajectory, right-hemispheric dominance of frontal alpha desynchronization may relate to prefrontal-amygdalae-hippocampal networks (Allen & Cohen, 2010; Henriques & Davidson, 1991; Jaworska et al., 2012). These brain regions have been repeatedly linked to high cortisol levels, stress and emotional withdrawal, and seem to underlie deficits in emotional and cognitive functions of individuals with maternal depression (Allen & Cohen, 2010; Coan & Allen, 2003, 2004; Field & Diego, 2008; Peltola et al., 2014; Thibodeau et al., 2006).

Differences in frontal alpha desynchronization might also reflect behavior and emotional tendencies (Smith & Bell, 2010). More specifically, right-hemispheric dominance of frontal alpha desynchronization might mirror internalizing disorders, withdrawal, increased negative affect, fear and depressive symptoms and left-hemispheric dominance of frontal alpha desynchronization might reflect externalizing disorder, such as impulsivity, hyper-activity, aggression, secondary related to diminish control approach behaviors and emotions, and anger (Davidson & Fox, 1989; Smith & Bell, 2010). Moreover, pre-school children with right-hemispheric dominance of frontal alpha desynchronization have externalizing behavior problems, more stressed behavior, decreased social competence and attentional problems (Ashman et al., 2008).

Even though there are many EEG studies investigating the relationship between right-hemispheric dominance of frontal alpha desynchronization and depression, there are still some inconsistent issues and a further research is needed. Some methodological issues such as an EEG reference and the duration of EEG recording might be addressed (Hagemann, 2004). Several studies recommended 60 seconds for duration of EEG for assessing the desynchronization (Allen & Cohen, 2010; Coan & Allen, 2003, 2004). Some studies used the vertex (Cz) (Field et al., 2010) as a reference electrode, other studies used the average (AV) (Jones et al., 2009). Additionally, as discussed above, individual difference, such as the severity of depressive symptoms and gender differences, might reflect the right-hemispheric dominance of frontal alpha desynchronization (Smith et al., 2017). Other studies, reported right-hemispheric dominance of frontal alpha desynchronization only for females (Hernandez-Reif et al., 2006; Wen et al., 2017).

In the BRISE project is possible to investigate the relationships between prenatal and postnatal maternal depressive symptoms and infants' neurophysiology. Depressive

symptoms before and after child birth were collected via self-reported questionnaires. Infants' alpha desynchronization was measured through spontaneous EEG at 8-months of infants' age.

Alpha asymmetry in this dissertation will be discussed in terms of alpha desynchronization and asymmetry and relative greater right frontal alpha asymmetry would be interpreted in terms of right-hemispheric dominance of frontal alpha desynchronization.

In the following, the impact of prenatal and postnatal maternal depressive symptoms on infants will be outlined. Neurophysiological changes related to spontaneous frontal alpha oscillations will be specially emphasized.

1.10 Prenatal and postnatal maternal depressive symptoms and infants' right-hemispheric dominance of frontal alpha desynchronization

Many studies have reported a high similarity between an illness-related frontal alpha desynchronization in depressed mothers and their infants (Field & Diego, 2008; Peltola et al., 2014; Van den Bergh et al., 2018). This similarity may be observed earlier than one week after birth and for at least 3 years of the child's life (Jones et al., 1997). The fact that newborns of mothers, who experienced depressive symptoms during and after pregnancy, have showed right-hemispheric dominance of frontal alpha desynchronization (Harmon-Jones & Gable, 2018), has inspired researchers to further examine this neurophysiological pattern. This section focuses on the studies

investigating the influence of maternal depressive symptoms on infants' neurophysiology that are relevant for the context of this work.

The right-hemispheric dominance of frontal alpha desynchronization is related to infants' withdrawal behavior, negative temperaments, having a preference for negative affect, increased distress during maternal separation and disturbed wake and sleep behavior (Davidson & Fox, 1989; Davidson et al., 2002; Field & Diego, 2008). A meta-analysis has pointed out the role of right-hemispheric dominance of frontal alpha desynchronization as an indicator for exposure to familial stressors and as a potential biomarker for the risk of developing a mood disorder in infants with history of maternal depression (Peltola et al., 2014).

Tiffany Field and their colleagues have published several studies investigated how maternal depression and infants' frontal alpha desynchronization (F3 and F4) are related. A cut-off for a clinical depression, reported according to mean Center for Epidemiological Studies-Depression scale (CES-D) scores for mothers in the depressed group, were larger than 16 (Field et al., 2010). EEG recordings were mostly referenced to Cz, with the exception of one study which used the average reference (AV) (Jones et al., 2009). In the studies of this research group, right-hemispheric dominance of frontal alpha desynchronization has been pronounced in newborns (Field, Diego, & Hernandez-Reif, 2004), and throughout early (Diego et al., 2006, 2010; Diego, Field, Jones, et al., 2004), to late infancy (Jones et al., 2009) for infants of depressed mothers in comparison with healthy controls. This pattern has seemed to correlate with the mothers' postnatal right-hemispheric dominance of frontal alpha desynchronization (Field, Diego, & Hernandez-Reif, 2004). Stability of this pattern during infancy has been confirmed in a longitudinal study testing infants prior to their 3rd week and again between 3 and 6 months of age (Diego et al., 2006) and a cross-sectional comparison in

a similar age range (Diego et al., 2010). One study, however, has reported right-hemispheric dominance of frontal alpha desynchronization only for newborn girls (Hernandez-Reif et al., 2006).

The studies have further emphasized important links between the brain difference in frontal alpha desynchronization of high-risk neonates and characteristics of their mothers' mood disturbances. Right-hemispheric dominance of frontal alpha desynchronization in newborns may be specifically affected by the severity of prenatal maternal depression (Diego, Field, Hernandez-Reif, et al., 2004; Field, Diego, & Hernandez-Reif, 2004). Prenatal to postnatal permanence of depression, elevated cortisol and norepinephrine, diminished serotine levels, and severity of anxiety symptoms of the mothers may increase the right-hemispheric dominance of frontal alpha desynchronization in newborns (Diego, Field, Hernandez-Reif, et al., 2004; Field, Diego, Hernandez-Reif, et al., 2004; Jones et al., 2009; Lusby et al., 2014, 2016; Soe et al., 2016).

During the course of development seem early experiences in mother-child-interaction to further shape the alpha desynchronization pattern for infants of depressed mothers. A withdrawn interaction style of mothers, characterized by flat affect, disengaged behavior, and rare touching and vocalizing, increased right-hemispheric dominance of frontal alpha desynchronization between the age of few weeks and 3-to-6 months after birth and an intrusive interaction style, including rough physical contact and loud, fast verbal behavior, decreased it (Diego et al., 2006). This indicated arising of neurodevelopmental changes due to environmental influences during infancy. In infants of depressed mothers increased maternal behavioral inhibition, maternal depression and pronounced right-hemispheric dominance of frontal alpha desynchronization might be interrelated (Diego et al., 2006). Contrary, breastfeeding, which necessitates stability of

positive mother-child interactions, mark how right-hemispheric dominance of frontal alpha desynchronization and highly reactive temperaments in infants of depressed mothers are diminished due to positive experiences (Jones et al., 2004).

Some studies have been investigated right-hemispheric dominance of frontal alpha desynchronization as an indicator of emotional processes in infants of depressed mothers (Diego et al., 2006; Fernandez et al., 2004; Hernandez-Reif et al., 2006). More specifically, they have utilized the model of right- and left-hemispheric dominance of frontal alpha desynchronization being indicative of emotional withdrawal or approach, respectively. In the newborns, right-hemispheric dominance of frontal alpha desynchronization was found when an instrumental lullaby was combined with vocals. This pattern changed when instrumental music only was presented (Hernandez-Reif et al., 2006). Diego and colleagues (2004) utilized a peek-a-boo game to present 3-to-6-month-old infants with happy, surprised and sad facial expressions of their mothers and a stranger. Right-hemispheric dominance of frontal alpha desynchronization was increased in infants of depressed mothers in comparisons to healthy controls for all conditions. Moreover, sad facial expression increased the tendency for right-hemispheric dominance of frontal alpha desynchronization, and happy facial expression increased the tendency of left-hemispheric dominance of frontal alpha desynchronization in all infants. The tendency for right-hemispheric dominance of frontal alpha desynchronization was further related to increased looking time and negative affect during surprise and sad facial expressions. Salvia cortisol increased in infants of depressed mothers from prenatal to postnatal testing.

As part of a longitudinal study focusing on infant's vulnerability, Lusby and colleagues conducted two studies with 3, 6 and 12-month-old infants of depressed mothers (Lusby et al., 2014, 2016). The right-hemispheric dominance of frontal alpha desynchronization

was assessed again in F3 and F4 electrode sites. These studies confirmed longitudinally that the pattern of right-hemispheric dominance of frontal alpha desynchronization in 3- and 6-month-old infant was associated to prenatal and postnatal maternal depression. With ongoing maturation may frontal alpha desynchronization become more variable. Higher negative affectivity in infants of depressed mothers was related to right-hemispheric dominance of frontal alpha desynchronization at 3-month-old infants and left-hemispheric dominance of frontal alpha desynchronization at 12-month-old infants (Lusby et al., 2016). Moreover, the study has established the frontal alpha desynchronization mostly over the right hemisphere as an early psychophysiological marker in infancy for trait-level negative affectivity temperament, i.e., the infants' tendency to engage reactive processes when confronted with negative emotions.

Wen, Soe and colleagues conducted a longitudinal study with community sample. Prenatal and postnatal maternal depressive symptoms were assessed by a self-reporting questionnaire Edinburgh postnatal depression scale (EPDS) (Soe et al., 2016; Wen et al., 2017) and frontal alpha oscillations were measured with several frontal electrode sites. The results revealed a relation between a postnatal increase of maternal depressive symptoms and right-hemispheric dominance of frontal alpha desynchronization at 6 months of age (Soe et al., 2016). In agreement to other studies (Hernandez-Reif et al., 2006; Jones et al., 2004; Diego et al., 2006) was this pattern exhibited only in females (Wen et al., 2017). Soe and colleagues (2016) also reported that an increase of postnatal depressive symptoms in the infants was related to lower right frontal connectivity at 18 months, marking lower neural information transfer.

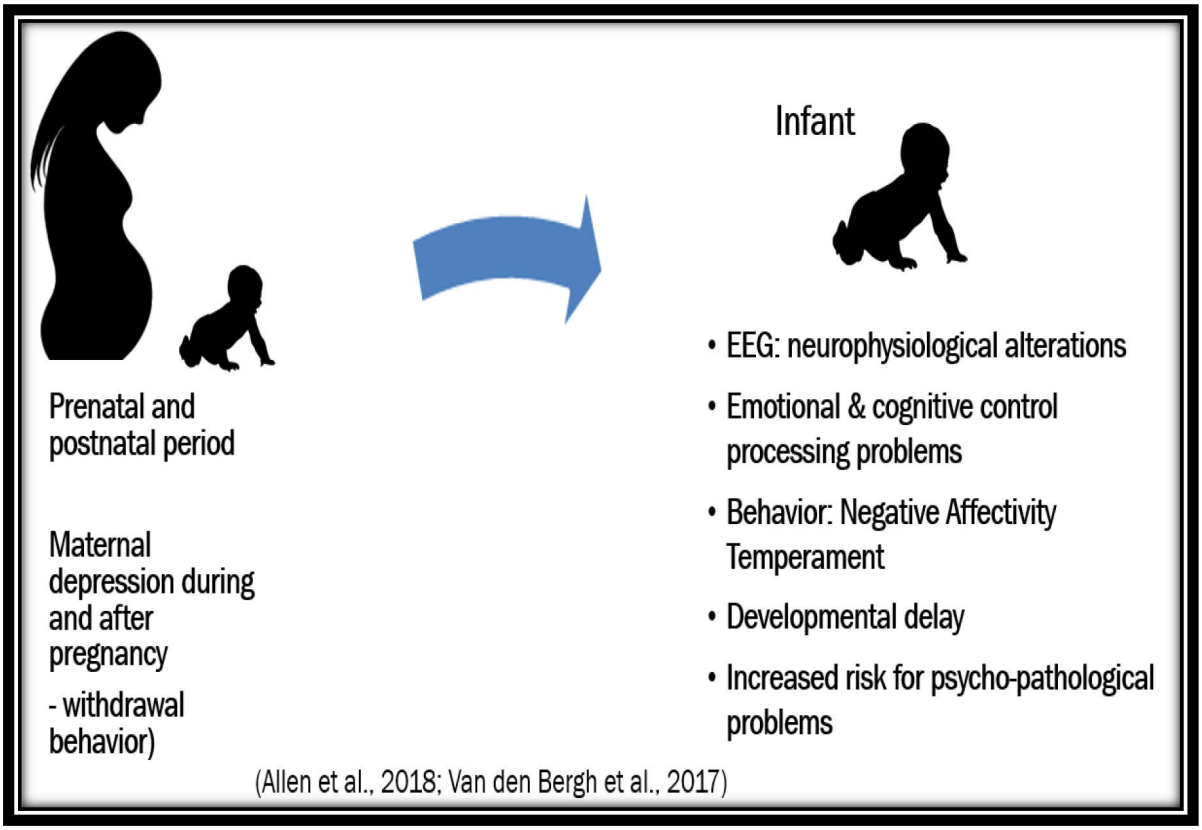
In summary, right-hemispheric dominance of frontal alpha desynchronization seems to be a neurophysiological marker of maternal depression early in infancy (Diego et al., 2006; Diego, Field, Jones, et al., 2004; Hernandez-Reif et al., 2006). As a marker, it is

stable over time and emotional states, especially in the first 6 months of infants' life. Moreover, a woman can transfer this pattern to her newborn and it can be observed earlier than one week after birth and for at least 3 years of the infants' life (Jones et al., 1997). It thus, creates a stable pattern of neural activation, which has consequences on infants' development (Field & Diego, 2008; Jones et al, 1997). In infancy, the right-hemispheric dominance of frontal alpha desynchronization is associated with dysregulation profiles like disturbed wake and sleep behavior, limited responsiveness on the Brazelton and facial and voice expression (Field & Diego, 2008). During late infancy, it is more variable and influenced by social experiences related to mother-child interactions (Diego et al., 2006). Right-hemispheric dominance of frontal alpha desynchronization, thereby, appears to be related to infants' emotion and negative temperament (Diego et al., 2006; Diego, Field, Jones, et al., 2004; Hernandez-Reif et al., 2006). The level of severity and timing of maternal depressive symptoms may also influence the degree of right-hemispheric dominance of frontal alpha desynchronization (Field & Diego, 2008). Some studies indicated pronounced right-hemispheric dominance of frontal alpha desynchronization only in girls (Hernandez-Reif et al., 2006). Moreover, right-hemispheric dominance of frontal alpha desynchronization might not appear only by depressive symptoms but it is relevant for other kinds of mental disorders as anxiety disorder as well (Field et al., 2010). Therefore, there is a consistency of the findings investigating the relationship between right-hemispheric dominance of frontal alpha desynchronization in infants of depressed mothers and impairments in emotional, cognitive processing and behavior during the infancy *(Figure 7)*. Hence, the right-hemispheric dominance of frontal alpha desynchronization in infants with maternal depression is a trait, persisting during time,

not just a state. This pattern might be used for identifying infants at risk as early as possible.

Figure 7

Overview of the influence on prenatal and postnatal maternal depressive symptoms during infancy



1.11 Hypotheses and research questions

This dissertation will be investigated the interactions between prenatal and postnatal maternal depressive symptoms, infants' neurophysiology, maternal educational status, stressful life events and distal protective factors. The hypotheses of this work are organized in two studies called *study one and study two* and are based on a diathesis model by Davidson and diathesis-stress model. More specifically, it will be tested the impact of prenatal to postnatal maternal depressive symptoms on infants' EEG alpha power and frontal and parietal alpha desynchronization, and then will be examined how prenatal and postnatal maternal depressive symptoms are related to stressful life events and distal protective factors. In both studies, the role of maternal educational status will be taken into consideration.

Firstly, in study one, the impact of prenatal to postnatal maternal depressive symptoms and educational status on infants' alpha power and frontal and parietal alpha desynchronization will be examined. In study two, the impact of different distal protective factors, such as overall social and partner social support, and proximal risk factors, such as stressful life events, on prenatal and postnatal maternal depressive symptoms will be interrogated. Thereby, various research questions will be investigated. Then, the methods, strategies and analyses will be compared and criticized.

This dissertation will be investigated five main hypotheses in two studies.

1.11.1 Hypothesis 1

Study one (Mothers-Infants)

Prenatal to postnatal maternal depressive symptoms and low educational status → infants' low alpha power

Firstly, according to Kan & Lee (2015) and Maguire & Schneider (2019), it can be hypothesized that prenatal to postnatal maternal depressive symptoms will be related to low alpha power in infants. Differences in alpha power between infants of depressed and non-depressed mothers and low-educated and high-educated mothers will be expected. Hence, low maternal education status will be supposed to impact infants' alpha power as well, possibly in similar manner as maternal depressive symptoms. The first hypothesis will be tested in study one.

1.11.2 Hypothesis 2

Study one (Mothers-Infants)

Prenatal to postnatal maternal depressive symptoms and low educational status → infants' right-hemispheric frontal and parietal alpha desynchronization

Secondly, according to Davidson & Fox (1989); Davidson et al. (2002); Field & Diego (2008) and Heller and Nitschke (1997), it can be hypothesized that infants from women with prenatal to postnatal depressive symptoms and low educational status will show differences in right- and left-hemispheric frontal and parietal alpha desynchronization during spontaneous EEG in comparison to infants from women without prenatal and postnatal depressive symptoms. Davidson's model hypothesizes that maternal

depressive symptoms were related to right-hemispheric dominance of frontal alpha desynchronization in infants. The model by Heller and Nitschke assumes right-hemispheric dominance of parietal alpha desynchronization. Both models will be investigated in this dissertation. Low maternal educational status will be supposed to be related to the right-hemispheric dominance of frontal and parietal alpha desynchronization, as well and in accordance with Feng et al. (2012) and Lopez-Duran et al. (2012). Previous research have indicated the impactful role of maternal education on offspring of depressed mothers (Feng et al., 2012; Lopez-Duran et al., 2012; Tomarken et al., 2004). Additionally, the quality of EEG data sets as an influencing factor will be taken into consideration as recommended by Magosso et al. (2019); Smith et al., (2017); Staudt (2014) and a sub-sample analysis would investigate whether there is a difference in the alpha desynchronization patterns from the participants with different EEG data quality (6-very good, 5-good and 4-satisfactory data quality; the categories were derived from the numbers of usable segments and artifacts). The second hypothesis will be tested in study one.

1.11.3 Hypothesis 3

Study two (Mother)

A reduction of maternal depressive symptoms over time ↓

Thirdly, in accordance with Soe et al. (2016) it can be hypothesized that maternal depressive symptoms will decrease over time. Previous research has shown that there is a deviation in depressive symptoms during the prenatal and postnatal periods (Soe et al., 2016). Mostly a reduction in postnatal depressive symptoms in comparison with

prenatal depressive symptoms was observed. The third hypothesis will be tested in study two.

1.11.4 Hypothesis 4

Study two (Mother)

Overall social and partner social support for the mothers →↓ prenatal and postnatal maternal depressive symptoms

Fourthly, in accordance with Gómez-López et al. (2019) it can be hypothesized that overall social and partner social support will reduce the self-perceived prenatal and postnatal maternal depressive symptoms. The increased prenatal and postnatal depressive symptoms in female participants are predicted to be related to less overall social and partner social support. The fourth hypothesis will be investigated in study two.

1.11.5 Hypothesis 5

Study two (Mother)

The occurrence of stressful life events and low maternal educational status →↑ prenatal and postnatal maternal depressive symptoms

Fifthly, in accordance with Glazier et al., (2004), it can be hypothesized that the more occurrence of stressful life events and low educational status will be associated with increased prenatal and postnatal depressive symptoms in female participants. The fifth hypothesis will be investigated in study two.

In the first study, it is assumed that prenatal to postnatal maternal depressive symptoms and low educational status will have a negative effect on infants' EEG alpha power and alpha desynchronization. In the second study, prenatal and postnatal depressive symptoms are predicted to be related to stressful life events and different protective factors, such as overall social support and partner social support. The two studies are addressed within the theoretical framework of both the diathesis-stress model and diathesis model by Davidson. The hypotheses are derived from studies on correlative and predictive relationships between infants' alpha power and frontal and parietal alpha desynchronization, maternal stressful life events, depressive symptoms and protective factors. It is assumed that increased right-hemispheric dominance of frontal and parietal alpha desynchronization will be observed in infants within the sub-clinical sample and their severity will correlate with increased maternal depressive symptoms. Moreover, the severity of stressful life events will be related to maternal depressive symptoms. Furthermore, it is expected that the prenatal and postnatal maternal depressive symptoms will be reduced by protective factors. The diathesis-stress model assumes that depressive symptoms interact with proximal risk and distal protective factors in a perplex way.

Even though there are some previous studies interrogating similar hypotheses, there are still uncovered questions and not enough studies with sub-clinical samples in both genders and within longitudinal design. This work would attempt to fill some of these gaps and clarified the untangled mechanisms behind them in infants and their mothers with sub-clinical level of depression. Additionally, the alpha desynchronization was not extracted only from the frontal brain areas but from the parietal brain areas as well, because there are just few studies investigating parietal brain areas. Information about the severity of the depressive symptoms and diagnostic tools will be provided as in

previous studies different procedures are used to identify a depression. In some studies (Lusby et al., 2014, 2016), diagnoses are made according to the diagnostic criteria of the DSM-V. In other studies, self-reported questionnaires such as EPDS are used for assessing depression (Soe et al., 2016; Wen et al., 2017). The symptoms' severity is important for the level of impact on EEG alpha desynchronization according to Davidson's model. It will be discussed how reliable findings may serve to monitor prodromal life trajectories and support prevention and intervention strategies. This dissertation, thereby, provides a better understanding of the complex interactions between the neurophysiology of infants, prenatal and postnatal maternal depressive symptoms, proximal risk and distal protective factors. Altogether, the main developmental life periods analyzed in the study are the processes during pregnancy, the transition phase and the dynamics after birth and early infancy.

2 CHAPTER TWO: METHODS

2.1 Participants and Inclusion criteria

The participants were the first 150 mothers and infants attending the longitudinal intervention study (BRISE, 1st wave) conducted by a research alliance under the main guidance of the University of Bremen and Leibniz Institute for Science and Mathematics Education affiliated to the University of Kiel. In Bremen (Bremen East, Bremen West, Bremen North and Bremen South), pregnant women and women who just gave birth to a child were invited to participate in the study. The BRISE sample includes more than 480 families (until 1st March 2022), who are living in the aforementioned districts in the municipality of Bremen. These specific districts were selected according to their social structure, such as unemployment rate, child poverty, according to their feasibility, e.g. existing infrastructure, sufficient number of child birth per year and according to the transferability of the results to a larger population. There were different strategies for recruiting families. However, families were recruited mainly through letters, which were sent to households with potentially pregnant women, or women, who have recently given birth. The BRISE recruitment process ended officially on the 31 st December 2021.

In addition, a network of people in the health care or social work sectors informed the potential families about the project and promoted it. Families, who live in one of the pre-determined districts, whose baby is not elder than 10 weeks, were included in the study, when at least one possible risk factor for the child development was presented in the family. These risk factors included low parental educational status, low family income, unemployment and migration status. The participants were divided, depending

on the district where they lived, into two groups: 1. intervention group and 2. control group. The mothers in the intervention group received information about specific programs related to childcare and child development. Most of the mothers and infants which were part of the intervention group participated in a series of intervention programs throughout this time. However, this doctoral project did not separate the participants according to these groups, because it investigated other research questions and as only the first 3 measurement points were included in the analyses, so that the long-lasting effects of the interventions possibly will not be followed. The families participated in the study after giving written, informed consent accordingly, the protocols approved by the Ethical Committee of the BRISE group. Compensations for the participation in data collection and experiments were provided. The doctoral project contained data from the participants who have completed the first interview during pregnancy (T0-prenatal) or 10 weeks after birth (T0-postnatal), the second interview after birth at 2 to 4 months from infants' life (T1) and the third interview after birth at 6 to 8 months from infants' life (T2).

Mothers were between 19-41 years old (M = 31.20, SD = 5.76 years), the mean week of pregnancy was M = 34.57, SD = 3.58 during the first visit, and 37% of the participants were expecting their first baby. 54% of the participants had no migration background; 21% were born in Germany, who either have one or two foreign-born parents; 25% were first generation immigrants. The 25% of participants who were born outside of Germany, were coming from each of the following countries: 4 mothers were from Syria, 2 mothers were from Iraq, Nigeria and Bulgaria and the rest participants were from: Egypt, Indonesia, Italy, Kazakhstan, Kosovo, Nepal, Nicaragua, Russia, Turkey, Ukraine and Venezuela. According to the CASMIN (Comparative Analysis of Social Mobility in Industrial Nations; Brauns et al., 2003; König et al., 1988) educational

classification, 70% of the participants were below the level of tertiary education (i.e. bachelor's degree).

2.2 Procedure

BRISE companions, who work in academic research at the University of Bremen visited the mothers and infants at home approximately two to three times a year. After signing consent forms, the interviews were conducted. The home visits contained questions about various topics, such as family environment, socio-economic factors, physical and psychological health, personality, family relationships, parenting and infants' development.

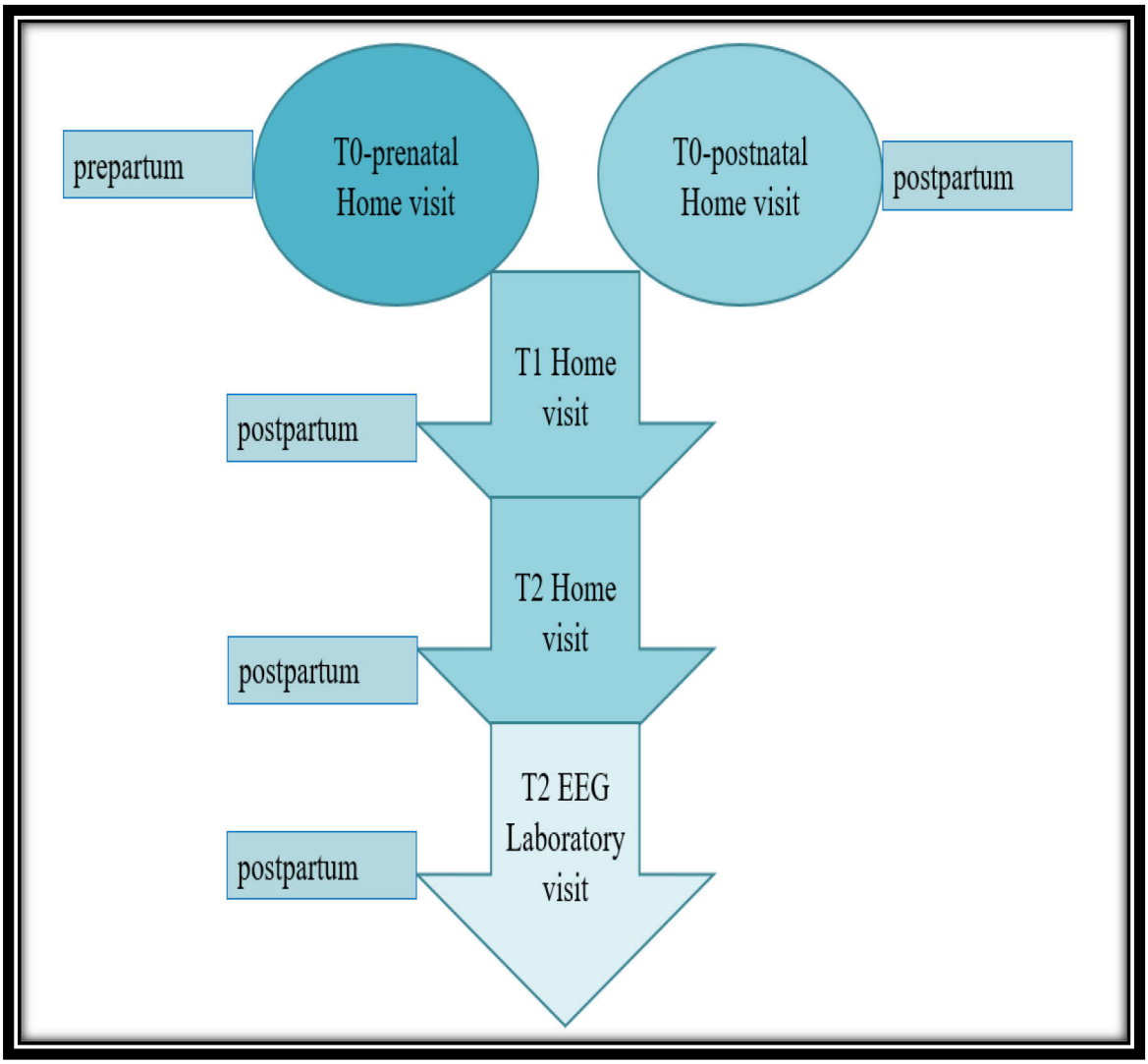
More specifically, the home visits contained questionnaires and different instruments for measuring various issues, such as pregnancy specific information, stressful life events, depressive symptoms, social experiences, social support, environment in family, health condition, family and partner relationships, parenting and different aspects for the development of the infants. At various time points, families were invited at the university laboratory for participating in different EEG sessions and other behavior experiments specially designed to investigate neurophysiology and neurocognitive functions of infants.

The participants were interviewed 1) after the 30th week of the pregnancy (T0-prenatal) or till the 10th week after child birth (T0-postnatal), and 2) between the 2nd and 4th month (T1), and 3) between the 6nd and 8th month after child birth (T2). The doctoral project contained data from the first 150 mothers-infants who have completed the interview during pregnancy or 10 weeks after birth and the next two visits after birth. *Figure 8* provides an overview of the procedure for these different measurement points

which will have been included in the study. For the doctoral project, the primary questions of interest from these three measurement points are the interactions between prenatal and postnatal maternal depressive symptoms, the occurrence of stressful life events, resources and infants' neurophysiological mechanisms assessed while spontaneous EEG session.

Figure 8

Overview of the procedure and different measurement points in the study



Note. Participants attended only T0-prenatal or T0-postnatal. T0-postnatal was submitted if the participants took part in the study after the birth of child.

2.2.1 T0-prenatal and T0-postnatal

The first interview during pregnancy (T0-prenatal) was conducted between the 30th and 40th weeks of pregnancy or till the 10th week after child birth (T0-postnatal). BRISE companions, who work in academic research at the University of Bremen, visited families at home, conducted a face-to-face interview with the women. This interview contained questions about the pregnancy, prenatal attachment, prenatal maternal self-efficacy, current emotions and thoughts about pregnancy and the baby, physical and psychological health, socio-demographics, family environment and family relationships, social and professional support and activities during pregnancy, etc. The partners filled by themselves the questionnaires including the same questions as for the mothers but in shorten version. This interview lasted around 1 to 1.5 hours. If the families were included in the project after delivery, the visit (T0-postnatal) took place during the first 10 weeks after birth and the same questions in shorten version were asked in a reformulated form. Understandably, the questions about prenatal depressive symptoms were not included in T0-postnatal because they could not be recollected in a reformulated form. 66 mothers did not complete the first interview (T0-prenatal) and in this way, the questionnaire data about prenatal depressive symptoms because they attended the program postpartum at T0-postnatal. Hence, only 84 mothers completed the first interview (T0) during the prenatal period.

2.2.2 T1 and T2

When the baby had a mean age of three (T1) and seven (T2) months, the families were visited again at home for the next interviews. Both of these visits again included face-

to-face interviews with the mothers and questionnaires for the fathers to fill out by themselves. For the first postpartum interview (T1), the families were visited when the infants were 2-to-4-month-old. The interview included questions about the delivery, nutrition, sleep and cry behavior, family environment and family relationships, maternal self-efficacy, physical and psychological health, social and professional support, milestones of early development, etc. Additionally, another interview to assess the socio-economical status (SES) of the household from the German Socio-Economic Panel (SOEP) was conducted.

The third visit (T2) took place between the 6th and 8th month of infants' life and the interview was again conducted in similar manner, using similar constructs, such as in T1, assessing: nutrition, sleep and cry behavior, family environment and family relationships, maternal self-efficacy, physical and psychological health, depressive symptoms, social and professional support, milestones of early development, etc.

The milestones of early development were assessed in two-to-four and in six-to-eight-month-old infants by the Milestones of Development in Early Years (MONDEY; Pauen, 2011) questionnaire. Both of these appointments took around 2 to 3 hours in total.

2.2.3 T2: EEG laboratory visit

The resting electroencephalogram (EEG) of the infants were recorded at the second postnatal measurement point (T2), when infants were approximately 8 months (±1 month), (M = 8.68 months, SD = 1.34 months). The experiment was conducted in a lightly illuminated and well-tempered room. During the spontaneous EEG, the infants were seated on the lap of the accompanying person (mostly the mother), with a view

onto a screen approximately 95 cm from the infants with two loudspeakers on each side. There were two experimenters available. The lead experimenter administered and maintained the task and conditions during EEG recording. The second experimenter was taking care after the infants and attended the recording only in case the infants became inattentive. The infants could play with silent toys or received snacks to keep themselves happy and quiet during test sessions. Extra breaks were offered when necessary. Stimuli (child short movie) were presented using Presentation® (Neurobehavioral Systems). The total duration of the child movie was 2 min and it was presented as the second of a set of four different paradigms. The movie was entertained in order to have a calm and relaxed effect on the infant and to avoid movements.

Caregivers were instructed to stay relaxed and were advised to avoid speaking or making noises. During the film, they were additionally informed that the infants were not required to watch the screen at all times. Attention getter use (a sound) was available. Moreover, pauses and interruptions were provided for the participants in case of restlessness.

2.3 Constructs

2.3.1 Prenatal and postnatal depressive symptoms

Prenatal and postnatal maternal depressive symptoms were assessed in BRISE with Edinburgh Postnatal Depression Scale (EPDS). The EPDS is a widely used screening tool for prenatal and postnatal depression (Bergink et al., 2011). It is a 10-item self-report questionnaire in which women are asked to rate how they have felt in the previous 7 days (Cox et al., 1987). The duration of the procedure is around 5 minutes. Different research groups have surveyed validation studies of the EPDS for prenatal and

postnatal periods (Bergink et al., 2011; Kozinszky & Dudas, 2015; Martin & Redshaw, 2018).

The EPDS has been translated in many languages including German. The German version of EPDS is proved to be a valid and reliable method tool assessing prenatal and postnatal depression (Bergant et al., 1998) and in this way, it is suitable for research use and for BRISE project.

Each question in EPDS is scored from 0 to 3 resulting in total score ranges from 0 to 30 and the whole procedure takes around 5 min (Cox et al., 1987). The standardization procedure as recommended by authors for summing the item responses (coding 0 to 3) was used and then dividing the total score by the number of items answered. There are 3 inverted items and their answers are coded from 3 to 0. With regard to missing values only participants who answered at least 70% of the questions were included in the analyses, e.g. at least 7 items answered, out of 10. Cut-off for "probable depression" has been suggested for total score at 12⁄13, and for "possible depression" at 9⁄10 (Cox et al., 1987) and are used as markers of possible major and minor depression, respectively.

EPDS questionnaire in BRISE was administered to mothers firstly, prenatally at T0: after 30 weeks of pregnancy, secondly, postpartum at T1: 3 months (±1 month) after delivery and thirdly, postpartum at T2: 7 months (±1 month) after delivery and used to quantify prenatal and postnatal levels of maternal depressive symptoms. Higher scores indicate a higher intensity of depressive symptoms. However, the EPDS is only a screening instrument and a subsequent clinical diagnosis was not conducted in BRISE.

Reliability analyses of EPDS at T0, T1 and T2 were performed by Cronbach's analysis. In this study, Cronbach's alpha for prenatal EPDS score was $\alpha = .90$ and for postnatal EPDS score (T1) and (T2) was 0.89 and 0.88, respectively. Test–retest reliability was determined by correlating scores of EPDS from the prenatal (T0) and postnatal (T1; T2)

periods using Pearson's correlation coefficients (two-tailed). The EPDS scores from T0 and T1 were highly correlated (r = .600, p = .00, N = 71). The EPDS score from the second postnatal period (T2) was also correlated with prenatal (T0) (r = .592, p = .000, N = 69) and postnatal (T1) (r = .618, p = .000, N = 128) EPDS scores. Pearson's correlations (two-tailed) were also used to examine the concurrent validity between the prenatal EPDS, and anxiety, somatization subscales (r = .655, p = .000, N = 75) and the stressful life events questionnaire (r = .379, p < .001, N = 75) also submitted in BRISE during the prepartum. The postnatal EPDS score only at T2 and not at T1 was also correlated with the stressful life events questionnaire (r = .259, p < .005, N = 114).

The same EPDS cut-off score of 9/10 in the prenatal and postnatal periods are used for the most optimal outcome of predicting the occurrence of depressive symptoms and for better group dividing. Hence, participants were grouped in a non-depressed group when they had lower than 10 total scores on the EPDS and to a depressed group when their scores were from 10 to 30. The prenatal and postnatal depressive EPDS scores were examined in study one and in study two as well. In study one, due to the big amount of missing data in the prenatal depressive EPDS score an average prenatal and postnatal depressive score (Mean from EPDS T0 and T1) was computed for some of further data analysis.

Table 1

Descriptive statistics for prenatal and postnatal maternal depressive symptoms at the first three measurements points (T0, T1, T2)

	Mean	Standard Deviation	Minimum	Maximum	N
EPDS T0	8.23	6.82	0	29	77
EPDS T1	7.06	5.82	0	24	140
EPDS T2	6.03	5.30	0	26	129

2.3.2 Educational status

Maternal educational status was assessed with the CASMIN (Comparative Analysis of Social Mobility in Industrial Nations; Brauns et al., 2003; König et al., 1988) educational classification. In CASMIN questions about the elementary, secondary and tertiary level of education were submitted. Score ranging from 1 (school) to 9 (higher tertiary education) is assigned to each maternal educational level. Thus, maternal educational status was scored on a 9-point ordinal scale created ad hoc and based on the German school system (M = 6.13, SD = 2.64, Min = 1 and Max = 9, N = 94).

The group division was made by means of a median split, which resulted that only mothers with university degrees were in the higher education group. According to the CASMIN, 70% of the participants had education below the level of tertiary education (i.e. bachelor's degree) and were assigned to the group with low educational status and 30% of the participants had education above the level of tertiary education and were

assigned to the group with high educational status. The educational status was investigated in study one and in study two as well.

2.3.3 Stressful life events

The stressful life events were assessed in BRISE with a self-created stressful life events questionnaire. This questionnaire is a list with 10-items that describes stressful life events that might have an influence on person life. Participants were asked with *yes* or *no* if any of the stressful life events occurred during the pregnancy period and only if it had occurred they rated how strong each event affected them on a 4-point Likert scale *1) was not at all stressful; 2) was somewhat straining; 3) predominantly burdening; 4) burdensome*. Examples of the stressful life events used in the questionnaire are an occurrence of death or diseases in the family, a loss of job, partner conflicts, divorce, etc. The stressful life events questionnaire in BRISE was administered to mothers after 30 weeks of pregnancy (T0-prenatal) till the 10^{th} week after child birth (T0-postnatal) and used to quantify the prenatal occurrence of stressful life events. A similar measure has been used in previous studies (Rai et al., 2012; Thomson et al., 2014). There are not inverted items and answers are coded from 1 to 4, creating a total score in the standardized way by summing the item responses and then dividing the total by the number of items answered. As controlling for missing values, only participants who answered at least 70% of the questions were included. Reliability analysis of the stressful life events questionnaire at T0 was performed by Cronbach's analysis. In this study, Cronbach's alpha for prenatal stressors was $\alpha = .70$, $M = 4.92$, $SD = 5.86$, $Min = 0$ and $Max = 33$, $N = 132$. Pearson's correlations (two-tailed) tested the concurrent validity between the stressful life events questionnaire and the prenatal and postnatal

EPDS, respectively (r = .379, p < .001, N = 75), (r = .259, p < .005, N = 114). The self-created stressful life events questionnaire was utilized in study two.

2.3.4 Overall social support and partner social support

The overall social and partner social support were measured with self-report questionnaires to assess the participants' subjective level of perceived support. Hence, in this dissertation was not assessed formal social support given from different interventions programs such as professional support providers but it was examined self-reported informal social support focusing on the specific behaviors that the participants reported to receive from their partners, families, friends and relatives. The overall social support was assessed in this study from a self-reported social support questionnaire focusing on the frequency and level of provided social and emotional integration from partners, relatives, families and friends. In addition, to assess levels of informal social support of partner, the study assessed the support received from partner during pregnancy and related to providing support and accompanying by pregnancy specific activity. The both questionnaires were submitted during pregnancy (T0-prenatal) or from birth to 10 weeks postpartum (T0-postnatal) and utilized in study two.

To measure Overall Social Support in BRISE a 14-item short version of the Social Support Questionnaire (F-SozU (K14); Fydrich et al., 2009) was submitted prenatally (T0-prenatal) or postnatally (T0-postnatal). The questionnaire assesses informal social support from partner, family, friends and relatives and social integration with a 5-point Likert scale. There are different items in the questionnaire assessing emotional support, practical support and social integration, they all express and reflect the level of self-perceived support. There are not inverted items in the short version and answers are

coded from 1 to 5. The total score is calculated in the standardized way by summing the item responses and then dividing the total by the number of items answered (Fydrich et al., 2009). To compensate for missing values the scale value was created only from participants who gave at least 70% responses. In this study, the Cronbach's α for social support questionnaire was α = 0.97 (*M* = 57.30*, SD* = 13,96*; Min* = 14 and *Max* = 70*, N* = 126*)*.

To assess partner social support, the mothers were asked whether their partners participated as accompanying person in any activities or programs related to pregnancy and child rearing, such as visiting mothers and children activity groups, sport courses specifically for mothers or a midwife or birth courses. It was a dichotomous scale, the participants answered the questions with yes or no. The reliability analysis was again performed by Cronbach's analysis. In this cohort, the Cronbach's α for partner social support questionnaire was α = 0.97 (*M* = 5.16*, SD* = 1.18*, Min* = 1 and *Max* = 6*, N* = 122*)*.

2.4 Statistical analysis

IBM SPSS Statistics (Version 25.0) were used for the descriptive statistics, correlations analyses between variables, t-test, ANOVA and regression analyses. Preliminary analyses proved different requirements for the further data analyses. As in the analyses, different types of scale variables were involved, such as nominal (e.g. age), ordinal (e.g. depressive symptoms) and metric (e.g. EEG alpha power and desynchronization), it was possible the above-mentioned statistical analyses to be performed and interpreted. The equality of variance and homogeneity for the variables included in the analyses and data distribution were confirmed with Levene-Test and Shapiro-Wilk Test, respectively. In order to investigate the interactions between prenatal and postnatal maternal depressive

symptoms, stressful life events, protective factors and infants' neurophysiology, possible confounders and predictors in regressions models were considered, only if they were significantly correlated with the above-mentioned variables. For all analyses, significance was inferred at p-value < 0.05.

For better differentiation, the description of statistical analyses performed in this dissertation will be presented separately in study one and study two. Moreover, the methods, results and discussion of these two studies: study one and study two will be introduced separately in the following two chapters: *chapter three: study* one and *chapter four: study two*.

3 CHAPTER THREE: STUDY ONE

The main aim of study one was to provide insights into the neurophysiological patterns in infants of sub-clinically depressed mothers from culturally and socially diverse population. More specifically, study one investigated on the first place, the influence of prenatal to postnatal maternal depressive symptoms and educational status on infants' alpha power and on the second place, on frontal and parietal alpha desynchronization. In the following paragraphs, the methods, results and discussion of study one will be described in details.

3.1 Methods: Study One

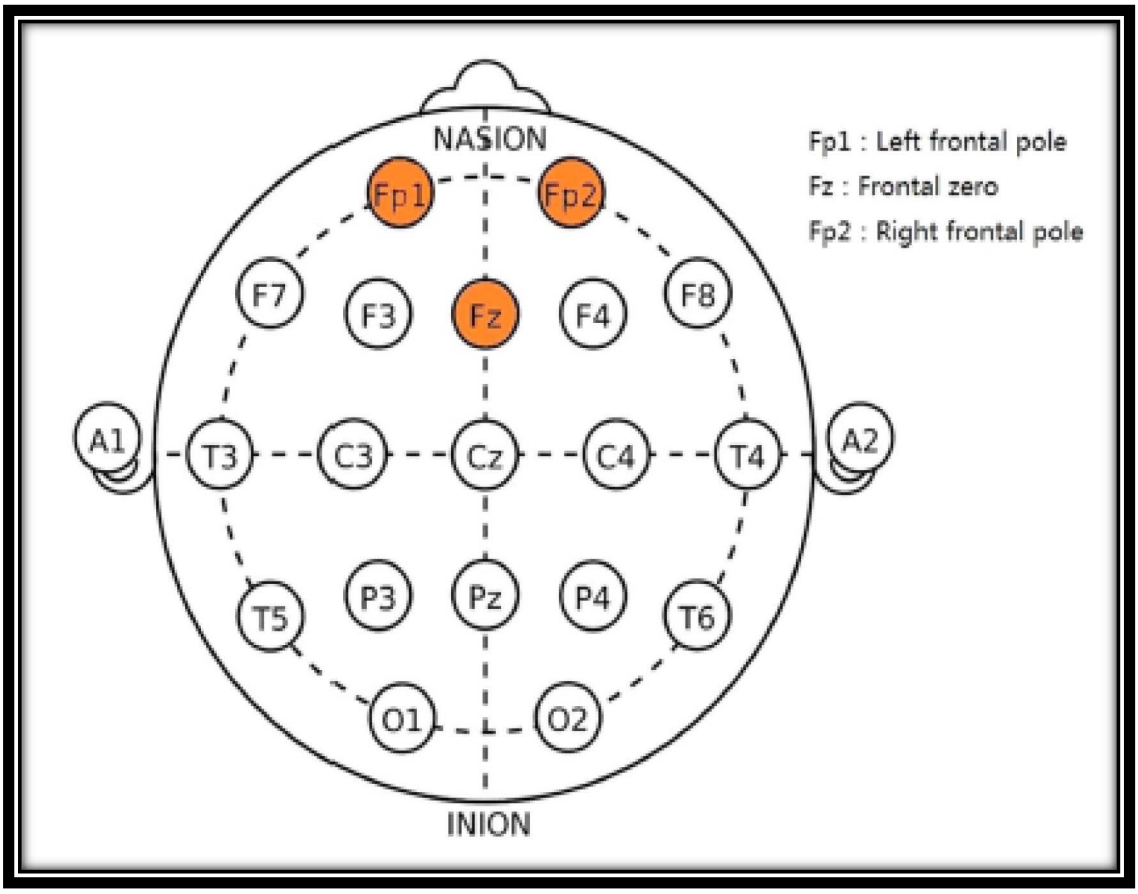
EEG

As mentioned before, the spontaneous EEG was recorded at the second postnatal measurement point (T2), when the infants had a mean age of approximately 8 months, (M = 8.68, SD = 1.34). EEG was recorded from 32 channels with a sampling rate of 512 Hz and online broadband filter of 140 Hz was derived towards to the vertex (Cz) using BrainVision professional Recorder (Brain Products GmbH). An actiCAP snap (EasyCap GmbH) with 32 channels (AG/AgCl sensors bedded into active electrodes) was applied to an actiCHamp device (Brain Products GmbH). According to the 10-20 system, the electrodes are placed at the same distance from certain fixed points of the head (e.g. between the nasion and the inion) in percentage terms (Zhao et al., 2021). Each electrode is designated with a letter for its location in one of the brain lobes and a number for the side on which it is located e.g. the designation "O1" describes the

electrode in the occipital lobe on the left side *(Figure 9)* (Zhao et al., 2021). To provide better contact between the head surface and electrodes, a warmed gel, as recommended by (Addyman & Mason, 2016), was carefully injected and applied to each electrode (SUPER-VISC, EasyCap GmbH) with a blunt dental syringe.

Figure 9

Placement of the EEG electrodes measured according to 10-20 system



Note. Figure after Zhao et al. (2021).

3.1.1 EEG pre-processing

For EEG pre-processing both BrainVision Analyzer Version 2.1.0 (Brain Products GmbH) and MATLAB Version 9.7, R2019b were used, e.g. the artefacts were removed in the BrainVision Analyzer.

First, the data were filtered within 0.5 and 48 Hz in order to examine the signals primarily attributable to brain activity (Sturm et al., 2009). Channels with noisy signals were interpolated. If more than three channels were interpolated, the dataset was excluded from further analysis. The average referenced was then applied. Infants' EEG is generally limited in recording time because it is more difficult for infants to be instructed to stay calm (Sturm et al., 2009). To enhance the accuracy of detecting the artifacts and through this to separate the brain signals from the other signal, a rough semi-automatic EEG artifact removal was conducted, removing all sections with min-max differences greater than 200 μV and jumps >50 μV. The main advantage of semi-automation is the time efficiency due to the combination of technical and human productivity (Tousseyn et al., 2014). The cleaned data were then divided into epochs of 2000 ms with a 50% overlap for better accuracy and data classifications. During this procedure, the fine artefacts check was conducted segment by segment to improve and precise the artifact rejection procedure. Hence, the individual, non-overlapping parts of the segments were free of artifacts. Only when the remaining segments in a data set were at least 17 (Allen et al., 2004; Diego et al., 2010), the data set will be included in the analysis and exported to MATLAB. Finally, the general signal quality was assessed and the data sets were coded for the further statistical analyses as follows: *6-very good data quality; 5-good data quality; 4-satisfactory data quality*, based on the level of quality and the epochs (the proportion of artifacts, signal-to-noise ratio (SNR) and the usable segments). The SNR is „the ratio of the power spectral density (PSD) of a signal (meaningful information) with respect to the power of the background noise" and it is characterized as „the gold standard for quantifying the performance of brain recording devices" (Suarez-Perez et al., 2018, p. 1-3). The cut-off criteria were at least 17 usable segments. As conducting EEG with infants was associated with more data loss than

adults because infants moved more, had small attention spans and were not able to follow instructions (van der Velde & Junge, 2020), sets with different EEG data quality were analyzed. Only in the two two-way ANOVAs the data of participants rated with 4-satisfactory data quality were excluded. In this analysis, it was not distinguished between the sets rated with 6-very good data quality and 5-good data quality. Thus, the analysis with a sub-sample of the participants was conducted. In the rest of the analyses, a group division was not made and all data sets rated with 6, 5 and 4, with at least 17 remaining segments were included.

3.1.2 EEG data analysis: Power analysis

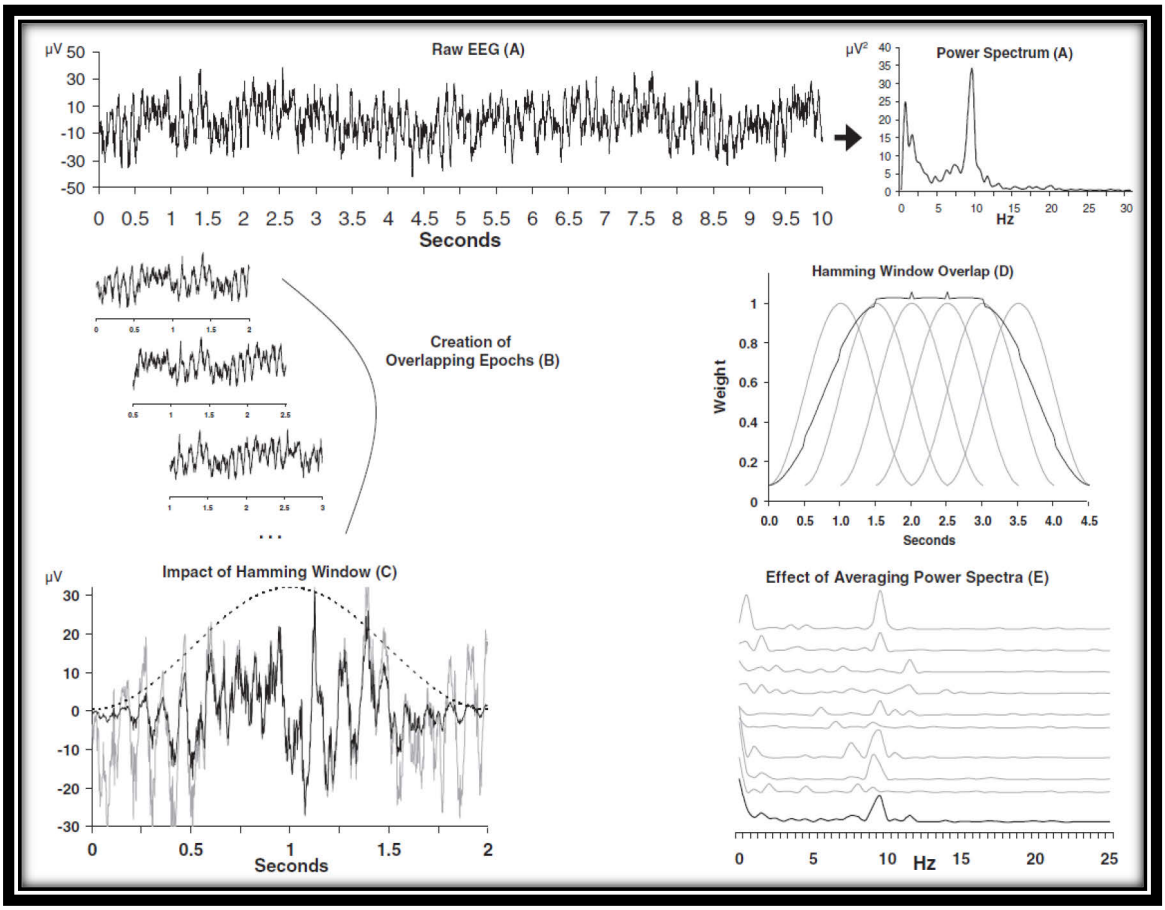
The data were analyzed as following the basic steps in transforming time domain to power spectrum in EEG alpha desynchronization research and in similar manner as recommended by (Allen et al., 2004; Smith et al., 2017) *(Figure 10)*. In MATLAB, a Fast Fourier Transform (FFT) was conducted. Because the data were collected from a continuous resting period, short epochs were created and converted to frequency spectrum (Allen et al., 2004; Smith et al., 2017). The main assumption of FFT is that each signal is periodic (repeats over intervals of time) and it should be decomposed into series of sine and cosine waveforms at many frequencies, with the beginning of each waveform having its own particular phase (Smith et al., 2017). For resting data, relative short epochs (1–2 s) are recommended for FFT because short epochs provide for further data analysis small segments of data with features that repeat in a highly similar way in the waveform (Allen et al., 2004; Smith et al., 2017).

As windowing has avoided the discontinuity but at the same time reduced the contribution of data near the end of the epoch to the power spectrum, overlapping

epochs helped the data to receive minimal weight near the end of epoch (Smith et al., 2017). Only the signal near the central portion of each epoch was highly weighted and the signal near the end of each epoch was reduced. Therefore, epochs were overlapped to ensure that all portions of the signal receive high weighting. Thus, windowing of the time-domain signal was performed to reduce the artifactual frequencies in the resultant power spectrum. Hence, a Hanning window was performed over the length of each epoch (2 seconds) in course of determining the power values (in μV^2 by summing all spectral points in the frequency range and dividing by the range in Hz) for the infants' alpha frequency band (Marshall et al., 2009; Saby & Marshall, 2012). The band was set at 3-12 Hz to reflect the current state of infants' alpha, considering EEG variability and for better detection of alpha band as the strongest frequency because there is a wide range of alpha frequency band in studies with infants of depressed mothers (Diego et al., 2010; Mizuno et al., 1970). The power spectrum was determined for each channel and epoch and the EEG signal was transformed into a frequency-domain representation in the form of a power spectrum. Power spectrum reflected the power in the signal for each infant at each frequency. Thus, with a two-second epoch, one infant had greater precision with values every 0.5 Hz (0.5, 1.0, 1.5 …). As mentioned before, the FFT converted each time-domain epoch to a power spectrum and the average of the power spectrum was ultimately taken as the basis for the analysis.

Figure 10

The basic steps in transforming time domain to power spectrum for alpha desynchronization research



Note. "Panel A": The signal is transformed into a frequency-domain representation in the power spectrum. "Panel B": The epoching of the longer segment into shorter overlapping epochs. "Panel C": The effect of the Hamming window (dotted bell curve). "Panel D": The net weighting of overlapping hamming windows. "Panel E: The effect of averaging power spectrum." Figure after (Allen et al., 2004, as cited in Smith et al., 2017, p. 101).

3.1.3 EEG data analysis: Calculation of the alpha desynchronization

For the alpha desynchronization calculation, the natural log of left hemisphere alpha power was subtracted from the right hemisphere alpha power (e.g. *log right alpha power minus log left alpha power*) (Coan & Allen, 2004; Reznik & Allen, 2018). In this way, the differences of alpha power (right minus left alpha power) from electrodes of the two hemispheres was compared to reflect alpha desynchronization. Hence, the alpha desynchronization score was created for each of the electrode pairs: medial-frontal F3/4; lateral-frontal F7/8; medial-parietal P3/4; lateral-parietal P7/8 as usually investigated in asymmetry research (Coan & Allen, 2004; Reznik & Allen, 2018). The alpha desynchronization was assessed within alpha frequency band and in the frontal lobe as mostly assessed (Jesulola et al., 2015). As a relative measure, positive value of alpha desynchronization score mirrors a relatively higher left-hemispheric alpha desynchronization and a negative value of alpha desynchronization score mirrors a relatively higher right-hemispheric alpha desynchronization (Coan & Allen, 2004). As such, a positive score putatively reflects a left-hemispheric dominance of alpha desynchronization and an negative score reflects a right-hemispheric dominance of alpha desynchronization (Coan & Allen, 2004; Reznik & Allen, 2018). Alpha desynchronization in depressed individuals is hypothesized to be presented as a right-hemispheric dominance of frontal alpha desynchronization (Jesulola et al., 2015). Hence, the hemispheric difference in EEG activity of depressed individuals is described in terms of *alpha desynchronization*, wherein a negative alpha desynchronization score is assumed to be associated with hyper-activity of the brain regions over the right hemisphere.

Statistical analysis

In study one, due to the big amount of missing values in the prenatal depressive EPDS score (T0), an average prenatal and postnatal depressive score (Mean from EPDS T0 and T1) was created for further data analysis. The use of this average score was possible because there were not significant differences between the both mean scores (*T0: M* = 8.09*, SD* = 7.02 and *T1: M* = 6.89*, SD* = 6.15) evaluated with t-test (p = .288). For all analyses, significance was inferred at p-value < 0.05.

3.1.4 Pearson Correlation analysis

To investigate the first two research hypotheses in study one, Pearson's correlation analyses (two-tailed) were used. Firstly, average prenatal and postnatal (T0 and T1) and postnatal maternal depressive symptoms (T2) scores were correlated to maternal educational status and infants' alpha power. Secondly, average prenatal and postnatal (T0 and T1) and postnatal maternal depressive symptoms (T2) scores were correlated with maternal educational status and infants' alpha desynchronization score.

3.1.5 Regression analysis

Series of separate linear regression analyses were used to interrogate the hypotheses of the study. After confirming the requirements for further analysis, such as distribution of variables which are measured on an incremental level and homogeneity test, the regression analyses were conducted. Additional requirements were the correlations performed before and described above to reached the level of significance ($p < 0.05$), then the variables were entered in the following linear regression models. The

aforementioned hypotheses were investigated in the entire sample using the regression models.

In study one, the influence of maternal depressive symptoms on infants' frontal and parietal alpha power and alpha desynchronization, including maternal educational status as a confounder, were examined within series of separate linear regression analyses. To test the first hypothesis, in each regression models, one of each EEG alpha power value: medial-frontal (mean score from F3, F4), lateral-frontal (mean score from F7, F8), medial-parietal (mean score from P3, P4) and lateral-parietal (mean score from P7, P8) was separately entered as a dependent variable and average prenatal and postnatal maternal depressive score (T0 and T1) was entered as an independent variable. In each regression models, testing the second hypothesis, one of each alpha desynchronization value: medial-frontal (F4-F3), lateral-frontal (F7, F8), medial-parietal (P3, P4) and lateral-parietal (P7, P8) was separately entered as a dependent variable and an average prenatal to postnatal maternal depressive scores (T0 and T1) was entered as an independent variable.

In all of the aforementioned regression models, maternal depressive symptoms were considered as continuous variables. The use of average prenatal and postnatal maternal depressive score for further data analyses was necessary due to the huge amount of missing data in the prenatal depressive score and was possible because there was almost no difference between the both depressive scores. Potential confounders were selected based on prior theory and empirical work, but were included in the analyses only if significantly correlated with the average prenatal or postnatal depressive score (T0 and T1) and infants' EEG outcome (alpha power or alpha desynchronization). As past research revealed that infant's gender and age might contribute on EEG outcome, these variables were considered as possible potential covariates (Field & Diego, 2008;

Hernandez-Reif et al., 2006). Thus, to determine which covariates should be included into the models, the relations between infants' EEG outcome, maternal depressive symptoms and potentially relevant variables, such as maternal educational status and infant's age and gender, were examined within correlation analysis. At the end, the whole process led to the inclusion only of maternal educational status as a covariate in the aforementioned regression analysis. In summary, there were eight regression models.

3.1.6 ANOVA with sub-sample

Two two-way ANOVAs were performed to understand if there were any interaction effects between the average score of prenatal and postnatal depressive symptoms (independent variable), maternal educational status (independent variable) and firstly, average medial-frontal F4/3 alpha desynchronization score (dependent variable) and secondly, average medial-parietal P4/3 alpha desynchronization score (dependent variable) with the small sub-sample of the participants. The small sub-sample of the participants included only infants with the EEG signal quality coded with 6-very good data quality and 5-good data quality. Hence, in this analysis, the data of participants rated with 4-satisfactory data quality were excluded to test if the quality of data set might be an influencing factor and whether there is a difference in the alpha desynchronization patterns from the participants with different EEG data quality, and because such analysis is suitable for infants (Magosso et al., 2019; Staudt, 2014). According to some authors, the quality of EEG data might be a reason for the alpha desynchronization to be observed or not (Magosso et al., 2019; Smith et al., 2017; Staudt, 2014). For the analysis, participants from the sub-sample were split into two groups according to the severity of their depressive symptoms assessed with EPDS as 1

non-depressed and 2 depressed group and according to their educational status assessed with CASMIN into 1 low-educated and 2 high-educated (Cut-off scores were described and aforementioned in *chapter two: methods*).

3.1.7 SEM model

Structural equation modeling (SEM) as implemented in the MPLUS software package (Muthén & Muthén, 2014) was used to investigate further the complex interactions between variables and provide another way of data presentation. Moreover, structural equation modeling was conducted because it simultaneously models all paths, giving more powerful, accurate and robust estimation of the effects than regressions and in this way, another data assessment and presentation (Muthén & Muthén, 2010). SEM modeling is also suitable for BRISE and this research project because it analyzes different kinds of data that contain missing values and because of the importance for modeling the latent variables in this project. Latent variables were used to represent different factors corresponding to the examined constructs (Muthén & Muthén, 2014). SEM was used to investigate the impact of prenatal to postnatal maternal depressive symptoms on infants' alpha power over the whole brain. Hence, there was one SEM model. The direct effects were assessed through general analysis and maximum likelihood estimation. To assess the goodness-of-fit of the model, model fit was evaluated by use of the Chi-Square statistic, the root mean square error of approximation (RMSEA, with values ≤ .08 indicating adequate fit), the Tucker–Lewis index (TLI, with values ≥ .95 indicating adequate fit) and the comparative fit index (CFI, with values ≥ .95 indicating adequate fit) (Muthén & Muthén, 2010; Hu & Bentler, 1995; Marino et al., 2019).

In the model, a latent variable called *prenatal to postnatal maternal depressive symptoms* which was created from prenatal depressive score (EPDS T0) and postnatal depressive score (EPDS T1), was used. The SEM model investigated the effects of maternal depressive symptoms on infants' EEG alpha power. For this purpose, latent variable called *infants' EEG alpha power* was build from average medial-frontal (F3, F4) alpha power, average lateral-frontal (F7, F8) alpha power, average medial-parietal (P3, P4) alpha power, average lateral-parietal (P7, P8) alpha power, average occipital (O1, O2) alpha power and average fronto-central and temporal EEG (FC1, FC2, FC5, FC6, T7, T8) alpha power. In this way, was possible to assess in one model more brain regions and to achieve a better picture of consequences of maternal depressive symptoms on infants' brain activity. These two latent variables were entered in the first SEM model and interrelated (based on regression method) with each other. Hence, a regression analysis was performed by using infants' EEG alpha power as the dependent variable and prenatal to postnatal maternal depressive symptoms were modeled as latent independent variable. Standardized estimates of path coefficients were reported in the model as well.

3.2 Results: Study One

Study one investigated the first two hypotheses of this dissertation, firstly, the impact of prenatal to postnatal maternal depressive symptoms and maternal educational status on infants' alpha power and secondly, on frontal and parietal alpha desynchronization were examined.

Out of 150 mothers and infants who participated in the study: 111 infants attended the

EEG session (43% females and 57% males) and 20 infants did not have usable EEG data (for example, data with motion and muscle artifacts). After controlling if these 20 infants were from mothers with increased depressive symptoms, no such connection was found. Hence, the full infants' sample of this study included 91 infants. As was mentioned before, the mean age of the infants was 8 months (M = 8.68, SD = 1.34) during the EEG scanning (T2).

3.2.1 The impact of prenatal to postnatal maternal depressive symptoms and educational status on infants' EEG alpha power

Hypothesis 1

To examine the first hypothesis, the impact of prenatal to postnatal maternal depressive symptoms and educational status on infants' EEG alpha power, different kinds of statistical analyses as described in the aforementioned section: *methods: study one*, were conducted. In the results section, firstly, correlation analyses between the study variables will be reported, secondly, a series of linear regression analyses will be reported and thirdly, a SEM model will be reported.

Infants in this study (N = 91) had values for alpha power ranged from *min* = 8.27 in the lateral-frontal electrodes (F7-F8) and *max* = 124.57 (P8-F7) at the lateral-parietal electrodes. It seemed that in the lateral-frontal areas, the EEG absolute alpha power was the lowest, and in the lateral-parietal areas, the EEG absolute alpha power was the highest. The mean power score at medial-frontal electrodes (F3-F4) was M = 24.71, SD = 12.59 and at lateral-frontal electrodes (F7-F8) was M = 22.52, SD = 11.36. The mean power score at medial-parietal electrodes (P3-P4) was M = 32.38, SD = 19.68 and at

lateral-parietal electrodes (P7-P8) was M = 40.42, SD = 19.34. The mean power score at occipital electrodes (O1-O2) was M = 41.08, SD = 16.86. The mean power score at fronto-central and temporal electrodes (FC1-FC2-FC5-FC6, T7-T8) was M = 23.74, SD = 10.52.

On the first place, series of correlation analyses demonstrated significant results. More specifically, pearson correlations with prenatal to postnatal (T0 and T1) maternal depressive symptoms and infants' EEG alpha power (3-12 Hz) in different brain regions reached a significance level (*Table 2)*.

Table 2

Pearson correlations for Study Variables: The relationship between prenatal to postnatal maternal depressive symptoms, educational status and infants' EEG alpha power

Variable	*N*	*M*	*SD*	1	2	3	4	5	6
1. Depressive symptoms (T0 and T1) [a]	146	7.53	5.68	-					
2. Infants' EEG alpha power (F3-F4)	91	24.71	12.59	-243* .020	-				
3. Infants' EEG alpha power (F7-F8)	91	22.52	11.36	-264* .012	.875** .000	-			
4. Infants' EEG alpha power (P3-P4)	91	32.38	19.68	-250* .017	.863** .000	.776** .000	-		
5. Infants' EEG alpha power (P7-F8)	91	40.42	19.34	−.218* .038	.678** .000	.697** .000	.696** .000	-	
6. Maternal educational status	94	6.13	2.64	−.358* .00	-.217 .085	-.158 .212	-.155 .220	−.366** .003	-

Note. [a] An average score of prenatal and postnatal maternal depressive symptoms,

*$p < .05$; **$p < .01$.

Increased prenatal to postnatal maternal depressive symptoms were related to low alpha power in infants' different brain regions. Additionally, prenatal and postnatal maternal depressive symptoms and infants' alpha power at lateral-parietal areas were significantly negatively correlated with maternal educational status. Higher maternal depressive scores were associated with lower educational status. There were no significant relations between maternal depressive symptoms at the second postpartum period (T2) and infants' EEG alpha power.

Regression analysis

Secondly, after the above-mentioned correlations revealed significance, separate regression models were conducted to evaluate the role of prenatal to postnatal maternal depressive symptoms on infants' EEG alpha power. Among the plausible covariates, such as infants' age and gender, mentioned in *chapter three (methods: study one)*, only maternal educational status ($r = -.358$, $p < .001$, $N = 94$) was found to significantly correlate with the study variables. Hence, only maternal education status was included in the analyses as a covariate. The results of these linear regression analyses are presented in *Table 3.*

Table 3

Linear Regression analysis: The effects of prenatal to postnatal maternal depressive symptoms and educational status on infants' frontal and parietal EEG alpha power

EEG alpha power	Medial-frontal F3, F4				Lateral-frontal F7, F8			
	ΔR^2	SE	ß	*P*	ΔR^2	SE	ß	*P*
Depressive Symptoms [a]		.329	-.310	.017*		.293	-.335	.011*
Educational status	.132	.707	-.321	.014*	.125	.630	-.270	.038*
EEG alpha power	Medial-parietal P3, P4				Lateral-parietal P7, P8			
	ΔR^2	SE	ß	*P*	ΔR^2	SE	ß	*P*
Depressive Symptoms [a]		.522	-.274	.038*		0.468	-.286	.021*
Educational status	.091	1.123	-.274	.061	.207	1.006	-.462	.000*

Note. [a] An average score of prenatal and postnatal maternal depressive symptoms, $N = 64$; $^{*}p < .05$; $^{**}p < .01$.

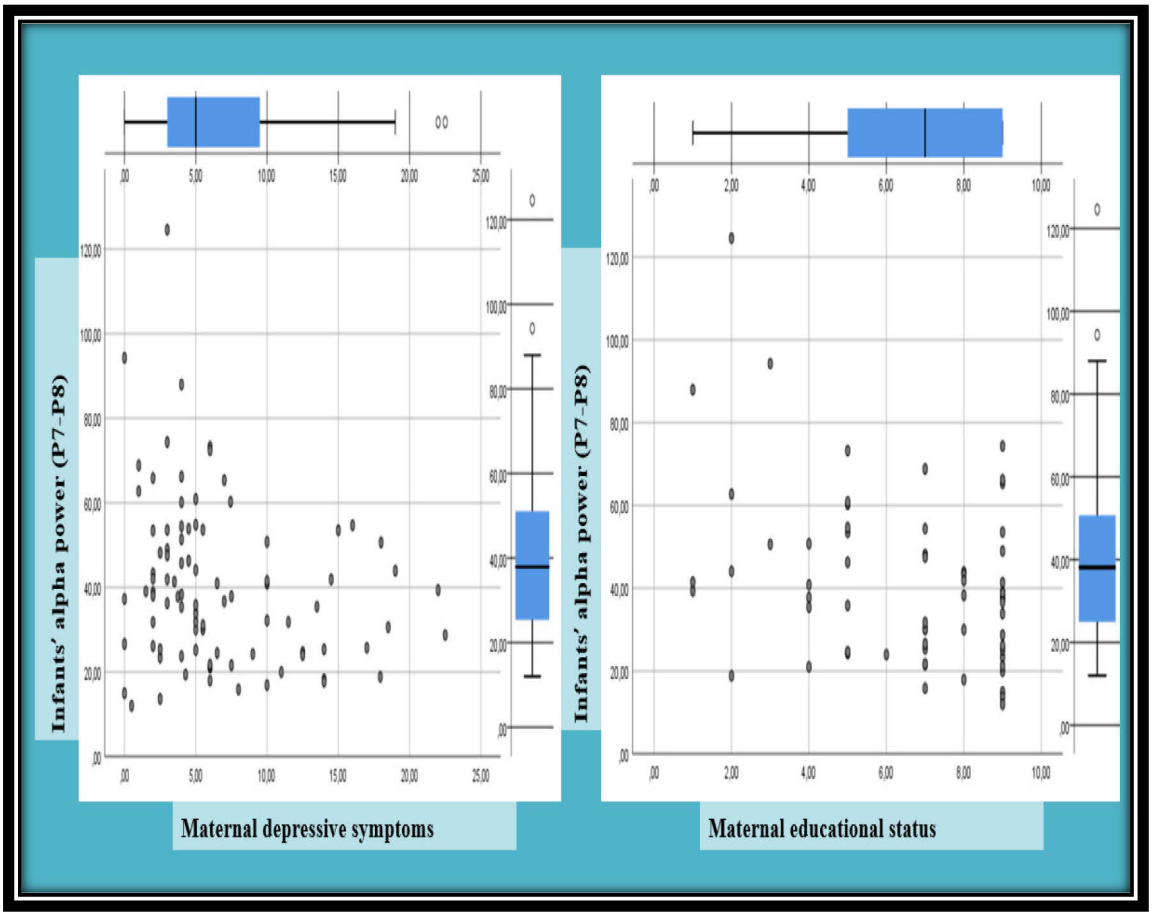
The results indicated that prenatal and postnatal maternal depressive symptoms and educational status both influenced infants' EEG alpha power. Increased prenatal to postnatal maternal depressive symptoms and higher maternal educational status predicted significantly lower frontal and parietal alpha power in infants *(Table 3)*. It seemed that higher the depressive symptoms at the prenatal and the first postnatal periods were and higher the maternal educational status was, lower the infants' EEG alpha power in frontal and parietal region were. Generally, the EEG alpha power was

lower in the infants of mothers with depressive symptoms in comparison with the infants of non-depressed mothers (*Figure 11).*

The *Figure 11* shows that the infants' alpha power has tendency to be lower within the depressed group of mothers. On the *Figure 11*, the EEG alpha power seems almost evenly distributed between the infants of mothers with low educational status and the infants of mothers with high educational status. The tendency revealed in the aforementioned analyses of alpha power to be only slightly higher in the infants of mothers with low educational status was hardly to be observed at the graph. The lateral-parietal areas have been shown on the graph because only the alpha power of these regions was highly correlated with maternal educational status but the proneness has been seen over the whole brain.

Figure 11

The relationship between prenatal to postnatal maternal depressive symptoms, educational status and infants' alpha power at lateral-parietal areas



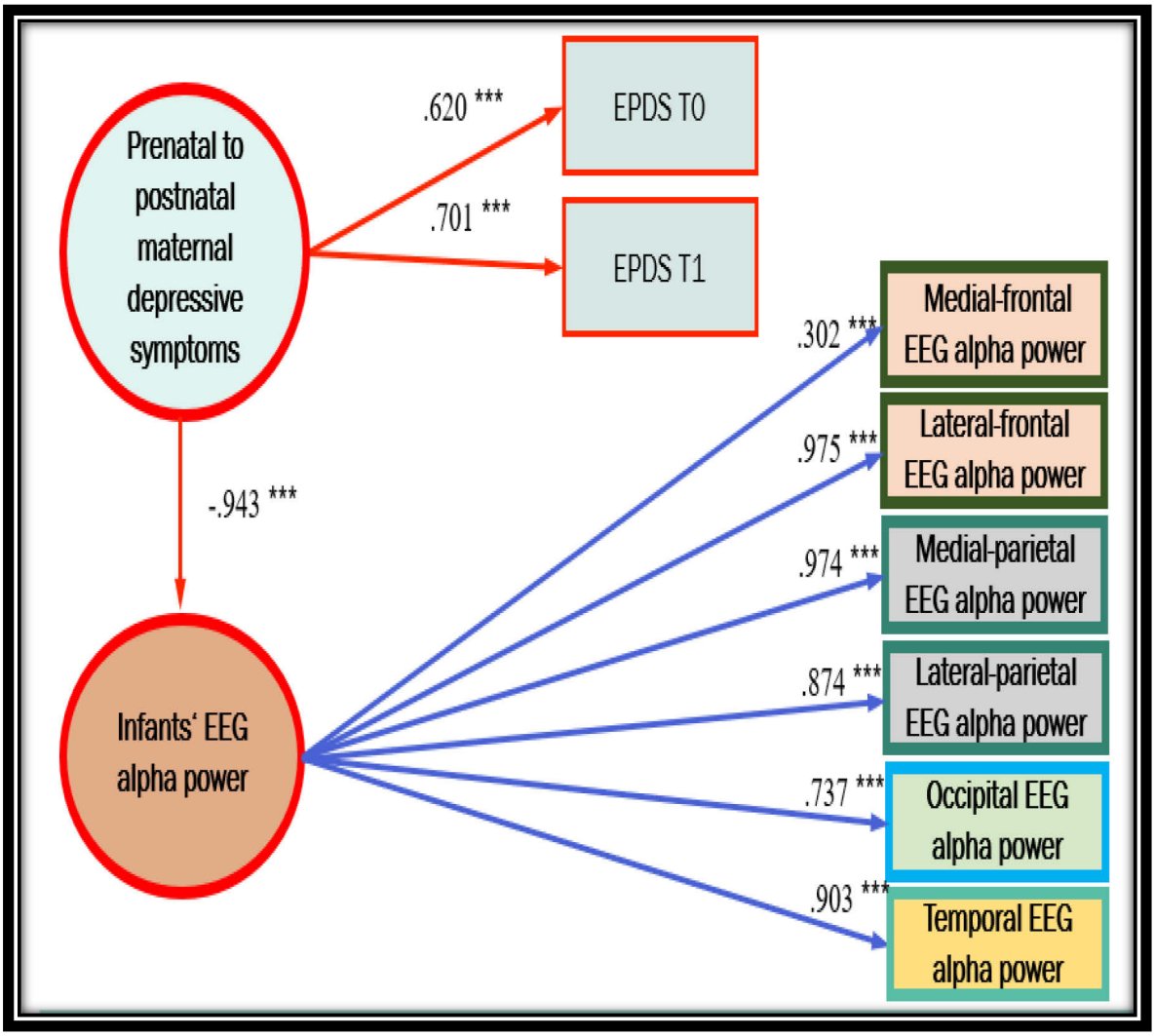
Note. In the two graphs, the vertical axis represents the infants' alpha power at lateral-parietal areas and the horizontal axis represents the average prenatal and postnatal maternal depressive score (T0 and T1) and maternal educational status, respectively. The increased alpha power is mostly observed in the infants of mothers with no or low depressive symptoms. The infants' alpha power tendency to be only slightly higher in the infants of mothers with low educational status is hardly to be seen at the graph.

SEM

Thirdly, for further analysis of the effects of prenatal to postnatal maternal depressive symptoms on infants' EEG alpha power, structural equation modeling (SEM) was conducted. Indexes were reported according to Marino et al., (2019) and Hu & Bentler (1995), reflecting different standards to judge model fit. The model provided a satisfactory, almost poor, model fit to the data ($\chi^2(3)$ = 115.796, p = .000; *RMSEA* = .187, *CFI* = .859; *TLI* = .792; N = 146) (Hu & Bentler, 1995; Marino et al., 2019). However, it seemed that the hypothesized model fit the data. The Chi-Square provided a significant value for evaluating the overall model fit which might be interpreted as "badness of fit" (Hu & Bentler, 1995). Nevertheless, RMSEA, as an absolute fit index, showed that parameter estimates managed to fit the populations covariance matrix, although the model is not perfect (Hu & Bentler, 1995). The incremental fit indices (CFI and TLI), compared on satisfactory level, showed the fit of this model with that of a baseline model (Hu & Bentler, 1995; Marino et al., 2019). The effect of the path from prenatal to postnatal maternal depressive symptoms to infants' EEG alpha power was significant (β = −.943; *SE* = .246; p = .000). Inspection of beta scores revealed that greater levels of prenatal to postnatal maternal depressive symptoms were associated with lower alpha power in infants. Hence, comparisons of beta scores demonstrated that prenatal to postnatal maternal depressive symptoms explained the low alpha power in their infants. Moreover, prenatal to postnatal maternal depressive symptoms predicted the lower alpha power over the whole brain, because the low alpha power was found in frontal, parietal, occipital and temporal brain regions of infants in relation to maternal depressive symptoms during and after pregnancy. *Figure 12.* shows the tested path diagram.

Figure 12

The impact of prenatal to postnatal maternal depressive symptoms on infants' EEG alpha power over the whole brain



3.2.2 The impact of prenatal to postnatal maternal depressive symptoms and educational status on infants' frontal and parietal alpha desynchronization

Hypothesis 2

To examine the second hypothesis, the impact of prenatal to postnatal maternal depressive symptoms and educational status on infants' frontal and parietal alpha desynchronization, firstly, series of correlations and secondly, series of regressions were performed. Additionally, a two-way ANOVA within a sub-sample of infants only rated with 6-very good and 5-good quality of EEG data was carried out to investigate the group differences and interactions within maternal depressive symptoms, educational status and infants' frontal and parietal alpha desynchronization. The ANOVA within a small sub-sample of infants was conducted as a contrast than the regression analyses within the whole sample as enabled grouping the infants according to maternal depressive symptoms, educational status and EEG data quality, and in this way, showed a different research design manner. In comparison with the investigation of the first hypothesis, in the second hypothesis no SEM was used because such model was not possible (no model fit was reached).

In general, infants in this study (N = 91) had positive or close to zero value for alpha desynchronization score, which meant that they exhibited slightly alpha desynchronization in the left hemisphere in comparison to the right. The alpha desynchronization values range from *min* = −.75 in the fronto-central electrodes (FC2-FC1) and *max* = .96 (P8-F7) at the lateral-parietal electrodes. At the fronto-central areas (FC2-FC1) was the alpha desynchronization score negative M = −.00, SD = .20 which

meant alpha desynchronization in the right hemisphere. The alpha desynchronization score at medial-frontal electrodes (F4-F3) was M = .04, SD = .19 and at lateral-frontal electrodes (F8-F7) was M = .04, SD = .20. The alpha desynchronization score at medial-parietal electrodes (P4-P3) was M = .03, SD = .24 and at lateral-parietal electrodes (P8-P7) was M = .15, SD = .27. The alpha desynchronization score at occipital electrodes (O2-O1) was M = .00, SD = .14. The alpha desynchronization score at fronto-central electrodes (FC6-FC5) was M = .05, SD = .22.

Firstly, maternal depressive symptoms, neither average score of prenatal and postnatal (T0 and T1) depressive symptoms, nor the depressive symptoms evaluated separately at T0 and T1, were significantly related to infants' parietal and frontal alpha desynchronization score. There was no statistically significant influence of maternal depressive symptoms at the prenatal (T0) and postnatal (T1) periods and educational status on infants' frontal and parietal alpha desynchronization. At the third measurement (T2) point, which was timewise the closest one to the infants' EEG assessment in comparison to the first two assessments, there were significant associations between maternal depressive symptoms and infants' parietal alpha desynchronization. Hence, the correlation analysis between maternal depressive symptoms during the second postpartum period (T2) and medial-parietal alpha desynchronization (P4-P3) revealed significance *(*r = .213, p = .048, N = 86). More specifically, higher postnatal maternal depressive symptoms were related to higher left-hemispheric parietal alpha desynchronization score.

As mentioned before, while investigating the *hypothesis 1*, there were also no significant effects of maternal depressive symptoms at the second postnatal period (T2) and maternal educational status on infants' EEG alpha power. At the second postnatal

period (T2), only maternal depressive symptoms and not maternal educational status influenced only infants' parietal alpha desynchronization.

Additionally, there was a negative relationship between depressive symptoms at the second postpartum period (T2) and maternal educational status *(r* = −.316, *p* = .002, *N* = 91). This relationship was in accordance with the negative correlation between the prenatal to postnatal maternal depressive symptoms at T0 and T1 and maternal educational status. This meant that increased prenatal to postnatal maternal depressive symptoms are associated with low educational status of the mother.

ANOVA with sub-sample within the group of infants

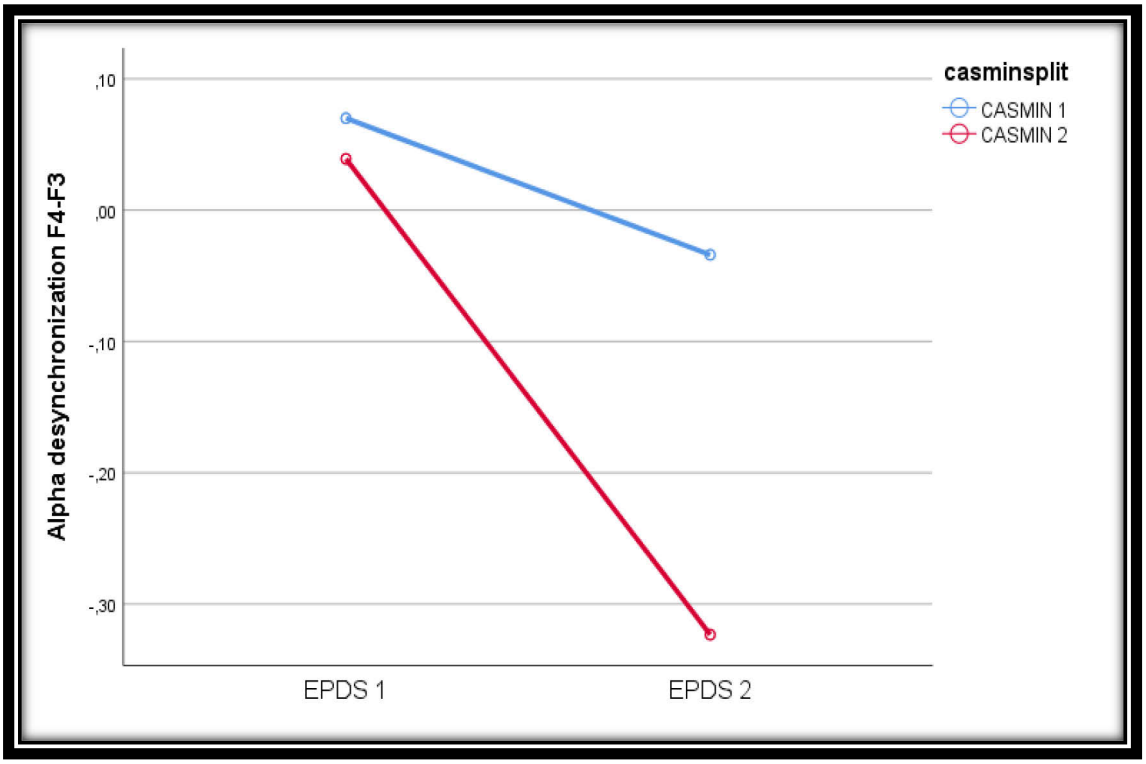
The analysis of the small sub-sample (*N* = 56) within the group of infants with EEG quality data rated with 6 and 5 was executed to investigate the group differences and interactions between prenatal to postnatal maternal depressive symptoms, maternal educational status and infants' frontal and parietal alpha desynchronization. The results revealed an increased difference between right and left medial-frontal alpha desynchronization (F4-F3) in infants within the group of mothers with elevated depressive symptoms whose mothers had higher educational status (*M* = −0.32, *SD* = 0.08) than in infants whose mothers had low educational status (*M* = −0.03, *SD* = 0.13). Both depressed groups exhibited right-hemispheric dominance of frontal alpha desynchronization. Within the non-depressed group, the group with low maternal educational status showed a tendency towards greater left-hemispheric dominance of frontal alpha desynchronization *(M* = 0.07, *SD* = 0.20) than the group with high maternal educational status *(M* = 0.03, *SD* = 0.16).

The two-way ANOVA model with frontal alpha desynchronization (F4-F3) showed

highly significant mean differences between the groups ($F_{(3)} = 5.166$, $p = .003$, $\eta_p^2 = .230$) with 23% proportion of the variance of frontal alpha desynchronization. The analysis revealed that frontal alpha desynchronization differed significantly, depending on maternal depressive symptoms ($F_{(1)} = 14.453$, $p < .000$, $\eta_p^2 = .217$), maternal educational status ($F_{(1)} = 6.813$, $p = .012$, $\eta_p^2 = .116$) and their interactions ($F_{(1)} = 4.437$, $p = .040$, $\eta_p^2 = .079$). Hence, prenatal to postnatal maternal depressive symptoms, maternal educational status and their interactions as well influenced infants' frontal alpha desynchronization and led to right-hemispheric dominance of frontal alpha desynchronization. It seemed that the alpha desynchronization value had the tendency to be more positive within the depressed group with low maternal educational status and more negative within the depressed group with high maternal educational status than in the non-depressed group. Infants whose mothers had increased depressive symptoms had negative alpha desynchronization value, indicating increased power in the right-hemisphere in comparison to the left. The group differences between maternal depressive symptoms and educational status for the infant's alpha desynchronization between the electrode pairs F4/3 (3-12 Hz) are displayed in *Figure 13*.

Figure 13

The estimated marginal means for the infants' alpha desynchronization between the electrode pairs F4/3 (3-12 Hz) and prenatal to postnatal maternal depressive symptoms and educational status

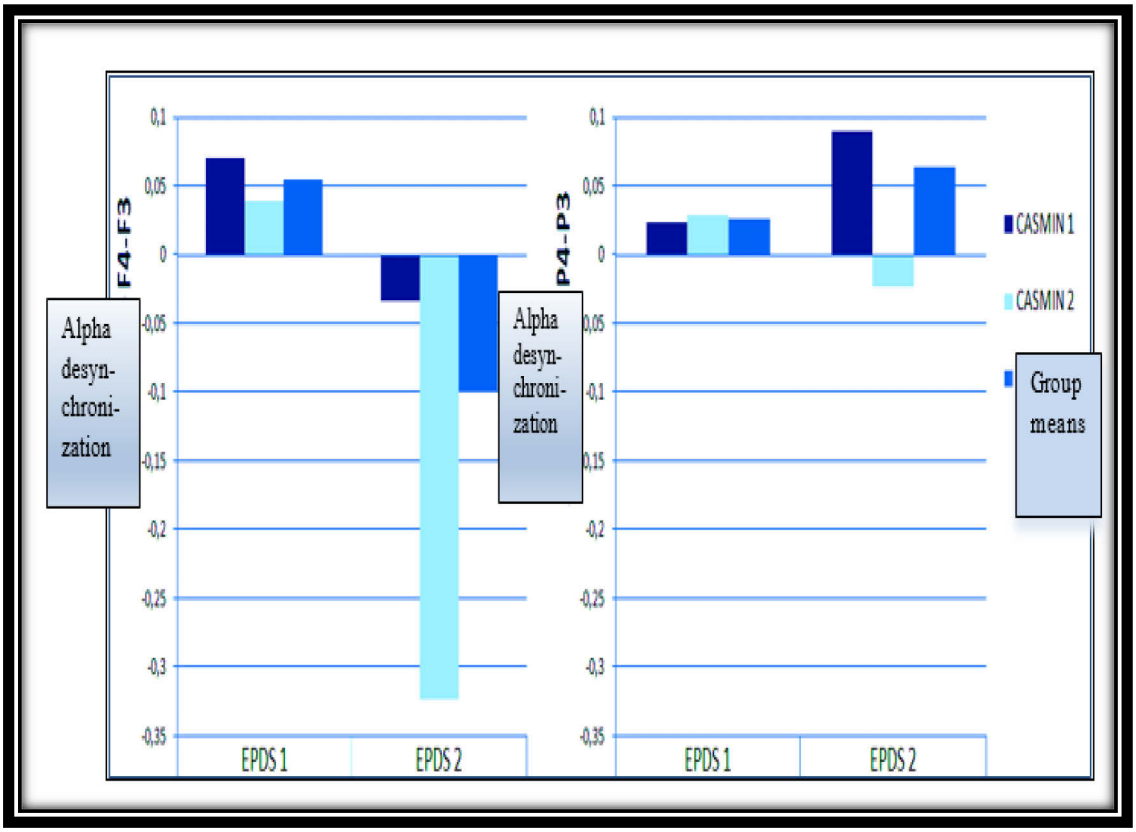


Note. EPDS 1 = non-depressed group, EPDS 2 = depressed group; CASMIN 1 = low educational status, CASMIN 2 = high educational status. The graph shows the tendency of the alpha desynchronization at medial-frontal areas (F4-F3) to be more positive within the depressed group with low maternal educational status and more negative within the depressed group with high maternal educational status.

The two-way ANOVA model with medial-parietal alpha desynchronization (P4-P3) revealed no group differences ($F_{(3)} = 0.191$, $p = .902$, $\eta_p^2 = .011$) and no interaction effect ($F_{(1)} = 0.343$, $p = .561$, $\eta_p^2 = .007$), neither with maternal educational status ($F_{(1)} = 0.293$, $p = .591$, $\eta_p^2 = .006$), nor with prenatal to postnatal maternal depressive symptoms ($F_{(1)} = 0.005$, $p = .942$, $\eta_p^2 < .000$). This meant that neither the depressive symptoms, nor the maternal educational level and their interactions influenced the medial-parietal alpha desynchronization in the sub-sample. The between group differences between the electrode pairs F4/3 and P4/3 (3-12 Hz) based on the group means of maternal depressive symptoms and maternal educational status are presented in *Figure 14.*

Figure 14

Group means comparison of the infants' alpha desynchronization between the electrode pairs F4/3 and P4/3 (3-12 Hz) and prenatal to postnatal maternal depressive symptoms and educational status



Note. EPDS 1 = non-depressed group, EPDS 2 = depressed group; CASMIN 1 = low educational status, CASMIN 2 = high educational status. The graphs represent that the depressed groups mostly with high maternal educational status exhibited right-hemispheric dominance of frontal alpha desynchronization at the medial-frontal areas (F4-F3) and at the medial-parietal areas (P4-P3), respectively. *SE* for EPDS 1 and CASMIN 1 = .037, *SE* for EPDS 1 and CASMIN 2 = .036, *SE* for EPDS 2 and CASMIN 1 = .053, *SE* for EPDS 2 and CASMIN 2 = .098.

3.3 Discussion: Study One

The aim of study one was to provide insights into the EEG patterns in infants of sub-clinically depressed mothers, and therefore, might show possible clues to the mother-child risk transmission and how maternal educational status might possibly affect them. As it was hypothesized above, the infants of prenatally and postnatally depressed mothers showed differences in their EEG patterns. In the study, infants of depressed mothers in prepartum (T0) and first postpartum (T1) periods demonstrated low alpha power over the whole brain, that was also additionally, related to maternal educational status. The maternal depressive symptoms at the second postpartum period (T2) were slightly associated with parietal alpha desynchronization presented mostly in the left hemisphere in infants. Furthermore, a right-hemispheric dominance of frontal alpha desynchronization was found within a sub-sample of infants of prenatally (T0) and postnatally (T1) depressed mothers. The findings support and extend the previous studies providing indications for the negative impact of depressive symptoms during and after pregnancy on infants' neurophysiology (Bruder et al., 2007; Diego, Field, Hernandez-Reif, et al., 2004; Field, Diego, Hernandez-Reif, et al., 2004; Kan & Lee, 2015; Lopez-Duran et al., 2012; Lusby et al., 2014; Tomarken et al., 2004; Wen et al., 2017). Even though in study one the two research hypotheses are partly confirmed, some of the results might be interpreted as controversial. For instance, according to the findings, prenatal to postnatal maternal depressive symptoms and low educational status seem to impact infants' alpha power and alpha desynchronization in different ways. As a further matter, in the overall sample the alpha desynchronization is presented mostly at the parietal regions in left hemisphere, whereas within the sub-sample the alpha desynchronization is observed on the frontal regions in right hemisphere. Future

investigation with larger sample is needed to clarify these issues and thus, makes these complex interactions easier to understand.

3.3.1 The impact of prenatal to postnatal maternal depressive symptoms and educational status on infants' EEG alpha power

Hypothesis 1

In the study, infants whose mothers reported depressive symptoms during the prepartum and postpartum periods had low EEG alpha power, especially in the frontal lobe where the alpha power was the lowest. Hence, low alpha power at the frontal and parietal lobes was related to prenatal to postnatal depressive symptoms. The findings suggested more explicitly that prenatal and postnatal maternal depressive symptoms predicted relative lower alpha power in infants. The path modeling revealed that infants of depressed mothers had reduced relative alpha power in the frontal, parietal, occipital and temporal brain regions. Thus, the effects of maternal depressive symptoms on infants' alpha power demonstrated several direct effects in the path model over the whole brain. Therefore, in the path model, indications towards associations between reduced alpha power in infants and prenatal to postnatal maternal depressive symptoms might be found. It can be argued that the prenatal to postnatal maternal depressive symptoms had a negative impact on infants' alpha power. It might be speculated that the adverse alpha power found in this study might be a predictor of alpha desynchronization, which might be later somehow shown but such suggestion is uncertain and might be further criticized.

Furthermore, the results of this dissertation reveal that the differences in infants' alpha power are not only observed in relation to maternal depressive symptoms but to

educational status as well. The infants of mothers with low educational status tended to have higher power in comparison to the infants of mothers with high educational status. Hence, low maternal educational status is associated with increases in infants' alpha power. On the contrary, prenatal to postnatal maternal depressive symptoms were related to decreases in alpha power. Besides, low maternal educational status influences the infants' alpha power in different manner than maternal depressive symptoms. Nevertheless, it should be noticed that alpha power was almost evenly distributed between the infants of mothers with low educational status and the infants of mothers with high educational status. Thus, a replication with larger sample size is required to provide more information about the direction of alpha power in infants of mothers with low educational status. According to the diathesis model by Davidson, other socio-economic factors might moderate the relationship between infants' EEG outcomes and maternal depressive symptoms. Other studies have also found evidence for the impact of maternal SES (Tomarken et al., 2004) and only educational status (Lopez-Duran et al., 2012), on maternal depression and offspring EEG outcome, mostly on offspring alpha desynchronization. Future research in the area is urgent and needs to account for the influencing role of maternal educational status.

The findings of this study are also consistent with previous studies that found a relationship between low alpha power and depression in adults (Gatt et al., 2008; Kan & Lee, 2015). Previous studies have observed that reduced resting EEG alpha power is related to maternal depression while a functional polymorphism of the brain-derived neurotrophic factor predicted depression via the mediating effects of alpha power (Gatt et al., 2008). Even though there are some studies which partly support the results of this study, it is hard to make conclusions about linking low alpha power of infants and prenatal and postnatal maternal depressive symptoms as there are almost no studies

within infants and there are some controversial and inconclusive reports from previous studies (Olbrich & Arns, 2013).

The findings from EEG studies about the differences between depressed and non-depressed participants in alpha and other frequency bands have been inconsistent. Previous studies reported enhanced alpha and theta power in association with depression (Olbrich et al., 2015), while others showed reduced alpha and theta in depressed adults (Roy et al., 2020), some studies did not find any differences at all (Korb et al., 2008; Newson & Thiagarajan, 2019). The elevations in theta power might be secondary related to alpha power, and related to a lack of cortical maturation (Klimesch, 1999). Several studies revealed that depression was related to an increase in the absolute power (Olbrich et al., 2015). However, these increases couldn't be observed when considering relative power where most studies failed to find any significant differences across any band (Korb et al., 2008; Newson & Thiagarajan, 2019).

Some studies have showed that power decreased across higher frequencies, such as alpha, beta and gamma, and increased across lower frequencies, such as delta and theta (Olbrich & Arns, 2013). Other studies have reported that at low frequencies the depressed adults showed lower alpha power and at higher frequency above 10 Hz the same group exhibited higher alpha power (O'Connor, Shaw, & Ongley, 1979). A possible suggestion might be that a resting-state condition, eyes-open paradigm, might not be the best to find oscillations differences especially in infancy. This assumption is postulated from other authors who examined oscillations in adults (Roy et al., 2020). Moreover, the differences are related to various brain regions (Gatt et al., 2008; Kan & Lee, 2015; Olbrich & Arns, 2013). It should be emphasized that there are almost no studies that investigate the infants' EEG alpha power in association with prenatal and

postnatal maternal depressive symptoms and maternal educational status, and future research should focus on filling this gap for better validation of this study findings.

In summary, this study indicates that both prenatal and postnatal maternal depressive symptoms and educational status influence infants' EEG alpha power. Across the entire cortex, infants' alpha power is reduced in relationship with maternal depressive symptoms, so low alpha power might be a neurophysiological marker of depression. Hence, the findings might provide a possible indication that the low alpha power in infants might be associated with prenatal and postnatal maternal depressive symptoms. Of course, this suggestion is speculative and future investigation is necessary to provide more information about the mechanisms behind low alpha power of infants in relation to maternal depressive symptoms and possible covariates such as maternal educational status. As EEG literature focused mostly on differences in the alpha power in the resting state condition across a depression (Olbrich et al., 2015), analyzing broad frequency bands in the EEG power spectrum delta, theta, alpha, beta, and gamma (Berger, 1929) should be recommended. Future studies may seek to interrogate, if infants' alpha power might be interrelated to maternal depressive symptoms and educational status.

3.3.2 The impact of prenatal to postnatal maternal depressive symptoms and educational status on infants' frontal and parietal alpha desynchronization

Hypothesis 2

As it was hypothesized before, the occurrence of maternal depressive symptoms in the prepartum and postpartum was associated with the alpha desynchronization in infants. In this study, the infants of depressed mothers in the second postpartum period (T2) demonstrated slightly parietal alpha desynchronization, presented in the left hemisphere. In the sub-sample, the right-hemispheric dominance of frontal alpha desynchronization was related to maternal educational status and prenatal to postnatal depressive symptoms. The last results are consistent with considerable amount of previous findings (Diego, Field, Hernandez-Reif, et al., 2004; Field, Diego, Hernandez-Reif, et al., 2004; Lopez-Duran et al., 2012; Lusby et al., 2014; Tomarken et al., 2004; Wen et al., 2017).

The impact of prenatal to postnatal maternal depressive symptoms and educational status on infants' frontal and parietal alpha desynchronization

No right- or left-hemispheric dominance of alpha desynchronization in the overall sample of infants was associated with maternal depressive symptoms and educational status at the prenatal period (T0) and the first postpartum period (T1). Hence, no interaction effect between maternal depressive symptoms and infants' EEG frontal or parietal alpha desynchronization during the first two periods was found. Therefore, the right-hemispheric dominance of alpha desynchronization has not been presented in the overall sampe of infants with maternal depressive symptoms at the prenatal and first

postnatal periods. A recent study has not found any link between right-hemispheric dominance of alpha desynchronization and depressive disorders in a multiverse analysis of five studies with sub-clinical sample as well (Kołodziej et al., 2021). Even though the authors have considered the probabilty that maybe there has been no relationship with depression and right-hemispheric dominance of alpha desynchronization, they explained their results with study limitations and anticipate the several previous reports which have been highlighted the right-hemispheric dominance of alpha desynchronization as marker of depression. Due to the small sample size and methodological constraints in measuring the alpha desynchronization leading to small effect size, it is possibly that the right-hemispheric dominance of alpha desynchronization is not observed (Kołodziej et al., 2021).

Some authors propose that right-hemispheric dominance of alpha desynchronization might be exhibited mostly by people with major depressive disorder (van der Vinne et al., 2017) and pose the question whether it might be observed within sub-clinical sample. Even though other authors claim that as a trait marker, right-hemispheric dominance of alpha desynchronization might also be presented without high depressive symptoms, within sub-clinical sample, especially in infancy (Henriques & Davidson, 1991). The participants of this study had sub-clinical mostly moderate levels of depressive symptoms.

Another possible explanation might be in accordance with the postulated diathesis model by Davidson. Thus, a right-hemispheric dominance of frontal alpha desynchronization might appear not only as a trait marker but also as a response to negative stimuli and in this way, as a condition might be related to negative affect and withdrawal behavior. According to the diathesis model by Davidson which was also considered in several studies, during resting EEG, left-hemispheric dominance of

frontal alpha desynchronization was associated with more positive affect and approach behavior, whereas relative right-hemispheric dominance of frontal alpha desynchronization was associated with more negative affect and withdrawal behavior (van der Vinne et al., 2017). Hence, alpha desynchronization might also result from an activation shift from approach behavior and positive affect or withdrawal behavior and negative affect of the mothers or infants (Diego et al., 2006) or when odors were presented (Fernandez et al., 2004). A shift in the alpha desynchronization from right to left might also be observed in depressed mothers who breastfeed their infants (Jones et al., 2004).

It seems that there are a different factors that could have an impact on the alpha desynchronization, especially in infancy when alpha desynchronization patterns show more variance (Field & Diego, 2008; Gotlib, 1998; van der Vinne et al., 2017). Possibly, there are other factors which influenced the infants' right-hemispheric dominance of frontal alpha desynchronization, such as the quality of EEG data, some situational factors during the EEG procedure e.g. the experimenter's characteristics and daytime (Magosso et al., 2019; Smith et al., 2017; Staudt, 2014) and some of them were not considered in this study. However, as right-hemispheric dominance of frontal alpha desynchronization makes a significant contribution to the prediction of depressive symptoms at an earlier stage, further investigation of the states and traits variance in EEG desynchronization is recommended.

The second postpartum period: T2

The participants in this study had positive or close to zero desynchronization values in relation to depressive symptoms emphasizing slightly a left-hemispheric dominance of

alpha desynchronization. Moreover, in the overall sample, only slightly alpha desynchronization at left parietal brain areas between the electrodes P4 and P3 was related to increased maternal depressive symptoms at the second postpartum period (T2). However, when alpha desynchronization is examined in infants without stable alpha desynchronization patterns, its relationship might reflect on left-hemispheric alpha desynchronization (van der Vinne et al., 2017). A possible explanation why this relationship appeared firstly during the second postpartum period might be that this period of the assessment of depressive symptoms was the closest to the EEG session. The maternal depressive symptoms at T2 were assessed in the time from 6- to 8-months after birth and the infants' EEG session took also place, when the infants were from 6-to-8-month-old, usually 2 weeks after the assessment of maternal postnatal depressive symptoms. It seems that maternal depressive symptoms should be investigated over a longer period of time to enable an observation of infants' right-hemispheric dominance of frontal alpha desynchronization. It should be noticed that a relationship between postnatal maternal depressive symptoms and infants' alpha desynchronization is found only at the parietal region and was not found at the frontal areas where alpha desynchronization is usually postulated by Davidson model to be presented. According to previous studies, many brain regions are related to depression and in this way, an EEG hemispheric dysfunctional patterns are found in frontal and limbic areas, such as dorsolateral prefrontal cortex (DLPFC), medial prefrontal cortex (mPFC), anterior cingulate cortex (ACC) and orbitofrontal cortex (OFC) (Gotlib, 1998; Korb et al., 2008). Davidson claims that the right-hemispheric dominance of frontal alpha desynchronization is related to depression, negative affect and withdrawal behavior (Henriques & Davidson, 1991). However, when the right-hemispheric dominance of frontal alpha desynchronization is investigated within the group of infants without

extreme depressive symptoms or stable alpha desynchronization, its relationship with depression is observed to weaken or patterns of dissonance might be related to left-hemispheric dominance of frontal alpha desynchronization (van der Vinne et al., 2017). Additionally, according to the model of Heller and Nitschke (1997), alpha desynchronization might be presented at the parietal regions between the electrodes P4 and P3 in relation to depressive symptoms. Hence, the aforementioned interpretations might be possible reasons why left-hemispheric dominance of parietal alpha desynchronization was found within the group of study participants. Moreover, the model by Heller and Nitschke (1997) points out the importance of the impact of depression and negative affect on parietal cortex region for alpha desynchronization. Generally, the model by Heller und Nitschke postulates the same assumptions as Davidson and Tomarken's model (1989), but reflects mostly on the parietal cortex region. It should be noticed that while frontal regions are used to test predictions from the Davidson and Tomaken model, the investigation of parietal regions as predicted by Heller and Nitschke model is usually only used to prove the specificity of frontal alpha desynchronization.

Moreover, the mechanisms behind the EEG alpha desynchronization might be limited not only to frontal regions but to parietal as well and in this way, a better prediction of the depressive symptoms over time might be possible. The findings of this dissertation are consistent with previous research on parietal alpha desynchronization (Bruder et al., 2005, 2007). Previous studies also reported that left-hemispheric dominance of alpha desynchronization was found in the parietal brain regions in children of parents and even grandparents with depression (Bruder et al., 2005, 2007). These studies highlighted the risk transmission, not only of parents to children, but of grandparents as well. Another study within a community sample provided an evidence for parietal alpha

desynchronization in 6-month-old infants of depressed mothers, indicating that changes in alpha desynchronization due to maternal depression may be not restricted to the frontal lobe (Marino et al., 2019). Hence, the findings of this dissertation that infants' hemispheric differences might be also observed at parietal regions in relation to maternal depression are consistent with previous studies revealing similar outcomes (Bruder et al., 2005, 2007).

An increased left-hemispheric dominance of frontal alpha desynchronization might predict behavior and emotional disturbances (Smith & Bell, 2010). Moreover, left-hemispheric dominance of frontal alpha desynchronization might be related to externalizing disorder, such as hyper-activity, impulsivity and aggressive behavior, which might be later associated with anger and diminished control approach behaviors (Davidson & Fox, 1989; Smith & Bell, 2010). It can be speculated that these findings might be transferred to left-hemispheric dominance of parietal alpha desynchronization as it can be argued that there are similar mechanisms behind alpha desynchronization in frontal and parietal areas.

The impact of prenatal to postnatal maternal depressive symptoms and educational status on infants' alpha desynchronization with sub-sample

As according to the diathesis model by Davidson, different factors might have an impact on the occurrence of a right-hemispheric dominance of frontal alpha desynchronization and it is possible that the quality of EEG data might be a reason for the right-hemispheric dominance of frontal alpha desynchronization to be observed or not (Magosso et al., 2019; Smith et al., 2017; Staudt, 2014). Hence, an analysis with the small sub-sample of infants with 6-very good and 5-good EEG data quality was conducted.

Infants in this sub-sample whose mothers reported more depressive symptoms had negative alpha desynchronization value at the frontal lobe, which meant a right-hemispheric dominance of frontal alpha desynchronization (Smith et al., 2017). The findings that the infants in the small sub-sample of depressed mothers exhibited a right-hemispheric dominance of frontal alpha desynchronization are in accordance with Davidson's model and previous results that reported the same findings (Diego, Field, Hernandez-Reif, et al., 2004; Field, Diego, Hernandez-Reif, et al., 2004; Lopez-Duran et al., 2012; Lusby et al., 2014; Tomarken et al., 2004; Wen et al., 2017). Moreover, several previous studies have been accentuated the relationship between maternal depressive symptoms during and after pregnancy on infants' right-hemispheric dominance of frontal alpha desynchronization (Field et al., 2006; Field, Diego, & Hernandez-Reif, 2004).

Furthermore, in the analysis with the small sub-sample, the right-hemispheric dominance of frontal alpha desynchronization was related not only to maternal depressive symptoms but to educational status as well. The maternal educational status influenced the right-hemispheric dominance of frontal alpha desynchronization. The infants whose mothers had high educational status showed right-hemispheric dominance of frontal alpha desynchronization, while the infants whose mothers had low educational status showed almost no difference and nearly a synchronization or symmetrical alpha activity. Thus, the low maternal educational status seems to impact the alpha desynchronization in the opposite way than maternal depressive symptoms and lead mostly to left-hemispheric dominance of frontal alpha desynchronization. These controversial results should be cautiously interpreted as the study sample size was very small and the participants were not equally distributed between the groups. However, the findings are in accordance with the assumption of

diathesis model by Davidson that other factors such as SES might moderate the infants' alpha desynchronization of depressed mothers. Other studies also found evidence for the impact of maternal SES (Tomarken et al., 2004) and educational status (Lopez-Duran et al., 2012) on offspring's right-hemispheric dominance of frontal alpha desynchronization. It seemed that not only maternal depressive symptoms but educational status as well might modified infants' EEG alpha desynchronization. As there are just few studies investigated the impact of both maternal depressive symptoms and educational status on infants' alpha desynchronization, it is hard to make conclusion about the direction of the alpha desynchronization, such as left- or right-hemispheric activity, in accordance with the modifing role of educational status.

In the small sub-sample, no group differences were found at the parietal brain regions. Moreover, the right-hemispheric dominance of parietal alpha desynchronization pattern didn't differ, neither based on maternal depressive symptoms, nor on maternal educational status. These findings are in accordance with some previous study which found right-hemispheric dominance of frontal alpha desynchronization in children of depressed mothers, but did not find any parietal alpha desynchronization patterns (Buss et al., 2003; Diego et al., 2010).

It should be emphasized once again that the findings revealed that right-hemispheric dominance of frontal alpha desynchronization was found only in the sub-sample of the infants with the data sets characterized with 6-very good and 5-good data quality. It can be argued that the level of quality and the epochs, such as the proportion of artifacts, SNR and the usable segments, possibly influenced the results. As conducting EEG with infants is more challenging than adults and related to more data loss (van der Velde & Junge, 2020), concentrating on the EEG data quality and performing additional analysis within a sub-sample are recommended (Magosso et al.,

2019; Smith et al., 2017; Staudt, 2014) and in accordance with the postulation of Davidson model. As stated in the diathesis model by Davidson, the quality of EEG data affected the detection of the alpha desynchronization (Magosso et al., 2019; Smith et al., 2017; Staudt, 2014) and therefore, the right-hemispheric dominance of alpha desynchronization might be easier to obtain. Even though the small sample size of this sub-sample analysis and the methodological limitations in defining precisely the EEG data quality criteria, the result revealed the occurrence of the right-hemispheric dominace of frontal alpha desynchronization in infants of sub-clinically depressed mothers.

Previous research showed that prenatal and postnatal maternal depressive symptoms have a negative impact on neurophysiology and cognitive abilities throughout infancy and during the development (Kinsella & Monk, 2009; O'Connor et al., 2014; O'Donnell et al., 2009; Van Batenburg-Eddes et al., 2013; Van den Bergh et al., 2017; Van den Bergh et al., 2018). Infants of mothers who experienced depressive symptoms during prenatal and postnatal periods often have difficulties adapting to new stimuli or stress, and are more likely to show reduced motor skills deficits and abilities in regulating their attention, behavior and emotions (Van den Bergh et al., 2017; Van den Bergh et al., 2018). Hence, easy-to-obtain markers predicting the aforementioned impairments and monitoring developmental trajectories during the prodromal stage might help implementing effective diagnostic programs. Moreover, right-hemispheric dominance of frontal alpha desynchronization and eventually parietal appears to be a marker of vulnerability for depression (Diego et al., 2006; Tomarken et al., 2004). Other studies have linked right-hemispheric dominance of frontal alpha desynchronization to emotional and cognitive control processing disabilities (Diego et al., 2006; Diego, Field, Jones, et al., 2004; Hernandez-Reif et al., 2006) and temperamental negative affectivity

(Lusby et al., 2016). Hence, infants of mothers with depressive symptoms indicate protracted neural development (Talge et al., 2007; Van den Bergh et al, 2005) and neurophysiological changes resembling pathological findings in depressed adults (Glover, 2014; O'Connor et al., 2014; O'Donnell et al., 2009).

Gender differences on EEG alpha desynchronization patterns of infants of depressed mothers

In this study, any gender differences in infants' alpha desynchronization were not found. Some studies revealed potential gender differences on EEG patterns of offspring of depressed mothers (Hernandez-Reif et al., 2006; Mennes et al., 2009;). One study reported right-hemispheric dominance of frontal alpha desynchronization only for female newborns (Hernandez-Reif et al., 2006). It seems that girls might be stronger affected from maternal depression than boys and through this under higher risk for developing behavior problems and a mood disorder later in life (Bress et al., 2013; Hernandez-Reif et al., 2006). As a consequence, the impact of maternal depression on infants' neurophysiology may relate to genetic dispositions in comparison with all other aspects and factors.

Other methods

Other methods such as magnetic resonance imaging (MRI), or combinations of various measurement tools should also be used to investigate the neurophysiological markers of depression during development and, thereby, the mother-to-child transmission risk. Magnetoencephalography (MEG) can also be implemented, it is a technique that provides a better signal for localization but it is much more expensive and technically more challenging than EEG (Sanjuan et al., 2016). For example, Sanjuan and colleagues

(2016) have conducted a MEG study with 6-month-old infants and showed delayed cortical maturation in infants due to perinatal post-traumatic stress disorder (PTSD). They provided an evidence for the neurophysiological consequences from PTSD of infants' abilities because the resting left anterior temporal/frontal theta power was associated with PTSD.

An integrated MRI and EEG follow-up study has interrogated three generations to find biomarkers for depression and demonstrated that offspring with two generations previously affected were at the greatest risk to develop depression (Talati et al., 2013). Thinning of the cortical mantle (MRI) and reduced resting-state activity (EEG) within the right parieto-temporal hemisphere differentiated high- from low-risk offspring, regardless of whether the offspring had depression, suggesting that these measures might serve as familial trait markers for depression (Talati et al., 2013).

Another possibility is the combination of EEG and Functional Near-Infrared Spectroscopy (fNIRS) methods, which enables researchers more easily to uncover the functioning of cortical mechanisms of infants because it requires less attention involvement (Grossmann & Johnson, 2007). Hence, other methods and a combination of different kinds of techniques might be very effective and should be further investigated, even though there is a longstanding tradition in the EEG use. This dissertation emphasizes that EEG has been established as a good method for supporting researchers to develop a better comprehension of the neural mechanisms behind depression and helping to find neurophysiological markers of depression.

Summary and future research directions

In study one, the results reveal significant associations between prenatal and postnatal maternal depressive symptoms and changes in alpha power and alpha desynchronization

patterns in infants. The infants of depressed mothers showed lower alpha power over the whole brain in comparison to the infants of non-depressed mothers. The relation between the infants' alpha desynchronization and maternal depressive symptoms was observed firstly in the second postpartum period in the overall sample. Additionally, among the small sub-sample of infants of depressed mothers, a right-hemispheric dominance of frontal alpha desynchronization was found. The findings about the precise impactful role of maternal educational status are inconclusive.

Prenatal to postnatal permanence of maternal depressive symptoms are associated with adverse alpha power and alpha desynchronization patterns in infants, which are also under higher risk to experience cognitive and emotional impairments and to develop depression (Field, 2017). When considering transmission pathways, the right-hemispheric dominance of frontal alpha desynchronization as a brain activity pattern should be highlighted, as it is found in both people with depression and in infants of depressed women (Field, 2017).

Nevertheless, the right-hemispheric dominance of frontal alpha desynchronization is more variable with age and is co-influenced by different factors (Diego et al., 2006; Tomarken et al., 2004). Hence, there are possibly moderators, such as the infants' characteristics, the timing of maternal depression and the role of father, interacting with the transmission mechanisms. Therefore, other psychosocial and physiological factors should be further investigated. Field and colleagues (2004), for instance, observed an association between maternal and child alpha desynchronizations patterns and maternal depressive symptoms. Other factors modifying the interactions between maternal depressive symptoms and infants' alpha desynchronization, together with other methods and biomarkers, such as elevated salvia cortisol, lower vagal tone and maternal EEG patterns, should be involved in future research.

4 CHAPTER FOUR: STUDY TWO

Study two aimed to increase the understanding of the interactions between maternal depressive symptoms, proximal and distal risk and protective factors in pregnant women. Study two investigated on the first place, the change of depressive symptoms over time, on the second place, the influence of different protective factors, such as overall social and partner social support, on prenatal and postnatal maternal depressive symptoms and on the third place, the influence of stressful life events and maternal educational status on prenatal and postnatal maternal depressive symptoms. In the following paragraphs, the methods and results of study two will be described in details. The findings of study two will be precisely summarized and will be argued in the discussion section of study two.

4.1 Methods: Study two

To examine the hypotheses in study two, the interactions between prenatal and postnatal maternal depressive symptoms, stressful life events and resources, firstly, series of correlation and secondly, series of regression analyses were conducted.

4.1.1 Pearson Correlation analysis

Pearson's correlation analysis (two-tailed) was used to assess the relationships between the study variables. Separately, the scores of prenatal maternal depressive symptoms (T0), postnatal maternal depressive symptoms (T1) and postnatal maternal depressive

symptoms (T2) were correlated with the scores of overall social and partner social support.

4.1.2 ANOVA

One-way repeated measures analysis of variance (ANOVA) was conducted to examine the change and deviation of maternal depressive symptoms in prenatal and postnatal periods within the same group of participants. After the assumptions, such as the significance of Maulchly's test of Sphericity and Wilks´ Lambda, were confirmed, pair-wise test to make post hoc comparisons between conditions were used for each paired analysis as a comparison between the prenatal EPDS score (T0), the postnatal EPDS score (T1) and the postnatal EPDS score (T2).

4.1.3 Regression analysis

In study two the interactions between prenatal (T0) and postnatal maternal depressive symptoms (T1 or T2) and different resources (such as overall social and partner social support), and stressful life events were investigated. After confirming the above-mentioned requirements, two series of regressions were performed within the entire sample. For the first one, the influence of overall social and partner social support on prenatal and postnatal maternal depressive symptoms were examined with three regression analyses. For this purpose, maternal depressive symptoms (prenatal (T0) or postnatal (T1) or postnatal (T2) maternal depressive scores) were separately entered as dependent variable and overall social and partner social support score were entered as independent variables. For the second series of regression analyses, separate regression models (three regressions) with depressive symptoms at the three different time points

(prenatal (T0) or postnatal (T1) or postnatal (T2) maternal depressive scores) as a dependent variable and stressful life events score as an independent variable were conducted. Thus, the role of stressful life events as a predictor of prenatal or postnatal maternal depressive symptoms were interrogated. The covariates were included in the first series of regression analyses only if significantly correlated with prenatal and postnatal maternal depressive symptoms and overall social and partner social support and in the second series of regression analyses only if significantly correlated with prenatal and postnatal maternal depressive symptoms and stressful life events. Hence, in the second serie of regression analyses, maternal educational status was included as a covariate. In both analyses, the influence of maternal age as a possible confounder was taken into account but the relationship turned out to be insignificant.

4.2 Results: Study Two

Study two investigated firstly, the change of depressive symptoms over time, secondly, the impact of different protective factors, such as overall social and partner social support, on prenatal and postnatal maternal depressive symptoms and thirdly, the impact of stressful life events and maternal educational status on prenatal and postnatal maternal depressive symptoms.

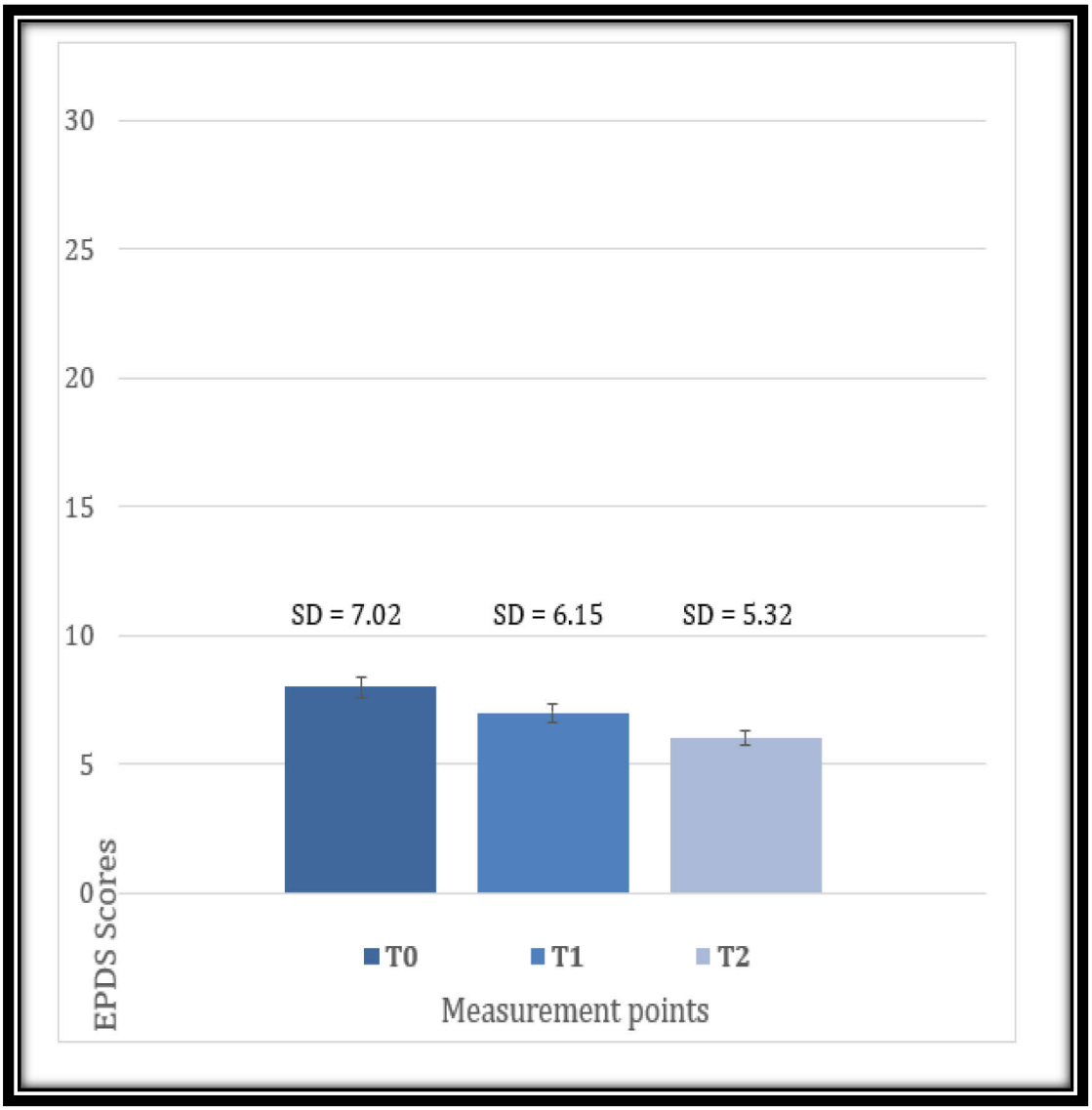
4.2.1 The differences between prenatal and postnatal maternal depressive symptoms

Hypothesis 3

Depressive symptoms in the study changed over time *(Figure 15)*. The women during the prenatal period reported higher levels of depressive symptoms (M = 8.09, SD = 7.02; Min = 0 and Max = 29, N = 68) than women during the first postpartum period (M = 6.89, SD = 6.15; Min = 0 and Max = 24, N = 68). The female participants during the second postpartum period (T2) experienced lower levels of depressive symptoms (M = 6.07, SD = 5.32; Min = 0 and Max = 26, N = 68) in comparison with the first postpartum period and with the prenatal period. The deviation over time in depressive symptoms was significant, Wilks´ Lambda = .889, F (2,66) = 4.102, p = .021, N = 68. The pairwise comparisons indicated that there was not a significance difference between the depressive symptoms in the prenatal (T0) and the first postnatal period (T1) (SE = .710, p = .288). On the other hand, depressive symptoms in the second postpartum period (T2) differed significantly in comparison with the prenatal period (T0) (SE = .703, p = .016) but didn't differ significantly in comparison with the first postpartum period (SE = .635, p = .584).

Figure 15

The diagram representing the change of depressive symptoms over time by T0, T1 and T2



Note. Women at T0 have higher levels of depressive symptoms (M = 8.09, SD = 7.02) than women at T1 (M = 6.89, SD = 6.15) and at T2 (M = 6.07, SD = 5.32), N = 68.

4.2.2 The impact of distal protective factors on prenatal and postnatal maternal depressive symptoms

Hypothesis 4

Hypothesis 4 investigated the interactions between prenatal and postnatal maternal depressive symptoms and distal protective factors. *Table 4* presents the correlations analyses between prenatal and postnatal maternal depressive symptoms and protective factors.

Table 4

Pearson correlations for Study Variables: The interactions between prenatal and postnatal maternal depressive symptoms and protective factors

Variable	*N*	*M*	*SD*	1	2	3	4	5
1. Depressive symptoms (T0)	77	8.23	6.82	-				
2. Depressive symptoms (T1)	140	7.06	5.82	-.600** .000	-			
3. Depressive symptoms (T2)	129	6.03	5.30	.592** .000	.618** .000	-		
4. Overall social support	126	57.30	13.96	-.420** .000	-.493** .000	-.345** .000	-	
5. Partner social support	122	5.16	1.18	−.318** .000	-.247* .008	-.257* .008	.357** .000	-

Note. $^{*}p < .05$. $^{**}p < .01$.

In the first place, increased prenatal (T0), first and second postnatal maternal depressive symptoms (T1 and T2) were associated with less maternal resources. Prenatal maternal depressive symptoms were significantly negatively correlated with overall social support and partner social support received by mothers. Postnatal maternal depressive symptoms (T1) were also negatively related to overall social support and partner social support. At the the second postpartum period, negative relationships between postnatal maternal depressive symptoms (T2) and overall social support and partner social support were again observed. Hence, the more depressed the mothers were, the less overall social and partner support they received and the less support they had, the higher the depressive symptoms were.

Regression analysis

After conducting correlation analyses, separate linear regression models were performed to examine the impact of overall social support and partner social support on prenatal (T0) and postnatal (T1 and T2) maternal depressive scores without the influence of any covariates *(Table 5)*. The maternal educational status was correlated neither with overall social support, nor with partner social support and it was excluded from the analysis.

The lack of overall social support significantly predicted the occurrence of maternal depressive symptoms during the prenatal, first postnatal and second postnatal periods *(Table 5)*. Overall social support also explained a significant proportion of variance in prenatal and postnatal maternal depressive symptoms *(Table 5)*. There was no significant influence of the partner social support on maternal depressive symptoms during the prenatal (T0), first postnatal (T1) periods. The predictive role of partner social support on maternal depressive symptoms was firstly shown at T2. The lack of

partner support at the prenatal and postnatal periods significantly predicted the occurrence of depressive symptoms firstly during the second postpartum period *(Table 5)*.

Table 5

Linear Regression Analysis: The effects of overall social support and partner social support on prenatal (T0) and postnatal maternal depressive symptoms (T1 and T2)

Dependent variable	Depressive Symptoms, T0, N = 75			
Predictors	ΔR^2	SE	ß	*P*
Overall social support		.056	-.332	.008*
	.194			
Partner social support		.665	-.188	.124
	Depressive Symptoms, T1, N = 115			
Overall social support		.034	-.517	.00**
	.323			
Partner social support		.405	-.139	.092
	Depressive Symptoms, T2, N = 112			
Overall social support		.038	− .336	.00**
	.193			
Partner social support		.438	− .204	.031*

Note. $^{*}p < .05$. $^{**}p < .01$.

In line with the presented above regression models, there were high significant contributions from protective factors for the mothers to prenatal and postnatal maternal depressive symptoms. The results showed that increased prenatal and postnatal depressive symptoms were associated with less overall social and partner social support.

4.2.3 The impact of stressful life events and maternal educational status on prenatal and postnatal maternal depressive symptoms

Hypothesis 5

To investigate the influence of occurrence of stressful life events on maternal depressive symptoms at the prenatal (T0), first postnatal (T1) and second postnatal (T2) periods, linear regression analyses were conducted. Maternal educational status was included in the analysis as a covariate, because it was significantly correlated with all study variables. In the *chapter two (methods: constructs)* were described the Pearson's correlations between the stressful life events and the prenatal (r = .379, p < .001, N = 75) and postnatal (r = .259, p < .005, N = 114) maternal depressive symptoms, respectively which were performed to investigate the concurrent validity for the used constructs.

The conducted three regressions analyses showed the predictive role of stressful life events only on prenatal depressive symptoms at T0 but did not on postnatal depressive symptoms at T1 and T2 *(Table 6)*. The occurrence of stressful life events predicted significantly the occurrence of depressive symptoms during the prenatal period *(β* = .379, p = 0.001, N = 75). Different stressors, such as divorce, job loss and etc., also

explained a significant proportion of variance in prenatal maternal depressive symptoms *(Table 6)*. Inspection of the regression coefficients scores revealed that increased levels of prenatal depressive symptoms were associated with both low educational status and more occurrence of stressful events in mothers' life, which, in turn, predicted depressive symptoms *(Table 6)*. Maternal educational status predicted increased depressive symptoms in both prenatal and first postnatal periods.

Table 6

Linear Regression Analysis: The effects of stressful life events and maternal educational status on prenatal and postnatal maternal depressive symptoms (T0, T1, T2)

Dependent variable		Depressive Symptoms, T0, N = 75		
Predictors	ΔR^2	SE	ß	*P*
Stressful life events		.138	301	.031*
	.248			
Maternal education		.371	-.309	.027*
		Depressive Symptoms, T1, N = 109		
Stressful life events		.109	.013	.907
	.056			
Maternal education		.252	-.232	.041*
		Depressive Symptoms, T2, N = 110		
Stressful life events		.098	.064	.574
	.093			
Maternal education		.230	-.203	.074

Note. $^{*}p < .05$. $^{**}p < .01$.

4.3 Discussion: Study two

The aim of study two was to provide insights into the interactions between maternal depressive symptoms, proximal and distal risk and protective factors during the prepartum and postpartum. In Study two the three research hypotheses are supported. Firstly, there was a significant difference between the prenatal and postnatal depressive symptoms as it was supposed in hypothesis 3. Secondly, as it was hypothesized in hypothesis 4, the lack of maternal resources such as overall social support was related to maternal depressive symptoms in both preparturm and postpartum. Thirdly, in accordance with the hypothesis 5, the occurrence of stressful life events and low maternal educational status predicted the occurrence of depressive symptoms during pregnancy.

The interactions between maternal depressive symptoms, resources and stressful life events during and after pregnancy seemed very perplex. The findings of the dissertation have supported the previous evidence on the negative impact of less social support on prenatal and postnatal depressive symptoms, and the negative impact of more stressors on the occurrence of depressive symptoms during and after pregnancy (Elsenbruch et al., 2007; Glazier et al., 2004; Gómez-López et al., 2019). The results are also theoretically consistent with the notion that women from socially and culturally disadvantaged background, living in diverse stressful contexts are more vulnerable to develop depressive symptoms due to proximal and distal risk factors during transition to motherhood. Focusing on bio-psycho-social aspects, this dissertation has been established links between increased internalizing symptoms among pregnant women and stressful life events, and emphasized the protective role of social support, but the complexity of the mechanisms that potentially explain these links remain not

completely uderstood. Future research needs to account for this complexity.

4.3.1 The differences between prenatal and postnatal maternal depressive symptoms

Hypothesis 3

Maternal depressive symptoms decreased after birth, in the first postpartum period and continued to decrease in the second postpartum period, when the depressive symptoms were the lowest. In the study, depressive symptoms during pregnancy seemed to be a predictor of depressive symptoms in the postpartum period as well. The difference between women's prenatal and postnatal depressive symptoms might be explained with the hormone prolactin and the different worries which women seem to have during these periods (Li et al., 2017; Stuebe et al., 2012). The release of the hormone prolactin which is responisible for milk synthesis might be a reason for the mothers to feel less depressed in postpartum as prolactin has been linked to maternal mood (Stuebe et al., 2012). However, in this study the maternal level of prolactin was not measured.

It seems that women worried more during pregnancy than during postpartum. According to Li et al., 2017, during prepartum, women distressed about their own health conditions, baby's health, birth and possible complications. During postpartum, women distressed about sleep, cry and nutrition their babies and if they are caring well for the infants (Li et al., 2017). In this study, the participants who experienced depressive symptoms during the prenatal period continued to show depressive symptoms in both postnatal periods at 3 months and at 7 months after child birth. Moreover, the women who reported higher depressive symptoms levels during pregnancy, remained depressed

during the first and second postpartum periods. Hence, in the study, prenatal depressive symptoms seem to be a predictor of postnatal depressive symptoms at 3 months and at 7 months after birth. This is consistent with the results of one previous study (Da Costa et al., 2000). The previous study highlighted the consonant connection between prenatal and postnatal depressive mood but pointed out the methodological limitations, for instance, a small sample size and different instruments being used to assess prenatal and postnatal depressive symptoms (Da Costa et al., 2000).

Being mother for the first time might be also very challenging for some women and might cause the development of depressive symptoms during prepartum and postpartum (Prinds et al., 2018). After controlling if the first-time mothers might experience more depressive symptoms, the results revealed non-significant effect. Hence, in this dissertation, being first time mother was not associated with the occurrence of depressive symptoms, possibly, because less than a half of the participants (37%) were expecting their first baby.

To examine the depressive symptoms at different periods and its relatioships with stressors and resources, and socio-economic factors, it is important to better understand the dynamical mechanisms of depressive symptoms during prepartum and postparum. Concurrently, there are less studies focusing on investigating the dynamic of depressive symptoms, most of the studies are concentrating on a single time period and it is unclear whether the effect is stronger during the pregnancy or during the postnatal period (Li et al., 2017). Obscure is also the impact of other psychosocial and biological influencing factors on the depressive symptoms over time. Some authors argued that prenatal depressive mood might be mostly influenced by biological factors and postnatal depressive mood might be affected by psychosocial factors or might be interpreted as a continuum of depressive symptoms during prepartum (Da Costa et al., 1999, 2000;

Glazier et al., 2004). This dissertation claims that stressful life events, resources and some socio-economic characteristics such as maternal educational status influence the dynamics of depressive symptoms in a complex way, while increase or decrease affective mood variability.

4.3.2 The impact of distal protective factors on prenatal and postnatal maternal depressive symptoms

Hypothesis 4

The results of the study have revealed that women during and after pregnancy might be more vulnerable to depressive symptoms when there is a lack of overall social support and partner social support. Moreover, the provision of social support has reduced depressive symptoms during the prepartum and postpartum. This findings are consistent with other reports which also emphasize the protective role of social support on women during the prenatal and postnatal periods (Glazier et al., 2004). In this dissertation, it was distinguished between overall social support provided from partner, relatives and friends and partner social support provided only from partner. Thus, it was found that partner social support seemed to have an influence on maternal depressive symptoms only in postpartum while the overall social support during both prepartum and postpartum. Moreover, women who reported that they received less partner social support already during pregnancy experienced more depressive symptoms during the second postpartum period. Therefore, in the study, the lack of partner social support had a negative impact on maternal postnatal depressive symptoms. More specifically, women who were less supported from their partners experienced depressive symptoms

mostly in postpartum. These findings are consistent with previous research which demonstrated that the lack of partner social support has been highlighted as a risk factor in women during and mostly after pregnancy (Martin et al., 2013; Nakamura et al., 2020). Hence, the social support provided from partners is an important resource for women in time of needs.

The absence of overall social support during and after pregnancy predicted the occurrence of depressive symptoms in the sample over time. Therefore, in the study within a longitudinal design, it has been shown that less overall social support was related to depressive symptoms at the prenatal period (T0), the first postpartum (T1) and the second postpartum period (T2). Furthermore, women who received more overall social support reported fewer depressive symptoms over time. Hence, participants who received insufficient social support from the partners, families and friends reported increased depressive symptoms.

The findings of this study reveal that social support sufficiently reduces prenatal and postnatal depressive symptoms in the mothers and in this way, supports the transitions from pregnancy to motherhood. Consistent with previous studies, it has been found that social support received from partners, families, relatives and friends is an efficient resource for women who reported depressive symptoms in the prepartum and postpartum (Elsenbruch et al., 2007; Glazier et al., 2004). Social support sources provided from partners, families and friends assist women to cope with depressive symptoms and to adjust to the motherhood successfully. Hence, social support sources reduce the risk for developing depression during the prepartum and postpartum. As indicated by this study, the partner social support received during pregnancy did not protect women from developing depression but during postpartum it did protect. Hence,

the findings suggested that a partner social support was a protective factor for the mothers during the postpartum rather than the prepartum.

Different kinds of social support in the period of pregnancy and the transition to motherhood should be further investigated because social support is a significant protective factor and should be implemented in future preventive programs. The findings of this dissertation have showed that the distal protective factors sufficiently provided support for depressed mothers. Moreover, it seems that maternal resources reduce effectively prenatal depressive symptoms and enable the transitions to motherhood in this sample. Furthermore, women who received sufficient support from their partners, families, relatives and friends reported lower depressive symptoms over time. Hence, overall social and partner social support are important protective factors for women during and after pregnancy. Thus, it should be encouraged the development of interventions concentrating on increasing social support through creating social networks for the reduction of prenatal and postnatal depressive symptoms and help especially women at risk to cope with the life changes.

4.3.3 The impact of stressful life events and maternal educational status on prenatal and postnatal maternal depressive symptoms

Hypothesis 5

The findings of this dissertation have showed that the occurrence of stressful life events during pregnancy was associated with depressive symptoms in the prenatal period, but the effects did not continue into the first and second postpartum period. Moreover, women in this study who reported more stressful life events during pregnancy were

more likely to experience depressive symptoms over time. A previous study revealed similar results (Da Costa et al., 2000). The sample of the above-mentioned study was relatively small but the authors also found the similar outcome, with no relations to other demographical factors such as age, as well. In a previous study of the same authors (Da Costa et al., 1999) has been shown that younger women seemed to worry more than older and experienced more distress facing different kinds of stressors. Younger women in their early twenties experienced more difficulties in adjusting to marital life and career during pregnancy than older women as well (Da Costa et al., 1999). A reason that the maternal age was not an influencing factor in this dissertation sample might be that the most of the participants were neither very young, nor very old, as having a mean age of 31 (M = 31.20, SD = 5.76 years).

The findings of this study showed that women who experienced more stressful life events during pregnancy were more likely to report depressive symptoms in the prepartum but not in the postpartum. One possible interpretation is that stressful life events assessed during pregnancy might be a predictor of prenatal depressive symptoms rather than postnatal depressive symptoms. It is possible that stressful life events occuring during pregnancy weaken the effects of depressive symptoms in postpartum because of the influence of the important life event of giving birth and the joy connected with the baby. However, the occurrence of different stressful life events acted as a predictor for the occurrence of depressive symptoms during pregnancy. A previous study reported similar results and accentuated the cumulative effects of the stressors on maternal well-being (Glazier et al., 2004). Even though, the life events scales for assessing different kinds of stress stimulus are widely used in the research and mostly a rule than an exception, they might have some limitations. Moreover, how precisely the scales assess the effect and the exact frequency of the stressful life events might be not

comepletely clear and in this way, it might be questioned if they might influence the results.

Concurrently, maternal educational status predicted the occurrence of depressive symptoms in prenatal and only in the first postnatal period, but did not in the second postnatal period. A possible explanation might be that women reported very low depressive score during the second postnatal period. Depressive symptoms in the sample decreased after birth in the first postpartum session and in second postpartum session when the depressive symptoms reduced slightly. Previous studies have pointed out that women with low SES experience more stressors and have less resources which affect their well-being and thus, increase the depressive symptoms (Levine et al., 2020). As maternal educational status is one of the components of maternal SES, the findings of this dissertation might be interpreted in the similar manner. Moreover, women with low educational status are more likely to experience stressors, such as job loss and financial problems (Levine et al., 2020), which might have a reflection on their depressive mood. Hence, low educational status of the women in this study appear to be a distal risk factor during pregnancy and in the first postpartum period, predicted the occurrence of prenatal and postnatal depressive symptoms. At the same time as already discussed above, the prenatal stressful life events also play a predicitive role for the occurrence of depressive mood during pregnancy.

In the study, there is a link between the timing of maternal depressive symptoms, stressful life events and maternal educational status. It seemed that within the group of participants, prenatal exposure to stressful life events, to depressive symptoms, and low maternal educational status increase the probability for women to remain depressed during postpartum period and through this have long-lasting effects on maternal well-being.

Summary and future research directions

Study two shows that depressive symptoms decreased from the prenatal to postnatal periods. The received overall social support and partner social support by pregnant women is a protective factor against developing depressive symptoms during the prenatal and postnatal periods. Stressful life events assessed in pregnancy and low maternal educational status predict the occurrence of prenatal depressive symptoms. According to the findings, there are significant effects of the stressors and resources on prenatal and postnatal maternal depressive symptoms. In the study, women who received less social support from partners, families and friends experienced depressive symptoms during the prenatal and postnatal periods, which were predicted from the occurrence of stressful life events. This dissertation accentuates the positive impact of maternal resources and at the same time the negative impact of stressful life events on the maternal depressive symptoms over time. The findings further extend the knowledge about the interactions between maternal depressive symptoms, resources and stressful life events in a dynamical context. These indications are in accordance with the evidence of previous studies (Glazier et al., 2004; Gómez-López et al., 2019; Levin & Defrank, 1988; Turner et al., 1999) and reveal that stressful life events and the lack of social social support are predictors of prenatal maternal depressive symptoms.

5 CHAPTER FIVE: GENERAL DISCUSSION

5.1 General discussion

Summary: study one

In the first study, how maternal indicators relate to infants' brain activity during a spontaneous EEG was examined. Based on this, the neurophysiological outcomes of infants born from depressed mothers were compared with those born from non-depressed mothers. Prenatal to postnatal maternal depressive symptoms have been found to be associated with low alpha power and alpha desynchronization in 6-8-month-old infants. During this period of an infants' life, the prerequisite for acquiring basic skills appear and the impairments seen in EEG patterns might be possible predictors for developmental delays, behavioural and emotional problems, and psychopathology later in life. Several studies accentuate the relations between prenatal and postnatal maternal depressive symptoms and infants' adverse neurophysiological mechanisms impairing cognitive and emotional processing in infants and might be related to delay in development (Glover, 2014; Kinsella & Monk, 2009; Van Batenburg-Eddes et al., 2013). By studying a young child's neurophysiology, it is possible to have a better understanding of changes with age and maturation in developing infants at risk. Investigating different brain regions enables to better comprehend the alpha power and alpha desynchronization over brain and to distinguish more precisely the neural mechanism in risk groups. Moreover, this study shows that alpha power and alpha desynchronization differ significantly between infants of mothers with and without depressive symptoms. Thereby, study one extends the knowledge on biomarkers associated with maternal depressive symptoms and the neurophysiological development

in infants. The first study highlights the importance of the neurophysiological markers of depression that predicted infants' susceptibility and showed mother-to-child transmission risks. The neurophysiological markers of depression might be a reliable diagnostic tool that might be crucial in the period of infancy. Consequently, results from the first project are able to give information for the provision of developmentally sensitive programs to prevent effectively malfunction and to promote positive outcomes in infants.

Summary: study two

In the second study, the cumulative effects on stressful life events and resources for prenatal and postnatal maternal depressive symptoms were evaluated. Maternal depressive symptoms have been assessed longitudinally providing a differentiation between prenatal and postnatal depressive symptoms and enable to identify the change of depressive symptoms over time. The findings of study two show that stressful life events increase the prenatal depressive symptoms and that overall social support and partner social support are important resources for depressed mothers. Moreover, stressful life events predict the occurrence of prenatal depressive symptoms and maternal educational status predict the occurrence of prenatal and postnatal depressive symptoms. Therefore, the investigation of the results shows that there is a positive impact of maternal resources on prenatal depressive symptoms and concurrently, stressful life events negatively affect the prenatal depressive symptoms. As discussed before, these findings have been shown in other studies as well (Glazier et al., 2004; Gómez-López et al., 2019; Levin & Defrank, 1988; Turner et al., 1999). Maternal resources, such as partner social support and overall social support, have been established as efficient protective factors. Furthermore, the protective factors might serve as preventive tools for mothers at risk and therefore, might be of crucial

importance. Results from the second project also call for the development of preventive programs including different resources that provide social support.

5.1.1 The interactions between prenatal and postnatal maternal depressive symptoms, distal protective factors, proximal and distal risk factors and neurophysiology in early infancy

The goal of this project is to show the interactions between prenatal and postnatal maternal depressive symptoms, protective factors, stressful life events and infants' neurophysiology. More specifically, it has been presented how prenatal and postnatal maternal depressive symptoms affected the infants' neurophysiological patterns and because depressive symptoms during prepartum and postpartum appeared to have a harmful effect on the infants' brain, then it was further investigated the dynamical interactions between maternal depressive symptoms, resources and stressors. In all of these interactions, the role of maternal educational status was taken into consideration.

As indicating in this study and in a previous study as well (Newman et al., 2016), there is a link between the timing (prenatal or postnatal) of maternal depressive symptoms and the occurrence of stressful life events. According to the findings of this dissertation, the occurrence of stressful life events and depressive symptoms during prepartum have increased the probability for the persistence of depressive symptoms during postpartum. Other authors claim that prenatal exposure to maternal depressive symptoms, together with different stressful life events might affect the infants more severely than postnatal exposure because of the increased cortisol level which is transferred to the baby (O'Donnell et al., 2009; Van den Bergh et al, 2005; Van den Bergh et al., 2018). High

levels of maternal cortisol may cause a developmental risk for infants' brain (O'Donnell et al., 2009; Van den Bergh et al., 2005) and transfer high sensitivity to stressful life events to the neonate (Van Den Bergh et al., 2018). This is particularly detrimental since the severity of the maternal illness seems related to higher incidences of stressful life events in their infants (Plant et al., 2015). The severity of maternal depressive symptoms and the occurrence of stressful life events might put the infants into high risk for developing different kinds of emotional, neurocognitive and behavior problems (van den Heuvel et al., 2018). That is why often the infants of mothers with increased depressive symptoms appeared to show more pronounced alpha desynchronization patterns. Hence, alpha desynchronization in infants might be specifically affected by the disrupted patterns of cortisol and severity of the mood disorder of the mothers (Field, 2017; Van den Bergh et al., 2018).

In this dissertation, both prenatal to postnatal permanence of depressive symptoms are associated with EEG alpha desynchronization patterns in infants (Diego, Field, Hernandez-Reif, et al., 2004; Field, Diego, & Hernandez-Reif, 2004; Field, Diego, Hernandez-Reif, et al., 2004). Changes in EEG alpha desynchronization in infants of depressed mothers seem related to lower cognitive abilities, emotional and behavior problems and potentially facilitate illness onset later in life (Field et al., 2010; Van den Bergh et al., 2018). Alpha dissonance patterns as neurophysiological markers might have a great importance to assess potential risk very early in life and in this way to support the identification of a mood disorder. Moreover, neurophysiological markers of depression might enable indicating women and infants at risk.

As the depressive mood during prepartum and postpartum impairs the infants' development, it is important to show which risk factors might predict the depressive symptoms and which factors might be protective for the women and infants at risk. In

this community sample, the lack of social partner and overall social support for the mother, the occurrence of stressful life events and the low educational status are predictors of maternal depressive symptoms. It seems that both stressful life events and resources influenced prenatal maternal depressive symptoms and should be further investigated. Furthermore, the role of social support received by partners, families and friends in the treatment of depression should be involved in possible programs for women and infants at risk.

Even though the investigated research questions were organized in two studies for better structural differentiation, there are some aspects and factors which should be discussed and suited together as their significance is relevant for the overall work, such as possible confounders as the role of maternal educational status. The low educational status might be interpreted as a distal risk socio-economic factor in the study's sample because it increased the risk for developing depressive symptoms during the prenatal and postnatal periods. The last finding suggests that mothers with low educational status are under higher risk for having depressive symptoms and therefore, their infants are possibly under high risk to have impairments in emotional and cognitive processing and psychopathology later in life. Besides, this dissertation shows that maternal educational status also somehow modified the relation between infants' alpha power and alpha desynchronization and maternal depressive symptoms. Thus, there is puzzled complexity behind the interactions between maternal depression, educational status and infants' alpha power and desynchronization, which have not been clearly understood. Moreover, maternal educational status seems to be an impactful variable almost in every analysis and it has significant influence on the interactions between prenatal and postnatal maternal depressive symptoms, stressful life events and infants' neurophysiology. How exactly the maternal educational status modifies these relations

should be further investigated in larger sample size as there are just few studies considering the influence of education.

Once the depressive symptoms occur, appears the question about the factors and causes behind them to discover the determinants of susceptibility. In this dissertation one proximal and two distal indicators are concerned as potentially relevant to the prediction of maternal depressive symptoms in the community sample. These factors might be behind the causal pathways of depressive symptoms, thereby, predicting and affecting depressive symptoms. Moreover, the causality of depressive symptoms might be interpreted as an interplay of the proximal and distal risk factors, embraced by both bio-psycho-social and through the social determinist frameworks. In addition to the risk factors investigated in this dissertation (stressful life events, lack of social support, low educational status), there are other risk factors, such as unintended pregnancy, single marital status, physical complaints during pregnancy and traumatic birth experiences, as well as the tobacco and alcohol consumption, that are also considered to be responsible causal aspects for the development of depressive symptoms during prepartum and postpartum (O'Donnell et al., 2009; Plant et al., 2015; Van den Bergh et al, 2005; Van den Bergh et al., 2018). Possibly, there is no single cause of depressive symptoms. Furthermore, probably, there are a variety of contributing factors and it must be hard to concretise the complex components and pathways for the causality of depressive symptoms.

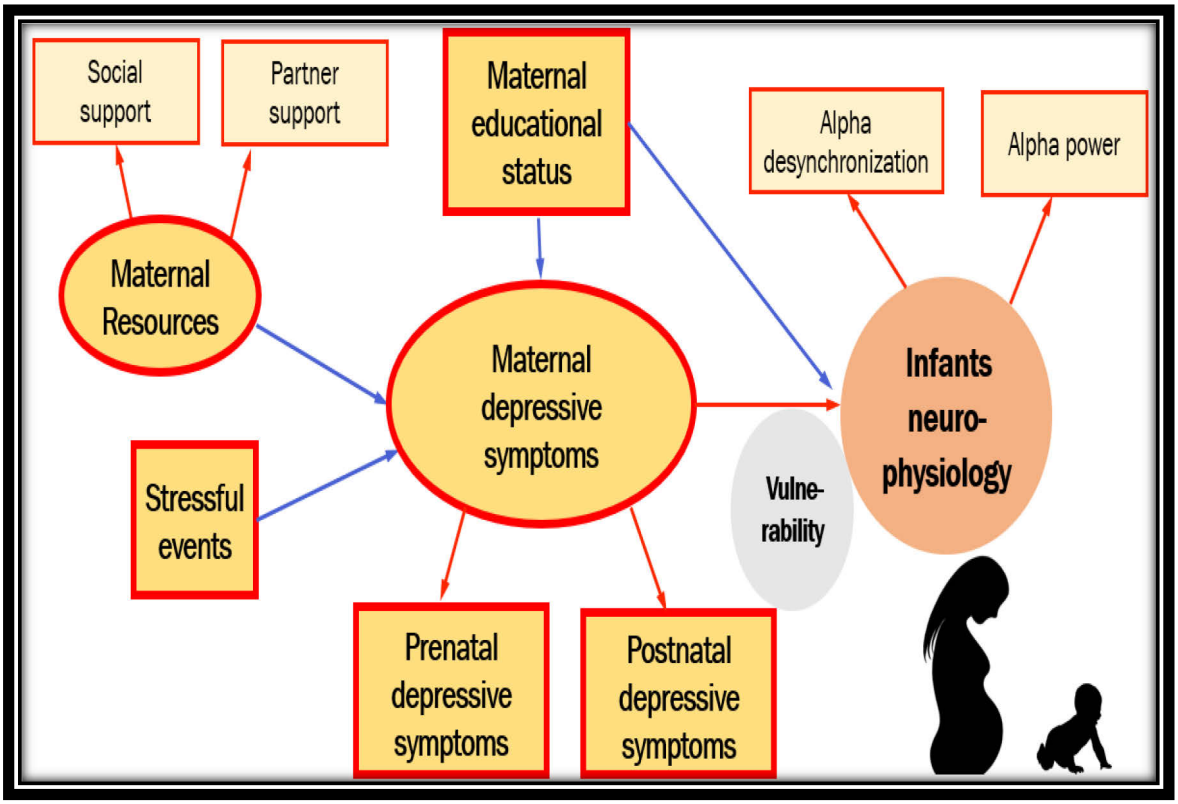
Besides, maternal depressive symptoms, there are other factors that can affect maternal health and consequently, the infant's development. For example, the consumption of toxic substances, such as alcohol and nicotine during pregnancy, impacts maternal health and poses risks for an infant development (Römer et al., 2020). Pathological outcomes in children that result from prenatal alcohol and nicotine exposure comprise

deficits in neurophysiological but in behavioral, cognitive and morphological development as well (O'Donnell et al., 2009; Plant et al., 2015; Van den Bergh et al., 2005; Van den Bergh et al., 2018). Moreover, maternal depressive symptoms and the resulting impairments in infants might be caused by stressful events, lack of social support or the consumption of toxic substances, which are also often associated with each other. Hence, the accumulation of factors that cause deficits in maternal mental health and, subsequently, in affected infants, represents a special burden for families.

The important linkage between both studies in this dissertation is to emphasize the need for prevention for risk groups. Thus, study one and study two both call for creating supporting programs for disadvantaged families. Furthermore, investigating the neurophysiological markers of depression, the dynamics of depressive symptoms at different periods and its relationships with risk and protective factors might help to detect women at risk and to motivate interventions, enhancing social support to reduce the depressive symptoms during the prepartum and postpartum and in this way, prevent impairments in infants. The benefits of social support should be applied in future interventions. In summary, with regard to the bio-psycho-social aspects, the results of this dissertation reveal that prenatal and postnatal maternal depressive symptoms, educational status, protective factors, stressful life events and infants' neurophysiology were thought to be strongly interconnected *(Figure 16)*. Using multi-method research, this dissertation addresses the interplay of maternal depressive symptoms with one proximal and two distal indicators, and predicts infants' susceptibility reflecting a pathology.

Figure 16

Overview of the interactions between prenatal and postnatal maternal depressive symptoms, educational status, protective factors, stressful life events and infants' neurophysiology



Note. Prenatal and postnatal maternal depressive symptoms, predicting from stressful life events and affecting from ressources, are responsible for the infants' susceptibility to have adverse neurophysiological outcomes. Maternal educational status affecting these impairments.

5.1.2 Other factors within the group of disadvantaged families

According to the diathesis-stress model, there are other factors together with maternal resources and stressful life events, such as demographic and socio-economic factors, which have significant contributions to the development of depressive symptoms. In this dissertation, it is hard to draw conclusions about the influences of different socio-economic factors on depressive symptoms. A previous study within a small sample did not find any relations between depressive symptoms, stressful life events and maternal resources, with other demographic and socio-economic factors, such as age and educational status, respectively (Da Costa et al., 2000). Concurrently, in this dissertation, prenatal and postnatal maternal depressive symptoms are related to low educational status. It is important to notice that the majority of the participants who are part of the BRISE longitudinal study belonged to socially and/or culturally disadvantaged families in Germany. It is assumed that disadvantaged families have less resources and are more vulnerable to stressful life events and thus to depression (Jennings et al., 2020; Levine et al., 2020).

Hence, the results of this dissertation should be sensitively interpreted as considering the uniqueness and specificity of the population participated in the project and the generalizations of the findings should be avoided. Moreover, the majority of the participants of this dissertation project were from low SES with almost half of them having migration background. It is supposed that these participants experienced more cumulative life stressors, such as financial and health problems. The occurrence of more stressful life events might be also associated with the need of more resources and social support opportunities. It should be noticed that the accessibility to different support structures seemed hard in the disadvantaged populations (Darling et al., 2019; Jennings et al., 2020). The disadvantaged people meet certain problems, such as lack of

approachability, language problems and awareness for care providers and programs (Darling et al., 2019; Jennings et al., 2020). This highlights the importance for more studies with socially and culturally disadvantaged populations to further investigate the complicated mechanisms behind the different risk factors and the need of variety of resources in hard times.

Another reason to motivate research within disadvantaged groups is to show different perspectives within a variety of populations and the specific risk and protective factors available for them. Most of the studies are concentrated on participants within middle and high socio-economic classes in the well-developed countries, which might be easily reached (Darling et al., 2019; Jennings et al., 2020; Levine et al., 2020). Hence, conducted a longitudinal study within the group of participants with low SES and motivated them to be part of the study for long-term is challenging but at the same time gives a unique contribution to the interrogated questions.

5.1.3 Intervention and Prevention

Even though this dissertation is not investigating the effectiveness of different preventions, it has some practical implications because it highlights the importance of neurophysiological markers as indicators for identifying depression and the role of social support as a protective factor for the reduction of depressive symptoms. Study one shows that prenatal and postnatal maternal depressive symptoms might pose a risk to the infants which has been manifested in the differences in alpha power and alpha desynchronization of the infants of depressed mothers in comparison with infants of non-depressed mothers. Therefore, the transmission risks for the infants might be then detected early through these EEG patterns which might be emphasized as markers of depression and interventions might be encouraged. Study two indicates that overall

social support and partner social support decrease prenatal and postnatal maternal depressive symptoms and through this accentuates the protective role of social support for women. Hence, the social support for the mothers from partners, relatives and friends is helping women in the management of depressive symptoms and might be preventing depression during the transition to motherhood. Some women found the birth of their first child to have established stronger relationships and ties with their partners, families and friends (Prinds et al., 2018). Moreover, supporting the mother in dealing with her depressive symptoms, while caring for her baby, might strengthen the attachment to the child and might decrease the risk for an altered infant's outcome. Therefore, it is necessary to examine which interventions are the most effective at decreasing the mothers' levels of depression and through this improving infants' outcomes. Due to different kinds of interventions, factors or conditions that depend on the social experience, maternal depressive symptoms and infants' outcomes might be positively influenced (Diego et al., 2006; Fernandez et al., 2004; Jones et al., 2004). The social experiences related to mother-child interactions decrease maternal depressive symptoms and improve infants' alpha desynchronization (Diego et al., 2006). Therefore, the approach interaction style might have a preventative effect for the infants. The infants' outcomes improved not only in infants whose mothers had approached interaction style but also through lavender and rosemary exposure (Fernandez et al., 2004). The stability of breastfeeding might be also used for the need of intervention because it might have a positive impact on depressed mothers and their infants (Jones et al., 2004). Massage and music sessions are also effective interventions for mothers and infants (Field & Diego, 2008).

In Germany, there are some center-based and home visit programs aimed to provide professional support for mothers during their transition to motherhood as well as for

disadvantaged families. Home visits might be a preventative approach to support socially and culturally disadvantaged families. The German home visiting program Pro Kind is closely based on the Nurse Family Partnership (NFP) program (Olds, 2006). In this program, professional home visitors offer support to mothers from pregnancy to the child's second birthday through regular home visits while counseling the mothers using theories of self-efficacy, attachment, and thus, aims at enhancing the mother-infant relationship (Jungmann et al., 2015). A recent study reports significant but small positive program effects on maternal health, parenting competencies and young children's cognitive and behavioral development (Jungmann et al., 2015). Other interventions may include mood induction, interaction coaching, stress reduction, counselling and natural buffers, such as non-depressed fathers and caregivers (Field & Diego, 2008). It is necessary to examine which interventions are the most effective in improving mother and child outcomes and to investigate the influence of partner, social and family supports on decreasing the level on depression.

5.2 Limitations

Even though this dissertation contributes to the understanding of mechanisms behind the interactions between prenatal and postnatal maternal depressive symptoms, resources, stressful life events and infants' neurophysiology, it has several limitations. Therefore, the results of this dissertation must be interpreted in the context of these limitations.

In the first place, there is a large rate of missing data (prenatal maternal depressive symptoms missing values and EEG missing values). Missing data from the questionnaire assessing prenatal maternal depressive symptoms were due to the fact that

less participants attended the first prenatal session (T0), in comparison to the two postnatal sessions. Due to the lack of data of depressive symptoms at T0, an average value from prenatal and postnatal maternal depressive symptoms scores, which were submitted in different time periods, was used in some of the analyses and in this way, prenatal and postnatal depressive symptoms were not separately addressed in these analyses. In the analyses, the term *prenatal to postnatal maternal depressive symptoms* was used as a reference to mark the difference. A better differentiation of the effects of prenatal and postnatal depressive symptoms (Soe et al., 2016) is recommended for future research.

EEG missing values were mostly due to infants' movements, agitation and therefore, interruption of the EEG recording session. Conducted EEG with infants was a challenging process because the infants were not able to follow instructions for the measurement (van der Velde & Junge, 2020). Hence, EEG missing data and prenatal depressive symptoms missing data and possibly other missing values may impact data analysis. Moreover, data were lost through grouping participants for further analysis, as some of the individuals had missing values for some of the used variables. Additionally, the ANOVA within the small sub-sample of the participants grouped by depressive symptoms and maternal educational status should be interpreted cautiously, as the participants are not equally distributed to the groups due to the missing values. With regard to the limitations of the missing values, a replication of the analysis with a larger sample size should be encouraged.

In the second place, the assessment of prenatal and postnatal maternal depressive symptoms, overall social and partner social support were based on the mothers' self-report assessment, which might have implied some biased scores i.e. social desirability (Larson, 2019). In this study couldn't be control if the participants were faking or

having a tendency for social desirability. It might be assumed that the women showed a tendency to report their depressive symptoms as less severe and their partners as more supportive (Konstabel et al., 2006).

In the third place, some limitations regarding the EEG analysis should be discussed. A very broad frequency band to assess the alpha power (3-12 Hz) was used. Some authors suggest the use of the frequency range between 6-9 Hz for assessing alpha frequency in infants (Saby & Marshall, 2012). Hence, the broad frequency band in which alpha is extracted might influence the power and desynchronization. Moreover, the broad frequency band was used to guarantee that alpha frequency is presented, because in the frequency of 6-9 Hz it was not possible to ensure that the alpha band could be found and it was dominant there. Individual differences should be emphasized and individual alpha frequency analysis should be recommended for better replication and interpretation (Klimesch, 1999). Spontaneous EEG was recorded while a child film condition and thus, emotional task was not involved. Some authors suggest the importance of different emotional task and mother child interactions task for better evaluating the alpha desynchronization score (Field & Diego, 2008). Moreover, the examination of test-retest reliability of the alpha power and desynchronization (Vuga et al., 2008) in a longitudinal design might be encouraged for better validation of the results and to prove whether alpha power and desynchronization remain stable or whether there is a variability due to some other factors. Furthermore, the parameterization of alpha desynchronization should be examined as there are different procedures for it. Concurrently, in the EEG analyses, sets with different EEG data quality were used. Within the ANOVA small sub-sample analysis, the data of participants rated with 4-satisfactory data quality were excluded. The procedure of analyzing and rating the EEG data sets quality might introduce possible constraints and

should be criticized, thus, further investigation is recommended.

In the fourth place, overall social and partner social support were assessed over a short perinatal period from late pregnancy to 10 weeks postpartum, thus, it is unknown whether there were changes at each of these stages. Future investigation should focus on the course of overall and partner social support at each stage of the perinatal period from early pregnancy to the first year postpartum.

Finally, it is possible that other potentially impactful confounders, such as socio-economic, demographic, etc., have not been included. The findings of this study, that frontal alpha power and right-hemispheric dominance of frontal alpha desynchronization are not only associated with maternal depressive symptoms but also with the educational status, provide an evidence for the complexity of the interactions. Besides, maternal educational status influences the interactions between prenatal and postnatal depressive symptoms and stressful life events as well. As maternal educational status plays such an important role, the impact of SES, especially in disadvantaged families should also be included. In this study, different kinds of contributing factors, for instance, the influence of overall social support and partner social support, and stressful life events are included. Nevertheless, other factors and socio-economic circumstances, such as an income and occupation, should be taken into account. Future replications with a control group is also recommended.

Strengths

On the one hand this dissertation has the mentioned before limitations, but on the other hand it has its advantages. Longitudinal measures are used, adding to the literature how prenatal and postnatal maternal depressive symptoms change over time. Hence, this research project observes over time the change of maternal depressive symptoms.

Furthermore, both frontal and parietal regions are investigated, providing a better understanding of the alpha power and alpha desynchronization over the brain, compared to most studies in infants, where only frontal regions were studied. This should be considered as a differentiated manner that should encouraged interrogating different brain regions. This dissertation uses more than one method to examine the research questions and thereby, it provides a multi-method research, enabling the investigations of the relatively complex interactions. Moreover, this multi-method dissertation is a combination of bio-psycho-social research that represents close interactions between the bio-psycho-social environment reflecting a psychopathology and adverse outcomes. These strengths should be considered as big advantages. Furthermore, the findings of this dissertation provide a link between the prenatal and postnatal maternal depressive symptoms, resources, stressful life events and infants' neurophysiology in the first year of life.

6 CHAPTER SIX: CONCLUSION

Despite the above-mentioned limitations, this dissertation has several strengths, such as longitudinal measures, combination of multi-method research inspired by bio-psycho-social model, and might emphasize the neurophysiological markers as indicators of transmission risk in socially and/or culturally disadvantaged families.

The main objective of the dissertation is to investigate how maternal indicators, prenatal and postnatal depressive symptoms, relate to infants' neurophysiological mechanisms, maternal stressful life events and resources. Moreover, this study shows that both alpha power and alpha desynchronization differ significantly between infants of mothers with and without depressive symptoms. This dissertation addresses the importance of neurophysiological markers as indicators for identifying depressive disorders. Moreover, EEG markers might support the detection of depression and therefore, they might have significant practical implications. Furthermore, resources for the mothers, such as overall social and partner social support, could be identified as efficient protective factors. In addition, stressful life events and educational status predicted prenatal maternal depressive symptoms.

The findings of this dissertation expand the knowledge about the impact of maternal prenatal and postnatal depressive symptoms and educational status on infants' brain EEG activity, including the study of neurophysiological markers, and about the influence of stressful life events and protective factors on maternal depressive symptoms by applying a bio-psycho-social model. Hence, this dissertation provides more knowledge about the reliability of the findings of previous studies investigating the interactions between prenatal and postnatal maternal depressive symptoms,

educational status, neurophysiological outcomes during infancy, stressful life events and protective factors. The relevance of doing research with culturally and socially varied individuals with differing social settings, which has an impact on the social environment and maybe on the transition to motherhood, is highlighted in this dissertation.

In summary, this study accentuates that there are differences in brain EEG alpha activity between infants of mothers with and without depressive symptoms and points out the importance of different stressful life events and protective factors during the period of pregnancy and early infancy. Consequently, results are able to provide information and call for the development of effective preventive tools for women and young children during the phases of pregnancy and early infancy.

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8 ABBREVIATIONS

ACC Anterior cingulate cortex

CASMIN Comparative analysis of social mobility in industrial nations

CFI Comparative fit index

DLPFC Dorsolateral prefrontal cortex

DSM Diagnostic statistical manual of mental disorders

EEG Electroencephalogram

EPDS Edinburgh postnatal depression scale

ERP Event-related potential

FFT Fast Fourier transform

fNIRS Functional near-infrared spectroscopy

GLM General linear model

HPA Hypothalamic-pituitary-adrenal

Max Maximum

M Mean

MEG Magnetoencephalography

Min Minimum

mPFC Medial prefrontal cortex

MRI Magnetic resonance imaging

OFC Orbitofrontal cortex

PSD Power spectral density

PTSD Post-traumatic stress disorder

RMSEA Root mean square error of approximation

ROI Region of interest

SD Standard deviation

ABBREVIATIONS

SEM Structural equation modeling

SE Standard error

SES Socio-economic status

SNR Signal-to-noise ratio

TLI Tucker–Lewis index

9 APPENDIX

Appendix A:

Constructs and used Questionnaires

1. Prenatal and postnatal depressive symptoms: EPDS (Edinburgh Postnatal Depression Scale; Cox et al., 1987): T0, T1, T2

Die folgenden Fragen beziehen sich auf die letzten sieben Tage. Bitte wählen Sie jene Antwort, die Ihren Gefühlen in den letzten sieben Tagen am nächsten kommt.

Ich konnte lachen und das Leben von der heiteren Seite sehen:

☐ überhaupt nie

☐ deutlich weniger als früher

☐ nicht ganz so wie früher

☐ genauso wie früher

Es gab vieles, auf das ich mich freute:

☐ fast gar nicht

☐ viel seltener als früher

☐ eher weniger als früher

☐ so oft wie früher

Ich habe mich unberechtigter Weise schuldig gefühlt, wenn etwas daneben ging:

- ☐ nein, nie
- ☐ ganz selten
- ☐ ja, manchmal
- ☐ ja, sehr oft

Ich war ängstlich und machte mir unnötige Sorgen:

- ☐ nein, nie
- ☐ ganz selten
- ☐ ja, manchmal
- ☐ ja, sehr oft

Ich habe mich häufig erschrocken und wurde panisch ohne wirklichen Grund:

- ☐ nein, nie
- ☐ ganz selten
- ☐ ja, manchmal
- ☐ ja, sehr oft

Mir ist alles zu viel geworden:

- ☐ ja, ich wusste mir überhaupt nicht mehr zu helfen
- ☐ ja, ich wusste mir manchmal nicht mehr zu helfen
- ☐ nein, ich wusste mir meistens zu helfen
- ☐ nein, ich konnte alles so gut wie immer bewältigen

Ich war so unglücklich, dass ich kaum schlafen konnte:

- ☐ nein, nie
- ☐ ganz selten
- ☐ ja, manchmal
- ☐ ja, fast immer

Ich war traurig und fühlte mich schlecht:

- ☐ nein, nie
- ☐ ganz selten
- ☐ ja, manchmal
- ☐ ja, sehr oft

Ich war so unglücklich, dass ich weinen musste:

- ☐ nein, nie
- ☐ ganz selten
- ☐ ja, manchmal
- ☐ ja, sehr oft

Gelegentlich kam mir der Gedanke, mir etwas anzutun:

- ☐ nein, nie
- ☐ ganz selten
- ☐ ja, manchmal
- ☐ ja, sehr oft

2. Educational status: T0-prenatal and T0-postnatal

Ausbildung

Wo haben Sie zuletzt die Schule besucht? War das…

☐ in einem Bundesland in der Bundesrepublik Deutschland?

☐ in der DDR?

☐ in einem anderen Land?

Welchen Schulabschluss haben Sie gemacht?

☐ Schule ohne Abschluss verlassen

|_____|

☐ Volks- oder Hauptschulabschluss (DDR: 8. Klasse)

☐ Realschulabschluss oder Mittlere Reife (DDR: 10. Klasse)

☐ Fachhochschulreife (Abschluss einer Fachoberschule)

☐ Abitur/Hochschulreife

☐ anderer Schulabschluss

|_____|

Haben Sie in Deutschland eine Berufsausbildung oder ein Studium abgeschlossen?

☐ Nein

☐ Ja

Was für ein Ausbildungs- oder Studienabschluss war das und in welchem Jahr haben Sie diesen Abschluss gemacht?

☐ Lehre, Facharbeiterabschluss;

Jahr: |___|___|___|___| Ausbildungsberuf: |_____|

☐ Berufsfachschule, Handelsschule, Schule des Gesundheitswesens

Jahr: |___|___|___|___| Ausbildungsberuf: |_____|

☐ Fachschule, z. B. Meister-, Technikerabschluss

Jahr: |___|___|___|___| Ausbildungsberuf: |_____|

☐ Beamtenausbildung

Jahr: |___|___|___|___| Ausbildungsberuf: |_____|

Laufbahn: |_____|

☐ Fachhochschule, Berufsakademie (früher auch: Ingenieurschule, Lehrerbildung, DDR: Ingenieur- und Fachschulabschluss)

Jahr: |___|___|___|___| Fachrichtung: |_____|

☐ Universitäts-, Hochschulabschluss

Jahr: |___|___|___|___| Abschluss: |_____|

Fachrichtung: |_____|

☐ Promotion

Jahr: |___|___|___|___| Abschluss: |_____|

Fachrichtung: |_____|

☐ Sonstiger Abschluss

Jahr: |___|___|___|___| Abschluss: |_____|

APPENDIX

Haben Sie in einem anderen Land als Deutschland eine berufliche Ausbildung oder ein Studium gemacht?

☐ Nein

☐ Ja

Was für eine Ausbildung war das?

☐ Ich wurde in einem Betrieb angelernt

☐ Ich habe in einem Betrieb eine längere Ausbildung gemacht

☐ Ich habe eine berufsbildende Schule besucht

☐ Ich habe eine Hochschule besucht

☐ Sonstiges _____

	Level	**Track** *gen/voc.*	**CASMIN**	**Description**
Tertiary	*High*		**3b**	Higher tertiary education: The completion of a traditional, academically oriented university education
	Low		**3a**	Lower tertiary education: Lower-level tertiary degrees, generally of shorter duration and with a vocational orientation
Secondary	*High*	Voc	**2c_voc**	Voactional maturity: Full maturity certificates including vocationally-specific schooling or training

		Gen	**2c_gen**	General maturity: Full maturity certificates (e.g. the Abitur, A-levels)
	Mediate	Voc	**2a**	Intermediate vocational qualification, or secondary programmes in which general intermediate schooling is combined by vocational training
		Gen	**2b**	Intermediate general education Academic or general tracks at the secondary intermediate level
	Low	Voc	**1c**	Basic vocational training above and beyond compulsory schooling
		Gen	**1b**	General elementary education
Primary		Gen	**1a**	Inadequately completed general education

Anmerkungen: gen = general (allgemein); voc = vocational (beruflich)

Hinweis: Die Antworten wurden gemäß CASMIN (Comparative Analysis of Social Mobility in Industrial Nations; Brauns et al., 2003, S.223; König et al., 1988) kodiert.

3. Stressful life events scale: T0-prenatal and T0-postnatal

Besondere Belastungen

Während der Schwangerschaft wie auch zu jedem anderen Zeitpunkt im Leben können verschiedene Ereignisse auftreten, die als belastend empfunden werden. Dazu gehören z. B. gesundheitliche Probleme, Partnerschaftsprobleme, finanzielle Probleme, Probleme am Arbeitsplatz, Krankheit oder Todesfälle in der Familie.

Sind bei Ihnen im Verlauf der aktuellen Schwangerschaft solche belastenden Ereignisse aufgetreten?

☐ Nein

☐ Ja

Welche Ereignisse sind aufgetreten und wie belastend haben Sie diese erlebt?

	gar nicht belastend	**etwas belastend**	**überwiegend belastend**	**stark belastend**
☐ finanzielle Probleme	☐	☐	☐	☐
☐ Belastungen durch die Wohnsituation (z. B. Hausbau, Umzug)	☐	☐	☐	☐
☐ Belastungen durch die Arbeitsplatzsituation (eigene/ bei Partner)	☐	☐	☐	☐
☐ Partnerschaftskonflikte	☐	☐	☐	☐

	gar nicht belastend	**etwas belastend**	**überwiegend belastend**	**stark belastend**
☐ Trennung von Partner	☐	☐	☐	☐
☐ Konflikte mit der eigenen Familie	☐	☐	☐	☐
☐ Todesfälle in der Familie/ nahestehender Personen	☐	☐	☐	☐
☐ Krankheiten in der Familie / von nahestehenden Personen	☐	☐	☐	☐
☐ Schwangerschaftsspezifische Belastungen (z. B. Komplikation, Ablehnung der Schwangerschaft)	☐	☐	☐	☐
☐ Gibt es außerdem etwas, das Sie als belastend erlebt haben? (Bitte angeben)	☐	☐	☐	☐

4. Overall social support and partner social support: T0-prenatal and T0-postnatal

Soziale Unterstützung

Inwieweit können Sie grundsätzlich auf die Unterstützung durch nahestehende Personen zählen? Bitte geben Sie an, in welchem Ausmaß die nachfolgenden Aussagen auf Ihre Situation zutreffen.

	trifft überhaupt nicht zu	**trifft eher nicht zu**	**teils-teils**	**trifft eher zu**	**trifft genau zu**
Ich finde ohne Weiteres jemanden, der sich um meine Wohnung kümmert, wenn ich mal nicht da bin.	☐	☐	☐	☐	☐
Es gibt Menschen, die mich ohne Einschränkung so nehmen, wie ich bin.	☐	☐	☐	☐	☐
Ich erfahre von anderen viel Verständnis und Geborgenheit.	☐	☐	☐	☐	☐
Ich habe einen sehr vertrauten Menschen, mit dessen Hilfe ich immer rechnen kann.	☐	☐	☐	☐	☐
Bei Bedarf kann ich mir ohne Probleme bei Freunden oder Nachbarn etwas ausleihen.	☐	☐	☐	☐	☐
Ich habe Freunde/Angehörige, die sich auf	☐	☐	☐	☐	☐

	trifft überhaupt nicht zu	**trifft eher nicht zu**	**teils-teils**	**trifft eher zu**	**trifft genau zu**
jeden Fall Zeit nehmen und gut zuhören, wenn ich mich aussprechen möchte.					
Ich kenne mehrere Menschen, mit denen ich gerne etwas unternehme.	☐	☐	☐	☐	☐
Ich habe Freunde/Angehörige, die mich einfach mal umarmen.	☐	☐	☐	☐	☐
Wenn ich krank bin, kann ich ohne Zögern Freunde/Angehörige bitten, wichtige Dinge für mich zu erledigen.	☐	☐	☐	☐	☐
Wenn ich mal sehr bedrückt bin, weiß ich, zu wem ich damit ohne Weiteres gehen kann.	☐	☐	☐	☐	☐
Es gibt Menschen, die Freude und Leid mit mir teilen.	☐	☐	☐	☐	☐
Bei manchen Freunden/Angehörigen kann ich auch mal ganz ausgelassen sein.	☐	☐	☐	☐	☐
Ich habe einen vertrauten Menschen, in dessen Nähe ich mich ohne Einschränkung wohl fühle.	☐	☐	☐	☐	☐
Es gibt eine Gruppe von Menschen	☐	☐	☐	☐	☐

	trifft überhaupt nicht zu	**trifft eher nicht zu**	**teils-teils**	**trifft eher zu**	**trifft genau zu**
(Freundeskreis, Clique), zu der ich gehöre und mit der ich mich häufig treffe.					

(F-SozU (K14); Fydrich et al., 2009)

Partnerschaft und Paarbeziehung

Nun möchte ich Ihnen gerne einige Fragen zu Ihrer Partnerschaft stellen.

Können Sie sich mit Ihrem Partner oder einer anderen nahestehenden Person über Ihre Schwangerschaft austauschen?

☐ Ja ☐ Nein

Begleitet Ihr Partner oder einer andere nahestehende Person Sie zu den medizinischen Vorsorgeuntersuchungen?

☐ Ja ☐ Nein

Begleitet Ihr Partner oder eine andere nahestehende Person Sie zu weiteren Angeboten im Rahmen der Schwangerenvorsorge und Geburtsvorbereitung?

☐ Ja ☐ Nein

Gib es jemanden, der Ihnen Tipps und Ratschläge für die Schwangerschaft, Geburt oder Alltag mit einem Baby geben kann?

☐ Ja ☐ Nein

Wird Ihr Partner oder eine andere nahestehende Person während der Geburt für Sie da sein?

☐ Ja ☐ Nein

Können Sie nach der Geburt auf die Hilfe Ihres Partners oder einer anderen nahestehenden Person bei der Versorgung Ihres Kindes zählen?

☐ Ja ☐ Nein

Declaration of authorship

Eidesstattliche Erklärung

gem. §6 Absatz 5 der Promotionsordnung Dr.rer.nat. des Fachbereichs 11

der Universität Bremen vom 06.07.2011

Hiermit versichere ich, dass die vorliegende Dissertation ohne unerlaubte fremde Hilfe angefertigt wurde, keine anderen als die angegebenen Quellen und Hilfsmittel benutzt wurden und die den benutzten Werken wörtlich oder inhaltlich entnommenen Stellen als solche kenntlich gemacht worden sind. Die zu Prüfungszwecken beigelegte elektronische Version der Dissertation ist identisch mit der abgegebenen gedruckten Version.

Bremen, 06.04.22

Polina Stoyanova