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BRIEF REPORT

Maternal eating disorder severity is associated with increased latency of foetal auditory event-related brain responses

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Abstract

Objective: Maternal eating disorders (EDs) are associated with adverse pregnancy and child outcomes. There is limited research investigating the influence of maternal EDs on foetal brain development.

Method: Using foetal magnetoencephalography (fMEG), an auditory sequence was presented for 10 min to assess brain response latencies in foetuses of mothers with (n = 12) and without (n = 11) a history of anorexia nervosa (AN) in the third trimester of pregnancy. ED history and severity were assessed using the structured clinical expert interview eating disorder examination (EDE) and the self-report questionnaire EDE-Q.

Abbreviations: AER, auditory event-related responses; AN, anorexia nervosa; DSM-5, Diagnostic and Statistical Manual of Mental Disorders, 5th edition; EDs, eating disorders; EDE, eating disorder examination; EDE-Q, eating disorder examination questionnaire; fMEG, foetal magnetoencephalography; GDM, gestational diabetes mellitus; SARA, SQUID array for reproductive assessment; SQUIDs, superconducting quantum interference devices.

Results: Foetuses of mothers with AN showed delayed foetal brain responses to auditory stimulation compared to foetuses of control women. Self-reported ED symptom severity explained 34% of variance in foetal brain response latencies in the AN group.

Conclusions: ED pathology was strongly associated with foetal brain response latencies in the third trimester with longer latencies in foetuses of women with a history of AN reporting more ED symptoms. Follow-up on the children is pivotal to investigate if fMEG outcomes are associated with later child development.

KEYWORDS
anorexia nervosa, brain, development, eating disorders, foetus, fMEG, pregnancy

Highlights

- Foetuses of mothers with lifetime anorexia nervosa (AN) showed a slower brain response to auditory stimulation compared to foetuses of control mothers, though, this difference was not statistically significant
- Self-reported eating disorder severity explained 34% of variance in foetal auditory evoked brain response latencies in the AN group
- A history of maternal AN might have an impact on the child's brain development, even in mothers who do not fulfil overall diagnostic criteria

1 | INTRODUCTION

Eating disorders (EDs) including anorexia nervosa (AN) run in families (Zipfel et al., 2015). Maternal AN is associated with increased risks for stillbirth, low birth weight, small-for-gestational age birth (Ante et al., 2020; Mantel et al., 2019) and adverse cognitive, psychological, feeding and eating development in the child (Martini et al., 2020). However, most evidence on pregnancy outcomes in women with a history of ED is retrospective and predominantly epidemiological in nature, originating from cohort studies and self-report data. Questions regarding mechanisms of transgenerational transmission of EDs remain largely unanswered (Micali, Stahl, et al., 2014). In utero risk mechanisms might contribute to cognitive (Barona et al., 2017) and mental health outcomes in children of women with EDs (Micali, Stahl, et al., 2014). More precisely, children of mothers with EDs display neurobehavioural dysregulation in the early postnatal phase and poorer language and motor development at one year (Barona et al., 2017). Since large-scale systems of the brain are established before birth (Vasung et al., 2019), foetal brain development might be affected by maternal malnutrition (Cusick & Georgieff, 2016; Freedman et al., 2018) associated with EDs (Setnick, 2010). Foetal magnetoencephalography (fMEG) is the main non-invasive method used to determine brain activity measurable as biomagnetic fields with high detection rates produced by the foetus in the maternal abdomen (Preissl et al., 2004). Foetal auditory event-related brain response (AER) latency is a quantitative and reliable marker for foetal brain maturation, both structurally and functionally (Preissl et al., 2005). Foetal AER latency declines with advanced gestational age (Sheridan et al., 2010). Shorter latencies indicate more mature brain responses (Holst et al., 2005; Schleussner & Schneider, 2004). For instance, recent fMEG studies in women with gestational diabetes mellitus (GDM) have demonstrated that postprandial foetal AER latency was slower in the offspring of women with GDM (Linder et al., 2015). Also intrauterine growth restriction was associated with delayed foetal AER latencies (Kiefer et al., 2008). We investigated foetal brain activity in foetuses of mothers with a history of AN compared to foetuses of mothers without EDs using fMEG. Since women affected by EDs show dysfunctional eating behaviours, that is, bingeing, purging or extreme caloric restriction which may lead to inadequate intakes of macro- and micronutrients in pregnancy (Dörsam et al., 2019), we expected foetuses of mothers with a history of AN to show longer AER latencies compared to foetuses of control women.

2 | METHOD

The data are part of a longitudinal parent-child study. Women with a lifetime ED diagnosis according to *Diagnostic and statistical manual of mental disorders*, 5th

edition (American Psychiatric Association, 2013) were recruited for the ED group. Women with no history of ED were recruited as controls. Ethical approval was obtained from the ethics committee of the Medical Faculty at the University of Tuebingen. Each mother and father provided written informed consent. Forty-three pregnant women participated in the parent-child study. fMEG data were recorded between week 27 and 37 of pregnancy with the SARA system (superconducting quantum interference devices [SQUIDs] Array for Reproductive Assessment, CTF MEGTM System, VSM Med. Tech) installed at the fMEG Center at the University of Tuebingen. The fMEG device is placed in a magnetically shielded room (Vakuumschmelze) to attenuate the influence of external noise. The main component of the fMEG device is a concave sensor array, which contains 156 magnetic sensors (superconducting quantum interference devices, SQUIDs) and 29 reference channels for noise detection (see Figure 1). Prior to and after the fMEG recording, the foetal head position was determined by ultrasound (Logiq 500 MD, GE Healthcare). During the measurement, the pregnant women sat on the device in an upright position and leaned their abdomen against the smooth sensor array as comfortably as possible to avoid any muscle activity, which might disturb the measurement. Foetal head position was marked by a localisation coil placed on the maternal abdomen. Another three localisation coils fixed on an elastic belt were placed on the mother's spine and on her left and right side to record maternal movement during the measurement. Using the Presentation program (version 12.2, www.neurobs.com) a frequent 'standard' tone (frequency 500 Hz) was presented for 75% of the time and an infrequent 'deviant' tone (frequency of 750 Hz, presented 25% of the time) randomly interspersed to prevent habituation to the standard tone. Tone duration was 500 ms, tone intensity was 95 dB and interstimulus interval was set to 1500 ms. Through the reduction of sound intensity by maternal tissue, the tones reached the foetus with an intensity of approximately 65 dB. The auditory sequence was presented for 10 min. The sound was generated by a speaker and was transmitted through a plastic tube to an inflated plastic balloon which was placed between the maternal abdomen and the sensor array. Reported results refer to the standard tone (for a detailed description of the auditory stimulation see Linder et al., 2015). A trained staff member performed the structured clinical expert interview eating disorder examination (EDE) (Fairburn & Cooper, 1993) to assess ED history of mothers; ED severity was additionally assessed using the self-report questionnaire EDE-Q (Hilbert et al., 2007).

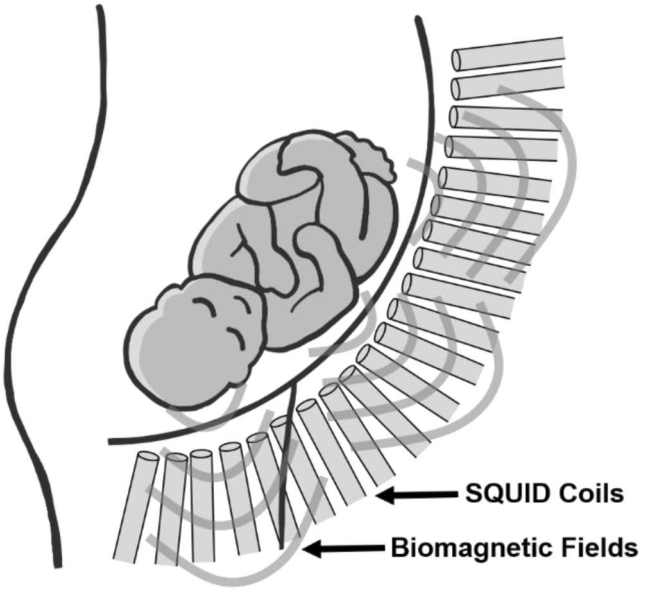

FIGURE 1 Schematic illustration of the foetal magnetoencephalography (fMEG) device (with kind permission of Julia Moser). SQUID, superconducting quantum interference device

Data management and analysis were performed using SPSS 27.0 (IBM Corp. Released 2020. IBM SPSS Statistics for Windows, Version 27.0., IBM Corp). Statistical significance for mean differences was analysed using *t*-test and Mann–Whitney *U*-test as appropriate. Linear regression analysis was applied to test for an association between foetal AER latency and ED severity. Missing values were excluded pairwise. All analysis with $p < 0.05$ were regarded as statistically significant. Foetal brain analysis was not possible for 20 participants due to weak foetal signals, maternal muscle activity or movement artefacts. Therefore, data from $n = 12$ women with a history of AN and $n = 11$ controls were analysed.

3 | RESULTS

Sample characteristics regarding pregnancy data are shown in Table 1. Nine women (75%) in the AN group were married and two women (17%) had a partner. One woman in the AN group had no permanent partner. In the healthy control (HC) group, seven women (64%) were married and four women (36%) had a partner. Except for the one woman without a partner, all women in the cohort lived together with their partner. All women in the AN group had German nationality, in the control group there were three women (27%) with other nationalities (Polish, Iranian, Spanish). The educational level of the cohort was very high: all women in the sample graduated from high school and 67% of the AN

TABLE 1 Sample characteristics

	Total sample	AN	HC	*p*-value
Sample size	23	12	11	-
Maternal age (years)	30.91 (4.66)	32.08 (4.93)	29.64 (4.20)	0.22[a]
Gravity (median; range)	1 (1–5)	2 (1–5)	1 (1–3)	0.06[b]
Parity (median; range)	0 (0–2)	0 (0–2)	0 (0–2)	0.29[b]
Gestational age (weeks)	30.91 (2.95)	30.42 (2.75)	31.45 (3.21)	0.38[b]
BMI pre-pregnancy (kg/m^2)	21.43 (3.34)	20.47 (3.61)	22.48 (2.80)	0.04[b]
BMI present (kg/m^2)	24.85 (3.55)	23.67 (3.81)	26.14 (2.86)	0.10[a]
Gestational weight gain/week (kg)	0.44 (0.21)	0.41 (0.22)	0.46 (0.19)	0.58[a]
EDE-Q global score	0.44 (0.50)	0.59 (0.58)	0.27 (0.33)	0.15[b]

Note: Data presented as mean (standard deviation) unless otherwise described.

Abbreviations: AN, anorexia nervosa group; BMI, body mass index; EDE-Q, Eating Disorder Examination Questionnaire; HC, healthy control group.

[a]Student's *t*-test.

[b]Mann–Whitney *U*-test.

group and 73% of the HC group had a university degree (bachelor, master, diploma). Five women in the AN group (42%) and two control women (18%) already had children at the time of the study. 83% of women with AN ($n = 10$) and 91% ($n = 10$) of control women became pregnant naturally.

3.1 | Characterisation of the AN group ($n = 12$)

On average, women in the AN group developed AN at 17.3 ± 4.8 years of age (range: 11–26 years of age). More precisely, 50% of the group developed the disease by their 15th birthday and 75% of the group developed AN before reaching the age of 20. The duration of the disease varied between 2 and 28 years with a mean of 10.8 ± 8.6 years. The number of years women spent in remission prior to study inclusion varied from 0 to 14 years, with AN defined as latently active in pregnancy in six women (three of the restrictive and purging type, respectively). In terms of lifetime AN diagnosis, four women reported AN purging type, seven women reported AN restrictive type, and one woman reported atypical AN (weight criterion not met). Two women (17%) in the AN group had not received any treatment and 58% ($n = 7$) had been treated as inpatients and/or outpatients for their AN. At the time of the study assessment, women with AN reported the following ED-specific behaviours: objective overeating ($n = 4$), objective binge eating ($n = 3$), loss of control over eating ($n = 4$), self-induced vomiting ($n = 1$) and driven physical exercise ($n = 3$).

3.2 | Latencies for foetal AERs

Latencies for foetal AER ranged from 103.2 to 342.4 ms, with a mean of 191.5 ms (71.5 ms). Foetuses of mothers with lifetime AN descriptively showed a slower brain response to auditory stimulation (212.6 ± 68.7 ms) compared to foetuses of control mothers (168.5 ± 70.4 ms), though, this difference was not statistically significant. The effect size indicated a moderately strong difference in foetal AER latencies between groups (95% CI = 0.1605–0.2224; $r = 0.36$; $p = 0.09$). In the AN group, there was a significant positive association between foetal AER latencies and maternal EDE-Q global scores, $F(1, 10) = 5.084$, $p = 0.048$, $R^2 = 0.337$, indicating that self-reported ED severity explained 34% of variance in foetal AER latencies (see Figure 2). For the HC group, the linear regression model for the EDE-Q global score and foetal AER latency was not significant, $F(1, 9) = 0.286$, $p = 0.605$, $R^2 = 0.031$.

4 | DISCUSSION

The aim of the study was to investigate whether foetuses of mothers with past or current AN show different latencies of auditory evoked brain responses compared to foetuses of mothers without EDs using fMEG. The current fMEG study found no statistically significant differences in mean AER latencies between foetuses of mothers with lifetime AN and foetuses of control mothers. However, self-reported ED pathology was strongly associated with foetal AER latencies in the third trimester with longer latencies in foetuses of women with AN reporting more

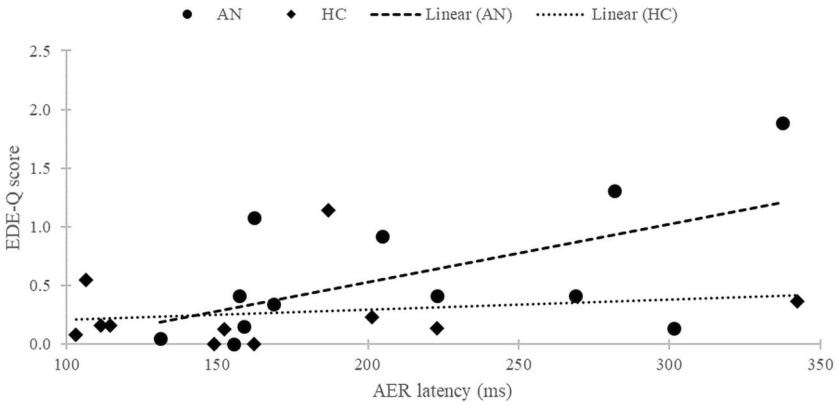

FIGURE 2 Linear regression model for Eating Disorder Examination Questionnaire global score and foetal auditory event-related brain response latency for the anorexia nervosa (AN) and the healthy control (HC) group; the Eating Disorder Examination Questionnaire (EDE-Q) score ranges from 0.0 to 6.0. AER, auditory event-related brain response

ED symptoms. This is a clinically relevant finding, as it suggests that a history of AN might have an impact on the child's brain development, even in mothers who do not fulfil overall diagnostic criteria, but who report ED-specific cognitions such as eating or weight concern. Human brain development is a vulnerable and highly complex process which sets the basis for cognition, behaviour, and emotions for the rest of one's life (Cusick & Georgieff, 2016; Vasung et al., 2019). Distorted eating behaviours, that is, bingeing, purging or extreme caloric restriction as expressed by EDE-Q scores, may be associated with inadequate intakes of macro- and micronutrients, which might affect neurodevelopment of systems controlling psychosocial behaviour, emotional function, appetite, and metabolism, for example, among others through epigenetic mechanisms (Freedman et al., 2018; Godfrey et al., 2016; Jones et al., 2017). Moreover, in a large population-based sample, ED symptoms, anxiety, and depression in pregnancy predicted psychopathology, that is emotional and behavioural problems, in children of women with EDs (Micali, de Stavola, et al., 2014; Micali, Stahl, et al., 2014). The interplay of maternal factors, such as malnutrition or stress, postnatal environmental factors, for example dysfunctional family feeding/eating practices (Kaß et al., 2021; Martini et al., 2018), and genes associated with ED risk (Watson et al., 2019; Yilmaz et al., 2015) might impact ED development in children of women with EDs (Jones et al., 2017). Separating the effects of these factors is complex since children born to mothers with EDs may have abnormal development due to in utero insults and/or a genetic risk for EDs (Barona et al., 2017).

4.1 | Strengths and limitations

This is to the best of our knowledge the first study which measured human foetal brain activity with a reproducible, non-invasive methodology in a sample of mothers with a history of mental disorders. Our project has been conceptualised as a pilot and feasibility study, therefore the sample size is small. The findings have to be replicated in larger samples across the ED spectrum (AN, bulimia nervosa, binge eating disorder, atypical EDs).

5 | CONCLUSIONS

Our pilot data derived from fMEG assessment suggest that a history of AN might have an impact on the child's brain development, even in mothers who do not fulfil overall diagnostic criteria. It will be pivotal to follow-up on the children and families to investigate if foetal AER latencies are associated with later child cognitive and behavioural development. Based on previous research, pre- and postnatal care is important for women with lifetime EDs, who show ED symptoms during pregnancy that might affect the developing child (Micali et al., 2007).

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CONFLICT OF INTEREST

The authors have no conflicts of interest relevant to this article to disclose.

AUTHOR CONTRIBUTIONS

Prof. Dr. Katrin Giel, Prof. Dr. Hubert Preissl, Prof. Dr. Nadia Micali and Prof. Dr. Stephan Zipfel conceptualised and designed the study, and reviewed and revised the manuscript. Annica Franziska Dörsam carried out the initial analyses, drafted the initial manuscript and reviewed and revised the manuscript. Julia Moser supervised data collection, drafted the manuscript and

critically reviewed the manuscript. Jana Throm carried out the initial analyses and reviewed and revised the manuscript. Magdalene Weiss collected data and reviewed and revised the manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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