Objective  To examine the relationship between the severity of infants’ congenital heart defects (CHD) and their mothers’ symptoms of depression and anxiety from pregnancy to 18 months postpartum.  Methods  Mothers of infants with mild, moderate, or severe CHD ($n = 162$) and mothers ($n = 44,400$) within the Norwegian Mother and Child Cohort Study were assessed with an eight-item short version (SCL-8) of the Hopkins Symptom Checklist-25 at the 30th week of gestation and at 6 and 18 months postpartum.  Results  Only the postpartum mental health trajectory of mothers of infants with severe CHD deviated from the mental health trajectory of the cohort at 6 and 18 months postpartum, showing significantly elevated levels of depression and anxiety symptoms.  Conclusions  The results elucidate the relationship between infants’ CHD severity and maternal symptoms of depression and anxiety, possibly identifying a specifically vulnerable patient dyad in need of postoperative interventions.

Key words  anxiety; cardiology; depression; mental health; motherhood.
prolonged hospital visits (Glaser, Harrison, & Lynn, 1964; Van Horn, DeMaso, Gonzalez-Heydrich, & Erikson, 2001). Parents often respond with shock when the diagnosis is first made and the postpartum parenting of a child with a chronic illness is considered to be a highly stressful for the parents and family (Cohen, 1999; Davis, 1993).

Even if treatment is successful and the infant eventually is discharged, many challenges might remain. The infant may show growth impairment and neurodevelopmental retardation, have respiratory problems, digestive problems, and/or breastfeeding problems (Clemente, Barnes, Shinebourne, & Stein, 2001; Dittrich et al., 2003; Saenz, Beebe, & Triplett, 1999) as a result of the cardiac defect, severe co-morbidities or repeated or prolonged surgical interventions. These challenges entail heightened levels of maternal stress that might complicate the postpartum period and affect mothers’ mental health.

In fact, a study by Lawoko and Soares (Lawoko & Soares, 2002) showed that when comparing differences in distress (e.g., depression, anxiety, and suicide ideation) among parents of children with CHD, parents of children with other diseases, and parents of healthy children, mothers of children with CHD were generally at higher risk, reporting the highest levels of distress compared to the mothers in the other groups. Furthermore, the results showed that 24% of the mothers in the CHD group reported global levels of distress within or above norms for psychiatric outpatients. CHD severity was shown to have a weak, but positive correlation with scores on depression, i.e., the more severe the defect, the higher the risk of maternal depression. A second study by the same authors (Lawoko & Soares, 2006) saw these findings withstanding the test of time within a longitudinal design, with a large proportion of the same sample reporting long-standing symptoms of depression, anxiety, and somatisation over a period of 1 year, indicating prolonged psychosocial morbidity. On a similar note, a study by Goldberg, Morris, Simmons, Fowler and Levinson (Goldberg, Morris, Simmons, Fowler, & Levinson, 1990) found that parents of infants with CHD reported the highest amount of stress within the child domain of the Parenting stress index compared to parents of children with cystic fibrosis.

On the other hand, Thompson and associates found that maternal adjustment in mothers of children with sickle cell disease was significantly more related to psychosocial processes, such as the presence of social support (Thompson Jr, Gil, Burbach, Keith, & Kinney, 1993). When incorporated in a transactional stress and coping model, the psychosocial processes accounted for variance in maternal adjustment over and above that explained by illness characteristics and/or severity. Moreover, a similar study by Uzark and Jones (Uzark & Jones, 2003) found that parents of children with heart defects were more likely than controls to report excessive parenting stress, but this was especially related to characteristics of the child that made them difficult to parent, not the heart defect severity in itself.

The studies by Lawoko and Soares (2002) and Uzark and Jones (2003) included parents of children with a mean age of 8 and 6 years, respectively.

Neither study provided information on the critical period from delivery to 6 months, where parents are most vulnerable (Cooper & Murray, 1998). Moreover, although information regarding the children’s CHD diagnosis was obtained, precise information regarding the classification criteria for CHD severity was not presented or deviated from the common CHD grouping of “mild”, “moderate” and “severe”, making it difficult to understand why only a weak association between CHD severity and parental distress was found.

Since depression and anxiety in mothers have previously been shown to interfere with the quality and responsiveness of maternal care (Downey & Coyne, 1990), one could speculate that the mothers’ symptoms of depression and anxiety could become an additional burden for infants with CHD and lead to disturbances in their development. It is therefore important to elucidate the above mentioned findings further in order to isolate the impact of illness severity, and to better understand the needs of mothers of infants with CHD with regard to future development of interventions.

Furthermore, most studies concerned with the prediction of maternal distress related to their children’s health have utilized retrospective or cross-sectional designs, whereas longitudinal designs with prenatal, maternal symptomatology baselines are missing. It is likely that psychological functioning during pregnancy account for the greatest amount of variance in postpartum functioning, and it is therefore important to assess mothers’ symptomatology both before and after childbirth. Only with this type of longitudinal design, can one begin to move from cross-sectional correlations to analyses that are temporally congruent with the causal ordering of the variables.

In light of the aforementioned, the aim of the present study was to chart symptom trajectories of depression and anxiety from pregnancy to 18 months postpartum in mothers of infants with varying degrees of CHD severity. A large pregnancy cohort served as controls, establishing a normative, postpartum mental health trajectory. We hypothesised that CHD severity would affect the mothers’ mental health, i.e. the more severe the defect, the higher the risk of maternal distress, and that mothers of infants with CHD in
general would be at greater risk of developing long-standing symptoms of depression and anxiety compared to controls within this especially vulnerable postpartum period.

Materials and Methods
Population
Data were obtained from the Norwegian Mother and Child Cohort Study (MoBa) conducted at the Norwegian Institute of Public Health. In brief, MoBa is a prospective pregnancy cohort study that was started in 1999 and ended in 2008 (Magnus et al., 2006). Pregnant women were recruited through a postal invitation after they registered for a routine prenatal ultrasound examination at their local hospitals at approximately the 17th gestation week. Participating women (45%) signed an informed consent to take part in a longitudinal study. The women then responded to mailed questionnaires at the 30th gestation week and at 6 and 18 months postpartum. Response rates among those who consented to join the study were 92, 87, and 77% at gestation week 30, and at 6 and 18 months postpartum, respectively. The Regional Committee for Medical Research and the Norwegian Data Inspectorate approved the study and the MoBa cohort was linked to the nation-wide Medical Birth Registry of Norway (MBRN) to capture health information related to the pregnancy, delivery, and the child’s status at birth.

In this study, mothers who (1) had identification information from the MBRN, valid MoBa questionnaires from gestation week 30, 6 months, and 18 months postpartum, and (2) had valid data for weight, gestational age, and questionnaire items concerning depression and anxiety symptoms were included. Among these women, mothers of infants with CHD were identified by means of the country-wide CHD registry at the Department of Pediatric Cardiology at Rikshospitalet, Oslo, Norway, using the unique personal identification number from the Norwegian National Population Register and information from the MBRN. All significant live-born pediatric heart defects cases in Norway are recorded in the country-wide CHD registry. The registry includes data on diagnoses, interventions, surgery, and detailed clinical outcomes obtained from hospital journals and information from other hospitals, out-patient clinics, and local physicians. All examinations, procedures, and contacts with patients with CHD are entered into the database with assigned dates. To ensure the quality of the registry, only senior pediatric cardiologists enter the data. To minimise errors in the present study, two research fellows cross-referenced diagnostic codes before entering them into the final analysis.

Procedures and Measurements
CHD severity classification: Classification of the severity of the cardiac defects was performed by two senior pediatric cardiologists based on previous published guidelines for grouping (Hoffman & Kaplan, 2002). Treatment aspects, however, were also included in the grading of the severity of pulmonary stenosis, aortic stenosis, and VSD. The children with CHD were assigned to the following three groups (see Supplementary Appendix, Diagnosis Charts 2 and 3 for details).

“Mild CHD” (n = 73). These children were generally asymptomatic and had defects that were left untreated. The defects usually resolved spontaneously.

“Moderate CHD” (n = 42). These children may be asymptomatic, but require treatment and follow-up through childhood. No one were treated more than once, and interventions were simple, e.g., balloon dilatation or closure of defects by catheter technique or surgery.

“Severe CHD” (n = 47). These children were generally symptomatic and in some cases severely ill in the newborn period or early infancy, and all required treatment. The VSD included in this group needed to be operated more than once. The pulmonary stenoses included were critical or in need of repeated or surgical treatment.

Symptoms of depression and anxiety: An eight-item version (SCL-8) of the Hopkins Symptom Checklist-25 was used to measure symptoms of depression and anxiety at gestation week 30 and again at 6 and 18 months postpartum. Short-form versions of SCL have previously been shown to correlate highly with the total score of the original scale and to have good psychometric properties (Strand, Dalgard, Tambs, & Rognerud, 2003; Tambs & Moum, 1993). Items in the SCL are scored on a Likert scale ranging from 1 (not at all bothered) to 4 (very much bothered) and, in the present study, Cronbach’s alphas for SCL-8 were 0.81, 0.83, and 0.83 for the three assessments (week 30 and at 6 and 18 months postpartum), respectively. The SCL-8 incorporates two subscales, four items each, allowing separate analysis of depression and anxiety scores (SCL-4Dep and SCL-4Anx), with Cronbach’s α’s of 0.72, 0.78, 0.81 and 0.73, 0.77, 0.77, 0.78, respectively.

Statistical Analysis
In order to avoid bias through listwise deletion of missing values and to preserve the size of the groups of mothers with CHD infants, we used maximum likelihood imputation procedures for missing data computation (Schafer & Graham, 2002). An Expectation Maximization algorithm (Dempster, Laird, & Rubin, 1977) was used to impute values for missing scores in SCL-8 by using SCL response
parameters from gestation weeks 17, 30, and 6 months and 18 months postpartum. The overall cohort missing rate for each assessment period was reduced from 10.9 to 0.1% for gestation week 30, 16.7 to 0.1% for 6 months, and 4.3 to 0.1% for 18 months in the final sample.

Logarithmic transformation was computed for each SCL item for all measurement times in order to minimise skewness (post transformation values = 1.74, 1.89, and 1.68) and kurtosis (post transformation values = 3.79, 4.42, and 3.41).

Mixed between-within subjects ANCOVAs with the between factor of Group (Control, Mild, Moderate, and Severe CHD) and the within factor of Time of measurement (gestation week 30, 6 months, and 18 months postpartum) for the dependent variable SCL4-Dep and SCL4-Anx were used to compute group differences in symptom trajectories over time. Birth weight and gestational week were included as covariates in all analyses, and Cohen’s $d$ was calculated for significant group differences at 6 and 18 months postpartum. Bonferroni corrected post-hoc tests were performed for each assessment period where a $p$-value of 0.05 was considered significant. Standardized regression coefficients and standardized marginal means were computed from the logarithmically transformed results before creating tables and figures in order to enhance readability. The Regional Committee for Medical Research and the Norwegian Data Inspectorate approved the study.

Results

Study Population

A case-match based on our previously mentioned inclusion criteria at 6 months postpartum initially generated a sample of 44,562 women. Within this sample 267 mothers of infants with CHD older than 18 months who were present in both MoBa and the CHD registry were identified. 53 of these mothers did not respond to the 18 months questionnaire and 1 infant died, leaving 213 mothers of infants with CHD in the sample. Co-morbid medical conditions (e.g., Esophageal atresia and Down syndrome) occurred among 46 of the 213 infants and these infants were excluded from the analysis (see Supplementary Appendix, Diagnosis Chart 1, “Excluded cases” for details). Due to missing values in variables of “birth weight” and “gestation week” five additional infants were lost, leaving a total of 162 mothers of infants with CHD in the final sample, as shown in Table 1.

CHD severity and maternal symptoms of depression (SCL-4Dep): Analysis of the four items measuring symptoms of depression separately showed a significant main effect of Group [$F(3,44556) = 7.136, p < .0005$] and a significant interaction between Group and Time of measurement [$F(6,89112) = 5.096, p < .0005$].

The overall effect of Time was not significant [$F(2,89112) = 0.805, p = .447$]. Parameter estimates revealed no difference in the mothers’ SCL-4Dep scores prenatally, but at 6 and 18 months postpartum, mothers of infants with severe CHD reported significantly higher scores than mothers in the control group, with standard deviations of 0.77 and 0.74, respectively, as shown in Table II. Standardized, adjusted marginal means are shown in Figure 1. Cohen’s $d$ for the adjusted marginal means showed a large effect at 6 months postpartum ($d = 0.76$) and a medium effect at 18 months postpartum ($d = 0.73$) when comparing the severe CHD group to controls.

Bonferroni corrected, pairwise comparisons further revealed that mothers of infants with severe CHD had significantly higher SCL-4Dep scores than both mothers of infants with mild and moderate CHD, with $p < .002$ and $p < .001$, respectively.

CHD severity and maternal symptoms of anxiety (SCL-4Anx): Analysis of the four items measuring anxiety separately showed no significant overall effects of Group [$F(3,44556) = 1.970, p = .116$] or Time [$F(2,89112) = 0.453, p = .636$].

However, the analysis still revealed a significant interaction between Group and Time of measurement [$F(6,89112) = 3.989 p < .001$]. Parameter estimates again revealed no difference in the mothers’ SCL-4Anx scores prenatally, but at 6 and 18 months postpartum, mothers of infants with severe CHD still reported significantly higher SCL-4Anx scores than mothers in the control group, with standard deviations of 0.47 and 0.43, respectively, as shown in Table II. Standardized, adjusted marginal means are shown in Figure 2. Cohen’s $d$ for the adjusted marginal means showed a small effect at 6 months postpartum ($d = 0.39$) and a small effect at 18 months postpartum ($d = 0.36$) when comparing the severe CHD group to controls.

Discussion

Main Findings

In slight disagreement with our a priori hypothesis, the results of the present study did not reveal a direct association between the severity of infants’ CHD and mothers’ symptoms of depression and anxiety. Only severe CHD in infants had prolonged effects on the mothers postpartum, as indicated by a significant increase in depression and anxiety symptoms for this group compared pregnancy cohort controls. Importantly, the mean scores of mothers...
of children with mild and moderate CHD did not deviate from the mean score of the pregnancy cohort, suggesting that these mothers were not at a greater risk of developing symptoms. These findings add to the existing literature on the co-occurrence of infants’ CHD and compromised maternal mental health by further elucidating the relationship between CHD severity and symptoms of depression and anxiety, possibly identifying a specifically vulnerable patient dyad.

When looking at symptoms of depression and anxiety separately, anxiety scores were lower than depression scores. This finding might suggest that the mothers’ anxiety was related to the initial shock of the diagnosis of severe CHD and the infants’ following hospitalization and treatments and already on the decline at 6 months, whereas symptoms of depression persisted. After 6 months the infant’s heart defect may no longer be perceived by the mother as life-threatening, reducing symptoms of anxiety, but at the same time, the infant may still show growth impairment or have respiratory or digestive problems that continue to cause elevated symptoms of depression related to the infant’s medical prognosis, psychosocial functioning, and future quality of life. This interpretation is partly supported by two related studies (Brandlistuen et al., 2010; Stene-Larsen et al., 2009) from our research group which reported infant motor impairment, social impairment and emotional reactivity in our CHD sample at 6 months. These impairments might also have complicated the postpartum period and affected the mothers’ mental health in a negative way, causing prolonged symptoms of depression. Further research is therefore needed in order to specify how, and to what extent, a range of illness related factors affect the mothers’ mental health.

**Strengths and Weaknesses**

Whereas the use of MoBa and Rikshospitalet’s large databases gave our study its main strength—allowing us to collect cohort data at a national level prior to and after delivery—it is also a limitation.

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**Table I. Characteristics of the CHD groups compared to the control group**

<table>
<thead>
<tr>
<th></th>
<th>Control group</th>
<th>Mild CHD (n = 73)</th>
<th>Moderate CHD (n = 42)</th>
<th>Severe CHD (n = 47)</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mothers’ education (years) M ± SD</td>
<td>14.6 ± 2.5</td>
<td>14.6 ± 2.5</td>
<td>14.7 ± 2.4</td>
<td>14.6 ± 2.6</td>
<td>.992</td>
</tr>
<tr>
<td>Mothers’ age (years) M ± SD</td>
<td>30.2 ± 4.4</td>
<td>31.1 ± 4.3</td>
<td>30.5 ± 4.6</td>
<td>29.6 ± 3.9</td>
<td>.312</td>
</tr>
<tr>
<td>Mothers’ Income (low/average/high %)</td>
<td>31.6/60.0/8.5</td>
<td>22.5/67.6/9.9</td>
<td>32.5/57.5/10.0</td>
<td>37.8/57.8/4.4</td>
<td>.310</td>
</tr>
<tr>
<td>Fathers’ Income (low/average/high %)</td>
<td>11.5/60.9/27.7</td>
<td>7.2/60.9/31.9</td>
<td>2.5/65.0/32.5</td>
<td>11.6/53.5/34.9</td>
<td>.260</td>
</tr>
<tr>
<td>Social Support (yes %)</td>
<td>96.7</td>
<td>100.0</td>
<td>97.4</td>
<td>93.9</td>
<td>.288</td>
</tr>
<tr>
<td>Birth weight (g) M ± SD</td>
<td>3,596 ± 576</td>
<td>3,682 ± 667</td>
<td>3,448 ± 848</td>
<td>3,313 ± 701*</td>
<td>.002</td>
</tr>
<tr>
<td>Gestational age (weeks) M ± SD</td>
<td>39.4 ± 1.8</td>
<td>39.4 ± 2.0</td>
<td>38.7 ± 2.9*</td>
<td>38.6 ± 2.3*</td>
<td>.001</td>
</tr>
<tr>
<td>Sex (girls %)</td>
<td>48.9</td>
<td>63.0*</td>
<td>42.9</td>
<td>46.0</td>
<td>.016</td>
</tr>
</tbody>
</table>

*Significantly different from the control group, assessed with parameter estimates or Pearson chi-square.

*p-values for test of between-subjects effects.

**Table II. Parameter estimates showing standardized regression coefficients (b) with 95% confidence intervals for SCL-4Dep, and SCL-4Anx across CHD severity (control, mild, moderate, severe) from gestation week 30 to 18 months postpartum**

<table>
<thead>
<tr>
<th>Time of measurement</th>
<th>Group</th>
<th>n</th>
<th>SCL-4Dep b (CI)</th>
<th>SCL-4Anx b (CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gestation week 30</td>
<td>Severe CHD</td>
<td>47</td>
<td>0.14 (–0.13 to 0.42)</td>
<td>−0.12 (–0.41 to 0.17)</td>
</tr>
<tr>
<td></td>
<td>Moderate CHD</td>
<td>42</td>
<td>0.12 (–0.16 to 0.41)</td>
<td>−0.10 (–0.40 to 0.21)</td>
</tr>
<tr>
<td></td>
<td>Mild CHD</td>
<td>73</td>
<td>0.00 (–0.22 to 0.22)</td>
<td>0.02 (–0.22 to 0.25)</td>
</tr>
<tr>
<td>Control</td>
<td>44,400</td>
<td></td>
<td>0b</td>
<td>0b</td>
</tr>
<tr>
<td>6 months postpartum</td>
<td>Severe CHD</td>
<td>47</td>
<td>0.77 (0.48 to 1.05)*</td>
<td>0.47 (0.20 to 0.75)*</td>
</tr>
<tr>
<td></td>
<td>Moderate CHD</td>
<td>42</td>
<td>−0.17 (–0.47 to 0.13)</td>
<td>−0.01 (–0.30 to 0.29)</td>
</tr>
<tr>
<td></td>
<td>Mild CHD</td>
<td>73</td>
<td>−0.04 (−0.27 to 0.18)</td>
<td>0.08 (−0.14 to 0.30)</td>
</tr>
<tr>
<td>Control</td>
<td>44,400</td>
<td></td>
<td>0b</td>
<td>0b</td>
</tr>
<tr>
<td>18 months postpartum</td>
<td>Severe CHD</td>
<td>47</td>
<td>0.74 (0.43 to 1.04)*</td>
<td>0.43 (0.14 to 0.72)*</td>
</tr>
<tr>
<td></td>
<td>Moderate CHD</td>
<td>42</td>
<td>−0.22 (−0.54 to 0.10)</td>
<td>−0.27 (−0.58 to 0.04)</td>
</tr>
<tr>
<td></td>
<td>Mild CHD</td>
<td>73</td>
<td>0.04 (–0.21 to 0.28)</td>
<td>0.02 (−0.21 to 0.26)</td>
</tr>
<tr>
<td>Control</td>
<td>44,400</td>
<td></td>
<td>0b</td>
<td>0b</td>
</tr>
</tbody>
</table>

*aSignificantly different from the control group, assessed with parameter estimates or Pearson chi-square.

bp-values for test of between-subjects effects.

The coefficients show deviations from the control mean in fractions of standard deviations.

This parameter is set to zero because it is redundant.

*p < 0.005.
First, and regrettably, our results cover mothers’ mental health state only and leave out fathers’ psychological reactions. In the MoBa cohort study, only one questionnaire targeted the fathers, and it did not include items related to paternal mental health or psychological functioning. Interest in fathers of sick children has recently increased in pediatric research, and several studies have shown that also fathers are affected by events related to pregnancy and delivery of a seriously ill child (Lawoko & Soares, 2006; Skari et al., 2006) and that fathers’ symptoms of depression might affect children’s development (Ramchandani, Stein, Evans, & O’Connor, 2005). Parallel SCL-8 scores for the fathers at all 3 assessment times would therefore have broadened the scope of our findings, adding more details to the picture.

The low percentage of respondents (initial recruitment rate of 45%, with a 77% follow-up rate at 18 months) should also be mentioned as a limitation. A study by Nilsen et al. (2009) found that participants in the MoBa study, compared to all women giving birth in Norway, differed on a number of exposure variables, but that associations of various exposures and outcomes were not affected. Therefore we do not expect the MoBa participation and attrition rates to have a significant impact on the generalizability of our data.

Second, in the present study we collected information on mental health using a shortened scale (SCL-8) of the Hopkins Symptoms Checklist, and in doing so we might have left out more detailed information that could have been obtained with the original full scale (SCL-90; Lipman, Covi, & Shapiro, 1979). Moreover, we did not assess symptoms of post-traumatic stress, which is known to occur in parents of children with a serious illness (Helfrich, Latal, Fischer, Tomaske, & Landolt, 2008; Landolt, Vollrath, Ribi, Gnehm, & Sennhauser, 2003). A measure of this type would have been desirable in order to obtain more detailed information concerning the impact of CHD severity and the consequences of the distress experienced by the mothers. On the other hand, the length of a questionnaire per se might affect the response rate, and there is a danger some respondents will take objection to a large number of very personal questions, which of course would be a threat to the validity of the study (Tambs & Moum, 1993). Still, additional clinical assessments of the mothers of children with CHD together with a matched control group would have been desirable for this study in order to investigate and validate the findings further. With the SCL-8, we only assessed depression and anxiety symptoms and can therefore not rule out that the elevated symptoms reflect a more general form of illness related distress.

A final limitation is related to the timing of the diagnosis and the severity grading of the different types of CHD. In the present study we did not obtain data on whether the CHD diagnosis was set pre- or postnatally. A measure of this type would have been desirable in order to obtain more detailed information concerning the impact of the timing of the CHD diagnosis. Previous studies have found high levels of anxiety symptoms both at the time of the test result and some weeks later in women who receive abnormal screening results on, for example, CHD or Down syndrome tests (Marteau et al., 1988; Rona, Smeeton, Beech, Barnett, & Sharland, 1998). An exact
measure of “time of diagnosis” could therefore have provided us with more detailed information when calculating the mental health trajectories of the mothers, especially in relation to possible, undetected peaks in anxiety symptoms.

Furthermore, the simplification of the complex diagnostic varieties of CHD into categories of mild, moderate, and severe might be an oversimplification in relation to maternal mental health. It is possible that this classification is not ordinal, i.e., the difference in impact between mild and moderate CHD may be very different from that between moderate and severe CHD. Within the category of “severe CHD”, there are also a wide variety of diagnoses that can range from moderate, reparable problems to life-threatening chronic conditions. Some of these infants will only have surgical palliation (surgery to allow the child to survive, but not restore the normal anatomic and physiological arrangement), while others will have repair, but still have residual anatomic or hemodynamic defects that require significant follow-up by the cardiologist.

How these different categories and illness related factors affect the mothers’ mental health is therefore not trivial. Future research should explore the different aspects of each category further in order to isolate key illness related factors and their effect on the mothers’ mental health. The case number within each category was also fairly low, and given, for example, the low number of specific complex CHD diagnoses (e.g., Hypoplastic left heart syndrome) in the severe CHD group, regrettably no specific conclusions can be made for any single specific CHD diagnose.

Policy Implications and Conclusions

Our findings suggest that simply having an infant with a CHD does not increase the risk of maternal depression and anxiety per se. Only when the child suffers from severe CHD might prolonged symptoms of depression and anxiety be present. Since depression and anxiety in mothers have previously been shown to interfere with the quality and responsiveness of maternal care, one could speculate, in the case of infants with severe CHD, that their mothers’ mental health could become an additional burden. Although the mothers’ symptoms might remit after 18 months, research has shown that infants’ cognitive development might already be affected at this stage and that mother–infant relationships often are characterized by insecure attachment and interaction difficulties (Coghill, Caplan, Alexandra, Robson, & Kumar, 1986; Cooper & Murray, 1998; Hay, 1997). Therefore, given early negative effects of maternal depression and anxiety, the clinical implications for infants with severe CHD might be an additional delay in development compared to children with less severe CHD and healthy children, especially if their mothers’ symptoms of depression and anxiety extend into the clinical realm. The link between severe CHD, maternal symptoms of depression and anxiety should therefore be identified by medical personnel as a risk factor for the health and well-being of both the mother and infant.

Considering this, the utilization of screening tools and treatment interventions targeting these mothers and their infants at an early stage is recommended. Future research should target screening and treatment programs that are suitable for immediate intervention, complementing already existing care. Evidence from controlled clinical trials suggests that such interventions should include programs teaching self management skills, patient education, and self-management education groups that bring together patients with similar problems (Bodenheimer, Lorig, Holman, & Grumbach, 2002). Further research is also needed in order to explore a possible negative reciprocal relationship between mother and infant in order to clarify the direction of their complex interaction and the underlying mechanisms that might be affecting both the mother and infant over time.

Supplementary Data

Supplementary data can be found at: http://www.jpepsy.oxfordjournals.org/.

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Conflicts of interest: None declared.
References


