Offspring of depressed/anxious patients: how do they fare after onset of a depressive/anxiety disorder?
ABSTRACT

Background
Although many studies have shown that offspring of depressed/anxious patients are at high risk to develop such a disorder themselves, knowledge on how these offspring fare after onset of a depressive/anxiety disorder is limited. This study aims to investigate the prospective course of depressive/anxiety disorders in offspring with a parental history of similar psychopathology.

Methods
Data on the six-year course of depressive/anxiety disorders were presented for 105 offspring with a lifetime diagnosis at baseline. Primary outcome was a depressive/anxiety episode during follow-up. Secondary outcomes included severity (i.e., depressive symptoms, anxiety symptoms, disability) and treatment indicators (i.e., antidepressant use, psychological treatment). Results were presented for all offspring and for offspring with and without a diagnosis separately. In addition, course trajectories in offspring were compared to those of controls without a parental history of these disorders.

Results
In our sample of offspring with a lifetime diagnosis 59% also experienced a depressive/anxiety episode during the six-year follow-up period and 41% had not. In offspring with and without an episode during follow-up symptom severity and treatment varied widely. No significant differences were found in course trajectories between offspring versus controls without a parental history (p≥.264), but the small comparison sample limits the ability to draw firm conclusions.

Conclusions
Previous literature clearly shows that offspring of patients with a depressive/anxiety disorder are at increased risk to develop these disorders themselves. This study shows that once the disorder has manifested itself, the course is variable. Results further indicate that fulfilling diagnostic criteria does not always go hand in hand with severe symptomatology and disabilities, and vice versa.
INTRODUCTION
The interest in the transgenerational transmission of depressive and anxiety disorders has grown rapidly in the last decades. Earlier studies have mainly considered the impact of a parental history of depressive and anxiety disorders on the onset of these disorders in their offspring.\textsuperscript{1-3} It has been amply demonstrated that children of depressed and/or anxious parents are at elevated risk of developing depressive and anxiety disorders and other adverse outcomes such as medical problems and impaired overall functioning.\textsuperscript{1,4} The risk of these offspring is substantial as shown by impressive incidence rates reported in longitudinal studies.\textsuperscript{1,2,5} For example, a recent study by our own group found that an estimated 65% of offspring of treatment-seeking depressed/anxious parents develops a mood/anxiety disorder before the age of 35 years.\textsuperscript{5}

While the high incidence in offspring is thus undisputed, few studies addressed how offspring fare after onset of a depressive or anxiety disorder. Focusing on the high-risk studies of high quality that determined psychiatric caseness by interviewing both parent and offspring\textsuperscript{6-10}, information on course has been provided on recurrence\textsuperscript{6-10}, severity\textsuperscript{6-10} and treatment outcomes.\textsuperscript{7} Studies like these are important as they can provide useful information regarding prognosis and can be helpful in treatment planning and (relapse) prevention. Overall these studies suggest that offspring risk is not temporary and many children display persisting problems. These studies were, however, hampered by a small sample of affected offspring (i.e., N≤23)\textsuperscript{8,10} or used relatively long time intervals between assessments\textsuperscript{6,7,9} which may have resulted in less reliable data on the course of disorder between assessments.

In addition, although previous studies did investigate the overall course in terms of, for example, recurrence rates, other relevant indicators of the precise course (e.g., symptom severity, disability or treatment) were not examined in offspring with and without a diagnosis separately. To gain a proper understanding of the course in offspring this information is of high importance as previous studies showed that syndromal recovery does not necessarily indicate restoration of psychosocial functioning and complete absence of symptoms.\textsuperscript{11-13} Bijl and colleagues\textsuperscript{11} for example, observed residual effects of mood and anxiety disorders as patients, although recovered, still experienced disabilities in everyday functioning.

This study reports on the course of depressive and anxiety disorders in 105 offspring of patients who had received specialized treatment for depressive or anxiety disorder. These offspring all had a lifetime depressive and/or anxiety disorder at baseline and were prospectively followed-up at biennial intervals for six years. Our first aim is to describe the overall course of depressive and/or anxiety disorders in offspring by considering the presence of a depressive/anxiety episode during follow-up (primary outcome). As secondary outcomes, we include both severity (i.e., severity of depressive symptoms, severity of anxiety symptoms, disability) and treatment (i.e. antidepressant use, psychological treatment) indicators. Our second aim is to further characterize the precise course by distinguishing those offspring with and without an episode during follow-up and, then, describing their course in terms of severity and treatment. A third aim is to examine whether the overall course of
depressive/anxiety disorders in offspring differed from that of persons without a parental history of these disorders. Comorbidity rates between depressive and anxiety disorders have shown to be substantial. The present study therefore takes a transdiagnostic approach and deals with the group of persons with a depressive and/or anxiety disorder further referred to as persons with depressive/anxiety disorders.

METHOD
Study sample
Data were from the (NESDA), which started in 2004-2007 and aimed to examine the long-term course and consequences of depressive and/or anxiety disorders (see Penninx and colleagues for a detailed description of the study design). The research protocol was approved by the Ethical Committee of participating universities and all participants provided written informed consent. Participants were recruited from several settings, including a subsample of 261 participants who previously participated in the Adolescents at Risk for Anxiety and Depression (ARIADNE) study (2000-2004). The ARIADNE study recruited offspring of patients who received specialized treatment for depressive and/or anxiety disorder at one of the 16 psychiatric services in the North of the Netherlands. Parental diagnoses were further confirmed by assessment with the Composite International Diagnostic Interview (CIDI). ARIADNE's study design had been previously described.

For the present study, the baseline, 2-year, 4-year and 6-year assessments of the NESDA study were used. The CIDI was conducted at all assessments and covered the period since the previous interview. For the present paper, we first selected 123 offspring (baseline age: 18-31 years) with a lifetime depressive (major depressive disorder, dysthymia) and/or anxiety (generalized anxiety disorder, social phobia, panic disorder, agoraphobia) disorder at the baseline assessment. We then selected all offspring who participated in the 6-year CIDI interview and for whom diagnostic information covering the entire six-year follow-up period was thus available, resulting in a sample of 105 offspring (response rate: 85.4%). Offspring who did not participate in the 6-year CIDI assessment reported higher depression severity scores (Inventory of Depressive Symptomatology; IDS) and higher disability levels (World Health Organization Disability Assessment Schedule; WHODAS) at the baseline assessment as compared to offspring of whom complete follow-up data was available. No differences were found for anxiety symptom severity (Beck Anxiety Inventory; BAI) and treatment indicators (i.e. antidepressant use and psychological treatment).

Comparison group
To examine whether the course of depressive/anxiety disorders in offspring differed from that of controls without a parental history (PH) of depressive or anxiety disorder, a comparison group was selected from the NESDA study. For this purpose, we first selected the 196 persons with a lifetime depressive/anxiety disorder in the age range of 18-31 years and who were recruited from a random sample of patients of 65 general practitioners and from a
population-based study (The Netherlands Mental Health Survey and Incidence Study). Of these persons, 49 (25.0%) had no PH as assessed with the family-tree method, that asks participants about the presence or absence of a depressive and anxiety disorder in their parents. Response categories were ‘yes’, ‘no’, ‘maybe’ and ‘don’t know’. PH was considered to be absent when there was no ‘yes’ answer for both parents. Of the 49 controls without PH, 33 (77.6%) had participated in the six year follow-up CIDI interview and comprised our comparison group.

**Overall course of depressive and/or anxiety disorders**
To describe the overall course of depressive/anxiety disorders in offspring, we considered the presence of a depressive/anxiety episode during follow-up as well as severity and treatment indicators.

**Presence of depressive/anxiety episode during follow-up (primary outcome)**
Diagnoses of depressive disorder (major depressive disorder, dysthymia) and anxiety disorder (generalized anxiety disorder, social phobia, panic disorder, agoraphobia) were established with the CIDI (version 2.1) at 2-year, 4-year and 6-year follow-up. Together, this information was used to determine whether an episode was present at any moment during the 6-year follow-up period. We additionally reported the type of disorder (i.e., pure anxiety disorder, pure depressive disorder, or comorbid depressive and anxiety disorder) and its persistence over time (i.e., single episode: diagnosis present at one follow-up assessment; chronic course: diagnosis present at two or three follow-up assessments).

**Severity indicators (secondary outcomes)**
*Depressive symptom severity* in the past week was measured with the 30-item Inventory of Depressive Symptomatology (IDS; self-report) at 2-year, 4-year and 6-year follow-up. To determine symptom severity, the highest IDS score over waves was selected.

*Anxiety symptom severity* in the past week was measured with the 21-item Beck Anxiety Inventory (BAI; self-report) at 2-year, 4-year and 6-year follow-up. To determine symptom severity, the highest BAI score over waves was selected.

*Disability* in the past month was measured using the 36-item World Health Organization Disability Assessment Schedule (WHODAS 2.0; self-report) at 2-year, 4-year and 6-year follow-up. The WHODAS inquiries about difficulties in six domains of life: cognition, mobility, self-care, interpersonal interactions, life activities and participation in society. To determine the level of disability, the highest WHODAS score over waves was selected.

**Treatment indicators (secondary outcomes)**
*Antidepressant use* was determined at 2-year, 4-year and 6-year follow-up based on drug container inspection of medication used in the past month and classified according to the Anatomical Therapeutic Chemical (ATC) classification. Antidepressants included selective
serotonin reuptake inhibitors (ATC code N06AB), tricyclic antidepressants (N06AA) and other antidepressants (N06AF/N06AX)\textsuperscript{22} and considered present when taken at least 50\% of the time and reported at any of the follow-up assessments.

*Psychological treatment* was assessed at 2-year, 4-year and 6-year follow-up. Participants reported whether they had received psychotherapy or counseling in the past six months. This information was used to determine whether participants had received psychological treatment during the 6-year follow-up.

**Precise course in offspring with and without a depressive/anxiety episode**

To further characterize the course of depressive/anxiety disorders, we distinguished offspring with and without an episode during six-year follow-up and, then, determined their precise course in terms of severity and treatment. For this purpose, we classified the severity measures into three groups. Severity of depressive symptoms was classified into no (IDS score<14), mild (score=14-25) and moderate/severe (score>25). Similarly, severity of anxiety symptoms was classified into no (score<9), mild (score=10-18) and moderate/severe (score>19). And finally, disability was classified into no (score 0; first tertile based on population norms\textsuperscript{23}, mild (score 1-6; second tertile) and moderate/severe (score>=7; third tertile).

**Potential covariates**

Potential covariates are gender, baseline age, parental educational level, lifetime diagnosis at baseline (pure depressive disorder, pure anxiety disorder or comorbid depressive and anxiety disorder), age of onset and recency of the depressive/anxiety disorder. Parental educational level was defined as highest education of the major breadwinner of household in youth. Scores ranged from 1 (i.e., elementary school not completed) to 9 (i.e., university education). Recency of the depressive/anxiety disorder was classified into past month, past year and longer ago based on the CIDI baseline interview.

**Statistical analyses**

First, we examined the overall course of depressive/anxiety disorders in offspring by considering the presence of a depressive/anxiety episode at any moment during six-year follow-up (primary outcome) as well as the severity and treatment indicators (secondary outcomes). In a second step, we further characterized the course in more detail by distinguishing offspring with and without an episode during follow-up and, then, describing the precise course by considering several severity and treatment indicators.

To examine whether the overall course of depressive/anxiety disorders in offspring differed from that of controls without PH, linear and (multinomial) logistic regression analyses were performed for each of the primary and secondary outcomes and with PH status (yes or no) included as predictor. Analyses were adjusted for those potential covariates that differed in offspring and controls at the $p<.10$ level. Analyses on the precise course in offspring with and without an episode separately were not performed due to the lack of statistical power.
caused by the small number of controls without PH (N=11 and N=22, respectively).

RESULTS

Overall course of depressive/anxiety disorder

In our sample of 105 offspring, 59.0% had a depressive/anxiety episode during follow-up and 41.0% did not (see Table 1). Of the offspring with an episode, 24.2% had a pure anxiety disorder, 30.6% had a pure depressive disorder and 45.2% had a comorbid depressive and anxiety disorder (i.e., 14.3%, 18.1% and 26.7% of the total offspring sample, respectively). Of the offspring with an episode, 56.7% had a persistent course with a diagnosis at two or three follow-up waves. Table 1 further shows the overall course in terms of severity and treatment.

Precise course in offspring with and without a depressive/anxiety episode

Table 1. Overall course of depressive/anxiety disorder in offspring

<table>
<thead>
<tr>
<th>Course characteristics</th>
<th>n(%)/mean (sd)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary outcome</td>
<td></td>
</tr>
<tr>
<td>Episode during follow-up (yes)</td>
<td>62 (59.0)</td>
</tr>
<tr>
<td>Secondary outcomes</td>
<td></td>
</tr>
<tr>
<td>Severity</td>
<td>18.7 (12.0)</td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>10.5 (8.5)</td>
</tr>
<tr>
<td>Anxiety symptoms</td>
<td>19.4 (14.6)</td>
</tr>
<tr>
<td>Overall functioning</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
</tr>
<tr>
<td>Antidepressant use (yes)</td>
<td>20 (20.0)</td>
</tr>
<tr>
<td>Psychological treatment (yes)</td>
<td>55 (55.0)</td>
</tr>
</tbody>
</table>

To further characterize the course, we considered severity and treatment in offspring who either had a depressive/anxiety episode during follow-up or had not (see Figure 1). Of the offspring with an episode during follow-up, 38.2% had moderate/severe depressive symptoms, 23.6% experienced moderate/severe anxiety symptoms and 87.3% reported moderate/severe disability. In addition, 28.3% used antidepressants and 70.0% received psychological treatment at any moment during follow-up. Of the offspring without a depressive/anxiety episode during follow-up, at least mild levels of depressive symptoms, anxiety symptoms or disability were reported by 43.2%, 18.9% and 94.4% of the offspring, respectively. In addition, 7.5% used antidepressants and 32.5% received psychological treatment at some point during follow-up.

Comparison between offspring and controls without PH

Table 2 presents the baseline characteristics of offspring versus controls without a PH of depressive/anxiety disorders. Offspring were younger (p<.001) and had a lower age of onset (p=.002) and further analyses on the course of depressive/anxiety disorders were therefore adjusted for these characteristics. To examine whether the overall course of depressive/anxiety
Figure 1. Precise course of depressive/anxiety disorder in offspring, separately for those with and without an episode during follow-up.
Table 2. Sample characteristics of offspring and controls without a parental history of depressive/anxiety disorder

<table>
<thead>
<tr>
<th>Baseline characteristics</th>
<th>Offspring (N=105)</th>
<th>Controls (N=33)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (female)*</td>
<td>74 (70.5)</td>
<td>27 (81.8)</td>
<td>.200</td>
</tr>
<tr>
<td>Age</td>
<td>22.8 (3.1)</td>
<td>25.6 (3.6)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Parental educational levelb</td>
<td>5.2 (2.1)</td>
<td>5.9 (2.3)</td>
<td>.125</td>
</tr>
<tr>
<td>Lifetime diagnosisa</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive disorder</td>
<td>21 (20.0)</td>
<td>7 (21.2)</td>
<td></td>
</tr>
<tr>
<td>Anxiety disorder</td>
<td>41 (39.0)</td>
<td>11 (33.3)</td>
<td></td>
</tr>
<tr>
<td>Comorbid disorder</td>
<td>43 (41.0)</td>
<td>15 (45.5)</td>
<td></td>
</tr>
<tr>
<td>Age of onsetb</td>
<td>14.9 (4.8)</td>
<td>17.9 (4.6)</td>
<td>.002</td>
</tr>
<tr>
<td>Recency</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Past month</td>
<td>57 (54.3)</td>
<td>13 (39.4)</td>
<td></td>
</tr>
<tr>
<td>Past year</td>
<td>22 (21.0)</td>
<td>8 (24.2)</td>
<td></td>
</tr>
<tr>
<td>Longer ago</td>
<td>26 (24.8)</td>
<td>12 (36.4)</td>
<td></td>
</tr>
</tbody>
</table>

*Based on chi-square statistics. *Based on independent t-tests

Table 3. Associations of PH status with the course of depressive/anxiety disorder*

<table>
<thead>
<tr>
<th></th>
<th>ORa</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary outcome</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Episode during follow-up* (yes)</td>
<td>0.75</td>
<td>0.31-1.83</td>
<td>.530</td>
</tr>
<tr>
<td><strong>Secondary outcomes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Severityb</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive symptoms</td>
<td>0.79</td>
<td>-4.44-6.03</td>
<td>.764</td>
</tr>
<tr>
<td>Anxiety symptoms</td>
<td>-1.77</td>
<td>-5.70-2.16</td>
<td>.375</td>
</tr>
<tr>
<td>Disability</td>
<td>-2.62</td>
<td>-9.19-3.96</td>
<td>.432</td>
</tr>
<tr>
<td><strong>Treatmentd</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antidepressant use (yes)</td>
<td>1.96</td>
<td>0.60-6.41</td>
<td>.264</td>
</tr>
<tr>
<td>Psychological treatment (yes)</td>
<td>1.01</td>
<td>0.42-2.41</td>
<td>.982</td>
</tr>
</tbody>
</table>

* PH=parental history of depressive/anxiety disorder; Odds and B’s are presented for offspring with PH, controls without PH were the reference category; Analyses are adjusted for baseline age and age of first disorder onset. * Based on logistic regression analysis. * Based on linear regression analysis

anxiety disorders in offspring differed from controls without PH, we considered both primary and secondary outcome measures (see Table 3). No significant difference was found in the presence of a depressive/anxiety episode during follow-up (primary outcome; p=.530), nor in the type of disorder (p=.442; not tabulated) and its persistence over time (p=.226; not tabulated). In addition, no significant differences were found in the course of any of the severity and treatment indicators (secondary outcomes; all p-values ≥ .264).
DISCUSSION

Main findings
This study shows that the clinical course of depressive/anxiety disorders is variable in offspring with a parental history of similar psychopathology. Of our offspring with a lifetime depressive/anxiety disorder at baseline, the majority also had an episode during the six-year follow-up period (59%). Although most of these offspring were severely impaired, a substantial proportion met diagnostic criteria at only one follow-up assessment (40%) or reported no or mild depressive (62%) or anxiety symptoms (76%). The remaining offspring did fairly well as they had no diagnosis during the entire follow-up period (41%). Among these, a small part reported suffering from anxiety symptoms (19%), but a large part still experienced at least mild levels of depressive symptoms (43%) or disability (94%). No significant differences in course trajectories were observed in offspring versus controls without a parental history of depressive/anxiety disorders. Notably, these results should be treated with caution as the small sample size of our control group limits our ability to draw strong conclusions.

Comparison with previous studies
In our sample of offspring with a history of depression/anxiety at baseline, 59% had experienced a depressive/anxiety episode during the six-year follow-up period. Comparison to previous studies is complicated by methodological differences, most importantly, the length and number of follow-up assessments, but can be best compared to studies that included a similar sample of offspring of treatment-seeking depressed patients.6-8 It should be noted that these studies focused on depressive episodes and did not report anxiety recurrences rates. Studies using this sample reported MDD recurrence rates of 17% at two-year follow-up8 and of 34% at 10-year follow-up.7 The higher rates for depressive disorder we found during our six-year follow-up period (i.e., 45%; 18% pure depressive disorder, and 27% comorbid depressive and anxiety disorder) may be due to the more closely spaced assessments that were performed in our study which makes failure to recall previous episodes less likely. Wickramaratne and colleagues6 focused on a subsample of offspring with a childhood or adolescent onset of MDD and estimated that 89% experienced a subsequent episode of MDD after age 20. These rates were remarkably higher as compared to the rates we found which may be explained by the increased risk for recurrences associated with early-onset cases.24

To our knowledge, this study was the first to provide a more detailed picture of the course of depressive/anxiety disorders in offspring with a parental history of similar psychopathology by separately considering, for example, its severity in those with and without an episode during follow-up. This is of high importance as previous studies in non-offspring samples indicate that no longer fulfilling diagnostic criteria does not always accompany the complete resolution of symptoms and disability.11-13,25 These residual symptoms are a clinically relevant state of illness as they have shown to be powerful predictors of relapse.26,27 Our study confirms that such an approach is valuable as we observed that many offspring who were diagnosis free during follow-up still experienced increased symptom levels and,
in particular, significant disabilities. And, conversely, we found that meeting criteria for a depressive or anxiety disorder does not always accompany severe symptomatology and high levels of disability. This is in line with studies showing high variability in symptom severity and functioning in depressed/anxious persons.\textsuperscript{13,28,29} In general, when studying the course of psychiatric disorders it is therefore valuable to look beyond the diagnostic labels and to also consider other outcome parameters as they may provide important additional information about patients’ wellbeing.

No significant differences were found in course trajectories in offspring of depressed/anxious patients and controls without PH. Notably, the small sample size of our comparison group of persons without PH limits our ability to draw firm conclusions. Previous high-risk studies as well as studies based on population and clinical samples produced inconsistent results with some studies suggesting that a parental or family history of depression/anxiety predicted recurrence of depressive or anxiety disorders\textsuperscript{8,30-32}, whereas others did not find such an association.\textsuperscript{6,8,24,33-35} Wilson and colleagues\textsuperscript{24} conducted a large community-based study and found that parental depressive disorder was not associated with recurrence after adjustment for earlier age of onset in patients with PH. When studying the impact of PH on the course of depressive/anxiety disorder it is thus useful to consider age of onset.

Another finding that deserves attention is the familial nature of depressive/anxiety disorders. Our study suggests that young persons with depressive/anxiety disorders without affected parents may be atypical as the comparison sample of controls without PH that we ended up with turned out to be rather small (i.e., 25% reported no PH). Previous investigators\textsuperscript{8,30} also came across this issue since they noted that statistical analyses could not be performed because the vast majority of young depressed offspring (i.e., younger than 23 or 16 years, respectively) had a depressed parent. From top-down studies, starting with a depressed/anxious parent and examining rates of psychiatric disorders in offspring, the high-risk status of offspring is well known.\textsuperscript{2,5,9} The current bottom-up finding of relative sparsity of healthy parents combined with affected offspring strongly supports that familial aggregation is the rule rather than the exception.

**Clinical implications**

This study showed that about half of the offspring with a history of depressive/anxiety will suffer from another episode later in life. Relapse prevention programs offer some promise in the prevention of relapse and recurrences\textsuperscript{37-40} and may be worthwhile to consider as structural part of treatment. In addition, the development of strategies to encourage treatment-seeking among young persons’ suffering from these disorders seem to be worthwhile as timely treatment may improve the long-term course.\textsuperscript{41,42} Adult mental health services where vulnerable offspring can be identified via their parents who receive treatment, may be an appropriate setting for implementing such strategies, as well as strategies focusing on prevention of a first episode, as we found that the vast majority of young depressed/anxious persons also have affected parents.
Strengths and limitations
The strengths of our study are the six year follow-up period with biennial assessments, the relatively large sample of affected offspring as compared to previous high-risk studies and the assessment of severity and treatment indicators in offspring with and without a diagnosis separately. However, there are some limitations to be recognized. First, the follow-up period did not necessarily cover the period immediately after first depression/anxiety onset. The ideal study design would be to prospectively measure first onset in both offspring and controls without PH followed by regular assessments of course indicators. However, the costs of such a prospective design are huge, as already illustrated by the relative rare combination of healthy parents combined with affected offspring. Second, the course may be worse than presented as offspring who dropped out of the study reported higher depressive symptom severity and disability levels at the baseline assessment as compared to offspring with complete data who were included in the present study. Third, in contrast to our course indicators based on the CIDI, the severity and treatment variables did not fully cover the entire period since the previous interview. Finally, we observed no significant differences in the overall course of depressive/anxiety disorders in offspring versus controls without PH, but the sample size of our control group was relatively small. Results should therefore be interpreted with caution and larger studies are required.

Conclusion
The prospective course of depressive/anxiety disorders in offspring of depressed/anxious patients is variable. More than half of the offspring followed a recurrent or chronic course while the remaining part had no diagnosis during follow-up. Note that symptom severity and disability levels varied widely between individuals within these two groups indicating that syndromal recovery does not necessarily go hand in hand with the complete resolution of symptoms and restoration of everyday functioning, and vice versa. Furthermore, we found no support for a worse course in offspring with PH as compared to offspring without PH.
REFERENCES
