Summary

The primary aim of this dissertation was to study genetic and environmental influences on problem behaviors in preschool children and adolescents. In chapter 1, it was argued that because of the continuous character of most child psychiatric conditions the methods of the quantitative genetic theory were appropriate, and that disorders should be assessed as quantitative variations of behavior rather than all-or-none categories. The dissertation was divided in two parts. The first part concerned genetic influences on problem behaviors in children and adolescents, and involved genetic analyses on a sample of 11- to 15-year-old international adoptees. The second part addressed problem behaviors in children of preschool age, and involved genetic analyses on a sample of 3-year-old twins.

Part 1. A short introduction to the methods that have been applied to study genetic influences on problem behaviors in children and adolescents, was presented in Chapter 2. This introduction was followed by a survey of findings from genetic studies of the commoner varieties of problem behaviors in children aged 4-18. The small sample sizes in most studies in this review, the different assessment procedures across studies, the use of possibly inappropriate genetic models, and the fact that estimates of genetic and environmental influences are population dependent, made it difficult to draw firm conclusions. To the extent that it was possible to draw general conclusions, it appeared that genetic influences were important to most problem behaviors. Evidence for shared environmental influences was found for antisocial behaviors. The too low DZ twin correlations found for social withdrawal and (hyper)activity, suggested that for these behaviors the commonly used model with additive genetic, shared environmental, and non-shared environmental influences may be inappropriate.

To obtain parental ratings of problem behaviors in the sample of international adoptees, the CBCL/4-18 (Child Behavior Checklist for Ages 4-18) was used. It was planned to use recently derived American CBCL/4-18 syndromes for the genetic analyses. Therefore, in chapter 3, the validity of these syndrome constructs was studied in the sample of international adoptees (N=2,148). Results were cross-validated on a clinical sample (N=1,387). Support was found for the validity of the constructs. However, in the adoption sample, the contribution of a number of items to the scales of the syndrome constructs was questionable. These items had very low variances, were not indicators of just one construct, or did not improve the reliability of the scale.

In chapter 4, American CBCL/4-18 syndromes, adapted to the Dutch sample of international adoptees, were used to study genetic and environmental influences on problem behaviors. The sample (mean age 12.43 years) comprised a group of biological siblings (111 pairs), a group of non-biological siblings (221 pairs), and a group of singletons (94). Non-shared environmental influences were most important. Genetic influences were substantial for externalizing behaviors, but unimportant for internalizing behaviors. For the CBCL total problem score, Attention Problems, and externalizing behaviors results were in agreement with findings from twin studies. The lack of genetic influences on internalizing behaviors was in contrast with results from twin studies. For the Externalizing grouping, Delinquent Behavior, and Aggressive Behavior, variances for singletons were significantly smaller than for siblings. Model fit indices indicated that these differences in variances are better attributed to smaller effects of factors associated with sibship size, than to active influences of siblings on each other. Significant sex differences were found for 7 of the 10 scales. The larger variances for boys on the Externalizing grouping and Aggressive Behavior were caused by genetic influences.

Part 2. For the genetic analyses on the sample of 3-year-old twins, the CBCL/2-3 (Child Behavior Checklist for Ages 2-3) was used to obtain parental ratings of problem behaviors. In chapter 5, the Dutch factor structure of the CBCL/2-3 investigated with three different samples - children referred to mental health services, children from the general population, and the sample of twin pairs. A series of exploratory and confirmatory factor analyses indicated a seven-factor model for all three samples. Syndromes were labeled Oppositional, Withdrawn/Depressed, Aggressive, Anxious, Overactive, Sleep Problems, and Somatic Problems. Internal consistency estimates, test-retest stability, and interparent agreement were moderate to high for the seven factors. Factor intercorrelations and a second-order factor analysis provided support for two groupings of problem behaviors - Externalizing and Internalizing.

In studying twin populations it is important to be able to generalize findings from the twin sample to the general population. The representativeness of the twin sample was studied in chapter 6. Maternal ratings of problem behaviors in twins, were compared with ratings of 2-3-year-old singletons whose mothers completed the CBCL/2-3. The twin sample consisted of 1281 twin pairs (407 MZ, 874 DZ), the singleton sample consisted of 420 children from the general population. Results indicated that the general level of problem behaviors in twins was broadly comparable to that in singletons. Five of the seven scales showed lower scores for DZ twins versus MZ twins and singletons. However, these differences were small. Standard deviations for 2 of the 7 scales were somewhat smaller for singletons than for twins. Higher means for boys were found for the total problem score, and the Aggressive and Overactive syndromes.

Chapter 7 reported, for the separate syndrome scales, the genetic analyses on the parental ratings of problem behaviors in their 3-year-old twins. The sample consisted of 218 MZ female, 189 MZ male, 233 DZ female, 252 DZ male, and 389 DZ opposite sex pairs. Both parents completed one CBCL/2-3 for each child. Model fit indices indicated that mothers and fathers assessed similar behaviors in their children. Genetic influences accounted on the average for 65% of the trait variance. Shared environmental influences accounted on the average for 12%, and non-shared environmental influences for 21% of the trait variance. Sex differences in genetic and environmental influences on problem behaviors were small. Evidence for sibling contrast effects was found for the Anxious and Overactive syndromes.

In chapter 8, multivariate genetic models were fitted to study patterns of problem behaviors in 3-year-old twins (446 MZ, and 912 DZ twin pairs). Fathers' and mothers' ratings of problem behaviors were obtained with the CBCL/2-3. A biometric model with two common genetic, one common shared environmental, and two common non-shared environmental factors fitted almost as well as the saturated unconstrained model for the genetic and environmental covariances. The common non-shared environmental factors produced externalizing/internalizing patterns of problem behaviors. One common genetic factor produced a clustering of the Oppositional, Withdrawn/Depressed, and Overactive syndromes with the Aggressive syndrome. The other common genetic factor produced a clustering of the Oppositional, Withdrawn/Depressed, and Overactive syndromes with the Anxious syndrome. A pattern of similar scores on all dimensions of problem behavior was most suggestive of the common shared environmental factor.

In the final chapter, chapter 9, results from the genetic analyses were discussed. Attention was paid to the interpretation of the findings, and issues concerning use and misuse of genetic findings were addressed. It was argued that heritabilities found in the present study imply that children show innate differences in liability to problem behavior,

and that misuse often is associated with misinterpretations of the quantitative genetic theory.