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Children of Alcoholic Parents. A Review

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Children of alcoholics are prone to genetic, environmental, and teratogenic risk factors. This review starts by outlining the developmental risks due to intrauterine exposure to alcohol. Furthermore, the overall findings from genetic research are summarized. A further section deals with the analysis of the environment of the family with an alcoholic parent. Within the section on psychopathology the special links to conduct disorders and delinquency, hyperkinetic disorders, substance abuse, anxiety and depression, and somatic problems are described. Special consideration is also given to the literature dealing with cognitive and neuropsychological functioning in the offspring of alcoholic parents. Finally, the limitations of current knowledge are emphasized.

Keywords: Alcohol Abuse, Child Psychiatry, Children, Parents, Offspring, Risk Factors

Introduction

Alcoholism represents one of the most common and deleterious psychiatric disorders throughout the world. Besides the devastating effects on the patients, the society, the community, and the more personal social environment of friends, relatives, and especially the immediate family suffer from enormous burdens inflicted by alcoholism. Children of alcoholics, who-according to some estimations in the United States of America-comprise at least 10 percent of the population (Sher, 1991), are specifically prone to a variety of risks.

First, largely but not entirely due to genetic risk factors, they may themselves develop alcoholism during their lifetime. Furthermore, they are exposed to an increased risk of developing other or comorbid psychiatric disorders. All these disorders, theoretically, manifest themselves as a consequence of genetic or environmental risk factors or an interaction of these two conditions. In the recent past it has become evident that, besides genetic and environmental factors, there is a third source of risk factors for the developing child of alcoholic parents that originates in the teratogenicity of alcohol. Thus, children born to alcoholic mothers are at specific developmental risk due to intrauterine exposure to alcohol. The following review will start with these effects of maternal alcoholism during pregnancy on the offspring and will then proceed to a brief sketch of the overall findings from genetic research followed by an analysis of the family environment with an alcoholic parent. Further sections will deal with the psychopathology and cognitive functioning in children of alcoholic parents.

The Effects of Prenatal Alcohol Exposure

The recent interest in the effects of intrauterine exposure to alcohol originated approximately two decades ago when Jones and Smith (1973) described a distinct pattern of abnormal morphogenesis and CNS dysfunction. The authors introduced the term "fetal alcohol syndrome" (FAS) to describe this pattern of features that is characterised by craniofacial malformations, stunted growth, delayed psychomotor maturation, and impaired intellectual development. The incidence of FAS is estimated at 1–2 per 1000 live births (Abel & Sokol, 1987). The less serious diagnosis of fetal alcohol effects (FAE) (Clarren & Smith, 1978; Rosett, 1980) is applied to patients exposed to alcohol in utero with some partial FAS phenotype

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and or CNS dysfunction but without sufficient features for a firm diagnosis of FAS. The less severe form – i. e., FAE – is estimated to occur several times more often than FAS.

FAS has been described in hundreds of clinical reports on individual children (for a review see Clarren & Smith, 1978). In the recent past, most notably European groups in Germany (Steinhausen et al., 1982a, b; Steinhausen et al., 1984; Spohr & Steinhausen, 1987; Majewski & Majewski, 1988; Spohr et al., 1993; Steinhausen et al., 1993; Steinhausen et al., 1994) and Sweden (Aronson et al., 1985), but also the Seattle group in the U. S. (Streissguth, 1992) reported on larger series of patients. All these reports contain a plethora of relevant paediatric data, which will not be summarised here. The focus will rather be on the psychiatric and psychological findings of these studies.

From its first description it was evident that FAS is associated with an extremely high rate of mental retardation and borderline intelligence. This was known even before children suffering from FAS were systematically studied. For instance, Hagberg et al. (1981) found alcohol fetopathy in 8% of an unselected series of 91 Swedish school children with mild mental retardation. In their series of 175 children with FAS of varying ages, including about 50 school-aged children, Majewski and Majewski (1988) described 83% as being mentally retarded. In the Berlin follow-up study of 158 children, including 70 school-aged children, we found 34% of the patients with borderline intelligence and 31% with mental retardation. In the Swedish series, one half of the children had borderline or retarded mental development and, in general, had significantly lower mental abilities than their mothers (Aronson et al., 1985). In the first major report on adolescents and adults with FAS the Seattle group (Streissguth et al., 1991) found that a little more than half of the patients were mentally retarded.

When describing the behavioural features of FAS, early reports always stressed the delayed development and a high frequency of hyperkinetic disorders in the afflicted children (Steinhausen & Spohr, 1986). Our own studies were the first to systematically assess developmental history and psychopathology in these children. Besides maternal and even paternal alcoholism, when analysing the developmental history, we found an increased rate of neonatal risk factors and indices of retarded development throughout infancy and the toddler period (Steinhausen et al., 1982a, b). Systematic psychiatric assessment in a control group design revealed that eating and sleeping problems, stereotypies, retarded speech and language development, hyperkinetic disorders, relationship problems, and emotional disorders were significantly more frequent in FAS children (Steinhausen et al., 1982a, b). These studies revealed that, in terms of dysmorphic features, the most severely damaged children were also the ones with the most marked psychiatric symptoms.

The Berlin study also comprises the largest cohort with the most extended follow-up periods that have been analysed so far. It was shown that in a 10-year follow-up the characteristic cranio-facial malformations diminished with time, but microcephaly and, to a lesser degree, short stature and underweight (in boys) persisted. The outcome was less favourable in terms of psychopathology and intelligence (Spohr et al., 1993; Steinhausen et al., 1993; Steinhausen et al., 1994). After extended follow-up periods extending into late adolescence in some cases, hyperkinetic disorders, emotional disorders, sleep disorders, and abnormal habits and stereotypies persisted over time. Even in the longitudinal perspective, the severe morphological damage was correlated with a high number of psychiatric symptoms and greater impairment of intelligence. There was no indication in the Berlin study that either postnatal milieu or remedial therapy contributed to the outcome.

Besides systematically assessing children who suffer from FAS or FAE, even the long-term consequences of prenatal exposure to lower levels of alcohol were analysed in various studies. Most notably, this was done in the Seattle Longitudinal Prospective Study on alcohol and Pregnancy (for reviews see Forrest et al., 1992; Streissguth, 1992).

This study on so-called "social drinkers" is based on a cohort of approximately 500 children who were examined at various ages and whose mothers were interviewed during pregnancy regarding alcohol and other drug use. Adverse effects of alcohol consumption during pregnancy were shown on various levels. In neonates it was manifested by poor new-born learning and behaviours, decreased sucking pressure, poor habituation, and low arousal. During infancy the signs were poor mental and motor development. At pre-school age the indictors were poor attention, slower reaction time, and a decrement of 5 IQ points in children of mothers drinking more than 250 grams of alcohol per week. At the age of seven years the adverse effects were still apparent in terms of poor atten-

tion, distractibility, slower reaction time, poor performance in further neurobehavioral tests, and a decrement of 7 IQ points when the mother drank more than 165 grams of alcohol per week. A recent further follow-up assessment of this cohort indicates that, even at the age of 14 years, prenatal alcohol exposure was significantly related to deficits in attention and memory in a dose-dependent fashion (Streissguth et al., 1994).

Whereas neonatal problems as a consequence of prenatal alcohol exposure were universally observed in other studies, too, the evidence for adverse effects beyond the neonatal stage is less convincing, as indicated by a recent analysis of the literature by Forrest et al. (1992). Based on this critique and aiming at studying the dose response curve at high consumption levels, a collaborative European study with nine participating centres, including more than 8400 subjects, recently studied the relation between maternal alcohol consumption and pregnancy outcome. This study showed an association between infant's body size and maternal alcohol consumption at levels of about 140 grams per week or more either before or in early pregnancy. From this study it was recommended that women abstain from drinking alcoholic beverages during pregnancy. If this is not possible because of social pressures, pregnant women should not drink more than one standard drink a day (Euromac Project Group, 1992).

Given these findings on the deleterious effects of prenatal exposure to alcohol, the question as to the confounding effects of cigarettes or other drugs has been advanced, because many alcoholics also abuse these substances. However, the issue of disentangling these various effects is very complicated. When looking at the effects of smoking cigarettes in general, various studies and reviews have concluded that exposure to nicotine during pregnancy is associated with deficits in physical growth, neurological functioning, cognitive development, school achievement, and even behavioural adjustment (Naeye, 1992; Tong & McMichael, 1992; Olds et al., 1994). However, no dysmorphic syndrome has been found to be associated with exposure to nicotine, and the cognitive and behavioural abnormalities are only slight.

Similarly, the effects of illegal street drugs is remarkably different from alcohol insofar as there are no clear teratogenic effects leading to a distinct dysmorphic syndrome. Whereas perinatal problems such as prematurity or small size for gestational age are quite common and certain drugs lead to withdrawal symptoms in the new-born, the effects on cognitive and behavioural functioning are again only slight. Current knowledge does not allow us to evaluate the long-term development of these children (Neuspiel & Hamel, 1991; Zuckerman & Bresnahan, 1992; Brooks-Gunn et al., 1994).

As with the studies on nicotine effects, it may be argued that confounding effects of prenatal exposure to alcohol has been ignored in most studies investigating the effects of illegal street drugs. Two recent studies tried to disentangle the differential effects of various substances and came up with rather conflicting findings. Fried et al. (1992) obtained an association between cognitive and verbal functioning and prenatal exposure to nicotine, whereas low levels of alcohol consumption and maternal use of marijuana were not found to be related to the outcome measures. In contrast, the Seattle Longitudinal and Prospective Study in the most recent analysis of attention and short-term memory in 14-year-olds reported that alcohol associations were not mitigated by smoking or other drug use during pregnancy (Streissguth et al., 1994). Thus, further studies on the interaction of various drugs and their effects on the foetus are clearly warranted.

In conclusion, there is overwhelming evidence that maternal alcoholism during pregnancy has devastating effects on child development. The affected children continue to manifest developmental disabilities, psychiatric disorders, and cognitive impairment as they mature. The more severely they are affected morphologically, the more they suffer from impaired development. Their attentional and cognitive deficits render them vulnerable for learning difficulties and poor school careers. Their psychiatric problems and deficits in adaptive behaviour impose specific burdens on their caretakers and high costs on the community at large. In the severely affected cases there is little evidence that postnatal environment or remedial therapy can reverse the adverse effects of prenatal exposure to alcohol.

The issue of the effects of maternal social drinking during pregnancy is less clear. Whereas there is no doubt that there are adverse effects on the neonate, so that drinking alcohol during pregnancy should be avoided, the development at later stages needs further studies. The alarming findings of the Seattle longitudinal study concerning later development require further replications.

Genetic Influences

There is strong evidence coming from a large series of studies that alcohol use and abuse are genetically determined. These findings will be only briefly summarised here (for reviews see Searles, 1988; Merikangas, 1990; Sher, 1991). The evidence of the familial transmission of alcoholism has been scientifically supported by family studies, twin studies, and adoption studies, thereby adding to a lot of historical and folklore type knowledge.

Family studies have shown that there is an average seven-fold increase in the risk of children of alcoholism among the first degree relatives of children of alcoholics as compared to children of non-alcoholic parents. The relative risk of alcoholism is greater in male than in female relatives. This sex difference obviously cannot be attributed to transmissible genetic factors.

Twin studies based both on either registries of normal twins or on pairs in which one member was identified through an alcoholism treatment program have shown that the concordance rates for monozygotic pairs are significantly higher than for dizygotic pairs.

Finally, adoption studies, most notably performed in Denmark, (Goodwin et al., 1974, Goodwin et al., 1977) and in Sweden (Bohman et al., 1981; Cloninger et al., 1981; Bohman et al., 1987) documented that there is a 21/2 times greater chance of an adoptee developing alcoholism-irrespective of exposure to the alcoholic parent-if a biological parent is alcoholic. The authors of the Swedish adoption studies delineated two subtypes of alcoholism based on their findings. Type I is comprised of equal number of males and females with onset after the age of 25 and is associated with anxiety and depression syndromes. In contrast, Type II predominantly affects men and is basically a heritable form with early onset heavy drinking and comorbid antisocial personality and criminality.

In summary, family, twin, and adoption studies suggest that a considerable proportion of the familial aggregation of alcoholism can be attributed to genetic factors. However, the specific inherited components of alcoholism are still unknown. The respective literature demonstrates a certain amount of inconsistencies, and a critical analysis of the findings suggests that environmental influences in their interaction with the genetic transmission have been under emphasised in their potential as further significant factors (Searles, 1988).

The Family Environment with an Alcoholic Parent

A large number of studies of both genetic and environmental factors in the development of alcoholism and other psychiatric morbidity indicate that psychosocial determinants are operant. However, the specific environmental variables that may contribute to alcoholism and other psychopathology have not been well delineated. Furthermore, certain characteristics of the home environment of the alcoholic parent do not necessarily imply causality in the development of disorders in the children. With this caveat in mind, the following description of the family environment of alcoholic parents addresses the most salient features that create further risk factors for the developing child.

Although a strong relation between family violence and parental alcoholism has been assumed by many clinicians, the empirical evidence is less convincing because of a number of methodological shortcomings pertaining to the ascertainment of violence and alcohol abuse, the sampling procedures, and the lack of adequate controls. From recent reviews it appears that the relation between alcoholism and spouse abuse is stronger than the relation to child abuse, including sexual abuse, and that this relation is stronger for the lower social class (Sher, 1991, 1992).

There is also a large series of studies using self-reports or behavioural observations to assess family interactions in alcoholic families. Reviews of these approaches (Jacob & Seilhamer, 1987; Sher, 1991, 1992) indicate that alcoholic families are characterised by higher levels of conflict and lower levels of cohesion, impaired problem-solving, and more negative and hostile communications relative to non-alcoholic families. However, this research also clearly shows a great deal of heterogeneity in the interaction patterns among alcoholic families and that disturbed family interaction is not specific to these families. Furthermore, it is difficult to generalise from these laboratory studies using volunteers to the community where the comorbidity is higher and the rate of intact nuclear families is expected to be lower.

So far, most of the research on family interaction in alcoholic families has not analysed the relation between family characteristics and offspring adjustment. From the limited knowledge, it seems that relapse of alcoholism-particularly in fathers, the ongoing stress due to a drinking parent, and the

severity of the alcoholism are crucial variables that are related to problems of adjustment in children.

Finally, it has to be assumed that modelling of parents' drinking could, in principle, represent an important factor of causality for at least some alcoholics. Parental alcohol use has been proven to be an important correlate of alcohol use in adolescence (Sher, 1991).

Although, in summary, there is some evidence that certain families with an alcoholic parent are marked by a negative milieu with adverse effects on the family members, one has to conclude that most of these effects are characteristic for the periods of active alcoholism. Recovering alcoholics do not have the same effects on family life. Active alcoholism leads to disturbed family interaction patterns that are also found in other kinds of parental psychopathology.

Psychopathology in the Offspring of Alcoholic Parents

Another large number of studies indicates that parental alcoholism is associated with a wide range of behavioural problems and psychopathology in the offspring. Recent reviews of this issue were provided by Earls (1987), West and Prinz (1987), von Knorring (1991) and Sher (1991, 1992). The studies show that the offspring are at risk for the development of a variety of disorders, although only a minority of all children of alcoholic parents are affected and such outcomes are not specific to this group of children. These disorders do not necessarily have to progress to adult manifestations of psychiatric disorders. Furthermore, little is known about protective factors that might explain why the majority of children of alcoholic parents do not develop psychiatric disorders. According to findings from the Kauai study, the offspring of alcoholics who did not develop any serious coping problems in childhood and adolescence were characterised by temperamental features that elicited positive attention from primary caretakers, higher intelligence, and communication skills, achievement orientation, a responsible and caring attitude, a positive self-concept, a more internal locus of control, and a belief in self-help (Werner, 1986).

The available literature shows that certain disorders are most likely associated with parental alcoholism. These types of psychopathology will be considered in the following and include conduct disorders and delinquency, hyperkinetic disorders, substance abuse, anxiety and depression, and somatic problems.

Conduct Disorders and Delinquency

There is a strong relation between parental alcoholism and conduct disorders, especially in sons (Rydelius, 1981; Steinhausen et al., 1984; Earls et al., 1988; Reich et al., 1993). However, the parental disharmony and the environmental disruption caused by parental alcoholism may be more influential than any direct influence of alcoholism. Similarly, adolescent delinquency in the children of alcoholics is more closely related to family discord (West & Prinz, 1987). In addition, there is a better chance that delinquent acts in these subjects are detected because of greater awareness of various social agencies supervising families with an alcoholic parent (Rydelius, 1981).

Hyperkinetic Disorder

Older family studies of hyperkinetic children have shown a higher prevalence of alcoholic and sociopathic fathers in the respective pedigrees, and a genetic relation has been postulated (Cantwell, 1976). However, the more recent studies came up with mixed findings. When studies began with alcoholic parents and then assessed the children, some association with hyperactivity was found. In contrast, this association did not emerge in studies in which the index sample consisted of hyperkinetic children (West & Prinz, 1987). None of the studies obtaining a positive association has controlled for prenatal exposure to alcohol and its strong association with hyperactivity. Furthermore, the lack of differentiation between hyperkinetic disorders with and without comorbid conduct disorders restricts clear conclusions. Several studies indicate that the association of conduct disorder with parental alcoholism is stronger than that of hyperkinetic disorders (Knop et al., 1985; Merikangas et al., 1985; Steinhausen et al., 1984).

Substance Abuse

The relation between parental alcoholism and alcohol abuse in adolescents has been documented in various studies (Rydelius, 1983; Merikangas et

al., 1985) but has not been confirmed in others (Knop et al., 1985; Johnson et al., 1989). On the other hand, in contrast to finding no increased rate of alcohol abuse, one study revealed that children of alcoholic parents were more likely to report abuse of various other substances, i. e., cannabis, speed, and cocaine (Johnson et al., 1989). In a recent study, Reich et al. (1993) also reported a higher use of alcohol and other substances but no abuse or dependence.

Emotional Disorders

In general, there is sufficient evidence from various studies indicating that children of alcoholic parents do suffer from an increased rate of emotional disorders and symptoms (for reviews see West & Prinz, 1987; von Knorring, 1991). Only a few studies have gone beyond the level of reporting a variety of emotional problems and have assessed the prevalence of emotional disorders as defined by the major international classification schemes. Accordingly, the most recent study reported an increased rate of over anxious disorders, as defined by DSM-III criteria, in the offspring of alcoholics, whereas the rate of depression did not differ from that of controls (Reich et al., 1993). In another study Steinhausen et al. (1984) found that 67% of the children with alcoholic mothers as having an emotional disorder according to ICD-9. The rate was 59% in children with two alcoholic parents and only 31% in the children with alcoholic fathers.

However, as in previously described psychopathology, one should caution against attributing emotional disorders in the offspring directly or entirely to parental alcoholism. Psychiatric comorbidity in the parents, disharmony in the family, and other environmental stress factors certainly have a confounding effect on the development of anxiety, depression, and further emotional problems in the offspring.

Somatic Problems

Only a few studies assessed the physical health status in children of alcoholic parents. Whereas Moos and Billings (1982) found only a trend for children of relapsed alcoholics to have a higher number of physical problems, there are three studies reporting a significant relation of impaired child physical health and parental alcoholism. In the study by Roberts and Brent (1982) female subjects had significantly higher physician utilisation rates and diagnoses than matched controls. Steinhausen et al. (1982a) found higher rates of outpatient therapy, eating problems, headaches, and sleeping problems in children of alcoholics when compared to children of epileptic or of healthy mothers, respectively. In the third study (Biek, 1981), which used a predominantly female sample, adolescent medical clinic outpatients with a problem-drinking parent were found to have nearly twice as many somatic complaints and health concerns as those without a problem-drinking parent. Again, these studies do not report on further family stressors that may play a role in the development of physical symptoms.

Cognitive and Neuropsychological Functioning in the Offspring

There are a number of studies reporting significantly lower IQ scores for children of alcoholics than for controls (reviewed by West & Prinz, 1987). However, the majority of these studies were undertaken to analyse the effects of prenatal exposure to alcohol, whereas others did not control for these effects. Unfortunately, this lack of control for prenatal exposure to alcohol also applies to a recent longitudinal, controlled population-based study in which children of alcoholic parents had poorer mental development up to the end of their fourth year of life (Nordberg et al., 1993). In other samples, there is less evidence of impaired intelligence. When Johnson and Rolf (1988) studied children of recovering alcoholics from a non disadvantaged background, they did not find statistically significant differences in intelligence. Similarly, Tarter et al. (1984) did not reveal differences in intelligence when they studied the adolescent sons of alcoholic fathers and matched controls. One may hypothesise that the neurotoxic effects of alcohol in prenatal life is the critical variable for any impairment of intelligence.

Beyond global measures of intelligence, the study of further cognitive and neuropsychological functioning has been the aim of various studies. As recently reviewed by Sher (1992), there is evidence that children of alcoholics have poorer verbal ability than controls, although the performance

is within normal limits and the deficit is only relative. Studies for a similar deficit in visuospatial abilities are less consistent, whereas learning and memory are not affected. Besides verbal ability, abstraction and conceptual reasoning are other areas in which deficits in children of alcoholics were detected. These deficits may contribute to school failure and, consequently, to an impairment in self-esteem and problems in behaviour and adaptation as well. In fact, several studies have shown that academic performance and school careers are more frequently negatively affected in children of alcoholic parents than in controls (for reviews see West & Prinz, 1987; Sher, 1992). However, the complex net of interactions between neuropsychological deficits, school failure, and behavioural problems in these children at risk has not been sufficiently disentangled.

Concluding Comments

Despite a number of inconsistencies resulting from the heterogeneity of the studies, and despite various methodological shortcomings that were precisely outlined in the recent monograph by Sher (1991), there is abundant evidence that children of alcoholic parents are children at risk. First, a large series of studies originating from the new field of behavioural teratology has convincingly shown that prenatal exposure to alcohol has devastating effects on development. Fortunately, this is also an area of research in which the consequences in terms of prevention and intervention in the fields of medical policy and practice are easy to delineate. This is by far more difficult for our current understanding of the findings coming from genetic research. Whereas there is no doubt that a significant proportion of the familial aggregation of alcoholism can be attributed to genetic factors, the specific components of alcoholism that may be inherited and the mechanisms for the transmission of alcoholism have yet to be identified. Thus, consequences for prevention are more difficult to delineate.

When looking at the environmental risks for the development of the offspring of alcoholic parents, it is by far more complicated to derive similar action-oriented conclusions from a complex field of research. Here we are confronted with a large body of both knowledge and ignorance. As previous reviews of the field have stated, there is considerable evidence that parental alcoholism is disruptive to family life and that a large proportion of children of alcoholic parents suffer from psychiatric disorders both of an externalising and an internalising type. However, as concluded by West and Prinz (1987) in their excellent review, more should be known about the individual differences and the impact of the developmental level and gender of the offspring of alcoholic parents. As in other fields of research in child psychopathology, comorbidity of disorders and specificity of disorders are largely neglected topics in the study of children of alcoholics. Similarly, a widely ignored area of research concerns the resiliency found in a large number of children who manage to cope positively, despite apparent distress from the environment.

So far, most of the research literature has ignored the fact that in a considerable number of families both parents are alcoholics. This may have potentiating adverse effects in terms of an extremely disorganised milieu with an increase of risk factors to the child. Besides the obvious environmental deficits in terms of negligent and abusive or violent rearing, an interaction with further biological risk factors is operant in these families. The latter include the consequences of in utero maternal ingestion of alcohol and may also be hypothetically extended to damage to paternal germ cells from alcohol with still unknown consequences to the child.

Currently, the most complicated issue for research in this area of studies on the children of alcoholics is the inference of causal pathways. Both mediating and moderating (buffering) factors have to be assumed and deserve carefully designed investigations. Mediating factors pertaining to the family – e.g., disrupted family interactions or marital discord-or characterising the sick parents – e.g., severity of the alcoholism or sex-specific drinking patterns-still have to be isolated when studying the development and outcome of children of alcoholic parents. Similarly, the potential moderating effects of social class, family life, social support, personality features, and other individual characteristics deserve further investigation. Scientific progress in the understanding of these complex issues will certainly contribute to interventions aimed at reducing the various risks that children of alcoholic parents are exposed to.

Résumé

Les enfants d'alcooliques sont exposés à des facteurs de risque génétiques, environnementaux et tératogènes. Cette revue commence par résumer les risques développementaux dues à l'exposition intra-utérine à l'alcool. Ensuite les résultats des recherches génétiques sont résumés. Un autre chapitre traite l'analyse de l'environnement de la famille avec un parent alcoolique. Dans le chapitre psychopathologie, les liens spéciaux avec les troubles des conduites et la délinquence, les troubles hyperkinétiques, la toxicomanie, l'anxiété et la dépression et les troubles somatiques sont décrits. La littérature traitant du fonctionnement cognitif et neuropsychologique des enfants de parent alcooliques fait l'objet d'une attention particulière. Enfin, on insiste sur les limites des connaissances actuelles.

Zusammenfassung

Kinder von alkoholkranken Eltern sind genetischen, umweltbezogenen und teratologischen Risikofaktoren ausgesetzt. Diese Übersichtsarbeit beginnt mit einer Darstellung der Entwicklungsriskiken durch die intrauterine Alkoholexposition. Ferner werden die Übersichtsbefunde der genetischen Forschung zusammengefaßt. Ein weiterer Abschnitt beschäftigt sich mit der Analyse der familiären Umwelt mit einem alkoholkranken Elternteil. In dem Abschnitt zur Psychopathologie werden die speziellen Verbindungen zu dissozialen Störungen und Delinquenz, hyperkinetischen Störungen, Drogenmißbrauch, Angst und Depression sowie somatischen Symptomen beschrieben. Besonders berücksichtigt wird auch die Literatur, die sich mit kognitiven und neuropsychologischen Funktionen bei den Kindern alkoholkranker Eltern beschäftigt. Schließlich werden die Grenzen des gegenwärtigen Wissens betont.

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