

*Recent Developments in the Effects of Early Parental  
Intervention and Subsequent Adaptation of Children, Adolescents  
and Adults with Commonly Diagnosed Mental Health and Cognitive  
Disorders: An Indictment of Poverty and Dysfunctional Parenting*



## **Centre for Family, Child & Adolescent Advancement**

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"On the basis of years of research, I am totally convinced that the first priority with respect to helping each child to reach his maximum level of competence is to do the best possible job in structuring his experience and opportunities during the first three years of life." (Burton White, 1975, p. 284).

### **Introduction**

*Early intervention.* While inevitably qualified by recent advances in our knowledge of child and adolescent development, Burton White's (1975) central claims, made thirty years ago, are essentially still valid today. White felt that the average American family has sufficient resources to complete this task of good parenting. But those parents who are poverty stricken and intellectually, neurologically or psychiatrically impaired are faced with an overwhelmingly daunting task. More often than not, such parents are incapable of providing adequate child care and require outside community assistance. Failure to intervene in a timely and effective fashion often sentences the developing child to needless intellectual and social decline, thus perpetuating an intergenerational pattern of functionally and economic dependency (Evans, 2004; McCain & Mustard, 1999; Gerber, 1988).

The long-term effects of such early parental privation are far-reaching in their implications for competent development. For our purposes, learning disorders are clinical conditions that impair thinking and learning. As we show, whether the learning disorders are in terms of mental *retardation* (*MR*) or developmental delay (*DD*), learning disabilities (*LD*), attention deficits (*ADHD*), fetal alcohol effects (*FAE*), or brain injury (*BI*), the quality of the

early parental environment and early clinical intervention is a vital consideration. While not discounting the legitimate need for respite care and community support for any overtaxed but otherwise competent parents, financially and socially stable families seem relatively more able to adequately cope with their children's and adolescents' learning problems. Such children tend to far much better as adults.

While the thrust of much of the recent literature supports White's (1975) claim, it is still true that the periods of childhood and adolescence certainly have their own potential vulnerabilities. However, the developmental risks and the most telling opportunities to overcome them occur most profoundly during the first few years of life. But overstating the impact of early experience is also misleading. It is patently incorrect and overly too simplistic to expect that quality care in the early years alone can sufficiently insulate children from subsequently emergent developmental problems, such as serious family crises. Hence, the larger issue of the prepotency of early or preschool intervention acting as a uniformly protective shield against educational decline and delinquency is now qualified by the nature of subsequent childhood and adolescent experiences (e.g., Kagan, 1996; Thompson & Nelson, 2001).

For example, Reynolds, Ou, and Topitzes (2004) found that the positive effects of early intervention with vulnerable low income minority children are heavily mediated by their subsequent enrolment in high quality elementary schools, lower mobility, higher parental involvement in school and low levels of child maltreatment. However, as Garber (1988) and White (1975) would have anticipated, the impact of the preschool experience is still quite telling. Or instance, Feinstein and Bynner (2004) found that, *while middle childhood*

*SES changes strongly affected adult outcomes, those children who escaped from the bottom SES quartile developed similarly to those who remained. Thus, people do not have clinically significant disorders functionally disconnected from the worlds in which they live (e.g., Reiss, Collins & Bersheid, 2000).* Parental neglect, maternal alcohol and drug abuse, and trauma all conspire with cultural and socio-economic marginalization to produce or aggravate chronic learning disorders. Thus, the practitioner in the field is often, if not usually, faced with clients having a number of presenting issues initially acquired within a compromised preschool learning environment.

*Bidirectionality.* It is very difficult to disentangle the familial and socio-economic environment from the disorder itself in assessing outcomes. Disproportionately, developmentally or neurologically compromised children ((e.g., with conduct disorders (CDs); attention deficit hyperactivity disorders (ADHDs) or learning disabilities (LDs)) are trying to function within functionally impaired parental environments (e.g., Anderson, Lytton & Romney, 1986; Smith, 2000). Delayed parents also compromise the parenting situation. The net result is a vicious circle: the parents often react to their dysfunctional and disruptive children in ways that aggravate instead of reduce the clinical problem. These *bidirectional* influences can be quite significant: parental harshness and impatience can easily be misread by the uninformed as causes rather than consequences of the child's unusual behavior. This it is less commonly the case that developmental disabilities are dealt with within a healthy and supportive family and school setting in which interventions are well-timed, targeted and efficient. It is far less likely that such resourceful families need significant interventions and their children's cognitive disabilities are more apt to have relatively optimum outcomes. Curiously, it is the middle income parent who is more

likely to take advantage of school contacts and community support (Evans, 2004). Those who need help the most are least apt to seek it.

*Poverty.* As we shall learn, however, it is often not easy to separate issues such as neglect and poverty from the presenting symptoms of a learning disorder. Our review shows that supportive and caring parents often do really quite well, for example, to their (*karotypically*) developmentally delayed (*DD*) children. However, those parents of children with acquired problems, such as those stemming from neglect and fetal alcohol syndrome (*FAS*), are significantly more often themselves deficient in basic parenting skills and are also often relatively more biologically and behaviorally vulnerable to having disabled offspring. Evans (2004) provided a stellar review of the damaging multidimensional effects of poverty on young children.

*Genetic damage.* In contrast to acquired disabilities, developmental delay (*DD*) caused by serious genetic damage (e.g., Down's syndrome (*DS*)) may be more independent of environmental contexts and is often also accompanied by physical problems. For example, accompanying physical problems of *DD* include mid-life osteoporosis (Center, Beange and McElduff, 1998). (Parenthetically speaking, we have less difficulty accepting a disability when it is a physical one, as the effects are obvious and indisputable. In this context, accompanying *DD* is somehow easier to accept.) While helpful, compensatory training is also less successful in altering overall functional outcomes with these populations as well. Tyler, Snyder and Zyzanski (2001) found that lower *Behavior Management Disorder (BMD)* quotients in preschoolers were better predicted by *DS* status than by socio-economic status (*SES*) or maternal age, sex, or race. Also, Nesor, Molteno and Knight (1989) found that, with age, preschool children with *DS* decreased in developmental

quotient (*DQ*) regardless of these other factors. While none of these findings are meant to suggest that useful interventions are not warranted for children with more severe genetically-based cognitive disabilities, they simply buttress the view that the milder forms of learning disorders or disabilities that are environmentally fostered are correspondingly more responsive to intervention.

*Intervention.* Moreover, such early parental and environmental deficiencies may masquerade as mere relative cultural and socio-economic differences. Of course, true cultural differences do flag some genuine differences in learning styles that are not due to true disabilities, but it must be stressed that significant intellectual and social decline is not a cultural virtue. This is particularly so with respect to Native peoples. Moreover, regardless of type of clinical disorder, the penultimate question is when and how to intervene and to determine to what degree selected interventions are beneficial. Indeed, there are hotly debated counter-arguments (e.g., Feinstein and Bynner, 2004; Kagan, 1996; Thompson & Nelson, 2001; Reynolds, Ou, and Topitzes, 2004) stressing the fact that older children and adolescents can indeed overcome the effects of earlier adversity. *However, effective preventative and clinical interventions for learning and behavioral disorders are best dealt with early and within the family context* (McCain & Mustard, 1999). And while we are ever mindful of these facts, interventions with adults are often also useful. But our expectations must be tempered with the realism that these later gains are more those of training specific adaptive skills than of making wholesale changes in level of ability.



The upshot of this realization is that early identification is essential in marshalling cost-effective community and clinical interventions. Early identification is also essential in targeting effective prevention programmes, especially where there is a distinct risk of the parents having additional offspring who are at risk. The practical issue is one of identifying appropriate intervention and in determining its usefulness. There was perhaps a time when clinicians thought that an ideal world was one wherein resources would be available with which to assist those for any developmental need. But more recently, such questions are severely edited by the fiscal realities of knowing how and when to apply such resources. Indeed, the penultimate question may be one of dealing effectively with poverty and its deleterious long-term effects, in particular the health and stability of the parental-child relationship within a supportive family environment.

Aside from the *war on poverty*, it is also essential to learn which families are more apt to cope with developmentally disabled children. Kwai-sang Yau and Li-Tsang (1999) reviewed the literature, concluding that families who cope well have a strong marital relationship, deal well with the specific characteristics of the disabled child (e.g., a milder communication impairment), have the availability of a parental support group and a small, intense social support network, have high *SES*, few children, and a supportive community. *Fractured, socially unsupported and isolated single-parent families are obviously at more risk of adjusting poorly with a child with a severe disability and are also more likely to foster disabilities in their offspring.*

*Native issues.* Our review will also embrace Native issues. Many of those having learning needs develop within an environment of poverty and marginalization. It is also true that what at first appears to be a learning deficiency is in reality a cultural difference. At this point in time, intelligence tests provide only token representation to Native populations, of which there are many. Without locally normed instruments and content-relevant test items, it is difficult to be confident that a true learning disability or mild intellectual deficiency is at play. Obviously, it is an easier task to determine a true deficiency when test scores are very low and school problems are serious. *It is crucial then to have suitably normed tools for assessment and remediation that separate the effects of culture and language from those of impoverishment and family instability.* And while not the exclusive preserve of Native culture, fetal alcohol syndrome (FAS) and poverty are substantially higher within these populations and FAS is the single most common contributing cause of DD generally and within this culture in particular.

***Early Childhood Vulnerability: The McCain and Mustard Report***

Moreover, regardless of type of learning disorder, the penultimate question is when and how to intervene and to determine whether selected interventions will be beneficial. Since the most common stand-alone learning disability stems from significantly low intelligence, and since low intelligence is connected to the appearance or aggravation of several other learning problems as well, it is essential to examine the evidence for the importance of the preschool environment. The single best synopsis of importance of these "early years" is the McCain and Mustard report (1999):

***Early Years Study: Final report: Reversing the Real Brain Drain. Effective***

*preventative and clinical interventions for learning and behavioral disorders are best dealt with early and within the family context* (McCain & Mustard, 1999). Later efforts, while helpful, are much less effective and expensive to administer.

McCain and Mustard (1999) reviewed a vast amount of demographic material surrounding the importance of early childhood as a crucial period of child development. Literature gleaned from the neurosciences, developmental psychology, social sciences and other relevant disciplines confirmed White's (1975) earlier claims. These authors highlighted the importance of the early years in forging the intergenerational quality of intellectual and interpersonal competence. *Brain development within the first six years is the basis of learning, behavior and health over the entire life span.* With ample evidence, these authors contend that the future societal costs of failing to intervene during the early years are much higher than marshalling the needed resources to take appropriate societal action when it is needed most - during preschool. For example, brain development is most extensive *in utero* and in the first few years of life. Nutrition, loving care and nurturing directly affect the development of neural pathways during this vulnerable period of life.

McCain and Mustard's (1999) position on the need for early intervention, while hard-hitting, is not new (e.g., White, 1975). As we reveal in summarizing *The Milwaukee Project* (Garber, 1988), *negative early experiences, such as severe neglect and lack of appropriate verbal stimulation, are likely to have irreversible and sustained long-term effects on development.* Learning, behavioral and health problems are also tied to adverse early conditions. Structurally, socioeconomic privation is strongly

associated with adverse early child experiences. Such adverse conditions include lack of training in vocabulary and nascent acquisition of mathematics within the household. In contrast, nurturing families provide the intellectual and emotional environments necessary for future academic success. Learning is actively promoted through responsive interpersonal interaction.

*Poverty.* Evans (2004) hammered home the multi-pronged adverse effects of childhood poverty. Complicating matters further for the clinician is the humbling realization that *many learning and behavioral problems are fostered by early maternal deprivation (Garber, 1988), which is usually accompanied by persistent poverty (Ackerman, et al., 2004).* The risks of poverty on children can be as serious as those attributed to adverse genetics. Using an ecological confluence model, Evans carefully documented the cumulative harmful effects of poverty, which were: negative, harsh and unresponsive parenting, exposure to aggressive peers, family instability and divorce, lack of parental monitoring, lack of maternal emotional support, weaker social ties, less cognitive stimulation, less parental involvement in school activities, significantly more residential instability, excessively crowded and noisy living arrangements, less nutrition, and greater exposure to environmental toxins, such as lead, tobacco and alcohol. Indeed, in a similar vein, Sameroff, Seifer, Baldwin and Baldwin (1993) found that as various risk factors increased, children's IQs from various cultural backgrounds decreased. Such risks were poor maternal mental health, high maternal anxiety, low maternal education, head of household unemployed or unskilled, father absent, minority-group membership, more than four children, and a high incidence of stressful events, such as job loss or death in the family.

Nevertheless, there is an economic cutoff point above which children do fairly well, and a point below which children typically do not acquire a readiness to learn (Ackerman, Brown & Izard, 2004; Mistry, Biesanz, Taylor, Burchinal & Cox, 2004). Ackerman et al. examined the effects of persistent poverty risk by following 40 Head Start disadvantaged preschoolers and their families, testing them over three intervals every two years until grade 5. Poverty was defined using an *income-to-needs* ratio which was based on the total earned annual income for the past year for all adult family members divided by need, which was defined in terms of the annual income averaged over the total number of children and adults living in the same household. Using annually adjusted federal poverty guidelines, the *cutoff point* was calculated as a per capita family income estimate divided by the poverty level. Thus a household *per capita* income that equals a *per capita* poverty level is 1.0, or the poverty threshold. An income-to-needs ratio of <1.0 is below the poverty threshold. The risk criterion for this income-to-needs ratio was the number of times the ratio was <1.0. On the one hand, risk indices for the income-to-needs ratio interval ranging from 3.0 to 4.0 were nonsignificant. On the other hand, recent and persistent risk effects occurred more frequently for income-to-needs ratios up to and including 2.0. Poverty occurring at grade 5 (about age 11) **what does this mean i.e. grade 5** **????** positively correlated with externalizing and internalizing behavior problems and negatively with academic performance. Relationship instability was positively correlated with externalizing problems negatively correlated with academic competence; police contacts with externalizing behavior; psychiatric morbidity with internalizing behavior; harsh parenting negatively with academic competence; cognitive ability with academic competence; and maternal schooling with academic competence. Cognitive ability and maternal

schooling were each correlated with academic competence. Although there was a trend in this direction, residential instability did not correlate significantly with risk outcomes.

The effect of income during the preschool years is even more telling. Mistry et al. (2004) took various measures, such as perceived financial maternal depression, maternal sensitivity, child cognitive and language development, and maternal reports of child behavioral and social problems and family income on 1,364 preschool children followed from 6-15 months to 24-36 of age selected from 10 hospitals during regular family visits for medical services. Thus, this sample was more typical of the general population than the disadvantaged sample used by Ackerman et al. (2004). Using the same formula as Ackerman et al., these authors found that an income-to-needs ratio up to and including 5 was linearly associated with better family processes and child outcomes. After this level, the benefits of income tended to asymptote. An income-to-needs ratio of 1 - the poverty level - for a family of 4 was \$18,392 (*USD*). Thus, an income-to-needs ratio of 5 meant that a minimally optimal income for a family of four was \$59,960, or five times the poverty level income. At this level, variability in family process but not income, predicted child adjustment. However, at the poverty level, family processes fully accounted for the effects of average income over this period of time.

Thus, it is family processes tied to basic income needs that connects to child outcomes within the preschool and subsequent years. That is, below this line (i.e., the *cut-off*), perception of financial resources was relatively dissatisfied more than half of the time. Family processes which were adversely affected when income fell below level 5 were maternal

sensitivity and depression, and adverse child outcomes such as cognitive and language development, lower social competence, and (less strongly) child behavior problems. Intriguingly, families with incomes 20 times the poverty level fared little better than those at or above level 5 on these important measures.

As a result of poverty, children often do not enter formal schooling with the minimum skills required to respond effectively to instruction. The quality of parenting is pivotal to normal development and is addressed systematically in *The Milwaukee Project* (Garber, 1988). Moreover, the stresses associated with poverty and deprivation during the prenatal and perinatal periods interfere with the formation of synapses, laying the groundwork for inattentiveness and impaired learning in general. As we see in the following review, inattentiveness is a common symptom of many learning disorders and is not by any means confined to primarily attention disorders. Inattentiveness, impulsivity, aggression, moodiness and low self-esteem in older children has also been associated with lack of parental involvement (Baumrind, 1991). Antisocial behavior, school failure, delinquency and crime are common outcomes of inattentiveness and hyperactivity (Garber). The upshot of this realization is that early identification is essential in helping to martial cost-effective community and clinical interventions. *Poverty and lack of parental intelligence conspire to produce crippling child outcomes.* Early identification is also essential where there is a distinct risk of the parents having additional offspring who are also at risk.

So, at higher income levels, positive family environments are not necessarily the result of additional money, but that lack of money, as actual threshold poverty is approached, is associated more directly with adverse

effects. Such poverty is often associated with maternal relationship instability, police contacts, psychiatric morbidity, harsh parenting, and low maternal schooling. Indeed, as with Evans (2004) and Sameroff et al., 1993, Burchinal, Roberts, Hooper and Zeisel (2000) found that such early risk factors are cumulative in their effects on young children. In particular, lack of maternal education, maternal depression, household size, lack of maternal involvement during mother-child interactions (MULTI-PASS; Harms, Cryer, & Clifford, 1990; Marfo, 1992), a poor child-rearing environment (HOME; Elardo & Bradley, 1981), and poor quality of centre-based care (ITERS; Harms, Cryer, & Clifford, 1990; ECERS; Harms & Clifford, 1980) were significantly correlated with poorer child outcomes by 36 months. Thus, chronic and persistent poverty has a telling longer-term impact on the intellectual and social-emotional lives of children. However, identifying the specific adverse mechanisms within the families of economically deprived families is quite a challenge.

### ***Developmental Delay***

#### **Mild Developmental Delay: *The Milwaukee Project***

As indicated above, the environmental background is of substantial importance, especially in the milder but most numerous forms of mental retardation *MR* or *DD* (e.g., Garber, 1988). As indicated above, other than the mechanisms underlying early deprivation (Garber, 1988), specific etiologies are more difficult to identify in the largest group of mildly retarded (i.e., *IQs* < 70(*APA*)/75(*AAMR*), > 50) patients, which constitutes 75% of *DDs* (Garber, 1988; Valente, 1989). Those within the *borderline* subgroup (*IQs* >60, <75) were not isolated but remain the most numerous within this



group. The relationship between *DD* of the psycho-cultural or cultural-familial variety and poverty and its concomitants has been underscored anthropologically by Edgerton (1984). Cultural and social differences need not be confused with hardship. Regardless of race, lower working class (*LWC*) parents of *DD* children themselves scored one or more standard deviations lower on adaptive behavior than did their lower middle class (*LMC*) counterparts (Slate, 1983). Thus, such references to deficiencies in adaptive behavior are really quite important, both diagnostically, and in terms of preparing *DD* patients for adaptive outcomes.

Even though the two factors are often connected, as indicated by Mistry et al. (2004), Garber (1988) did not automatically equate poverty and lack of sufficient maternal intelligence. Poverty as a proxy for maternal developmental delay is simply too crude a measure to be clinically practical. What is more, it was necessary to identify the at-risk parental mechanisms that underlay maternal developmental delay (*DD*) if suitable interventions are to be effective. Low maternal intelligence is associated with the inter-generational transmission of *cultural-familial* or mild developmental delay. ((Intelligence measured within this range (*IQs* 50-75) accounts for 70-80% of mild *DD*, and most of these cases fall within the *borderline* range (*IQs* 60-75).)) Mild intellectual delay is almost always associated with adverse environments, such as poverty, and is usually accompanied by impaired functioning. Deficits in adaptive behavior are reflected in inadequate parenting.

*Unfortunately, by the time mild DD is identified in the school system, it is fairly resistant to remediation* (Garber, 1988). As a result of this fact, even mild *DD* is all but impossible to reverse once adulthood is

reached. So, early preschool identification of risk and a plan of effective intervention at this time are crucial. *The Milwaukee Project* (Garber) began in the 1960s and culminated in the 1988 report, which focused on the family dynamics of low *IQ* mothers and their children. It was initially found that the risks to the children were most pronounced when the mother's *IQ* fell below 80. Using a *social deprivation hypothesis*, compensatory preschool interventions were attempted, using a non-at-risk contrast group and at-risk control and experimental groups. Garber identified parental-child experiences important in facilitating normal development.

As a proxy for middle class experiences associated with long-term improvements in social and academic performance, Garber (1988) identified proximal home variables gleaned from the extant literature on child development, focussing on the fact that maternal low intelligence is the single best predictor of a child's later low intelligence. Within this social deprivation model, adaptive parents transfer their own cognitive structures to their children as they converse and interact with them in the early years. There are significant social class differences in the language and teaching strategies used by mothers and lower class parenting styles are related to less sophisticated verbal and problem solving skills. This parental micro-environment also differs qualitatively for at-risk children, regardless of income. That is, there are still some poorer families who function reasonably effectively with their children. Garber addressed this problem.

Garber (1988) selected 88 at-risk *DD* (*IQs* < 75) mothers who had at least one six-year-old child, with a total of 586 children. Within this group, it was found that 45.4% of the mothers with *IQs* < 80 accounted for

78.2% of children with *IQs* < 80. Fathers tended to be equivalently impaired and lacked stable employment. Children of these low parents had a progressive decline in *IQ* over time until at least age 14. That is, the effect was permanent. Moreover, the lower the maternal *IQ*, the lower the child's *IQ*. *Compared to normals, when maternal IQs fell below 67, there was 14 times the chance of having a six-year-old with an IQ < 75.*

The intervention experiment ultimately dealt with groups of twenty children each. There were relatively few dropouts. An *Infant Stimulation Centre* was used for short visits and the mothers were enticed by paying them a small hourly rate for having their children involved. Daycare services were also involved for preschools. All at-risk infants began enrollment by six months of age. Stimulation began at the rate of five days a week at seven hours per day. Maternal rehabilitation included refresher courses on reading, writing and math skills for four days over four weeks. Discussions were held on mothering, child-care and home management. Lessons on remedial education, motivational counseling, help with household tasks, and on-the-job training were held for 26 weeks for three days each week. This early infancy phase involved the training in perceptual and motor skills training. The next phase of intervention occurred at 24 months to four years and involved structured small group activities. These activities addressed cognitive and language skills tailored to school readiness and age level by qualified teachers. Special stress was given to encouraging verbal interactions (e.g., labeling, pairing, listening, echoing, expanding, and responding) with infants, even prior to talking. Interventions expanded during the toddler and childhood phases to include imitation, memory, vocabulary, concept formation, predictive labeling and memory skills training. Parents were instructed in listening, speaking, demonstrating, helping, listening,

conversing, and vocabulary skills. Parents were also trained in basic knowledge and verbal problem solving skills, culminating in school readiness instructional tasks.

Assessment and follow-up measures were significant in demonstrating a causal linkage between early stimulation and progressive improvement of the children's IQs. Controls began to decline measurably by 22 months. Experimental or intervention children were comparable in most ways to the low-risk contrast children in terms of motor, adaptive, personal-social and performance tasks, but were actually superior in measured language skills. Moreover, the comparisons widened over time when measured at 72 months. Grammatical comprehension was a notable success. Results persisted and grew by age 10. The control at-risk group continued to deteriorate over time, well into adolescence. Interventions done *post hoc* strongly suggested that interventions attempted after school entry were relatively ineffective at arresting this insidious intellectual decline.

### **Moderate and Severe Developmental Delay**

Although there are overlaps in IQ scores, the more severe types of developmental delay (*DD*) are often associated with genetic and physical disorders such as Down's syndrome (*DS*), phenylketonuria, Williams' syndrome, or Fragile X syndrome. This is a short-list, as there are more than 1000 known causes of *DD* (Dykens, 2003). It should also be emphasized that an India study (Kishore, Nizamie, Nizamie & Haque, 2004) found about 60% of patients with mental deficiency were also comorbid with other psychiatric disorders as defined by the *ICD-10*.

What is more, the quality or profile of intellectual challenge varies with type of *DD* diagnosis, with genetically identified groups often responding less well to training. Consistent with the *social deprivation hypothesis*, Neufeldt (1966) found that cultural-familial *DDs* have relatively more difficulties with short-term memory (*STM*) than do their *CA*, *MA*, and 'organically' *DD* (i.e., *IQs* < 70) matched controls. These findings are very useful when we consider whether children have cultural and educational deprivation or attentional deficit disorders (*ADD*) and learning disabilities (*LD*). In contrast, Clausen (1968) found that, when matched for *IQ*, mental age (*MA*), and chronological age (*CA*), Down's syndrome (*DS*) patients were more impaired in terms of sensory acuity and perceptual speed. Down's syndrome children seem to have more problems in communicating with others (Johnson & Abelson, 1969) and have a decreased developmental quotient (*DQ*) with increasing age (Neser, Molteno & Knight, 1998), with this diagnosis having no significant associations with maternal age, sex, social class or race of individual children and their development. Likewise, Smith (1987) found that *DS* toddlers were deficient in terms of vocalizations and gestures with mothers' utterances and these children preferred gestures instead of sounds. In contrast, Williams sufferers are well known for their keen social sensitivity and high linguistic responsiveness to their caregivers. However, visual-spatial functioning is typically impaired (Dykens, 2003) in spite of relatively intact ventral stream functioning related to well developed facial recognition skills. Grammatical development is also affected and is typically accompanied by an enhanced affinity for musical sounds.

Those with genetically based intellectual disabilities are also at particular risk for a number of life-style related health problems, such as bone loss and obesity. It is well known that Down syndrome children are more

likely to have respiratory and circulatory problems. Wells, Turner, Martin and Roy (1997) examined 120 intellectually disabled adults in Birmingham and found elevated incidences of cardio-vascular problems associated with adverse lifestyles, such as diet, smoking, alcohol consumption, and lack of physical activity, producing elevated blood pressure and an elevated body mass index. Milberger, LeRoy, Lachance and Edelson (2002) examined 23 postmenopausal women with *DS* and found that they were largely inactive and often had coexistent conditions such as thyroid disease and seizure disorders. As a result of being treated for these other conditions, compared to women of the same age and ethnicity, there was a significant elevation in osteopenia or osteoporosis. Fully 87% had such conditions. *Inactivity is strongly related to obesity and in DD adults in particular.* Gibson (1997) found that, of 1,021 *DD* adults receiving habilitative services, 19.2% were obese, which was significantly higher than national and state norms. Multivariate analyses also showed that age, gender, severity of *DD*, drug therapy and the presence of clinical syndromes affected obesity levels.

### **Adaptive Behavior and Developmental Delay**

*Mild DD.* The connection between intellectual ability and adaptive behavior, including socially adaptive behavior, is far from perfect for milder forms of *DD*. For example, Kicklighter, Bailey and Richmond (1980) examined the relationship between academic status and social competence in a group of 7-10 year-olds. Compared to the (*borderline*) slow learners (*SLs*), the authors found that the overall correlation was .51, with the educable or mildly mentally retarded (*EDD*) children scoring lower on all adaptive domains. Adaptive behavior is indeed multidimensional. That is, intelligence accounted for only about 25% of the adaptive variance.

*Adulthood adjustment.* Moreover, Nihira (1969) factor analyzed a behavior rating scale on 1232 retardates from preadolescence to adulthood, finding that personal independence, social maladaptation, and personal maladaptation are relatively independent dimensions. For a diagnosis of *DD*, deficiencies in adaptive behavior are required, in addition to intellectual deficiencies. The question of course then arises as to whether significant problems in any one of these three domains is diagnostically sufficient. As cautioned earlier, interventions during the later years, while less effective than during the early years, may still be beneficial. For example, adults with *DD* have been noted to increase their adaptive functioning after group home placement (Maisto & Hughes, 1995). However, Sandler and Thurman (1981) cautioned that it is first necessary to ferret out the specific characteristics in various residential environments which affect patients with *DD*, rather than to generalize that residential placement is always beneficial.

Although adaptive training for *DD* adults is more frustrating than with younger children, gains in personal adjustment have been noted for on-the-job training using a developmental approach for sensory-motor stimulation, concrete and practical instruction and vocational training (Doll, 1967). Sadly, Llewellyn (1995) followed six intellectually challenged couples for two years and found that parenting support was not usually forthcoming or seen as useful. Such parents sought out their partners before turning to their families and then to professionals. Considering workshop placements, Heller, Berkson and Romer (1981) found that clients, who were largely *DDs*, *improved their sociability in response more to the social milieu of the vocational programme they were placed in rather than the time spent in the*

*programme itself*. For severely retarded individuals, the question is far from clear as to whether institutionalization uniformly affects adaptation (Hodap & Zigler, 1985). In particular, using case histories, Hemming (1982) found that *DD* adults had significantly more adverse scores on the *Adaptive Behavior Scale*, especially in terms of aggression, after three months upon their return to a large institution after a placement from a large to a small unit.

Hemming cautioned that there are potentially harmful effects for transferring unsuitable *DD* adults from larger institutions to smaller units and back again. Rebellious and untrustworthy behaviors were cited as reasons for return. Using questionnaires such as the *Devereux Behavior Rating Scale*, Willer and Intagliata (1982) found that *DD* persons placed from large institutions into homes showed no differences in self-care skills when placed either in either group homes or family-care homes. However, maladaptive behavior was better controlled in family-based homes. As one would expect, community living skills improved in the group homes. Friendships and family relations were equivalent in both settings. These authors concluded that the type of home should be tailored to the kind of adaptive problems that need improvement. Using 72 adults with *DD*, Taylor (1976) found that trustworthiness was a significant predictor of group home success, apart from any language deficiencies. Broad generalizations about the potential benefits of institutional care should therefore be avoided.

More research on adaptation and training of *DDs* is certainly needed. Given the constraints imposed by their disability, any ethical constraints as to understanding the nature of any planned research and what it means to them and their rights of refusal can legitimately be called into question



(Arscott, Goodmayes, Dagnan & Kroese, 1998). *Perhaps the use of legal substitute decision makers would help considerably to close this ethical gap. Given important policy implications which affect the quality of life of our disabled residents, this is an area of considerable growth in terms of research ethics.*

*Social adjustment.* Lack of success with one's peers is very important in terms of negative life events, such as delinquency (e.g., Bigelow, 2000). Indeed, such at-risk children are at high risk for acquiring anti-social peer influences. Happily, early interventions with parents of inner-city preschoolers have been linked with reductions in delinquency - a high risk for multi-stressed families (Miller, 1994) - as well as significant reductions in borderline mental deficiency (Garber, 1988). We should not be surprised at this finding as Garber (1988) found that, in terms of intelligence and social behavior, the intervention group was marginally more intelligent than the normal control group.

While there is no direct connection between intelligence and social competence, this is often an area of often of considerable difficulty with intellectually delayed children. For example, as noted above for normal *IQ* children with social acceptance problems, Leffert, Siperstein, Gary and Millikan (2000) found that children with *DD* resembled younger children without *DD* in terms of their ability to correctly interpret another child's intentions (i.e., as "being mean"). Mentally retarded children also had more difficulty attending to multiple social cues and selecting appropriate social strategies for them.

Lack of social integration is perhaps the most serious adverse effect of most any disability or disorder as it alienates the person from valuable and necessary forms of information and self-validation. It also goes without saying that a host of occupational, educational, and personal adaptations are predicated upon social skills acquired chiefly through the peer group. Consistent with the work of Dodge (e.g., Crick & Dodge, 1994), *socially maladjusted normal IQ children often have inaccurate perceptions of other children's intentions, misperceiving untoward motives and acting accordingly.* In other words, such children are more apt to interpret entirely neutral occurrences (e.g., accidentally bumping into one) as aggressive acts and to act accordingly but inappropriately.

Santich and Kavanaugh (1997) found that partial integration of 32 school children from grades 3-6 can have negative consequences for them in terms of lack of social acceptance, stemming from higher levels of their inappropriate behavior. These mildly intellectually disabled students were also less likely to be preferred for play or selected as a best friend. In fact, Taylor, Asher and Williams (1987) found that, compared to controls, mildly *DD* children were generally rejected by their peers. They were perceived as more shy and avoidant, less cooperative and less leadership-like. One subgroup was aggressive. Of keen interest, social rejection was not confined to the peer group, as Leyser and Abrams (1982) found that *teachers as well were least accepting on a social distance scale of those students who were mentally retarded, emotionally distant or delinquent, compared to those who were normal or gifted.* Students who were perceptually and physically handicapped fell in-between.

Social skills training of such rejected and intellectually challenged children seems warranted and the work of Dykens and Cohen (1996) has been encouraging in this regard. Compared to controls, Dykens and Cohen found that *Special Olympics* involvement significantly improved participants' self-esteem. Level of time in *Special Olympics* was also the most powerful predictor of social competence. Indeed, Harter (1990) has found that the peer group is a main source for self-esteem in the elementary school years. Whether directly enhancing *DDs'* self-esteem fosters social skills remains to be seen. *Certainly, sharing common activities over time in an effort to integrate the developmentally disabled elderly, using sensitive and flexible leaders, has shown promise in improving feelings for both disabled and nondisabled groups* (Zimpel, 1990). Intriguingly, even Yoga training has shown promise in increasing *IQ* and social adaptation for *DD* people of all ages (Uma, Nagentra & Nagaratha, 1989).

The quality of early placement leaves its mark in terms of subsequent social adjustment. Indeed, for educable (i.e., mild) retarded India Indian children from different placement settings, such as residential, integrated programmes, and daycares, the social maturity scores were significantly higher only in the daycare group (Nalwa & Shanti, 1984). The initially deinstitutionalized group experienced the higher level of subsequent adaptation than did the previously non-institutionalized group. Thus, and not surprisingly, better social skills may well have been acquired within the more supervised settings and put into practice where peer social skills can be practiced. This finding should come as no surprise as Youniss (1980) reported that children need to learn social skills from supervision by parents but need to practice them with peers.

As expected, (e.g., Garber, 1988), it would appear then that milder forms of *DD* are more amenable to learning adaptive behavior. At first glance, interventions designed to train adaptive functioning in retarded children, as measured by the *Vineland Social Maturity Scale*, have yielded mixed results. Upon closer inspection, McIntosh (1969) found that, of 22 children enrolled in individualized or school programmes, only seven of these made gains, and only within the first year. Interestingly, of the seven cases showing significant declines, three were degenerative cases and four were *DS*. Controlling for *IQ* and *CA*, Vogel and Kun (1968) found that *DD* patients enrolled in special education or individualized classes made significant gains over a four-year period in terms of personal responsibility of self and possessions, but not in terms of social and emotional behaviors. Consistent with Garber (1988), Haring and Krug (1975) found that *economically disadvantaged special education students could acquire the basic skills to assist them in remaining within a regular classroom over a one-year period.*

As noted above (Vogel & Kun, 1968), *DD* children are quite prone to problems of social adaptation, but Guralnick (2002) investigated parental facilitation of peer relations in particular. Apparently, there were no significant differences between *DS* children and other mildly *DD* children in terms of maternal effectiveness in trying to prevent their children's social isolation from their peers. However, compared to matched controls, the maternal arranging of their offsprings' peer contacts was understandably rated more highly by mothers of *DS* children. Not surprisingly, it has been a general observation that parents rate their *DD* children as more adaptive on a wider basis than teachers do (Mealor & Richmond, 1980). Interestingly, Zigler (2002) found that Japanese *DD* students' adaptation within inclusive classrooms depends more on the personality and motivation of the student than

on the students' IQs. Cross-cultural comparisons may help to determine whether personal motivation and personality are more salient in noncollectivist cultures, such as North America.

*Parental adjustment.* Normal families of DD children tend to fare relatively well, especially if the child is older. However, as expected, problems are more significant when the DD offspring are younger (ages 6-8) and male (Robert, Hulgus & Beavers, 1988). Helping parents to cope better with their children's mental deficiencies is a daunting task, as most intellectually disabled children are within the borderline range and often stems from parental intellectual challenge. As such, even in Russia, their parents are often also within this range and have had broken homes and are uneducated, making for difficult interventions (Raku, 1983), a point also underscored by Garber (1988) in Milwaukee.

Maternal stress associated with having an intellectually challenged child is typically high, especially when the child has a pervasive developmental disorder such as autism. Weiss (2002; 2000) found that the mothers of DD as well as normal and autistic children fared best when they are emotionally hardy and have good social supports. The *Hardiness Test* (Maddi, 1986) is a 50-item self report measure, tapping into feelings of control, commitment and challenge. Women who express hardy beliefs also tend to perceive that supports are available. Hardiness was predictive of less depression, anxiety and depersonalization (i.e., burnout). Using 71 completed surveys, Lustig (1999) found no support for this pattern when the offspring are adults with DD, citing a family's sense of coherence and cohesion as more related to family adjustment than were measures of adaptability, social support, and maladaptive behavior. In an Italian study

(Clerici et al., 1998), institutionalized *DD*'s relatives had qualitatively higher expressed emotion (*EE*) scores, as did family members who had schizophrenia, but without the higher level of hostility with schizophrenia. High *EE* scores generally reflect lack of adaptability. Clerici et al. concluded that *family adjustment to a member with a cognitive disability is often difficult and problematic*. It is not surprising therefore that Kwai-sang Yau and Cecilia's (1999) literature review of parental adjustment and adaptation revealed a beneficial pattern of two-parent families who have higher *SES*, adequate crisis-meeting resources, and a supportive community. Such a pattern is far less forthcoming in intellectually challenged parents.

Based on self-report, Hara and Nishimura (1998) found that the psychological adjustment of Japanese siblings to their siblings' mental and physical disabilities revealed little maladjustment. However, Japanese families are noted for their comparative literacy, family coherency and collective culture (McCain & Mustard, 1999), so these results may not generalize too readily to North American populations. To illustrate, Herman and Marcenko (1997) found that maternal depression was significantly reduced when quality respite services were offered, so that the mother's perception of time resources were managed better. Such time resources are more constrained and therefore more stressful when the disabled child requires more time, when money is tight and when baby sitting resources are unavailable. It is not surprising then that Lustig (1997) recommended that vulnerable families which are more apt to be incoherent should receive interventions that help strengthen their ability to act on their own behalf. Japanese families are significantly more coherent (McCain & Mustard, 1999), and this may well account for siblings' lack of adverse reactions to an intellectually disabled brother or sister.

### ***Attention Deficit Disorders***

Aside from developmental delay and academic under-performance, the single most common behavioral and learning problem, in North America at least, is Attention Deficit Hyperactivity Disorder (*ADHD*), occurring in 3-7% of school-age children (*DSM-IV-TR*). This disorder is developmental in origin, usually identified by age 7 and is often first detected when the child must function within the strictures of a school setting. Presenting problems include lack of attention to details, careless in work, lack of sustained attention, inability to follow through with instructions, disorganized behavior, disliking of tasks requiring sustained attention, often losing things, distractibility and forgetfulness. Often school work begins to suffer as seat work becomes too difficult to sustain. Classifications may be of the inattentive (*ADD*), hyperactive (*ADHD*) or combined types and often also include an additional diagnosis of Oppositional Defiant Disorder (*ODD*) or Conduct Disorder (*CD*). McCauley (2001) found that Tourette's syndrome and Turner's syndrome (*TS*) are often also co-morbid with attention disorders. Developmentally, the prevalence of externalizing symptoms, such as hyperactivity and acting out often attenuates during adolescence and adulthood, but cardinal symptoms such as distractibility, impulsivity, inattentiveness, and inner restlessness (e.g., Atkins, 1997) often remain. Indeed, Wolf (2001) examined ten adult *ADHD* clients and found that these adults were less hyperactive but had more subtle executive function (*ED*) deficits.

For children and adolescents, an assessment needs to identify the occurrence of several features of *ADHD* within two or more settings. The

strictures of formal school attendance often trigger attentional difficulties not fully appreciated at home, but the other symptoms (e.g., excessive forgetfulness and impulsiveness) still apply. One of the most common instruments used to assess internalizing and externalizing behavioral problems in children of all age levels in several settings is the *Achenbach Child Behavior Checklist (CBCL)*; e.g., Achenbach & Rescorla, 2000) which uses report forms for teachers and parents. A convergence of significantly elevated subscales in more than one setting indicates a problem that is not simply situationally triggered.

Conduct problems are often simply documented by various credible witnesses and also recorded on the *CBCL* report forms, but evaluating a *CD* is a specialized enterprise in itself. Because of its potential legal and mandatory reporting ramifications, the diagnosis of *CD* needs to be done with an objectivity that extends beyond the normal clinical work-up. Another useful recording assessment tool is by Barkley, Edwards and Robin (1999), which delineates *ODD*, *ADHD*, and *CD* from multiple reporting sources with systematic suggestions for intervention. Another popular assessment tool also used for this purpose is the *Conners' Rating Scales* (1990). These multiple-source reporting scales measure conduct, learning, psychosomatic, impulsive-hyperactive, anxiety, and hyperactivity problems.

Galili-Weisstub and Segman (2003) flagged *ADHD* as a common idiopathic childhood neurodevelopmental disorder, which impairs schooling and social adaptation and is often accompanied by depression, *CD*, early school leaving and substance abuse proneness. This disorder has a known heritability involving dopaminergic (DRD4) genes. This is likely the reason that many *ADHD* adolescents, like their adult counterparts, present with smoking and



drinking problems as well. Other neurotransmitter systems (e.g., serotonergic, noradrenergic, cholinergic) may also be involved.

*Adapting to ADD/ADHD.* The behaviors of children and adolescents with emotional and behavioral disorders, such as *ADHD*, can be very demanding to parents and teachers alike. Rosenzweig and Brennan (2002) conducted a focus group study in which they found that parents usually tended to their offsprings' difficulties by adapting their work responsibilities and day care arrangements to make a better fit between their own and their children's needs. Work adjustment also involved taking jobs which were less demanding and which required fewer hours of work. None of these caregivers placed their children in a day care or an after school centre. It was usually the case that the affected children were cared for within the home by siblings, the parent, or an adult specifically hired for the purpose. It is worth noting that these parents were normal, not parentally challenged.

Rosenzweig and Brennan (2002) noted that lack of knowledge of the school personnel and the parents' work supervisors were key sources of frustration for such parents. Evidently, these parental perceptions of lack of support were not without a factual foundation. Matlock (2000) found that *teachers of ADHD elementary school children who were trained in appropriate teaching strategies and interventions and who were more controlling had more ADHD knowledge than those teachers without such training.* Interestingly, level of teaching experience alone did not differentiate knowledge in dealing with *ADHD* students.

How families cope with *ADHD* in their children is essential in planning any suitable intervention. In a qualitative interview study of 17 families

with children who have *ADHD*, Segal (1998) found that families of *ADHD* children changed their family routines to different scripts in order to foster their children's occupational success. These parents used strategies such as making a game of getting dressed, and delaying their own grooming until the evening period. Segal (2000) found that mothers of *ADHD* children often coped by multi-tasking (i.e., temporal enfolding) and by chunking or delegating work. Holderness (1998) followed 25 pre-adolescent boys diagnosed with *ADHD* and followed up on 18 of them five years later. It is important to note that these boys did not have *CD*, *ODD*, or other disorders complicating the picture. Compared to normative families, these target families were highly adaptive, but were understandably more highly stressed over time. As one might well expect, family cohesion, communication and good problem solving skills were related to lower levels of problem behaviors and higher self-concept scores. What is not known is whether higher family cohesion stems from less disruptive forms of *ADHD* behavior, but one can generally expect this to be the case (Anderson et al., 1986).

*Comorbidity.* The overlap of *ADHD* with mood disorders and depression in particular, is as high as 30% (Schmidt, 2002). Schmidt found that when children and adolescents from ages 7 to 17 were evaluated, along with reports from their parents and teachers, the comorbid group had no greater degree of automatic depressive thoughts than did the depressed group. However, the comorbid group had more negative views about the world, the future and a more negative attributional style.

Also, Carter et al. (2000) found that children and adolescents who also suffered from Gilles de la Tourette's syndrome (*TS*) had significantly more internalizing, externalizing and social problems if they also had comorbid

*ADHD*. Thus, when in the context of *ADHD*, depression, behavioral and social problems are significantly more entrenched. This connection of behavioral difficulties with *ADHD* was also evidenced in a Quebec five-year high school longitudinal study. Royer, Couture, Fortin, Potvin and Marcotte (2000) found that the *hyperactive ADD school children were significantly more depressed, had lower achievement scores and had more disciplinary and social problems, including higher rates of school absenteeism. It is thus very unfortunate that parents of ADHD children often feel that they cannot rely on extra-family supports* (Rosenzweig & Brennan, 2002), as Frame (2002) found that preadolescent support groups over eight sessions significantly enhanced participants' feelings of social acceptance, physical appearance, athletic competence, and global self-worth. Such therapeutic improvements are bound to reduce parental stresses as well.

*Etiology*. It appears that *ADHD* has many potential contributors; that is the principle of *equifinality* is alive and well. The etiology of *ADHD* has a known familial-genetic linkage (e.g., Galili-Weisstub & Segman, 2003) but there is also an interesting connection between physical and sexual abuse and neglect. While Garber (1988) found clear evidence that maternal neglect is causally linked to children's longer-term intellectual and social impairment, found. In a prospective study, Sexton (1999) also found that abuse and neglect are also linked to a variety of mood, learning and conduct problems, such as depression, thought disorders, *CD* and *ADHD*. Moreover, Sexton found that abuse coupled with neglect had the most deleterious outcomes. One must also be mindful of the increased stresses that such disorders have on parents who are also having difficulties coping. While more about this topic will be said later on in this review, it is also remiss not to address an etiology of *ADHD* with maternal use of ethyl alcohol. That is, a disorder, such as *ADHD*,

may have a number of different contributors, but alcohol is potentially one of them. Given the fact that *ADHD* and *FAS* share core symptoms such as impulsiveness, inattention and hyperactivity does not necessarily mean that treatments will be equally effective.

### ***Learning Disabilities***

Operationally defining Learning Disabilities (*LDs*) is often very difficult, but the prevailing administrative one is appealing. This definition identifies *LD* as a significant problem in processing information (usually linguistic) consistent with expected grade level - usually of at least 1 ½ to 2 academic years - not primarily due to low intelligence or sense organ impairment, such as deafness or blindness. An *LD* is not primarily associated with an emotional disorder unrelated to the impairment itself. At least one author (Daniel, 1996) views *LDs* as essentially language-based neurological disorders. However, the prevailing notion is that *LDs* are either verbal or nonverbal organizational informational deficits of comprehension (i.e., receptive) or production (i.e., expressive) in educational, employment and social settings (Weller, Watteyne, Herbert & Crelly, 1994).

It is interesting to note that while sense organ impairments are normally exclusionary conditions for *LD*, it is important to take special care in avoiding unwarranted conclusions that a learning impairment is due to lack of vision, rather than a stand-alone *LD*. Layton and Lock (2001) found that an undiagnosed *LD* may arise when sight-impaired children fail to respond effectively to specialized instruction. Indeed, Erin and Koenig (1997) found that between 14% and 65% of students with visual disabilities also have coexisting *LDs*. Obviously, care must be taken not to automatically exclude

those with sense organ impairments from *LD* consideration and investigations of the hearing impaired are also warranted along these lines. Obviously it is also *de rigueur* to assess blind and deaf clients in terms of specialized techniques of communication.

Learning disabilities were historically referred to as either Minimal Brain Dysfunctions (*MBDs*) or Specific Learning Disabilities (*SLDs*). Common *LDs* may involve problems in understanding (i.e., receptive aphasia), expressing (expressive aphasia), an inability to read (dyslexia), or specifically in terms of mathematical ability (i.e., *discalculia*). Prevailing theory is in terms of problems inherent in the sequencing or organization of information that one is attempting to comprehend or communicate. When the *LD* is secondary to another medical, psychiatric or pervasive developmental disorders (e.g., Asperger's Syndrome (*AS*)), then this disability is generally referred to as a communication disorder secondary to a developmental or other disorder.

Layton and Lock (2001) also addressed the issue of dual diagnoses in *LD* children. As with other learning and behavioral disorders, dealing with comorbid conditions is essential. To illustrate, McGrother et al. (1996) measured 2,117 *LD* adults, finding that behavioral and psychological problems, including epilepsy, were common concerns. It should also be made clear that an *LD* is not simply academic underachievement due to lack of sufficient training. Many students have learning problems or educational underachievement and do not have a *LD*. These students need effective compensatory education. Clues here are academically unsupportive parents and long-standing attendance problems, coupled with a normal *IQ* and few or no

problems with solving basic intellectual problems not requiring specialized instruction.

Learning disabled children often have social adjustment problems. Social difficulties are not the exclusive domain of children and adolescents with *ADHD*. Gadeyne, Ghesquiere and Onghena (2004) examined *LD* in first-graders, defining specific *LDS* in terms of achievement falling 1.65 standard deviations or more below IQ expectations and whose math or reading scores were significantly discrepant from each other. Along the same lines as O'Brien (2003), learning disabled children were far more likely than their low achieving counterparts to have deficiencies in attention, total behavior and total problems recorded on the *CBCL*. Learning disabled children were different from those who were simply low achievers in that the low achievers tended to exhibit lower self-concepts, whereas the *LD* children were more apt to have identifiable social difficulties.

*Adaptation.* Adjusting to the diagnosis of *LD* is of course crucial to its remediation, especially as it is likely a life span problem (Smith, 1988). Sorensen et al. (2003) examined the psychosocial adjustment over a two-year period of 100 children with *LD*. Even though academic difficulties were understandably chronic, it was the contextual factors that were most important in determining adaptation and adjustment that affected psychosocial outcome, as measured by the *Behavior Assessment System for Children (BASIC)*. Moreover, different factors were relatively more important for parents, teachers and children. Rigazio-DiGilio and Cramer-Benjamin (2000) found that *the family's stress experienced as a consequence of the child's LD was a function of the child's age and the level of uncertainty of the diagnosis. An early and definitive diagnosis is related to lowered parental stress.*

Reinforcing Rigazio-DiGilio and Cramer-Benjamin (2000), Morrison (1997) noted that *LD* itself does not necessarily imply that the affected individual will necessarily have serious problems, such as poor emotional or family functioning, dropping out of school, delinquency, and abusing drugs. Indeed, it is the presence of the affected person's "internal" characteristics coupled with the characteristics of the family, school and community that serve to be protective. Also, Werner (1993) investigated the development of 22 *LD* Kauai children from birth to age 32, and found that most of them had successful adaptations to adult life. While employment, marriage and divorce rates were comparable to age cohorts, mental health and criminal problems declined from adolescence to adulthood. Reminiscent of Garber (1988), in addition to temperamental, efficacy, planfulness, and self-esteem factors, resilience was related significantly to the availability of supportive and competent adult care givers.

Provocatively, McBurnett (1991) argues that, particularly if it is moderate to severe, *ADHD* actually interferes with the psychological processes necessary for learning and is therefore a type of *LD*. Attention Deficit Hyperactivity Disorder may actually interfere with rule learning. In a clinical ABAB/BABA design, Bicard (2002) found that five *ADHD* children adapted well to the rules for learning math problems, but the rules had to be consistent and that this stability of contingencies may slow adaptation to the ebb and flow of the real world.

It should come as no surprise therefore that *LDs* are common in delinquent populations. Zinkus, Gottlieb and Zinkus (1979) found that of their 55 male delinquents, 73% were two or more grades below expectation in

reading and 87% in spelling and arithmetic. In addition, about 80% of these subjects had auditory or visual-spatial processing deficits, such as errors on the *Bender-Gestalt* and mixed eye-hand dominance. Learning disabled children also have more difficulty acquiring grammatical morphological rules, such as the third person singular, possessives, and adjectival inflections. Moreover, Kravetz (1999) found that *LD* children in the 4<sup>th</sup> and 5<sup>th</sup> grades were significantly lower in interpersonal understanding and social adaptation in the classroom.

*It should therefore not be at all uncommon to find that such LD children often adapt poorly to their peers (e.g., Santich & Kavanagh, 1997) and often have early onset CD. Hatzichristou and Hopf (1993) also noted such social lack of acceptance, as well as academic difficulties. Moreover, it should not be counter-intuitive to learn that such adolescents gravitate to imitating deviant behavior with other delinquents. Indeed, this is precisely what a recent review (Bigelow, 2000) of the trajectory of delinquency found. Consistent with the commonly disruptive family origins of LD, CD and ADHD children, Sexton (1999) found that maltreated children are also at risk for developing relative underachievement and LDs. Even nutritional deficiencies, such as in terms of iron and calcium, in fetuses and infants, may be associated with LD (Haas, 1977). The bidirectionality issue is again very pertinent, as parents without a history of social support and effective models of parenting may be substantially more vulnerable to the cumulative effects of stress and far less able to nurture and protect their offspring. Adding to this behavioral brew, parental hyperactivity and impulsiveness and abuse may result.*



Those caring for these affected persons, particularly those who are informal carers, have financial, support and housing needs, including the need for respite care (McGrother et al., 1996). Interestingly, these informal carers investigated by McGrother et al. had 40% more health concerns than the normative population and had four times the incidence of depression. *Depression is a known risk factor for neglectful parenting, raising the spectre of child neglect and abuse amongst these cognitively disabled children*, who are themselves inherently more challenging to these parents. While other disorders, such as autism, are even more stressful to care-givers (e.g., Wadden, 1995), given the importance of family supports in dealing effectively with *LDs* (Rigazio-DiGilio & Cramer-Benjamin, 2000; Cramer-Benjamin, Morrison, 1997), this study underscores the need to take better care of our care-givers.

Those affected by *LD* seem to be particularly self-defeating. Hoehn (1999) found that community college students who qualified for support services and who viewed their disabilities as being more entrenched, global and stigmatizing were significantly less likely to seek out help from their professors and were more likely to give up. What is more, Biderman (1997) found that lower functioning adults with *LD* had fewer children and used support services less than did other adults their own age. Watts (1986) discussed this defeatist sense of discouragement and the way of life that tends to impair adaptive metacognitive skills in adolescents with *LDs*. Focussing on one's strengths may reduce the negative impact on one's sense of self. *Indeed, teacher and peer rejection of the failure-prone LD adolescent may foster a delinquent identity*. The future holds a lot of promise that assistive technologies will help to improve *LD* students to better achieve and to gain more social acceptance (Bryant & Bryant, 1998). Classifying *LDs* can

present problems, especially when attempting to distinguish *LDS* from mild developmental delay (*MDD*). As Scott, Fletcher, Jean-Francois, Urbano and Sanchez (1998) reported, this kind of discrimination is vital in prekindergarten and kindergarten children, so that early targetted intervention can be effective and timely.

### **Aboriginal Issues and Cognitive Disabilities**

As will be discussed, Aboriginal peoples in North America are at particular risk for learning and behavioral disorders as a product of maternal alcohol use during pregnancy, as well as a product of being marginalized culturally, with the poverty and stress that often accompany this unfortunate status. In fact, Rudmin (2003) reviewed the prevailing theories of acculturative stress and concluded that there is no convincing evidence that biculturalism is adaptive, whether this is in terms of Natives, immigrants, refugees or sojourners. Thus, practitioners working with Natives need to be very cautious in making diagnoses of intellectual delay and other disorders.

As Allen (2002) claimed in terms of *American Indian* and *Alaskan* Native settings, test administration and the lack of local norms and the problems of adapting psychiatric diagnoses to local cultures can hamper valid interpretations. In a particularly spirited account, Daron (1992) described to his dismay how he discovered that a Canadian group of Native counsellors he was teaching had children who were tested with the *WISC-R*, in spite of lack of established validity of this test on aboriginal populations. Using examples taken from the *Otis-Lennon* (1967) test, depressed verbal scores may be an artifact of *Algonkian* language transfer errors and lack of item

relevance. In contrast, Mueller, Mulcahy, Wilgosh, Watters and Mancini (1986) found that the *WISC-R* was reasonably internally consistent for *Kitimeot* and *Keewatin* Natives of the *North West Territories*. But for these *Inuit* children, fully one third of test items fell within extreme ranges, with Verbal items proving too difficult and Performance items too easy, causing these researchers some interpretive and diagnostic concerns.

Such concerns were not realized in work with Native American elderly in North Dakota (Ferraro, Bercier, Holm & McDonald, 2002). Neuropsychological testing, including the Boston Naming test, the Logical Memory Test, the *WAIS-R*, and the *WAIS-R* Digit Symbol Subscale, was completed equivalently well by Natives and non-Natives alike. On the other hand, Salois (1999) found that, compared to the norm, *Northern Plains* Native American Indian children's performance on the *WISC-R* was significantly lower for Full Scale and Verbal IQ scores. While Performance Scale IQs were not significantly different as a whole, visual-perceptual and visual-spatial-motoric abilities were particular strengths. In particular, Picture Completion and Mazes were significantly higher than the norm. Simultaneous as opposed to sequential reasoning was predominant. Congruent with this pattern, Wright, Taylor and Ruggiero (1996) found that, when compared to White Quebec norms, Inuit Quebec children (ages 4-6) performed significantly better during the first two years of formal education on the Raven's Coloured Progressive Matrices (*CPM*). These authors also found that *CPM* scores were not affected by the ethnic mix of the children's parents, whether they were both Native or of mixed Native/white heritages. The language of instruction and teachers' ethnicity were also unimportant in affecting scores. These authors concluded that *academic underachievement is produced by not allowing these children to capitalize on their strengths in learning specific material.*

St. John, Krichev and Bauman (1976) found that *WISC* Performance IQs were within the norms for *Cree* and *Ojibwa* children but their Verbal IQs were in the deficient or dull-normal range. Also, Atkinson (1995) found that Native American children who were referred for speech and language or general purposes were significantly lower on the *Wechsler Individual Achievement Test (WIAT)*. Moreover, using the *Clinical Evaluation of Language Fundamentals-Revised (CELF-R)*, volunteer children had better receptive than expressive language skills, but referred children were depressed in both. Non-referred volunteer children had scores equivalent to national norms. Similarly, Greene, Kersey and Prutsman (1973) found that *Seminole* school children, who live in the Florida Everglades, scored over one standard deviation below the norm on the *Wide Range Achievement Test (WRAT)*, and this difference widened with age. Especially with children, it is apparent that while the *WISC-R* has good internal validity, it is also apparent that *Native children are at a distinct disadvantage with commonly used IQ tests*. Therefore, identifying *DD* and *LD* native children should be approached with caution, using local norms wherever possible, or by interpreting *IQ* scores and subtest profiles with extreme caution.

Placement of Native children in foster homes is a contentious and even explosive issue, given the tragic history of residential schools. Cundcik, Gottfredson and Willson (1974) found that Native children enrolled at least five years in an Indian education programme had educational achievement scores (*Iowa Test of Basic Skills* and *California Test of Mental Maturity*) that declined with successive years of participation. *These fostered children did no better in these schools than did those who remained at home*

and educated by public schools. Also, their IQ scores did not change. It is commonly known that IQ is partly a product of formal education (Ceci, 1991).

### **Fetal Alcohol Syndrome and Effects**

Unfortunately, disentangling cultural differences from brain damage associated with drug and alcohol abuse, especially during pregnancy, is a serious issue. For example, Binns (2001) found that primary and preschool teachers of Alaskan Native ethnicity scored significantly lower on knowledge and awareness of FAS. It should be stressed that Native norms for drinking vary widely by community and subgroups within each community (May, 1991). Integrating education within a supportive Native community is the obvious goal. As is reported below, FAS is at alarming levels within many Native communities.

Drinking alcohol during pregnancy has become one of the largest sources of preventable contributors to developmental delay and behavior disorders. Olofsson (2000) claimed that fetal alcohol syndrome (FAS) is the most frequent cause of DD in Denmark and Niccols (1994) cites FAS as the most commonly known cause of DD in America. Phelps and Grabowski (1992) reported that FAS exceeds Down's syndrome (DS), cerebral palsy (CP), and spina bifida (SB) as a cause of DD. Conroy (1990) reported that in rural and Native communities where maternal alcohol abuse is a known concern, prevalence rates of FAS/FAE are estimated as being about 190/1000 children, instead of the normative 0.4 to 3.1/1000. That's 112 times the norm! FAE is a milder manifestation of the disorder in which - depending on the teratogenic timing - facial dimorphisms may or may not be in evidence and the infant or child may display some but not all of three cardinal behavioral features, such a

*DD*, inattentiveness or hyperactivity. Kavale and Karge's (1986) review showed that alcohol consumption during pregnancy was six times higher in lower class than middle class mothers. In a literature review, Niccols reported that during the first 4-10 weeks after conception, ethanol is a cytotoxin which causes excessive cell death in the central nervous system (*CNS*) and abnormal nerve cell migration, leading to disorganized tissue structure. Early identification is essential, as recurrence in additional siblings is 77% (Abel, 1990). In a Scottish study, Plant (1984) controlled for age, smoking and legal and illegal drug use, finding that there was a significant correlation between self-reported maternal alcohol use and previous experiences of pregnancy termination or having a physically or mentally handicapped child or having a spontaneous abortion.

The *CNS* damage to the fetus as a result of *FAS* results in varying degrees of developmental delay, as a function of dosage, timing, duration and the uterine, placental, and health status of the particular mother. Holzman (1982) believes that damage sustained in the first trimester is more telling, although there is no known safe period during gestation. In addition to *DD*, such infants tend to have delayed growth, short eyelid slits, abnormal jaw protrusion, altered palmar crease patterns, wide-spaced eyes, a low forehead, joint and cardiac anomalies, and fine motor dysfunction (Furey, 1982). As Streissguth (1983) noted, such infants are often tremulous, irritable, and hypersensitive to sound, and have a weak sucking response. Streissguth and La Due (1987) described facial and cranial abnormalities in *FAS* children, such as microcephaly, short palpebral fissures (i.e., open eye width), flat midface, indistinct philtrum (i.e., upper lip), thin upper lip, epicanthal folds, low nasal bridge, minor ear abnormalities, short nose and micrognathia (i.e., small chin).

Using the *Developmental Behavior Checklist*, Steinhausen, Willms, Metzke and Spohr (2003) found that, compared to controls ( $n = 15$ ), infants, children and younger adolescents afflicted with *FAS* ( $n = 12$ ) and fetal alcohol effects (*FAE*) ( $n = 26$ ) were more disruptive, self-absorbed, anxious, antisocial and communication disordered. These findings underscored the fact that hyperkinesis is a major characteristic of these children. Spatial memory may be a particular problem with *FAS* children (Uecker, 1998), although Kaemingk and Halverson (2000) found that such deficits are due more to deficiencies in perceptual and verbal memory. Long-term outcomes for such children include psychopathology, hyperkinesis, emotional disorders, sleep disorders, and abnormal habits and stereotypes (Steinhausen, Willms & Spohr, 1993). Even eye problems, such as optic nerve hypoplasia and retinal dystrophy and reduced visual acuity, have been identified with this population (Stroemland, 1990). In an interesting comparison, Nanson (1988) found that *FAS* children, by middle childhood, are similarly affected by inattention and behavior problems as are *ADD* children, apart from any intellectual decline.

While the direct teratogenic ethanol effects on the developing *CNS* are well known, specific teratogenic mechanisms are still being discovered. Ethanol administered to pregnant rhesus and cynomolgus monkeys has been found to interrupt feto-placental circulation, producing severe anoxia (Mukherjee, 1982). West, Hodges and Black (1981) also found that ethanol changes the hippocampal mossy fibers in rats. Tsuji, Guizzetti and Costa (2003) suspected that microencephaly associated with *FAS* is associated with ethanol-induced interrupted glial cell proliferation. These investigators found a dose-dependent decrease in phosphoproteins (i.e., MAPK and p70S6) necessary for neonatal cell proliferation. Stoltenburg-Diginer and Spohr (1983) found

that in rats prenatally exposed to alcohol, the parietal spine distribution of proximal apical dendrites of layer V pyramidal cells were abnormal at 12 and 40 days postnatal, resulting in long, thin and entangled spines and a decreased number of normal stubby and mushroom-shaped spines.

There is a saying that there are many ways to get to Rome (i.e., *equifinality*) and so it seems to be the case with *FAS* and cell injury, as these findings resembled those of normal karyotype *DD* children. Olney, Wozniak, Jevtovic-Todorovic and Ikonomidou claimed that ethanol has the potential to create widespread apoptosis (i.e., cell death) during the synaptogenesis period of gestation. Two processes, NMDA glutamate receptor blockade and excessive GABA-sub (A) receptor activation cause this cell death. Glutamate is the most common neuro-transmitter. Cell death is not confined to the gestation period, as Bellinger, Davidson, Bedi and Wilce (2002) found that neonatal rat pups had significant glutamate changes due to daily prenatal alcohol exposure, resulting in decreased brain and body weight and microencephaly. Glial cells and synapses normally proliferate during the postnatal period.

Of course, in practice, it is difficult to identify the direct teratogenic effects of alcohol on the human fetus, as it is often the case that pregnant mothers are abusing other illicit substances as well. To underscore the potential complexity of the diagnosis, Nanson (1992) found six cases where children were comorbid with *FAS* and autism. Asher (2002) found that cocaine may result in congenital abnormalities as well, such as stillbirth and interuterine growth retardation. Heroin reduces fetal growth and increases the rate of fetal death. Even small amounts of alcohol can produce developmental delay and intellectual impairment (i.e., *FAE*) but with



few and minor dysmorphic features, compared with *FAS* associated with heavy drinking. Asher reported that dosage and duration are important, especially during organogenesis. This is the embryonic period from 18 -60 days after conception, during which maximum cell damage is realized. During the fetal stage, growth is affected, especially to the brain.

*Low birth weight.* Low birth weight is the most consistent finding of alcohol-related birth defects (Sokol & Abel, 1992). Since heavy drinking is also associated with nutritional deficiencies (Able, 1980), mothers who are heavy drinkers are also more apt to deliver smaller babies (Landesman-Dwyer, 1982). Low birth weight is a significant developmental risk. For example, Anderson and Doyle (2003) examined 568 8-year-old children who weighed 1,000 g or less at birth. Significantly lower *IQs* were noted, as was the tendency to repeat a grade, to receive educational assistance, and to have an *LD*. Moreover, these children were rated by their parents and teachers as less adaptive.

*Nonspecific environmental factors.* There is a complicated etiology of *DD* in vulnerable children (Abel; Neugut, 1982). For example, Taylor and Hall (1979) described the profile of mothers with previous stillborns who had inadequate opportunities for mourning or supportive help. Such mothers had unwanted babies, a lack of hospital rooming, were pregnant as teens, had emotional illnesses and were dependent on their parents. They also had lengthy separations from their newborns, lacked father support and had a lower educational level and an iron deficiency. These *FAS/FAE* promoting mothers had a significantly more likely chance of having premature or clinically small babies requiring perinatal care. These authors also found that the most severe effect of alcohol on the fetus was spontaneous abortion.

*Genetics.* There are also quite demonstrable genetic vulnerabilities involved in alcohol exposure, as fraternal twins are not equally likely to have comparably adverse outcomes. In addition, *people of colour or Black Americans are seven times more vulnerable to alcohol-related damage to their babies.* The need for special after-care is especially important in such high risk births, as timely and continued support is essential in helping to prevent *DD* even in non-*FAS/FAE* infants. Ramey and Gowen (1986) found that systemic and intense intervention helps to prevent mild *DD* if administered before the second year, as indeed Garber (1988) found. As well, the later-borns of alcoholic mothers are more severely affected, which is possibly related to greater blood alcohol concentrations as a result of liver decline and lower kidney clearance.

Of course, the literature is still largely silent on the confounding factors of early childhood deprivation (e.g., Garber, 1988) coupled with alcohol use during pregnancy. Although Jones and Smith (1973) discovered *FAS* even in mothers with an adequate diet, social class is an essential consideration. Bingol et al. (1987) revealed that *lower socioeconomic status (SES) chronically alcoholic mothers (n = 48) were substantially more likely (71.9%) to have hyperactive offspring than were chronically alcoholic upper middle-class mothers (n = 36, 4.5%).* Moreover, hyperactivity was also more likely (71%) in the lower *SES* mothers than in the upper middle-class mothers (21%). The higher-class women ate regularly, had a more balanced diet, and supplemented with vitamins and minerals. Congenital malformation, failure to thrive and *DD* were also higher in the lower *SES* group.

It remains to be determined whether genetic selection, chronic stress, or the protective and buffering effects of antioxidants and other factors, such as nutrient malabsorption, differentiate socio-economically more vulnerable women from this serious problem. Perhaps the stresses associated with poverty help to account for such findings. Indeed, there seems to be good evidence for this. Chen, Langer, Raphaelson and Matthews (2004) administered ambiguous (e.g., browsing in a store with an attentive saleswoman watching) and negative (e.g., other students teasing you) videos to high school students. Those students from lower *SES* backgrounds were significantly more likely to perceive threat during both the ambiguous and negative videos and had greater diastolic blood pressure and heart rate reactivity. According to Taylor, Branch, Van Zuylen, and Redel (1988), stress elevates corticosteroids, which when chronic and excessive, damages brain cells and produces similar behavioral problems as with *FAS*. Paradoxically, fetal alcohol exposure is hypothesized to activate stress hormones, which may actually aggravate the effects of alcohol in the economically deprived population. Interestingly enough, Taylor et al. (1986) showed that there was a consistent pattern of enhanced challenge-induced neuroendocrinal and behavioral responses in adult rats, which were ethanol-exposed, *in utero*. Perhaps mothers of alcoholic parents respond more stressfully to poverty than do controls, perhaps making them more vulnerable to alcohol abuse and to having *FAS* offspring.

*Interventions.* Working with *FAS* children is very challenging, especially given the pernicious and pervasive nature of this disorder. However, Coe, Sidders, Riley, Waltermire and Hagerman (2001) have had some success using a variety of psychotropic interventions, such as stimulants, SSRIs, mood stabilizers and neuroleptics. Aggressive treatment was

advocated. If treatment is very difficult, then prevention is equivalently disconcerting. Murphy-Brennan and Oei (1999) found that *while FAS prevention programmes were good at raising awareness training, such programmes did not translate into lower alcohol consumption in the high risk, high consumption, group.*

### **Acquired Brain Injury**

Brain injury, of course, derives from several sources, including teratogenic, perinatal, genetic, and acquired. The current section focusses on acquired brain injury (*ABI*), which is sustained through trauma, either closed- or open-head trauma. In a literature review, Kreutzer, Witol and Marwitz (1997) learned that in the U.S.A. alone, 400,000 people suffer from brain injury (*BI*) annually, with 44,000 in the severe range, with an incidence of 1-1.8 million-lifetime accumulation. Brain injury is the leading cause of death under the age of 45, with the 15-24 age group at even higher risk, and the 5-14 age group having the highest risk overall. Ethanol pre-injury use is 51% and in *BI* survivors, 35% were still moderate-to-heavy drinkers, placing them at further risk for such accidents. Learning disabled youngsters are also at higher risk for *BI*. One of the most attended to signs associated with acquired brain injury (*ABI*) is loss of consciousness. For example, Talbot (1994) examined seven adults in acute and long-term care who had sustained "altered states of consciousness" or *ASC* and the severity of the head injury were measured by time in coma and scores on the *Glasgow Coma Scale* and *Coma Near Coma Scale*.

*Prevailing wisdom in treatment is that cell death occurs over a period of months after the initial injury is sustained, requiring interventions to be prompt.* Indeed, Talbot found exactly this, in that sense organ

stimulation therapy improved perceptual and motoric skills when introduced within the first 24 months of the injury. The sample size was too small to determine whether even earlier interventions are preferable, but this is a reasonable inference. This earlier-the-better hypothesis also has some support. Bond and Brooks (1976) found that the greater part of recovery following *TBI* is within the first six months and that subsequent recovery is likely due more to adaptation to fixed mental and physical deficits.

According to Ewing-Cobbs, Fletcher and Levin (1995), nearly 50% of severely *BI* children have lesions extending to the subcortical white matter and 51% had abnormal frontal lobe (*FL*) signal intensities. Diffuse cerebral injury at the moment of impact is the primary cause of damage due to the closed head injury (*CHI*). Degeneration and demyelination of white matter was frequently asymmetrical. *At injury, adolescents had more severe and persistent problems with verbal learning and memory than did children aged 6-12.* Closed head injury often involves destruction of white matter, producing the nonverbal learning disorder (*NLD*) syndrome in children (Rourke, 1995). Nonverbal Learning Disability syndrome involves deficits in tactile, visual, complex psychomotor and novel material, with secondary deficits in memory, concept formation and problem solving, with numerous academic and social adaptation difficulties.

In a very small but important intensive study, Gorman, Dayle, Hood and Rumrell (2003) found that cognitive rehabilitation helps *BI* patients to achieve better functional, personal, and social interactions. Compensations for such deficits in their experiences can be enhanced significantly through the use of assistive technology devices (*ATDs*). With proper selection and training in the use of such technologies, a level of independence can be

achieved that is not otherwise possible. Obviously, dependency on a human service provider is costly and discourages autonomy. A larger study ( $N = 53$ ) by Stern, Jeaco and Millar (1999) reinforced the notion that computers can help *BI* patients recover significantly from their cognitive and linguistic deficits by teaching them how to practically overcome everyday issues. Motivation is a crucial factor in post-injury therapy. Cleveland (1998) learned from 64 TBI patients that *(prompt) engagement in a rehabilitative programme is essential to recovery*. The contextual variables of perception of self, perception of recovery, vision, and personal interactions all contributed synergistically to the patient returning to community life.

Due to the frequent personality and functional changes that the brain injured patient frequently suffers from, *the social and personal impact on immediate family members is often traumatic and overwhelming* (Larkin, 2002). This is especially so in terms of the patient's' affective and behavioral functioning and its impact on family functioning (Kosciulek & Lustig, 1998). For example, Marsh, Kersel, Havill and Sleigh (2002) examined caregivers and 52 *BI* patients ranging from adolescence to older middle age, finding that by six months post-injury, over one third of the caregivers reported clinically significant symptoms, such as anxiety, depression and poor social adjustment. This symptom picture remained the same after a full year, in spite of some level of increased adaptation to the *BI*. However, the level of subjective distress tended to decline over the first year.

Apparently, over time, *the cognitive and social impairments associated with BI play a larger role than any physical impairment the BI person may have*. Indeed, Carnes (2001) found that family members are vulnerable to stress provoked by the emotional and behavioral changes often exhibited by

the *BI* patient. Family resources, including financial ones, and coping and appraisal skills influenced the stresses experienced. Underscoring this stress, Wade, Taylor, Drotar, Stancin et al. (1997) found that *TBI* produced more family stress than did orthopedic injuries not involving the central nervous system (*CNS*) in children and adolescent *TBI* victims. As can be expected with *TBI* patients themselves, a review of this literature shows that severe *TBI* predisposes family members more so to stress who have poor preinjury functioning and psychological disorders (Wade, Drotar, Taylor & Stancin, 1995). Moreover, in a large ( $N = 465$ ) study, Deloche, Dellatolas and Christensen (2000) found that the appraisal of the patient's difficulties was not significantly different when reported by the patients themselves, the relatives of the patient or the clinician. Common problems noted were impaired executive functions, depression and social adaptation problems.

It should not be very surprising then to note that the single major adjustment problem after one year post-injury is that the brain-injured person has socially inappropriate interactions (Kaplan, 1988). Pre-injury family functioning was highly predictive of post-injury return to work. Family subjective stress in reaction to a patient's *TBI* is also associated with irrational beliefs. Stebbins and Pakenham (2001) conducted a mail-in survey of 116 caregivers of those with *TBI*. *Those who worried the most were those who tended to have the greatest degree of irrational beliefs, opening the door for cognitive therapy for the caregivers as well as the patients of TBI.*

A common misperception is that any impairment that the *BI* patient has is necessarily due to the injury itself, when this is not altogether correct. Macmillan, Hart, Martelli and Zasler (2002) found that 45 adults ranging from

age 18 to 61 who sustained *BI* two years earlier were more likely to have employment and to abstain from substance abuse if their premorbid functioning was free of psychiatric disturbance and substance abuse. *Premorbid coping is then related positively to post-injury coping and recovery.* However, location of the injury is of course significant in this regard, as personality changes associated with social and emotional changes are often due to early acquired or genetic brain damage within the right hemisphere (e.g., Manoach, Sandson & Weintraub, 1995). A sense of loss of self can also provoke a grief-like reaction, due to damage sustained in the temporalfrontal regions and the paranormal religious experiences that often accompany this diffuse damage (Persinger, 1993).

Monitoring therapeutic progress for the brain injured is a difficult task, as most psychometric instruments are of the self-report variety and are incorrectly presumed to have little external support for their validity. However, as one may well expect, measures inherently closer to the source of the problem document brain activity, such as frontally generated electrophysiological responses elicited during a single attentional task, have been found to have superior associations with daily planning and initiation than do traditional paper-and-pencil measures of adaptive behavior. Indeed, self- and family-reports of adaptation in everyday life are reported (Dywan & Segalowitz, 1996) to be closer to such physiological measures and are essential in helping to evaluate the behavioral sequelae of *TBI*. Indeed, except for issues addressing sexuality and interpersonal functioning (Gaudet, 1996), family members' and patients' self-reports of symptoms and difficulties of *TBI* are equally valid.



## Adaptive and Challenged Families

### *The Mentally Ill Parent: Competence Under Siege*

The scope of this problem is quite large. For example, at least one empirical study (Gewurtz, Krupa, Easterbrook & Hogan, 2004) found that almost 84% of their mentally ill sample receiving services did not live with their children and many seldom get to see them. While parental dysfunction is a major issue with mental illness, an unavoidable part of the problem is legal. *Parens patriae* is the legal doctrine that recognizes the state's role in intervening with a parent's right to parent when necessary to ensure the welfare of children (see Grisso, 1986). Part of this legal issue is practical and part is purely diagnostic and evidentiary. On the practical side, severe mental illness impacts on cognitive and emotional processes, producing functional impairments not unlike those of developmental delay (e.g., Garber, 1988). Thus, we should not find it surprising that mental illness is often associated with lack of parental responsiveness and structure. This is precisely what is found. Competent, active, and responsive parenting is interfered with by the intrusion of psychiatric symptoms. For example, Egeland and Sroufe (1981a, 1981b) and Kochanska (1991) identified parental neglect as a serious issue in depressed parents. Social withdrawal is a cardinal negative symptom in schizophrenia. However, as with *DD*, the practical side of things is that a diagnosis of mental illness does not necessarily imply that impaired parenting is an inevitability. Thus, each parent with mental illness needs to have their parenting abilities functionally assessed. Oftentimes, however, courts accept a diagnosis of mental illness as sufficient grounds for government intervention (i.e., *parens patriae*) without functionally connecting child problems to parenting skills. This issue can be very prejudicial to the

mentally ill parent. As we indicate below, this legal dilemma is also very telling for parents with *LDs* and *BIs*.

Having a mental illness does not necessarily spell disaster for parenting. For example, in a large Dutch study, Overbeek, Vollebergh, Meeus, Graaf and Engles (2004) found that adults who recollected their childhood experiences with their mentally ill parents in terms of loving, caring and non-intrusiveness, were less likely to experience internalizing symptoms during their present adult functioning. The risk to the child with a cold and overprotective mentally ill parent is higher for the development of emotional problems in adulthood. The presence of anxiety and mood disorders was associated with such low-quality parental bonds. Using structured interviews, more evidence for impaired parenting was obtained by Diaz-Caneja and Johnson (2004). *Such mothers feared that their illness adversely affected their parenting, but also feared that fully disclosing these concerns to mental health services would jeopardize access and custody. In fact, these concerns are well-grounded in reality (Ackerson, 2003; Gewurtz et al., 2004).* An interview excerpt is very illustrative of the problem:

*"When I am really ill, I wish the children were not there so I wouldn't have that burden to bear. I can't cope with them; their demands are too much for me. And I keep on struggling until eventually I just break down."* (Diaz-Caneja & Johnson, 2004, p 476.)

Hence, dissatisfaction with the lack of interest shown by mental health workers was due, at least in part, to their own reluctance to talk about their parenting problems, which in turn provoked fear of loss of custody and access to their children. In a large UK study (Howard, Shah, Salmon, &

Appleby, 2003), the risks associated with social services involvement after a psychiatric hospital stay included social class (i.e., semi-skilled, unskilled, never employed), being single, having a behavioral disturbance and the partner also having a psychiatric disorder. In particular, the highest risk for social services involvement was for parental schizophrenia and personality disorders. In particular, of the 22% of mothers who were under supervision upon their discharge from hospital, thoughts of self-harm were pivotal in arriving at this decision. Presumably owing to social withdrawal, these authors speculated that the negative symptoms of schizophrenia are especially important predictors of poor parenting.

Thomas and Kalucy (2003) underscored the fact that mental illness alone does not necessarily undermine affective parenting. Indeed, Benjet, Azar, and Kuersten-Hogan (2003) advanced the position that assumptions of a necessary link between mental illness and faulty parenting are often biased. Such a bias view has also been identified by Ackerson (2003). **Functional and social skills pertaining to parenting are better predictors of good parenting than are diagnoses.** While a major red flag, automatically linking mental illnesses to incompetent parenting is akin to automatically linking poverty to incompetent parenting (Garber, 1988). These classifications, while generally useful, are too crude for clinical applicability. **Rather, Thomas and Kalucy felt that the motivational status of the parent is crucial. That is, adverse effects on children tend to result when the illness is chronic and is accompanied by lack of emotional involvement. In particular, these authors suggest that frontal lobe damage or impairment is associated with such lack of motivation, apathy, and mood problems, including lack of initiation.** *Everyday parenting requires planful, goal-directed behavior, which is often absent or severely impaired in mentally ill parents. Neglect of parenting*

*results.* Using semi-structured interviews with 35 mentally ill parents, these authors found that about half of these parents had little interaction with their children, relied heavily on available supports, isolated their children, and that their children had little understanding of their parents' illness. Such amotivational problems lingered beyond a few weeks after returning home from the hospital for about 20% of these patients. **These authors recommended that the provision of parental training should be provided after hospital discharge and before welfare agencies have been notified of any suspected abuse or neglect.**

Perhaps an alliance with mental health services can overcome some of the legitimate custodial fears that mentally ill parents have (Diaz-Caneja & Johnson, 2004). **Nicholson and Blanch (1994) reviewed the availability of parenting programmes for people with mental illness and found that the majority of the 69 programmes identified did not focus on the specific needs of the mentally ill. Only nine programmes addressed these needs. Specific unmet needs included decision-making training regarding reproductive issues and whether to retain or seek to resume custody, with particular emphasis on rehabilitation plans. Other areas of concern focus on medications and their impact on parenting. How to talk to the children about mental illness was also addressed. Moreover, Ackerson (2003) underscores the fact that this fear of loss of parenting rights may be well-grounded, as judges frequently associate a diagnosis with poor parenting. Ackerson advocates a comprehensive assessment of parenting which includes the severity of the illness, assessment of strengths and competencies, contextual observations in and out-of-home, and the availability of social supports needed by the whole family.**

While it is difficult to separate the effects of genetic concordance between mental illness in parents and children from the effects of adverse parenting, Mowbry, Oyerman, Bybee and Macfarlane (2002) highlighted the fact that parenting skills often suffer when mental illness is involved. In particular, the extensive literature on this topic underscores interruptions in the formation of secure attachments and neglectful parenting as the primary problem with maternal depression and unipolar depression in particular. However, whether such effects are more pronounced with schizophrenia is still somewhat controversial and to some extent irrelevant. To illustrate, Mowbry et al. interviewed 379 mostly symptomatic women from community mental health agencies in Michigan. Results showed a significant main effect on parenting of diagnosis. Specifically, diagnoses were predictive of parental stress, lower nurturance, and lower satisfaction with the relationship with the child, especially with schizophrenia. Women with schizoaffective disorder had higher parenting stress scores. Interestingly, hospitalized women had lower parenting stress scores. Symptomology was almost entirely related to the level of parental stress reported. Obviously, hospitalization serves to reduce symptoms. Lower nurturance scores were also reported by women with schizoaffective disorder. Hospitalizations were positively related to relationship satisfaction, which was rated more highly by African-American mothers. *The upshot of these findings was that symptomatology was significantly more pertinent than the specific diagnosis in terms of its impact on parenting.*

Reminiscent of Garber's (1988) findings, poverty severely impacts the degree to which psychiatric symptoms impact parenting. In a British study, Bifulco et al. (2002) showed that of 276 vulnerable (i.e., single, low SES) inner-city mothers were four times as likely to have sustained a yearly

psychiatric disorder and twice as likely to have experienced childhood adversity, such as severe neglect or abuse. Physical abuse in particular was more common in this vulnerable group. Neglect or abuse of children was the single best predictor of children's disorders than was the presence of maternal depression. These results highlight the significance of childhood neglect and abuse rather than mental illness diagnoses as the more salient issue in preventing childhood disorders. Nearly all types of disorders, such as depression, anxiety, and substance abuse/dependence, were higher in the presence of poverty. This poverty-related effect was particularly felt for depression and substance dependency. Child early disorder connected to chronic lifetime maternal depression in 33% of these parents, but neglect and abuse was the model that best fit the data a pattern also identified by Jacobsen and Miller, 1997.

In line with the persistent poverty hypothesis, Oyserman (202) found that the best predictors of effective parenting attitudes were maternal stress and current mental health. Parental involvement was best predicted by parenting attitudes, educational level and support. Mowbray, Oyserman, Bybee, MacFarlane & Rueda-Riedle (2001) found that poverty is strongly associated with significant stresses in clinically involved mothers and depression is the single most common disorder. Of 68.1 mothers below the poverty line, 39.8% were depressed. Little is currently known about effective response to medication and child-rearing skills.

### ***Parental Developmental Delay***

Parental adaptation of normal parents to the child with developmental delay is more charted waters, but encompasses family stress and the family's

coping resources, especially the supportiveness of their ecological environments (Crnic, Friedrich & Greenberg, 2002). As can be imagined, this need for such support is even truer for the issue of otherwise normal children adapting to parental *DD*. Tebes, Kaufman, Adnopoiz and Racusin (2001) examined children's resilience (i.e., successful adaptation despite adverse circumstances) in the face of their parents' mental disorders, **finding that family psychosocial processes are more important to the children's adaptation than are parental psychopathologies. Parental performance, rather than familial stress and the parent-child bond, was related to child adaptation. Thus, the tasks of parenting count for a great deal. Garber (1988) found that when maternal IQ is at or below 80, most of these parents are generally incapable of minimally acceptable basic parenting and require assistance.** Unassisted, such parents often tend to have children who are also developing borderline *DD*. It is relatively unusual for a normally functioning child to have an unassisted parent with *DD*. Notwithstanding this fact, normal and bright children of *DD* parents - who presumably and speculatively may well have had familial supports - are also at high risk for adaptation problems, such as taking on a pseudo-parenting roll (O'Neal, 1985). Obviously more work needs to be done to learn whether the presence of non-*DD* spouses and other healthy adult family members can help to ameliorate any adverse parenting affects due to parental *DD*.

The literature dealing with *LD* is fraught with definitional issues. While American studies define "*LD*" in terms of specific information processing problems impacting on academic achievement for those who are otherwise intellectually normal, British studies (e.g., Mencap, 1996) refer to *LDS* in terms of intellectual deficiencies which onset before birth, at birth, or during infancy or adolescence as indexed by low *IQs*. As such, these *LD* parents are really developmentally delayed (*DD*) in American terms.

(James' (2004) U.K. literature review of *LD/DD* parents identified significant risks to children.

*As is also true in terms of parental mental illness and developmental delay, Booth (2004) noted in Britain that LD/DD parents face a higher risk of losing their children to the authorities under parens patriae. Such parents often have difficulties accessing antenatal care and expose their own children to intellectual and linguistic delay, as well as including behavior problems. Because of the LD/DD, such parents often have trouble comprehending advice that they receive from others and translating it effectively into practice. Compounding the problem, and commensurate with Garber's (1988) data, when both parents have LD/DD, the risk is 40% that their children will also have LD/DDs. Obviously, given Garber's (1988) findings, the greater the parental DD, the higher the risk to the children. As the usual primary caretaker, the risk of intellectual delay is understandably greater when the mother has a LD/DD. As also indicated above with respect to DD and mental illness, LD/DD parents often have poverty-linked problems as well. Poor housing and social isolation are often involved. Behavioral problems are particularly noted for boys.*

*As noted by James (2004), the essential problem for LD/DD parents is one of ineffective discipline. Thinking from the child's perspective is a core parenting skill and is particularly difficult for such parents, as this perceptual leap requires adequate comprehension and communication skills - the very capacities that are impaired in an LD/DD. Garber (1988) also identified a lack of appropriate verbal interaction between DD parents and their children as central to the development of mild developmental delay. Moreover, James noted that LD/DD mothers are particularly vulnerable to child*



abuse from their male partners. As noted above, ADHD-like symptoms may accrue from a history of child abuse. At the core of the discipline problem is the more restrictive and punishing parental style that frustrated LD/DD parents often resort to in trying to communicate effectively, especially when their children have higher intellectual abilities than their own. As one would expect, social networks of such parents tend to be smaller but contrary to social support theory, coping is better when the families are smaller, not larger. Perhaps such parents are less distracted and more focused on parenting tasks when the family is smaller. Studies need to be done on single-parent LD parents to determine whether a healthy spouse assists in child management.

Booth (2003) described LD/DD [note in UK, LD is often used as a label for developmental delay] parents in Britain as a "stolen generation" as they needlessly and frequently fall victim to the child protection system. He argues that LD/DD parents need education, training and support and instead of getting this assistance, are placed at higher risk (40-60%) of losing their children to the authorities. In Booth's view, parental competency is often judged far too strictly for such parents. Actual documented child abuse by LD parents is actually rare. As the Milwaukee Project (Garber, 1988) clearly showed, since LD parents have the potential to learn new skills to improve their parenting, not providing them with such training generates what he calls a "calculus of despair". Such pervasive feelings of vulnerability are understandable when one is identified with specific needs. Woodhouse, Green and Davies (2002) noted that most of their 32 British children of LD/DD parents were subject to some form of government sponsored intervention and had intellectual delay. But with the right opportunities to learn skills, and provided with suitable support, the parents were capable of "good enough"

parenting. Aside from the intensive multimodal interventions documented by Garber (1988), there are currently no specifics as to such training. Borrowing from the field of parental brain injury, which often causes intellectual and emotional problems, may prove to be useful, as discipline problems are central here as well.

### ***Parental Learning Disabilities***

Although with us since recorded history, diagnosis of *LDS* has not been routine until somewhere in the mid-1970s. Accordingly, identifying parenting problems for parents with *LDS* is a fairly recent issue. However, Clarke (2002) had 15 genuinely *LD* parents provide qualitative data dealing with their experience of parenting. While these parents felt protective of their children's self-esteem and academic success, problems with time management and the impact of their own emotional reactions surrounding their own *LDS* generated feelings of inadequacy, guilt, regret, frustration and the need to become self-reliant. As with so much of the literature reported here on learning disorders, poverty is often a cause and a result of such problems. Indeed, *LDS* undermine children's self-concepts and erode social skills (Gadeyne, Ghesquire et al., 2004). *LDS* do not disappear after formal schooling is complete. Considered together with the risk of co-morbidity with *ADHD* (e.g., *McBurnett, 1991*), *LD* parents may then be inattentive, and relatively inept at social rule learning. At its core, competent parenting is an exercise in social rule transmission to offspring. Added to this burden, adolescent delinquents are usually *LD* (Watts, 1986; Zinkus et al., 1979). What is more, the social supporters of *LD* adolescents are at high risk for burnout (McGrother et al., 1996). Thus, *LD* parents are apt to have fewer social supports, such as a competent extended family, to draw on in buttressing their own need for parenting assistance.

Teenage parenting is a risk factor in modern western society and this social issue is more marked for those minority adolescents who are at risk for economic and educational disadvantage (Sommer, Keogh & Whitman, 1995). It is also more likely that *LD* adolescence are over-represented in this population, as they are more likely to drop out of school and to have lower incomes. Watts (1986) examined the social lives of *LD* adolescents. Assuming that *LD* adolescents are of normal intelligence and are commonly deficient in general learning strategies, such as listening, attention, expression and comprehension, Watts found that they have problems in their occupational and marital roles. Leaving school, they have fewer social involvements and are, as a group, relatively unpopular. In Watts' words: "Inevitably, then, many a learning disabled youngster - voluntarily or involuntarily - finds himself past school age and looking for a job. (pp. 263)". Dropping out of school and involving oneself in crime are popular attractions for *LD* adolescents. But, according to Watts' review, so is early marriage for young women. The military becomes relatively more attractive for *LD* young men. Thus, poverty, marital stress and harsh and ineffective parenting are likely the result. The connection of *LDS* and parenting difficulties is a field ripe for investigation.

### ***Parental Brain Injury***

Parental *BI* affects parenting. Passar (1993) investigated the effects of parental *BI* on the behavior of both parents and children in 24 families and 52 children, where one parent had *BI*. The uninjured parents reported that most of their children experienced at least some negative behavior change after the parents' injury. Ten of these families had significant

problems, such as a poor relationship with the injured parent, acting out and emotional problems. Interestingly, compromised parenting was noted for both injured and noninjured parents. Comparing 16 parents with *BI* and 16 parents without *BI*, Uysal, Hibbard, Robillard and Pappadopoulos (1998) found that *BI* parents had a number of parenting issues. They set fewer goals for their children, expressed less encouragement of their skill development, had less emphasis on obedience to rules and order, less stress on work, less nurturing, and had lower levels of active involvement. In addition, noninjured spouses expressed less warmth, love and acceptance toward their children. Not surprisingly, the children of both parents in the *BI* group viewed both parents as lax. Curiously, although both parents and children in this group were slightly (i.e., subclinically) more depressed, there were no significant differences in actual behavior problems between the two groups.

Uysal et al. (1998) noted that the cognitive sequelae of *TBI* often affect parenting skills such as attention to detail, ability to divide attention, memory for events, time management, ability to organize activities, irritability, aggression, mood swings, apathy, social withdrawal and depression. The noninjured parents have an increased child care burden, feeling depressed, isolated and lonely. Income stresses and weakened social supports are common. Accordingly, the children may mirror their parent's altered behavior, thus developing emotional, behavioral and cognitive problems of their own, such as reverse parenting. Depressed parents become detached, unresponsive and self-absorbed. The results of *BI* parenting include children having somatic symptoms, dependency, argumentativeness, aggression, over-activity, dependency, and school failure. The *BI* parents use less praise and encouragement, more yelling, impatience, and engage in less fun and positive activities. Thus, acting out and disobedience are core

problems tied directly to ineffective parenting skills linked directly to the symptoms of TBI. These effects were seen after at least two years post injury, with an average of nine years.

On a more encouraging note, more effective parenting can be taught to BI parents. Ducharme, Spencer, Davidson and Rushford (2002) trained 12 oppositional children (2-7 years) of BI parents with cognitive deficits, impulsiveness, and emotional instability. There were generalized and durable increases in observed child compliance after parenting treatment. Parental self-esteem improved too. Live child compliance data were obtained or videotaped on 120 tasks, such as hygiene, dressing and leisure activities. Parents were trained to deliver selected requests over several days in a polite but firm tone of voice. Immediate repetitions were avoided and noncompliance was not given consequences. Parents learned to give their children enthusiastic praise and physical contact after each compliance response. Ultimate compliance was increased 33% over baseline. Most interestingly, the most improvement occurred for the most severe noncompliance.

## Summary and Conclusions

While openly admitting the futility of dealing effectively with all the unanticipated exceptions to the rule, it would still be remiss not to underscore the preeminent importance of the early years as vital to normal child and adult development. *Nearly all types of disorders, such as depression, anxiety, and substance abuse/dependence, are higher in the presence of poverty.* For example it is patently obvious that even with wholesale upward mobility during the preschool years, those children who escape from the bottom *SES* quartile developed similarly to those who remain. Poverty is also a huge risk factor for staying poor. **What is equally telling is that the risk factors for adapting sub-optimally to parental developmental delay, BI and parental mental health symptoms are seated in poverty, which debases early childhood experiences and has lifelong effects. Thus, effective interventions for learning and behavioral disorders are best dealt with early and within the family context. It is also evident that fractured, socially unsupported and isolated single-parent families are obviously at more risk of adjusting poorly to a child with a severe disability and are also more likely to foster disabilities in their offspring. Fortunately, training dysfunctional parents how to be more effective disciplinarians is very feasible and needs to be attempted for parents with all types of cognitive and psychiatric impairments.**

For special populations, such as Natives, it is crucial to have suitably normed tools for assessment and remediation that separate the effects of culture and language from those of impoverishment and family instability. Poverty and ethnic marginalization hugely exacerbate the powerful effects of alcohol on fetal development. Furthermore, brain

development within the first six years is the basis of learning, behavior and health over the entire life span. Destroying brain structure and function in these early stages is much more likely to happen in the midst of poverty and marginalization. Negative, neglectful early experiences, such as lack of appropriate verbal stimulation, are likely to have irreversible and sustained long-term effects on development. Thus, placing Native children in foster families has not been shown to improve matters at all. Early experiences stressing strengths and cultural values are much more effective than forcing them to conform to a school system in which the norms of instruction clearly may be at variance with their own culture. Obviously as well, finding the magic formula that arrests maternal drinking in high risk populations continues to be a major challenge.

Many learning and behavioral problems that would occur within any competent family are aggravated by early maternal deprivation, which is usually accompanied by persistent poverty. Nevertheless, there is an economic cutoff point approximately 4-5 times the government definition of the individual poverty level above which children do fairly well, and below which children typically do not acquire a readiness to learn. Parents are less depressed and more responsive and children's language and thinking improves as income moves toward the threshold. Income stability is also more important as the poverty line is approached. As a society, we simply cannot afford to go on like this, pretending that poverty, intellectual decline, and mental illness are somehow unrelated. Clearly they are not. Indeed, indulging in separate diagnostic entities tends to cloud the major picture of poverty and despair. The usefulness of nurturing social stimulation cannot be over-emphasized. For example the quality of the social milieu is more

effective for the mentally retarded than sheer amount of time spent in a remedial programme.

As for mentally ill, LD/DD and possibly BI parents, the dilemma of child protection pits therapists at opposite poles from being true help agents. Clearly this impasse places children at increased risk and should be stopped. In all cases, functional parenting capacity assessments should be done and followed up by suitably fair periods of remedial mediation and training. Mentally challenged parents are also in an additional quandary, as they may not clearly understand the nature of the services offered to them and their children. Lack of accessibility to services for all kinds of learning disorders is impeded when poverty is involved. Perhaps the use of legal substitute decision makers would help considerably to close this ethical gap. Given the important policy implications which affect the quality of life of our disabled citizens and their children, this is an area of considerable potential growth in terms of research ethics. The application of the legal doctrine of *Parens Patriae*, where the state intercedes to help protect children's interests, should be formally modified to incorporate clinical parenting interventions as a mandatory first step instead of reflexively placing children in custody simply on the strength of a parental diagnosis.

One of the most intriguing aspects of early deprivation is the distorted perception that these children tend to acquire regarding their social lives and their peer group in particular. Socially maladjusted normal IQ children often have inaccurate perceptions of other children's intentions, misperceiving untoward motives and acting accordingly. The risks for mental retarded children are even greater. Complicating matters considerably is the



troubling fact that teachers were least accepting of those students who were mentally retarded, emotionally distant or delinquent. Indeed, teacher and peer rejection of the failure-prone LD adolescent may foster a delinquent identity. Clearly, cognitively and behaviorally disabled children have an uphill struggle. But there is hope. Sharing common activities over time has shown promise in improving feelings for both disabled and nondisabled groups alike. Such integration can indeed be risky when antisocial behaviors are afoot, but economically disadvantaged special education students can acquire the basic skills to assist them in remaining within a regular classroom. Integration is also assisted by having teachers trained in specific knowledge.

Hyperactive *ADD* school children are significantly more depressed, have lower achievement scores and have more disciplinary and social problems, including higher rates of school absenteeism. It is thus very unfortunate that parents of *ADHD* children often feel that they cannot rely on extra-family supports. Parents respond well to a clear and early diagnosis. Hence, delays are very counterproductive both in terms of helpfulness and in terms of containing costs. Such delays place LD children in a position of adapting poorly to their peers and they often have early onset *CD*. Depression is a known risk factor for neglectful parenting, raising the spectre of heightened child neglect and abuse amongst these cognitively disabled children.

Alcohol and poverty are strongly linked. In rural, marginalized and working class populations and Native communities there is a shocking elevation of *FAS*. What is even more alarming is the fact that *FAS* prevention programmes are good at raising awareness training for middle class mothers

but do little for those in the high risk, high consumption, impoverished group. Addressing the symptom overlap of *FAS/FAE* and other learning disorders is crucial to clearing up treatment effectiveness and dual diagnostic issues.

Brain injury is also very demanding of prompt attention. Prevailing wisdom in treatment is that cell death occurs over a period of months after the initial injury is sustained. Given the social and personal impact on immediate family members is often traumatic and overwhelming, we must intervene with remedial family-based cognitive and social programmes as soon as possible after brain injury is sustained. Those who worried the most were those who tended to have the greatest degree of irrational beliefs, opening the door for cognitive therapy for the caregivers as well as the patients. **What is more, the earlier the intervention, the better the likelihood of restoring competent parenting practices.** The overall financial and humanitarian savings to the system of effective early family intervention on all fronts is staggering.

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