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Minor Physical Anomalies and Behavior in Children: A Review

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The recent literature concerning minor physical anomalies (MPA) and their relation to behavior is reviewed. Research seems to indicate that for males there is considerable consistency in the results but the finding with females is tenuous at best. It appears that a high number of MPA are evident in several pathological groups of boys, as compared with normal controls. In addition, there is a suggestion that MPA are correlated with severity of hyperactivity, IQ, and school achievement. Furthermore, there is also a relationship between a high number of MPA and obstetrical complications. The etiology of MPA and their utility in predicting pathological behavior is discussed.

INTRODUCTION

Recent attempts to delineate etiological factors in childhood psychopathology, as well as interest in the development of reliable diagnostic tools, have led to considerable study of minor physical anomalies (MPA). These MPA—head circumference outside the normal range, malformed ears, large gaps between the toes—to mention only a few, are frequently associated with Down's syndrome. However, without associated major morphological aberrations these MPA are of little medical or cosmetic concern and cannot be discerned unless systematic and accurate measures are taken by trained individuals (Waldrop & Goering, 1971). Those studied have included groups of newborn children (Burg, Hart, Quinn, & Rapoport, 1978; Burg, Rapoport, Bartley, Quinn & Timmins, 1980; Quinn, Renfield, Burg, & Rapoport, 1977; Waldrop, Bell,

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McLaughlin, & Halverson, 1978), school-age children (Rosenburg & Weller, 1973; Waldrop, Bell, & Goering, 1976; Waldrop & Goering, 1971), schizophrenic and autistic youngsters (Campbell, Geller, Small, Petti, & Ferris, 1978; Goldfarb & Botstein, 1967); Links, Stockwell, Abichanandi, & Simeon, 1980; Walker, 1977), mentally retarded children (Firestone, Peters, River, & Knights, 1978; Smith & Bostian, 1964), psychoneurotic children (Firestone and Prabhu, 1983; Steg & Rapoport, 1975), learning-disabled children (Steg & Rapoport, 1975), speech- and learning-impaired children (Waldrop & Halverson, 1972), hyperactive children (Firestone, Lewy, & Douglas, 1976; Firestone et al., 1978, 1983; Rapoport, Quinn, & Lamprecht, 1974; Rapoport & Quinn, 1975; Waldrop & Halverson, 1971), and inhibited children (Waldrop et al., 1976; Waldrop & Goering, 1971).

The first significant investigations concerned with the relationship of MPA and disordered behavior were published in the mid-1960s. Goldfarb and Botstein (1961) discovered that schizophrenic and behavior-disordered children manifested more MPA than normal controls.

Smith and his associates studied the incidence of both major and minor physical anomalies in normal (Marden, Smith, & MacDonald, 1964; Smith & Bostian, 1964) and pathological groups of children. They report relatively few MPA in normals compared to 10% of a group of cleft lip and palate children, 18% of children with septal defect, and 92% of a group of retarded children. The implication was that defects in brain functioning may also be a congenital abnormality and that MPA may act as marker of these disorders.

THE MEASUREMENT OF MPA

The majority of recent studies have employed the list of minor physical anomalies developed by Waldrop and Halverson (1971), developed from that of Goldfarb and Botstein (1967) by selecting those anomalies that significantly differentiated schizophrenic from normal children (see Table I): fine electric hair, head circumference out of normal range, epicanthus, hyperteliorism, low-seated ears, adherent earlobes, high-steepled palate, curved fifth finger, and third toe longer than second toe. Most anomalies were assigned a weight of 1 or 2 according to the degree of deviation from the normal. Those anomalies that were more frequent in schizophrenic than in normal children, but not significantly so, were assigned a weighted score of 1. More than one hair whorl, soft pliable ears, and smooth-rough spots on the tongue either showed no incidence at all or were more common in normal than in schizophrenic children, and were noted but were assigned a score of 0.

Table I. Minor Physical Anomalies and Scoring Weights

Anomaly	Weight
Head	
Fine electric hair	
Very fine hair that will not comb down	2
Fine hair that is soon awry after combing	1
Two or more hair whorls	0
Head circumference outside normal range	
1.5	2
1.0 1.5	2
Eyes	
Epicanthus	
Where upper and lower lids join the nose, point of union is	
Deeply covered	2
Partly covered	1
Hypertelorism	
Approximate distance between tear ducts	
1.5	2
1.0 1.5	1
Ears	
Low-seated ears	
Point where ear joins the head not in line with corner of eye and nose bridge	
Lower by .5 cm	2
Lower by .5 cm	1
Adherent ear lobes	
Lower edge of ears extend	
Upward and back toward crown of head	2
Straight back toward rear of neck	1
Malformed ears	1
Asymmetrical ears	1
Soft and pliable ears	0
Mouth	
High-steepled palate	
Roof of mouth	
Definitely steepled	2
Flat and narrow at the top	1
Furrowed tongue (one with deep ridges)	1
Tongue with smooth-rough spots	0
Hands	
Curved fifth finger	
Markedly curved inward toward other fingers	2
Slightly curved inward toward other fingers	1
Single transverse palmar crease	1

Table I. Continued

Anomaly	Weight
Feet	
Third toe longer than second	
Definitely longer than second toe	2
Appears equal in length to second toe	1
Partial syndactylia of two middle toes	1
Big gap between first and second toes	1

Despite the fact that the scoring procedures developed by Waldrop and Halverson (1971) have been adopted by most researchers, several problems do exist. Many researchers tend to modify the list or scoring procedures to suit their purpose or because of the unique population being studied. Thus, for practical reasons some reports do not, for example, include measures of fine electric hair (e.g., in newborns or when the population is made up largely of black children), while others have dropped certain anomalies because of their frequent occurrence in population—Jacklin, Maccoby, and Halverson (1980) did not include the epicanthal measures. Some investigators have also added certain anomalies—for example, Walker (1977) included shortened fifth fingers as an anomaly. These differences in scoring procedures, in addition to the fact that a breakdown of scores for individual anomalies is often not published, make comparisons between different studies difficult and may account for the large variation in MPA means found in various studies. Examples of this can be seen in MPA scores for normal elementary school children, which is reported as 1.4 by Firestone et al. (1978) and 2.88 by Steg and Rapoport (1975) for boys only. For girls, Rosenberg and Weller (1973) report means of 2.61, while Waldrop and Goering (1971) submit a mean of 3.54. Mean MPA scores for both sexes together range from 2.7 (Halverson and Victor, 1976) to 3.53 (Walker, 1977).

MPA scores in clinical populations also seem to vary considerably among different research locales. For example, in autistic children MPA scores are reported as 4.25 by Steg and Rapoport (1975), 5.76 by Walker (1977), and 6.24 by Links et al. (1980). The differences among various research teams studying hyperactive children do not seem as great—3.58 by Rapoport et al. (1974) and 4.00 by Firestone and colleagues (Firestone et al., 1976, 1978). Despite the interlocation differences in MPA scores, there seem to be consistently high interrater reliabilities reported within projects that are generally between .75 and .90.

STUDIES OF NONPATHOLOGICAL GROUPS

In an initial study of normal 2½-year-old children, Waldrop, Pederson, and Bell (1968) assessed children without a history of pregnancy and/or birth complications for the presence of MPA. These analyses disclosed that MPA were significantly correlated with frenetic, restless, and aggressive behaviors and more specifically with an inability to delay gratification, nomadic play, spilling and throwing behaviors, perseveration, and oppositional behaviors toward peers. The authors suggested that this cluster of behaviors can be identified as those commonly associated with children diagnosed as "hyperactive."

Waldrop and her associates replicated the initial study with children who had not been screened for pregnancy and birth complications. The correlations between MPA and behavior in this study were very similar to those obtained in the initial investigation for boys. However, the "hyperactivity" factor found to characterize high-anomaly girls as well as boys in the initial investigation was replaced by a factor characterized as "intractibility-inhibition" for girls (Waldrop & Halverson, 1971). A follow-up study to determine the stability of both MPA and behavior was conducted when the children were 7½ years old. Both MPA and behavior that can be characterized as hyperactive were found to be stable over this 5-year period. Also discovered was the fact that children with high MPA tended to be clumsy and have lower IQs, suggesting a central nervous system aberration (Halverson & Victor, 1976).

Rosenberg and Weller (1973) found that verbal performance (Peabody Picture Vocabulary Test) was significantly negatively correlated with the number of anomalies in normal first-grade children. However, the presence of anomalies was not related to Draw-a-Man IQ, impulsivity, or teacher evaluations of classroom behavior. A very strong relationship was observed between the presence of more than five anomalies and teachers' recommendations that children repeat grade 1. The inconsistent result on the intellectual and behavioral measures, in contrast to the marked difference in global school failure, prompted these authors to speculate that more precision may be required to isolate the finer aspects of intellectual behavioral functioning that are related to both the MPA and academic failure. This study also revealed that twice as many boys as girls had more than five MPA, but the relationship between sex and academic failure is not reported.

It is of interest to note that other papers have found a significant inverse relationship between MPA and IQ (Waldrop & Halverson, 1971; Waldrop et al., 1976; Firestone and Prabhu, 1983) and between MPA and academic achievement (Halverson & Victor, 1976).

SEX DIFFERENCES

Further clarification of the previous findings was attempted by comparing MPA in children chosen by their teachers as being the most hyperactive with those chosen as exhibiting behavior in the normal range. This investigation revealed that "hyperactive" boys exhibited more anomalies ($\bar{X}$ = 5.59) than "normal" boys ($\bar{X}$ = 3.00), but this relationship did not hold for girls ($\bar{X}$s = 3.50 and 3.54, respectively; Waldrop & Goering, 1971).

Waldrop et al. (1976) confirmed that girls considered to tbe inhibited had more MPA than their outgoing counterparts ($\bar{X}$s of 4.85 and 3.38, respectively). Furthermore 33% of inhibited, but only 8% of outgoing girls, also had lower IQ's, poorer coordination, and a greater number of days absent from school. The suggested explanations for these differences between the sexes were that (1) there may be fundamental central nervous system structures and/or behavioral differences mediating motoric and expressive behaviors, (2) these basic differences could lead to a differential susceptibility to genetic or teratogenic agents, or (3) early socialization of the sexes into appropriate sex-role behaviors results in the observed differences.

In a series of factor-analytic studies of normal preschoolers, O'Donnell and his associates, using the Behavior Problem Checklist, found that MPA were correlated with distractibility for boys but not for girls (O'Donnell, O'Neill, & Staley, 1979). They suggest this finding supports the position of sex differences as one factor in the multiple congenital determination of problem behavior in children (O'Donnell & Van Tuinan, 1979).

Only one study has reported that MPA are related to high activity levels in girls rather than boys (Jacklin et al., 1980). This finding contradicting previous research may be attributable to the brief observation periods used in the study, and/or the situational specificity of the behavior observed.

MPA IN BEHAVIOR-DISORDERED CHILDREN

Several studies have been conducted with clinically referred children using the Waldrop scale. Most of the research to date has been conducted with children diagnosed as "hyperactive," with some recent interest in children exhibiting autism and childhood schizophrenia.

Quinn and Rapoport (1974) studied 81 elementary school boys diagnosed as hyperactive and further subdivided them into classifications of unsocialized aggressive, overanxious reaction, or hyperactive reaction. All children were measured for the presence of MPA and rated by both parents and teachers using the Conners behavior rating scales (Conners, 1969,

1970). The mean MPA score for the group as a whole was 3.45, with the unsocialized aggressive, hyperactive reaction, and overanxious reaction subgroups showing means of 4.00, 3.58, and 2.53, respectively. A low but significant correlation ($r = .38$) was reported between hyperactivity as measured by the Conners Teachers Rating scale and MPA, and 41% of boys who scored 9 or higher on the Conners Scale exhibited an MPA score of 5 and above. The majority of children with MPA scores greater than 5 had an onset of symptoms prior to age 3 (22/25). The presence of MPA was not associated with IQ or number of soft neurological signs. These authors suggest that their findings support a view of biological differences between neurotic and antisocial aggressive children, who are considered to be at the extreme of the "hyperactive" population (Quinn & Rapoport, 1974).

In a further study, the majority of these boys were tested for plasma dopamine-B hyroxylase (DBH) activity. This enzyme is involved in the biosynthesis of norepinephrine and is thought to effect peripheral sympathetic nervous system activity. DBH activity was found to correlate with MPA ($r = .38$) but not with teachers' ratings of behaviors. A subgroup of these children were selected with either a high (≥ 6) or low (0-3) anomaly score and matched on initial score of the Conners hyperactivity factor. This subdivision did not predict improved behavior when these children were treated with stimulant medication.

Firestone et al. (1978) compared the incidence of MPA in hyperactive, mentally retarded, and normal boys and their families. The hyperactive and mentally retarded boys exhibited significantly more anomalies than normal boys ($\bar{X}$s = 4.00, 4.67, and 1.40, respectively), but there was not a significant difference between the pathological groups. Furthermore, the siblings of the two pathological groups had MPA scores as high as the probands and significantly higher than the siblings of the normal controls. There was no evidence of psychopathology in the sibling groups. This finding questions the utility of MPA as markers of pathological behavior. It appears that there would be an unacceptably higher number of false positives using MPA as the criterion.

Steg and Rapoport (1975) compared normal, psychoneurotic, learning-disabled, and inpatient boys in a residential treatment center for autistic-like children. The autistic and learning-disabled boys exhibited mean MPA scores of 4.25 and 4.22, respectively, while the normal and psychoneurotic boys had means of 2.88 and 2.57. As there was not a significant difference between psychoneurotic and normal boys on MPA, the two groups were combined. Both the autistic and learning-disabled boys were found to have more MPA than the combined group. The percentages of boys with an MPA score of 5 or above were 19.4%, 19.2%, 43.4%, and 46.4% for the normal, psychoneurotic, learning-disabled, and autistic

groups, respectively. A nice feature of this study was that individual anomalies were reported. Only head circumference beyond normal range and high arched palate were found to significantly distinguish the learning-disabled or autistic groups from normals and psychoneurotics. It is interesting that these two anomalies were the most frequently found in all four groups. MPA was not correlated with IQ or linguistic competence for any group, and there was no evidence of a cluster of MPA that might differentiate one clinical group from another.

Firestone and Prabhu (1983) also studied hyperactive psychoneurotic and normal control children and their families and found that hyperactives had more MPA than normal controls, but neither of these groups differed from the psychoneurotic group ($\bar{X}$s of 3.48, 1.58, and 2.42, respectively). When the psychoneurotic groups and normal control group were combined, the hyperactives had significantly more MPA. Also found was a significant correlation between MPA and Peabody Picture Vocabulary IQ in the probands (no IQ scores were available for siblings).

Walker (1977) reported a high incidence of abnormal head circumference and arched palates in groups of normal and autistic children. Nevertheless, the autistic children demonstrated a higher incidence of low-seated ears, hypertelorism, and syndactylia. The mean MPA score for autistic children was 5.76, compared to 3.53 in normals. The usually higher MPA scores in this study were attributed to differences in subject selection, but they may also have been due to the addition of a shortened fifth finger to the Waldrop list of anomalies. The presence of a high palate and low-seated ears tended to be associated, and a significant disassociation between adherent earlobes and low-seated ears and between syndactylia and abnormal head circumference was noted (Walker, 1977). This is the only study to date to report the clustering, association, or disassociation of anomalies. The finding of significantly higher MPA in autistic children than in normal controls was further confirmed by Campbell and her associates (Campbell et al., 1978). Anomalies were analyzed separately for each anatomical region, but only anomalies of the mouth and ears were found to differentiate significantly between autistic and normal children.

It appears quite clear that, as a group, hyperactive, learning-disabled, retarded, autistic, and schizophrenic children demonstrate numerous MPA. Although other clinical groups (e.g., antisocial or aggressive children) may also show this high incidence of MPA, a lack of controlled studies precludes further comment. The relationship of MPA to hyperactivity is strengthened by several investigations that have found significant correlations between MPA and behaviors usually associated with hyperactivity in large groups of clinically undifferentiated boys (Firestone et al., 1976; Halverson & Waldrop 1976; Waldrop et al., 1968) and within groups of boys diagnosed

as hyperactive (Quinn & Rapoport, 1974; Rapoport & Quinn, 1975). At the same time, however, the correlations are generally quite low (r = .25), suggesting that MPA may play a rather minor role and that other factors may be more important.

INFANT FOLLOW-UP STUDIES

Two long-term studies have assessed the ability of MPA in predicting behavior in childhood from MPA measured in infancy. Rapoport, Pandoni, Renfield, Lake, and Ziegler (1977) appraised 193 newborns and grouped them on the basis of a high (5), middle (3-4), or low (0-2) MPA score. At 5 months of age children were rated by their mothers on the Carey Infant Temperament Scale, but none of the infant temperament variables were found to correlate with MPA.

Of these infants, predominantly from the high and low MPA groups, 123 were examined at 1 year of age (Quinn et al., 1977). MPA were found to be reliable and stable from birth to 1 year of age, and there was a low but significant correlation between MPA and irritability. Of the 9 infants considered to be markedly irritable at 1 year, 8 were in the high-anomaly group. Analysis by sex showed that high-anomaly females were less active and more withdrawn, whereas high-anomaly males showed more negative mood and a higher threshold, and were less adaptable at 1 year of age (Burg et al., 1978). At 2 years of age high-anomaly girls exhibited more negative emotional tone, while high-anomaly boys were less cooperative during psychological testing and their mothers reported them to be more irritable and suffering a greater incidence of night awakening. The authors suggest that the findings support those of Waldrop and her colleagues, who reported a relationship between MPA and "difficult" behavior, with differential patterning being observed for males and females (Waldrop & Halverson, 1971; Waldrop et al., 1976). However, they also point out that a high anomaly score does not inevitably predict the development of later behavior problems (Burg et al., 1980).

A subsample of the boys from the longitudinal study (N = 23) were seen as part of a large observational study in a research nursery school setting. The anomaly score for this subsample was found to be stable from birth to 3 years. Factor analysis of 23 behavioral categories yielded factors labeled as short attention span, peer aggression, and impulsivity. These three measures were significantly related to the presence of MPA. Combined into a composite score, they yielded a still higher correlation (r = .67) with MPA score (Waldrop et al., 1978).

It thus appears that MPA are stable and, at least with males, can be used to predict to certain behavior patterns in nonclinical groups of preschoolers. The behaviors that correlate with MPA are attention span, hyperactivity, impulsivity, and aggression. However, the correlations remain rather weak. Predicting problematic preschool behavior on the basis of newborn MPA would yield a high rate of false positives. There would also be, to a lesser extent, a sizable number of false negatives.

THE ETIOLOGY OF MPA

Several investigators have attmpted to clarify the role of genetic and traumatic factors involved in the morphogenesis of MPA. Rapoport and her associates in their studies of hyperactive boys found that 19 of 76 mothers reported obstetrical complications that included bleeding, toxemia, premature birth, and Cesarean section. Ten of those 19 mothers had children who have 5 or more MPA. However, a score of more than 5 anomalies was also associated with a history of childhood behavior disorders in the fathers of these children. It was noted, however, that only 3 boys from the sample of 76 presented with a history of both paternal hyperactivity and obstetrical complications. These findings prompted Rapoport and her colleagues to suggest that MPA may be associated with a genetic disorder that can be phenocopied by traumatic obstetrical events (Rapoport et al., 1974).

Investigating MPA in normal, neurotic, learning-disabled, and autistic children, Steg and Rapoport (1975) and Rapoport et al. (1977) found that obstetrical complications including bleeding, hypertension, toxemia, and birth difficulties tended to be higher in learning-disordered children and normal control children with an anomaly score of 5 or more, but that this relationship did not hold true for the other pathological groups.

Firestone and Prabhu (1983) found more pre- and perinatal complications in hyperactive children than in a combined group of psychoneurotics and controls ($\bar{X}$s of 2.95 and 1.95, respectively). There were no differences in the incidence of such complications among the siblings of these groups. The hyperactive probands also evidenced more obstetrical difficulties than their brothers and sisters ($\bar{X}$s of 2.95, 1.67, and 1.33), but only the difference between the hyperactives and their sisters was significant.

Firestone et al. (1978) compared the frequency of MPA in hyperactive, idiopathic retarded, and normal control children as well as their parents and siblings. The hyperactive and retarded children had more MPA than the normal controls. This was also true for the families of the

pathological groups compared with the families of controls. The within-group analyses revealed no differences in MPA.

Firestone and Prabhu (1983) also found that hyperactive children and their families had more MPA than a combined psychoneurotic and normal-control group of children and their families. Once again, within families the number of MPA was constant. Furthermore, the results indicated that MPA were significantly correlated with teachers' ratings of hyperactivity and with Peabody Picture Vocabulary IQs. Interestingly, chi-square analyses revealed that the combination of high MPA and a high number of pregnancy and birth complications significantly increased the probability of a child being diagnosed as hyperactive. Any combination of low MPA and/or low obstetrical complications was not predictive of hyperactivity.

Several studies have indicated that children with a history of numerous obstetrical complications or older mothers have increased MPA. This has been found with normal (Quinn et al., 1977), learning-disabled (Steg & Rapoport, 1975), and autistic children (Links, 1980), supporting the hypothesis that insults to the fetus during pregnancy may increase MPA. In addition, it appears that the average number of MPA within families is quite similar, supporting genetic transmission. Also, the combination of numerous MPA and a history of pre- and perinatal complications has shown to increase the probability of children developing hyperactivity (Firestone & Prabhu, 1983; Rapoport et al., 1974, 1977). Together these findings support the suggestion that MPA may be genetically determined or that they can be phenocopied by traumatic obstetrical events, and the MPA may be indicative of an aberrant central nervous system development. Nevertheless, the support for either of these theories is not strong. It is the trauma during the first trimester of pregnancy that should have the greatest impact on MPA, but four studies addressing the question have not found this (Campbell et al., 1978; Firestone & Prabhu, 1983; Links et al., 1980; Quinn et al., 1977), even though total pre- and perinatal scores do correlate with MPA.

The genetic hypothesis, although not refuted, would benefit from further exploration. It would be buttressed by data indicating that specific MPA run in families, not only the weighted scores. In addition, twin studies would allow for the relative weights accorded to genetic and fetal environment factors.

CONCLUSIONS

It is quite clear that MPA occur in a large portion of the population and fall along a continum. It has also been aptly demonstrated that the

average number of MPA are significantly higher in several groups of children exhibiting deviant behavior, compared with normal controls. However, the topography of the deviant behavior of the groups of children with high MPA cannot as yet be predicted. For example, retarded, autistic, hyperactive, and learning-disabled children have more MPA than do groups of normal children, but it appears that individual MPA features (e.g., hyperteliorism or low-set ears) do not distinguish between pathological and normal groups. To date, the statement that there is no association between the type or frequency of MPA and various disorders may be premature since there are few data comparing MPA in large numbers of children with varying diagnostic labels. Clinically, therefore, MPA demonstrate little utility. Even though MPA can distinguish groups of pathological children from normals, there would be many false positives and false negatives if one were to separate children into normal and abnormal groups based solely on MPA.

There may be several reasons for the rather strong relationship between MPA in groups of pathological children, especially males, but there is still a lack of predictive power for individuals. It may be that our knowledge concerning the specific behaviors or characteristics that result in any one child being diagnosed as deviant is incomplete, resulting in unreliable diagnoses in individual cases. Another problem is that the measurement of anomalies has been shown to vary considerably across locations. More universally accepted and reliable measures need to be established before large-scale studies are undertaken. The understanding of the significance of individual MPA is still far from complete, and conceivably the MPA being studied are the wrong ones or they are correlated with yet another biological feature that has not yet been recognized. There is also some suggestion that there are additional MPA that require identification (Durfee, 1974; Lerer, 1977).

It may well be that the search for any one "marker" for deviant behavior is futile. Several long-term investigations such as the Kauai study (Werner & Smith, 1977) have indicated that several factors in conjunction may lead a "high-risk" child to develop deviant behavior and that any one factor alone may not be sufficient. Although several studies have tried to relate pregnancy and birth complication with anomalies and behavior, there is a paucity of data investigating these features in probands, siblings, and parents utilizing systematic procedures and well-validated medical and psychological tools (e.g., objective ratings scale and personality measures).

Finally, a serious deficiency in this research is the lack of study of environmental factors such as familial stability or parenting style and how they combine with MPA to predict difficult children. For example, Nichols and his associates (Nichols, 1976) have shown that prenatal factors may in-

crease the likelihood of MBD from 2 to 5%, but the Kauai study shows that lack of adequate social and emotional systems may increase the likelihood of pathology developing in such at-risk children by 100 to 400% (Werner & Smith, 1977). Thus, it is not inconceivable that MPA, in conjunction with certain "psychonoxious" environments (Bergin, 1971), may predict atypical behavioral or emotional development. However, until such relationships are established, any suggestion of mass screening for MPA as predictors for "high-risk" children is unwarranted.

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