Acta Psychiatr Scand 1989:79:103–107

Key words: congenital anomaly; longitudinal study; minor physical anomaly; violence; recidivism; criminal behavior.

# Minor physical anomalies and recidivistic adult violent criminal behavior

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ABSTRACT - Minor physical anomalies (MPA) result from disruptions of gestation, and may be used as signs of central nervous system defects in development. Utilizing a Danish birth cohort, we tested the hypothesis that MPA predict adolescent and adult recidivistic violent criminal behavior. The number of MPA was measured at 11 to 13 years of age and police records of criminal behavior were ascertained at 20 to 22 years of age. Recidivistic violent offenders evidenced an elevated level of MPA compared with subjects with one violent offense or subjects with no violent offenses.

*Received August 14, 1988; accepted for publication September 3, 1988*

A very small proportion of the criminal population is responsible for a disproportionate number of crimes – especially violent crimes (1, 2). If the causes of recidivistic violent offending in this small group of men were understood, it would have implications for prevention.

One theoretical explanation of violent behavior emphasizes impaired neurological functioning (3–11). Several studies have noted an elevated level of central nervous system (CNS) deficits in violent offenders (3–5, 11). This neurological impairment may be a consequence of violent behavior or may be part of the cause.

Aggressive behavior in early childhood is a very good predictor of later delinquency (12). This suggests that a part of the cause of delinquency and criminal violent offending may lie in early life events. In view of the neurological findings cited and the early childhood origins of aggression, it seems plausible that CNS deficits leading to aggressive behavior may be partly caused by prenatal and/or perinatal factors. Evidence has been presented suggesting that disruptions of fetal neural development may increase the predisposition to adult mental illness (13). In this article, we explore the possibility that disturbances in fetal neural development may be associated with later violent behavior.

Disturbances in fetal neural development may be caused by genetic or teratogenic factors that are difficult to ascertain. There is, however, an indirect index of prenatal neural disturbances that is observable by trained individuals: small aberrations in external physical characteristics, known as minor physical anomalies (MPA). As an example of how such MPA occur, let us consider the fetal development of the ears. The ears begin low on the neck of the fetus and slowly drift upwards into their proper position. If a teratogenic event or substance impinges itself on the fetus, the development may be slowed or stopped and the ears' drift upward may terminate prematurely, resulting in low-set ears. Firestone & Prabhu (14), Rapoport et al. (15), and

Waldrop & Halverson (16) have noted that MPA are strongly associated with disorders of pregnancy (for example, rubella during pregnancy or bleeding during the first trimester).

The same agents that cause the formation of externally visible MPA may also alter critical CNS development. One study found that in schizophrenic subjects, the higher the number of MPA, the greater the neuropsychological impairment (17). In Long-Evans rats, teratogens administered pre- and postnatally resulted in both external and CNS anomalies (18). Many MPA suggest several teratogenic events and perhaps repeated interference with neurological development.

A number of studies have found that children with many MPA evidence high levels of hyperactivity, a condition usually ascribed to minimal brain damage (19, 20). In one study, MPA explained almost half the variance in hyperactivity at age 3 (21). Researchers in the field of hyperactivity have hypothesized that the factors operating during pregnancy probably influence both the formation of MPA and CNS abnormalities that produce a predisposition for impulsive, fast-moving behavior (22, 23). This same impulsiveness seems to operate to increase the risk that hyperactive boys will develop into serious, delinquent offenders (24).

The research suggests that MPA reflect disturbances in fetal neurological development. Perhaps the resulting brain anomalies are reflected in hyperactivity, impaired impulse control, or other functional disorders that increase the likelihood of violent criminal behavior. If an increase in violent behavior is partly caused by CNS deficits, it seems reasonable to expect that it will be part of a pattern of poorly controlled behavior, rather than manifested as an isolated instance. We hypothesize, therefore, that higher levels of MPA will be related to elevated levels of recidivistic violent behavior.

## Material and methods

### Subjects

The subjects in this study were drawn from a Danish perinatal study examining all 9125 children born between September 1, 1959, and December 31, 1961, at Rigshospitalet in Copenhagen (25, 26). Because of the extensive perinatal and one year data, 265 members of this cohort were selected to participate in a prospective study of children at high risk for delinquency. These 265 children consisted of 4 groups: children with schizophrenic mothers or fathers ($n=72$); children with character-disordered mothers or psychopathic fathers ($n=72$); and controls, children of parents without a psychiatric history ($n=72$). These 3 groups were matched for (a) sex of ill parent; (b) sex of child; (c) race; (d) multiple birth status (e.g., twins); (e) pregnancy number; (f) social class; (g) mother's age; (h) mother's height; and (i) father's age. The fourth group ($n=49$) were special controls for the children of schizophrenic parents; they were matched, individual for individual, for events in the pregnancy and delivery.

### Minor physical anomalies

As many as 87 MPA have been studied. Most research, however, focuses on a list of 18 MPA: head circumference beyond normal range; more than one hair whorl; fine electrostatically charged hair; epicanthus; hypertelorism; malformed ears; low-set ears; asymmetrical ears; soft pliable ears; no ear lobes; high steepled palate; furrowed tongue; smooth tongue with rough spots; curved fingers; single palmar crease; wide gap between the first and second toes; partial syndactyly of toes; and third toe longer than the second (16).

Waldrop et al. (21) found that for any individual the number of MPA remains stable over time. For this study, MPA were assessed at age 11 to 13 by an experienced pediatric neurologist (NM). The presence of the 18 MPA listed above was assessed for each subject. In view of the high correlation between a simple frequency count of MPA and a weighted score (20), we used a frequency count in this study. Because frequency counts require less qualitative judgement (16), they are likely to be more reliable than weighted counts (19).

The mean MPA count for all 265 subjects was 3.4 (SD = 1.84, range = 0–9). Subjects with 4 or more MPA are considered high in MPA; those

Table 1
Number of arrests for violent offenses as a function of minor physical anomalies

| | | Number of minor physical anomalies | | |
|---|---|---|---|---|
| | | Low | High | Total |
| Number of arrrests for violent offenses | 0 | 164 | 82 | 246 |
| | 1 | 5 | 4 | 9 |
| | ≥ 2 | 3 | 7 | 10 |
| Total | | 172 | 93 | 265 |

3.8% of the entire sample (1.7% of the low-MPA group and 7.5% of the high-MPA group) was recidivistically violent. Low MPA = < 3; high MPA = ≥ 4.

Table 2
Number of arrests for property offenses as a function of minor physical anomalies

| | | Number of minor physical anomalies | | |
|---|---|---|---|---|
| | | Low | High | Total |
| Number of arrrests for property offenses | 0 | 145 | 72 | 217 |
| | 1 | 10 | 5 | 15 |
| | ≥ 2 | 9 | 5 | 14 |
| Total | | 164 | 82 | 246 |

Low MPA = < 3; high MPA = ≥ 4.

with 3 or less, low. This split has been used in past research on MPA (15, 19, 27, 28).

## Criminal records

Wolfgang (29) remarked that Danish criminal recordkeeping is "probably the most comprehensive and accurate in the western world". In this study, the National Police Register (NPR) arrest record for the 265 subjects was ascertained in 1981, when they were between 20 and 22 years old. Therefore, all criminal data referred to in this article reflect registered criminality. Violent offenses included: domestic violence, disorderly conduct, illegal possession of a weapon, threats of violence, robbery, armed robbery, assault, and murder. Property offenses included: stealing for temporary use, minor larceny, illegal handling of stolen objects, shoplifting, buying or selling stolen goods, car theft, breaking and entering, and purse snatching.

## Results

According to the NPR criminal records, 19 subjects in this cohort had been arrested for at least one violent crime. Of these, 10 had been recidivistically violent (arrested for 2 or more violent crimes).

Table 1 presents the cross-tabulation of violent offenses and MPA. As can be seen, subjects with no violent offenses or only one violent offense tend to fall into the low MPA group. However, 7 out of 10 subjects with 2 or more violent offenses are characterized by high MPA. The difference in these cells is statistically significant ($\chi^2 = 6.03$, $P < 0.05$).

There are several potentially confounding variables that might explain the pattern observed above. These variables include socioeconomic status (SES) (30), age, sex, and parental psychiatric diagnosis. It is important to examine whether MPA have an effect on violent behavior even when the effects of these other variables are accounted for. In order to do this, we entered these other variables into a logistic regression analysis (BMDP) predicting violence prior to the entry of MPA. The results of this logistic regression demonstrated that the level of MPA significantly predicts violent offenses, even when all of these other variables are controlled for simultaneously ($\chi^2 = 3.94$, $P < 0.05$).

In order to determine whether the relationship between recidivistic violent offenses and MPA was specific to violent offenses, we examined the relationship between MPA and recidivistic property offenses. Property offenders who had also been arrested for violent offenses were excluded from this analysis. Table 2 presents the cross-tabulation of MPA and property offenses. There was no relationship between recidivistic property offenses and number of MPA.

## Discussion

The results of this study tentatively support the hypothesis that CNS malformation caused by the disruption of fetal neural development (indicated by minor physical anomalies) is related to adult recidivistic violent behavior. This relationship does not hold for recidivistic property offenses, and it cannot be explained by SES, age, sex or parent's psychiatric diagnoses.

The following limitations to this study should be considered:

1. The sample was selected to be at high risk for delinquency and is therefore not representative of the general population. There were almost no violent offenders in the control groups, so the results may be specific to high-risk children. Unfortunately, the number of violent offenders in the sample is too small to evaluate this hypothesis.
2. There were only 19 individuals in the entire sample who were registered for violent offenses. This low number is a consequence of not beginning with a sample selected for violent offending, such as a prison sample. This selection method has an advantage in that it minimizes Berkson's fallacy (31).
3. The MPA were assessed by an experienced pediatric neurologist (NM); however, no measure of the reliability of his rating is available. Past research (32) showing very high interrater reliability in frequency counts of MPA ($r = 0.95$) suggests that this is a reliable measure.
4. The number of MPA has been shown to be stable up to the age of 7.5 years (16,21), but it has not been empirically demonstrated to be reliable up to the age of 11 to 13, when our measure was assessed. It is not likely, however, that physical characteristics (such as low-set ears) would undergo significant change between the ages of 7.5 and 11–13 years.

While only 1.7% of the low-MPA group became violent recidivists, 7.5% of the individuals with high MPA did. These results suggest that individuals with more than 3 MPA are 4.4 times as likely as those with 3 or fewer MPA to become violent recidivists. Because the MPA developed during gestation, before any criminal activity commenced, this observation does suggest disturbances in fetal neural development are among the set of factors that might be etiologically related to recidivism in violent offenses. The relationship between MPA and recidivistic violent offenses cannot be accounted for by differences in the demographics or parental psychiatric diagnosis.

The results in Table 1 make it clear, however, that most individuals with high levels of MPA were not recidivistic violent offenders. There are many possible reasons for this finding. First, MPA are but an indirect, external correlate of neurological developmental anomalies. Perhaps some MPA may occur without neurological maldevelopment, and therefore may not be associated with poor impulse control or later recidivistic violent offenses. Second, in Denmark most violent offenses are committed by individuals between 18 and 25 years of age, and the rate of violence remains steady across those years (33). In this study violent criminal offenses were ascertained when the subjects were between 20 and 22 years of age. It is very possible, therefore, that others in the high-MPA group evidenced recidivistic violent offenses after age 22. Finally, while CNS dysfunction may be an etiological factor in violent recidivistic behavior, it is probably most salient in combination with or in interaction with other factors.

It is very likely that on some occasions in their lives almost all of the subjects in this study have been frustrated or angry and have felt the inclination to strike out, at objects or at people. Most subjects, however, were able to inhibit this inclination enough to avoid a police record for violence. Recidivistic violent offenders may suffer neural development disturbances which, in combination with other factors, may compromise their ability to inhibit impulsive, aggressive behavior.

## Acknowledgement

This research was supported by United States Grant 87-IJ-CX-0063 from the National Institute of Justice.

## References

1. Mednick SA. A biosocial theory of the learning of law-abiding behavior. In: Mednick SA, Christiansen KO, eds. Biosocial bases of criminal behavior. New York: Gardner Press, 1977: 1–8.

2. Wolfgang ME, Figlio RM, Sellin T. Delinquency in a birth cohort. Chicago: University of Chicago Press, 1972.
3. Elliot FA. Neurological aspects of antisocial behavior. In: Reid WH, ed. The psychopath. New York: Bruner/Mazel, 1978: 146–189.
4. Fitzhugh KB. Some neurological features of delinquent subjects. Percept Mot Skills 1973:*36*:494.
5. Krynicki VE. Cerebral dysfunction in repetitively violent assaultive adolescents. J Nerv Ment Dis 1978:*166*:59–67.
6. Pontius AA, Ruttiger KF. Frontal-lobe system maturational lag in juvenile delinquents shown in narratives test. Adolescence 1976:*11*:509–518.
7. Spellacy F. Neuropsychological differences between violent and nonviolent adolescents. J Clin Psychol 1977:*33*(4):966–969.
8. Spellacy F. Neuropsychological discrimination between violent and nonviolent men. J Clin Psychol 1978:*34*(1):49–52.
9. Tarter R, Hegedus AM, Winsten NE, Alterman AI. Neuropsychological, personality, and familial characteristics of physically abused delinquents. J Am Acad Child Psychiatry 1984:*23*:668–674.
10. Yeudall LT, Fromm-Auch D. Neuropsychological impairments in various psychopathological populations. In: Gruzelier J, Flor-Henry P, eds. Hemisphere asymmetries of function and psychopathology. New York: Elsevier/North Holland Biomedical Press, 1979.
11. Yeudall LT, Fromm-Auch D, Davies P. Neurological impairment of persistent delinquency. J Nerv Ment Dis 1982:*170*(5):257–265.
12. Olweus D. Stability of aggressive reaction patterns in males: a review. Psychol Bull 1979:*86*:852–875.
13. Mednick SA, Machon RA, Huttunen MO, Bonett D. Adult schizophrenia following prenatal exposure to an influenza epidemic. Arch Gen Psychiatry 1988:*45*:189–192.
14. Firestone P, Prabhu AH. MPAs and obstetrical complications: their relationship to hyperactive, psychoneurotic, and normal children and their families. J Abnorm Child Psychol 1983:*11*(2):207–216.
15. Rapoport JL, Quinn PO, Lamprecht F. Minor physical anomalies and plasma dopamine-g-hydroxylase activity in hyperactive boys. Am J Psychiatry 1975:*131*:387–390.
16. Waldrop MF, Halverson C. Minor physical anomalies and hyperactive behavior in young children. Except Infant 1971:*2*:343–380.
17. Guy JD, Majorski LV, Wallace CJ, Guy MP. The incidence of minor physical anomalies in adult male schizophrenics. Schizophr Bull 1983:*9*(4):571–582.
18. Mankes RF, Rosenblum I, Benitz K, Lefevre R, Abraham R. Teratogenic and reproductive effects of ethanol in Long-Evans rats. J Toxic Environ Health 1982:*10*(2):267–276.
19. Fogel CA, Mednick SA, Michelsen N. Hyperactive behavior and minor physical anomalies. Acta Psychiatr Scand 1985:*75*:551–556.
20. Waldrop MF, Goering JD. Hyperactivity and minor physical anomalies in elementary school children. Am J Orthopsychiatry 1971:*41*:602–607.
21. Waldrop MF, Bell RA, McLaughlin B, Halverson CF. Newborn minor physical anomalies predict short attention span, peer aggression, and impulsivity at age 3. Science 1978:*199*:563–564.
22. Quinn PO, Rapoport JL. Minor physical anomalies and neurological status in hyperactive boys. Pediatrics 1974: *53*:742–747.
23. Waldrop MF, Pedersen FA, Bell RQ. Minor physical anomalies and behavior in preschool children. Child Dev 1968:*39*:391–400.
24. Satterfield JH. Childhood diagnostic and neurophysiological predictors of teenage arrest rates: an eight-year prospective study. In: Mednick SA, Moffitt TE, Stack SA, eds. The causes of crime: new biological approaches. New York: Cambridge University Press, 1987: 146–167.
25. Mednick SA, Mura E, Schulsinger F, Mednick B. Perinatal conditions and infant development in the children of schizophrenic parents. Soc Biol 1971:*18*:5103–5113.
26. Zachau-Christiansen B, Ross EM. Babies: human development during the first year. New York: Riley, 1975.
27. Schexnider V, Bell R, Shebilshke W, Quinn P. Habituation of visual attention in infants with minor physical anomalies. Child Dev 1981:*52*:812–818.
28. von Hilsheimer B, Kurko V. Minor physical anomalies in exceptional children. J Learn Disabil 1979:*12*:38–45.
29. Wolfgang ME. Foreword. In: Mednick SA, Christiansen KO, eds. Biosocial bases of criminal behavior. New York: Gardner Press, 1977: v-vi.
30. Svalastoga K. Prestige, class and mobility. Copenhagen: Gyldendal, 1959.
31. Berkson J. The statistical study of association between smoking and lung cancer. Proc Staff Meetings Mayo Clinic 1955: 319–348.
32. Steg JP, Rapoport JL. Minor physical anomalies in normal, neurotic, learning disabled, and severely disturbed children. J Autism Child Schizophr 1975:*5*:299–307.
33. Guttridge P, Gabrielli WF, Mednick SA, Van Dusen K. Criminal violence in a birth cohort. In: Van Dusen K, Mednick SA, eds. Prospective studies of crime and delinquency. Boston: Kluwer-Nijhoff, 1983: 211–224.

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