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Summary and discussion

The existence of reliable and stable individual differences in performance on psychometric intelligence tests or IQ, is beyond any doubt. Theories on the structure of intelligence give little information about underlying processes that may cause these individual differences (Brody, 1992). The search for biological determinants of intelligence that might explain differences in cognitive functioning has long been the subject of experimental psychology and can be divided into two main approaches. One explores the relation between general intelligence and the speed of execution of elementary cognitive tasks (reaction time tasks), based on ideas that can be traced back to Galton (1883). Another approach is to study individual differences in intellectual abilities in relation to physiological measures.

The existence of biological determinants underlying cognitive functioning gains much evidence from results of behavior genetic research, which has shown that genetic factors account for 50-60% of the phenotypic variance in psychometric intelligence (Bouchard & McGue, 1981). Beyond showing evidence for some genetic determination of phenotypes, quantitative genetic methods can also reveal the nature of the relationship between phenotypes. The correlation between phenotypes may be entirely environmental in origin or may be caused by underlying common genetic influences. Genetic analyses, thus, are of importance when interpreting the correlations between biological and behavioral variables.

Biological determinants of cognitive functioning may be translated into neurophysiological and biochemical processes in the central nervous system. Among a great number of biological variables, peripheral and central nerve conduction have been investigated in the search for biological determinants underlying the individual differences in psychometric intelligence. Reed (1984) hypothesized that, as far as individual differences in IQ are genetically determined, they can be attributed to genetic variability in the structure and amount of 'transmission proteins', which determine information processing rates and, consequently, intelligence. This is called the 'neural efficiency model of intelligence'. The established relationship between intelligence and Reaction Times on elementary cognitive tasks (a behavio-

ral manifestation of this neural efficiency) may provide additional evidence for this model (Vernon, 1993).

In the present study the relative contributions of genetic and environmental factors to the relation between peripheral nerve conduction velocity, reaction times and IQ was investigated. These characteristics were measured longitudinally in a group of 213 twin pairs. At the first test occasion the mean age was 16 years and at the second, 17.5 years.

In this general discussion, first, results are summarized of the phenotypic and genetic factor structures underlying the individual differences in WAIS subtest scores (test occasion II). This kind of genetic analysis on the WAIS subtest scores was only rarely conducted. Also, the results of the genetic association between the WAIS subtests and the Raven are discussed. Next the nature of the RT - IQ relationship and the longitudinal analysis results of RTs on test occasion I and II are discussed. In the next section special attention is drawn to the results on the genetic architecture of peripheral nerve conduction in humans. Finally, the results of the PNCV - IQ relationship of test occasion I and II (chapters 3 and 4) are summarized and a theory is suggested to account for the apparently contradictory findings of test occasion I and II.

Multivariate analysis of the WAIS subtests and the Raven

The factor structure underlying individual differences in WAIS subtest scores was explored by means of multivariate genetic analyses. The associations among the subtests were decomposed into parts due to genetic and environmental influences. Additive genetic and non-shared environmental influences accounted for the phenotypic covariance, whereas shared family background did not. The matrix of genetic correlations suggested a General factor, a Verbal factor, a Performance factor and Specific factors. The matrix of environmental correlations among subtests did not bear any evidence for a separate Verbal and Performance factor, the covariance structure indicated a General factor and Specific factors. Thus the regular subdivision of the WAIS (Wechsler, 1955) into a Verbal and Performance scale reflects genetic rather than environmental covariance between the subtests.

Association among WAIS subtests showed the significance of a General intelligence factor (*g*) which was more dominant in the genetic matrix. The construct *g* refers to the positive manifold, the presence of positive intercorrelations among diverse tests of cognitive abilities. The General genetic factor was predominantly tapped by the verbal subtests. In addition to the genetic General and Group factors,

Specific factors were observed, accounting for the unique characteristics of each subtest, not shared by the other subtests. Multivariate genetic analyses, thus, can help solve the question about the nature of *g* (Jensen, 1993). The establishment of a genetic basis of *g* supports the notion of a biological basis of *g*.

A special feature of chapter 2 was the inclusion of the Raven (Raven, 1958) in the multivariate analyses. The Raven is a nonverbal test of reasoning and does not rely heavily on acquired knowledge. The association with the WAIS and other tests of mental abilities is expected to be the result of loadings on a General factor of intelligence *g*. With a multivariate genetic model it was possible to address the question whether this association is mediated by the General genetic factor or General environmental factor. The covariance of the WAIS subtests and the Raven was solely accounted for by the General genetic factor.

The finding of nonsignificant common environmental (family background) influences of the present study fits the idea that non-shared family environment becomes more important once children start their formal education (Scarr & McCartney, 1983, Thompson, 1993, Boomsma, 1993). Adoption studies indicate that younger children, regardless of genetic relatedness, resemble each other intellectually because of similar rearing environment. Adolescents are able escape the influences of the family by actively selecting their own environment and, therefore, resemblance will exists only if they share genetic background (Scarr & Weinberg, 1983).

Intelligence Quotient in the Dutch population

In the present study, individual differences in WAIS Full-Scale IQ score were shown to be highly heritable. The heritability estimate of 82% is higher than values reported for adult IQ (70%) as measured in reared apart MZ twins (Bouchard *et al.*, 1990) and higher than meta-analyses results of 50% to 60% (Bouchard & McGue, 1981). This difference remains even when the lower bound of the 95% confidence interval of our estimate is considered (75%). The upper bound was estimated to be 87%. The question is how this high heritability estimate in the Dutch population can be explained. When cultural influences on a phenotype are important and relatively homogeneous, environmental variance decreases and heritability will increase (Bouchard *et al.*, 1990). Tambs *et al.* (1984, 1986) argued that the high genetic variance for WAIS IQ observed in Norwegian twins could be a results of the rather egalitarian Norwegian society. Higher genetic variance was particulary observed in younger generations (Sundet *et al.*, 1981), which probably is an effect of developing social homogeneity. This was also demonstra-

ted by the fact that, compared to the US population, a smaller proportion of Norwegians was doing extremely poor on the test. A possible explanation for the high heritability for individual differences in IQ in Norway and The Netherlands could be that genetic and environmental influences upon intelligence are correlated. Genotype-environmental correlation refers to the fact that the environments which individuals experience may not be random, but are caused by their genes and the genes of their parents. It is possible that these higher heritability estimates are caused by the fact that social equality creates optimal educational conditions for all individuals and an equal chance to choose among a variety of secondary schools and educational institutions. Therefore, selection or choice of schools and length of education may be (actively) influenced by genotypes of individuals (rather than e.g social economical status of the parents) and, thus, a balanced, random sample of adolescents is likely to exhibit large genetic influences on IQ. This is called active genotype-environmental correlation.

Despite the higher heritability estimate for IQ in our study, the common finding of higher shared-family environmental influences on IQ in children compared to adolescence and adults, is also observed in the Dutch population (Boomsma & Van Baal, in press). This could be an effect of the rather uniform elementary school education which does not permit a large influence of (genotypically driven) choices of parents or children.

The action of specific mechanisms by which differences in genotypes in human behavior are expressed in phenotypic differences are still unclear. It is hypothesized that genetic influences may work indirectly by determining the effective psychological environment of the developing child (Scarr & McCartney, 1983). This is called passive genotype-environmental correlation or cultural transmission. Another explanation for the high heritability for individual differences in IQ a population could be that genetic differences might affect psychological differences indirectly, by influencing the effective environment of the developing child. An example is when higher than averaged ability children, in addition to the inherited genes, also benefit a more enriched environment of books and education etc. from there parents. Positive genotype-environmental correlations will increase estimates of all the genetic components of variance in phenotypes (Neale & Cardon, 1992). The genotype-environmental correlation declines from infancy to adolescence and the importance of the active genetic-environmental correlation (e.g. selection of schools and experiences) increases and exhibit stronger effects (Scarr & McCartney, 1983).

One way to disentangle genetic and environmental influences is by studying reared apart MZ and DZ twins. In studies of reared apart MZ twins, 70% of the

variance in IQ was found to be associated genetic variation. On other psychological traits reared apart MZ twins were as similar as MZ twins reared together. Correlated placement (if the adoptive homes were selected to be similar in trait-relevant features) did not seem to be the cause of psychological similarity. Social economic status effects on IQ in adoption studies have been found in children but not in adults. Another way to estimate the effects of genotype-environmental correlation (cultural transmission) can be accomplished by including parents' data in the twin model. However, no evidence for possible cultural transmission was found in parents and their children in a series of studies conducted in the Colorado Adoption Project (e.g. DeFries, Plomin, & LaBuda, 1987).

An earlier large Dutch study on hereditary and environmental influences upon intelligence was conducted by Vroon, de Leeuw and Meester (1986). Vroon *et al.* analyzed IQ as measured by the Raven Progressive Matrices and measures of educational and professional level in a sample of 2,847 fathers and sons, both recruited for military service in the Netherlands between 1945 and 1982. A father-son IQ correlation of .34 was reported. From a path analytic model in which educational level and IQ of the father predicted IQ of the son, it was concluded that only 3% of the variation in son's IQ was explained by these variables. Neither hereditary nor SES of the father was found to be responsible for IQ of the child. However, the path analytic model used by Vroon *et al.* failed to correctly specify the genetic relationship between parent and child. The observed correlation of .34 reported by Vroon *et al.* was in agreement with the DZ correlation for the Raven score of the present study (r = .39, for 111 twin pairs). This correlation also was in close agreement with the meta-analyzed father-son weighted correlation of .38, based on 2,843 pairings (Bouchard & McGue, 1981), and, thus, was not specific to the Raven test. In a paper by Rowe and Hay (1988) the method and conclusions of the Vroon *et al.* study were criticised. Data from nuclear families, especially when taking into account only one relation, are not sufficient to disentangle genetic and environmental influences since parents and children share both genes and environment. Fathers' educational level was seen as an environmental variable, but this variable can also contain genetic components. Contemporary behavior genetics uses more sophisticated methods and has gone beyond examining parent-child relationships to infer heritability estimates of behavioral traits.

From large studies conducted in the Netherlands it was concluded that IQ does not seem to predict occupational success. The concept 'Emotional Intelligence' (EQ), which is much more difficult to measure, was introduced to stress the importance of psychological stability and social skills in social economic achievement. IQ was claimed to account for just 20% of success in life. The other

factors important for predicting success in life vary from 'SES' to 'happiness'. Results indicated that high IQ university students were not socially more successful at middle age, compared to less skilled students (.

The relation between IQ and speed-of-information-processing

A theoretical explanation for the relationship between Reaction Times and IQ was given by the neural efficiency model (Jensen, 1982; Vernon 1993), in terms of three characteristics of Short Term Memory or 'working memory' in which basic cognitive operations are carried out. One of these is the limited storage capacity (of approximately seven units of information) for which not much variation is seen between individuals. The second is the rapid decay of information in absence of continuous rehearsal and the third is the trade-off between the amount of stored information units and the amount of information that can be processed at the same time. A fourth property is proposed as a solution for the limiting characteristics: the speed-off-information-processing. Because variation among individuals in the three other properties is limited, it is reasonable to assume that most variation will be observed in speed-of-information-processing. Individuals differ in the speed with which basic cognitive operations can be executed, a property that is related to individual differences in intellectual functioning.

On test occasion I, substantial heritabilities were observed for the Reaction Times (50% to 60%). The mean phenotypic correlation between the RTs and Raven test score was -.24 and was exclusively mediated by genetic influences. On test Occasion II, lower heritabilities were observed for the Reaction Times (32% on average). The mean correlation with the WAIS subtests (-.18), lower compared to test occasion I, was again solely determined by common underlying genetic factors. This RT-IQ relation, therefore, does not seem to be based on correlated environmental factors like training or practice effects on some kinds of test characteristic. The RT-IQ relationship was independent of the nature of the test (verbal or performance). In contrast to what was assumed by Vernon (1989), heritability estimates were observed not to depend on the extent to which they tap either a phenotypic General Speed factor and a General genetic factor. As loadings on the General genetic factor were more or less equal for all RT tasks on test occasion II, RT heritability estimates were more a function of the loadings on the genetic Reaction Time factor. Specific genetic influences were low for all RT tasks on both test occasions.

Longitudinal analysis of Reaction Times

Genetic and environmental influences, which were expressed at test occasion I and also effective at test occasion II, were significant for all RT tasks. There seemed to be a trend of significant new genetic effects specific to test occasion II for the simple RTs, whereas these specific genetic effects were less significant or not significant for the more complex RTs.

On the second visit, subjects were rewarded for each correct response, but were also speeded up by feedback on their response time and were encouraged to be faster than an established target reaction time value. This may have caused different response strategies, for example faster responding with a bigger chance of operating the wrong key. Evidence for this effect was shown by the lower mean reaction times (faster responses) and a higher percentage of errors on all tasks. The 'environmental' pressures on test occasion II (as a consequence of the modified administration procedure) could have increased the unique environmental variance observable in the individual differences in reaction times. This may be an explanation of the significant decrease in heritabilities for the RTs (except Simple RT) on test occasion II.

The relation between IQ and peripheral nerve conduction velocity

Test Occasion I

No significant correlation between PNCV and Raven test score was observed at test occasion I (mean age, 16 years). Heritability estimates for Raven and PNCV were 65% and 76%. Reed (1993), observed increased brain nerve conduction and PNCV in mice as a result of environmental enrichment and physical exercise. He suggested that physical exercise level might increase PNCV and therefore should be taken into account when studying the relationship between IQ and PNCV. Physical activity scores from questionnaire data on sports participation did not correlate with PNCV in our sample.

The experimental conditions of accessing PNCV were in close agreement with the Vernon and Mori (1992) studies, which observed substantial correlations between PNCV and IQ. Temperature, the main confounder of PNCV, was experimentally controlled for and supramaximal nerve stimulation was applied. Supramaximal stimulation ensures stimulation of all (slow and fast) fibres in the nerve bundle (chapter 3). The other PNCV - IQ studies which failed to replicate the results of Vernon & Mori (Reed & Jensen, 1991 and Barrett *et al.*, 1990) had also administered the Raven. An IQ test that more resembled the MAB (employed

in the Vernon & Mori studies) was suggested to replicate the positive correlation between PNCV and IQ.

Test Occasion II

A low but significant correlation was observed between WAIS Full-Scale IQ score and PNCV (r = .16). Heritability estimates for WAIS IQ and PNCV were 81% and 66%, respectively. The Raven score of test occasion I also showed a low but significant correlation with the PNCV of test occasion II. This correlation was almost as high as the correlation between the WAIS and PNCV of test occasion II, suggesting that the lack of correlation between IQ and PNCV of the first measurement was not due to the use of the Raven.

PNCV heritability estimates on both test occasion were high but the test-retest correlation between PNCVs of test occasion I and II turned out to be very low, although no changes were made in experimental procedures. Lack of PNCV stability caused by statistical artifacts as non-binormality were ruled out (chapter 4). The question, then, was what other factors could have caused this observed instability of the PNCV measure. One possible factor causing this instability, could have been an unreliable PNCV acquisition procedure. However, the twin correlation patterns of both occasions suggest otherwise. Measurement errors and technical pitfalls (causing unreliable acquisition) would have been evenly distributed among all subjects and all groups. The high MZ correlation (on both test occasions) suggests that the lack of test-retest correlation was not caused by measurement error and the relatively low DZ correlations suggest that the high MZ correlations are not a result of correlated measurement errors. Additional evidence for the reliability of the PNCV acquisition procedure is provided by the high split-half correlation for the 3 latencies (obtained from two nerve action potentials) for both test occasion I and II.

It was speculated that the lack of correlation between PNCV at age 16 and age 18 could be explained by ongoing maturation in this age interval. We therefore investigated what is known about maturation in human peripheral nerves in this age interval. Also, from the longitudinal PNCV measures difference scores between occasion I and occasion II were computed for all subjects and similarity of this difference score between MZ and DZ twins was explored.

Maturation of peripheral nerve conduction in humans

There are only a few clinical maturation studies on PNCV reporting data of sensory and mixed nerve conduction velocity in infants and children. In a study by Gamstorp and Shelburne (1965), median and ulnar sensory PNCV (digit-wrist) was studied in 72 normal children, aged 2 weeks to 15.5 years. The nerve conduction velocity of young infants was roughly 50% of the adult value. A rapid increase during the first few years was followed by small changes through later childhood and adolescence. The changes were interpreted as a functional expression of increasing thickness and myelination of the nerve fibres.

In a second study by Cruz Martinez *et al.* (1978), normal values of sensory PNCV in distal and proximal segments of median, ulnar and sural nerves were determined in 76 normal subjects from newborns to children of 14 years of age. From infancy to age 14, sensory (digit-wrist) and mixed PNCV (wrist-elbow) can be considered an index of maturation of the sensory peripheral nerve fibres. At about 3 months of age the values were below 50% of adult values. PNCV develops in logarithmic function with age: PNCV increases about a 100% during the first year of life and it continues to increase with a progressively slower rate. In young adults normal sensory values are reached earlier in lower than in upper limbs, and also earlier in proximal (wrist-elbow) than in distal (digit-wrist) segments in the upper limbs. This is an indication that myelination starts proximally. Maximum sensory PNCV from the proximal segment is higher than in the distal segment. This difference increases with maturation of the peripheral nerve fibres. There are indications that maturation in sensory fibres is slower than in motor fibres.

Oh (1993) summarized the results of PNCV maturation studies as follows: The changes in PNCV are most profound in the first few years of life. PNCV in all fibres is about 50% of the normal adult values in the full-term newborn baby, reaching about 75% of the adult value at 1 year of age, and about 100% at 4-6 years of age. Peripheral nerve conduction is suggested to increase in a logarithmic function and is likely due to: (1) the increase in the number of large fibres between birth and 8 years of age, when the number is the same as in adult nerves, and (2) the complete myelination of nerve fibres by 5 years of age. No further increase in PNCV between age 6 and 16 was noted (Oh, 1993). In adults PNCV decrease with age, possibly caused by an increased loss of large fibres or segmental demyelination after age twenty. For mixed nerve conduction, the rates of decrease are 4.0 m/sec in the median nerve per decade.

The main disadvantages of these PNCV maturation studies are the use of rather small samples with a broad age range to establish normative values. It is possible that subtle changes in PNCV were left undetected by the design of these

studies. On the basis of our own results, we propose that PNCV might still undergo maturation processes between age 16 and 18.

The maturation hypothesis of the relation between PNCV and IQ

To investigate the hypothesis that differences in maturation may have caused the low test-retest correlations, difference scores in PNCV were computed (measure assessed on occasion II - measure assessed on occasion I). PNCV did not increase or decrease systematically, there were positive and negative difference scores. Positive difference scores can be an indication that PNCV is still increasing and has not yet reached the highest value, whereas negative scores might reflect a phase beyond this point in which PNCV is decreasing.

Strikingly, the MZ correlations for this difference score were very high as opposed to the relative low DZ scores. These results indicated that the causes for the changes in PNCV in the age interval 16 - 17.5 years were more alike for MZ twin pairs compared to DZ pairs. These causes could be ongoing maturation processes controlled by genetic influences. A reasonable assumption would be that, with respect to PNCV development, individual growth curves show the same morphology but slightly different slopes, indicating individual differences in speed of maturation. Figure 8.1 shows a hypothetical PNCV growth curve for 3 individuals. In the next step we may assume that these maturation processes (biologically determined) might be more alike in MZ twins compared to DZ twins. Genetic analyses of the difference scores revealed this measure to be highly heritable (86%). This means that PNCV value at age 18 is better predicted by the rise or fall in PNCV of the co-twin than the own score at age 16.

It is possible that the variance in IQ determined by PNCV is only fully observable when PNCV has reached its peak value as a consequence of new genetic effects. This additional genetic variance in PNCV could be responsible for the additional genetic variance in IQ and is supported by the increase of IQ heritability in adults (Bouchard, 1993).

For intelligence there seems to be an increase in heritability from infancy to childhood and a decrease in common environmental influences during adolescence (Thompson, 1993). As children become older and enter schools and other social institutions, the effects of common (parental) environment decrease and the effects of genetic factors may, thus, increase. Longitudinal twin and family studies may reveal the age-dependent changes in the relative contributions of genetic and environmental effects to individual differences in IQ. A longitudinal study from adolescence to early adulthood was conducted in a sample of Swedish male twins

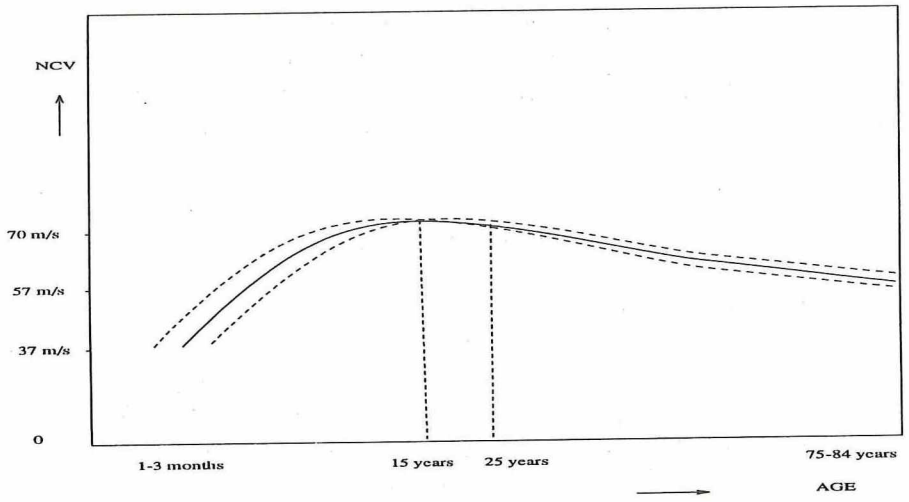

Figure 8.1
Hypothetical PNCV growth curves for three individuals. All three show the same morphology, but slightly different slopes, indicating individual differences in speed of PNCV maturation.

between 12 to 18 years of age (Fishbein, 1979). Verbal ability and inductive reasoning skills were examined. No longitudinal analyses were conducted, but correlations for verbal ability were reported to increase slightly from .70 to .78 for MZ twins and decreased from .60 to .50 for DZ twins. For inductive reasoning tests, correlations increased for both MZ and DZ twins, from .59 to .78 and from .46 to .56, respectively. Heritability, thus, for males seems to increase from around 20 to 40%.

Another way to derive heritability changes in IQ is by cross-sectional studies. In a meta-analysis on age and development of IQ based on 103 studies published between 1967 and 1985, a tendency of greater decrease in DZ compared to MZ twin correlations and, consequently, an increase in heritabilities is noticed (McCartney *et al.*, 1990). Analysis of adoption data from the Colorado Adoption Project indicates that genetic influence on IQ increases steadily between infancy and middle childhood (Fulker *et al.*, 1988). Although little adult twin IQ data are available, MZ correlations appear to peak at about 16-20 years (Bouchard, 1993).

Thus, the increase in heritability of IQ in adolescence and adults, may provide some evidence for additional genetic variance in IQ as a result of NCV maturation that probably results from increasing thickness and myelination of the nerve fibres not only centrally, but observable in the periphery as well. There are theories

suggesting that higher intelligence might be associated with higher central conduction speed. Miller (1994) proposed that this association could by related to central myelination. The observed positive correlation between brain size (as measured by Magnetic Resonance Imaging) and IQ (e.g. Wicket *et al.*, 1994, Willerman *et al.*, 1991, Schultz *et al.*, 1993) also supports the myelin hypothesis. Thicker myelin sheathed nerves are faster, more accurate in signal processing and might therefore be associated with faster information processing and higher scores on IQ tests (Miller, 1994).

This myelin hypothesis is supported by recent studies on age-related changes in cognitive functioning. The long-standing believe that cognitive changes in the normal healthy elderly were caused by widespread neuron death is challenged by recent findings based on more sophisticated neural imaging techniques (Wickelgren, 1996). The higher densities observed in young brain tissue compared to old tissue (interpreted as cell loss) could have been an effect from commonly used methods for preparing brain tissue for microscopy study in which young cortical tissue simply shrank more than old tissue. New studies indicate no age-related differences in cell number and some imaging studies indicate that age-related brain shrinkage is almost exclusively due to loss of white matter, probably caused by shrinkage of myelin.

Recent studies in monkeys suggest that a breakdown of myelin may account for the cognitive changes in aging. No age-related differences in the volume of the animals' grey matter were observed. It was theorized that myelin breakdown slows neural conduction along an axon and may influence problem solving speed (Peters, 1996). This theory is in congruence with the observation that PNCV decreases with age, as a possible cause of an increased loss of large fibres and segmental demyelination after age twenty.

Final remarks

Traits that are influenced by multiple factors (like variation in intelligence) are unlikely to show large correlations with a single causal factor. Consistently replicable small significant correlations between psychometric and biological variables may be of theoretical importance. This is especially true when these correlations are shown to be genetically determined. Genetic analyses by means of twin studies, thus, may play an essential role in the theoretical interpretation of the relationships of biological and behavioral variables (Jensen & Sinha, 1993). The small genetically mediated correlation in our study between PNCV and IQ

contributes to theoretical knowledge in terms of the 'neural efficiency' model. The fact that this relationship was not observed at the first test occasion was explained by ongoing maturation processes in PNCV between age 16 and 18. So, the correlation between PNCV and IQ, as initially reported by Vernon and Mori (1992) has been replicated and this correlation is indeed genetically mediated.

In accordance with other studies (Ho, Baker & Decker, 1988; Baker, Vernon & Ho, 1991) the moderate correlations between Reaction Times and IQ, were entirely mediated by genetic influences. There was no relationship between PNCV and Reaction Times, which both seem to be independent correlates of human intelligence. This observation, to some extent, agrees with the suggestion of Vernon and Mori (1992). They found both RT and PNCV contributing to the prediction of IQ in two studies (multiple R's = .53 and .57) and hypothesized that the relationship between IQ and speed-of-information-processing was, at least in part, a function of each one's correlation with PNCV. However, the partial RT - IQ correlations remained significant after PNCV was controlled for. Vernon and Mori concluded that intelligence and speed-of-information-processing may be thought of as two types of related cognitive abilities (e.g. verbal and quantitative abilities), which are both correlated with PNCV, but their relationship is not attributable to PNCV.

The next question that rises is of what interest (theoretically or practically) the observed correlation between PNCV and IQ can be? Multivariate genetic analyses of cognitive abilities suggest a substantial overlap of genetic influences and imply a common set of genes associated with these traits (Plomin, Owen & McGuffin, 1994). Molecular techniques are now applied to identify multiple loci affecting normal variation in psychometric abilities. The first allelic association studies to identify quantitative trait loci (QTL) associated with intelligence have been conducted (Plomin *et al.*, 1994, Plomin *et al.*, 1995) using markers related to genes which are likely relevant to neural functioning. Two of the three initially identified markers of the Plomin *et al.*, 1994 study (alcohol dehydrogenase 5 and the beta polypeptide of nerve growth factor) yielded results in the same direction but were not significant in the replication sample (Plomin *et al.*, 1995). This was possibly due to limited statistical power. The third marker (EST00083) of mitochondrial origin was also significant in the replication sample and the technique was described in Skuder *et al.* (1995).

Recently, the specific hypothesis of the hierarchical genetic structure of cognitive abilities (as suggested by multivariate quantitative genetic research) was supported by means of QTL analyses. In cognitive abilities the genetic effects are largely general, but some genetic effects are specific to certain abilities. Four

markers were identified which showed similar predicting effects across the ability scales (Verbal, Spatial, Perceptual Speed and Memory), suggesting that they are related to general cognitive ability (*g*). These associations became negligible when the effects of *g* (WISC-R IQ score) were removed. Three other markers continued to be significantly associated with specific cognitive ability scales after the effects of *g* were removed (Petrill *et al.*, in press). QTL research concerning cognitive disabilities did yield promising findings in the context of reading disabilities. A QTL on chromosome 6 was identified to be associated with dyslexia (Cardon *et al.*, 1994). Recently, this result was replicated in a study by Grigorenko *et al.* (1996) in which word segmentation (a basic process involved in reading) was linked to markers on chromosome 6, whereas single word reading (presumably a higher-order process) was linked to markers on chromosome 15. These findings may in the future contribute to early diagnosis and intervention.

QTL studies with regard to PNCV have not yet been carried out. A well established genetic correlation between PNCV and IQ could, from a speculative point of view, make PNCV an additional (relatively easy obtainable) measure to (physiologically) select extreme phenotypes and investigate linkage to markers associated with genes which are thought to be important for myelination and neural functioning. In this way a more particular set of genes (markers) may be identified which can increase the power to detect QTLs for cognitive abilities.