Development and Behavioral Genetics

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1. Unifactorial Methods

Unifactorial genetic analyses tend to focus on single gene effects whereas multifactorial (Quantitative Genetic) approaches focus on multiple gene effects.

Inbred strain analyses in animals (e.g. mice) compare an inbred strain with outbred strains that carry a known single gene mutation or two inbred strains are compared that differ in one allelic variation at a location on a chromosome.

There are two types of problems with these studies for determining specific effects of genes

1) Not all aspects of an organism’s environment are easily controlled, for example, intrauterine, birth, and maternal effects.

2) Genes can have pleiotropic effects, which means that mutations can be highly disruptive in behavior(s), but their specific causal role is not necessarily inferable. (see figure below)

PKU (phenylketonuria): Is a condition that leads to severe intellectual impairment of human functioning. The condition occurs when there is a single recessive gene present in both sister chromosomes that play a role in the liver’s production of the enzyme phenylalanine hydroxylase. Without this enzyme, the amino acid phenylalanine is not converted to tyrosine. The consequence of building up phenylalanine is structural malformations of the brain, resulting in children that are mentally retarded and irritable. However, by modifying diet, to greatly lower the intake of phenylalanine, this condition can be largely avoided.

The important point is that while the presence of this defective gene on both chromosomes can have catastrophic effects on mental function, it does not cause this condition. It is one factor that is necessary but not sufficient for this condition. If other factors are present, such as low phenylalanine diet, then the condition is eliminated or largely so.
Knockout and Transgenic Organisms

A knockout organism, e.g., a mouse is created by disabling a specific gene (a brief description of the procedure Video part 1, Video part 2). This type of research is very promising, but still has serious issues:

1. Typically produced in inbred animals.

2. As we would predict, genes expressed earlier in development, when knocked out, are often lethal.

3. Knocking out a gene is somewhat analogous to making a part of a machine non-functional. It is relatively easy to see how the machine breaks down, but not as easy to identify the functional role in a complex regulatory system.

This type of genetic engineering has often been more focused on creating organisms that have some technological use such as disease resistant plants or the production of an important compound such as insulin.

A. An Example of a Natural Knockout: Vasopressin Deficiency and the Brattleboro Rat

The Brattleboro, di/di, rat strain was discovered in the laboratory of Henry A. Schroeder in Brattleboro, VT, USA in 1964 who cloned the normal rat vasopressin (AVP) gene and then identified the mutation responsible for AVP deficiency in these rats (Valtin H. The discovery of the Brattleboro rat, recommended nomenclature, and the question of proper controls. Ann New York Acad Sci, 1982; 394: 1-9.).

It turned out to be a single base deletion at nucleotide 1552 in a conserved region of exon B resulting in a frame shift mutation that produces an altered amino acid sequence (Schmale H, Richter I. Single base deletion in the vasopressin gene is the cause of diabetes insipidus in Brattleboro rats. Nature, 1984; 308: 705-709).

Immunohistochemical and in situ hybridization studies showed that the vasopressin gene is transcribed and translated in di/di rats, but with an impaired response to physiological stimulation (McCabe JT, Morrell Jl, Ivell R, Schmale H, Richter D, Pfafp DW. Brattleboro rat hypothalamic

Brattleboro rats develop diabetes insipidus at least by the time of weaning with indications of AVP deficiency even earlier in development (Dlouha H, Nrecek J, Sicha J. Postnatal development and diabetes insipidus in Brattleboro rats. Annuls of the New York Academy of Science, 1982; 394: 10-20).


**ASD may be associated with vasopressin resistance due to hyporesponsiveness of AVP V1a receptors** (Boso, et al, 2007).

Knockout mice lacking V1a receptors have **deficits in social recognition resembling aspects of ASD** (Bielsky IF, Hu SB, Ren X, Terwilliger EF, Young LJ. The V1a vasopressin receptor is necessary and sufficient for normal social recognition: a gene replacement study. Neuron, 2005; 47:503-513).

AVP deficient Brattleboro rats (di/di), **exhibit abnormalities in emotional reactivity** (Williams AR, Carey RJ, Miller M. Altered emotionality of the vasopressin-deficient Brattleboro rat. Peptides, 1985; 6(Suppl. 1): 69-76),

**memory and attention** (Jentsch JD, Arguello PA, Anzivino LA. Null mutation of the arginine-vasopressin gene in rats slows attentional engagement and facilitates response accuracy in a lateralized reaction time task. Neuropsychopharmacology, 2003; 28: 1597-605.),

Indeed, AVP was first implicated in behavioral deficits in studies that found avoidance learning and memory deficits in di/di rats (Williams AR, Carey RJ, Miller M. Altered emotionality of the vasopressin-deficient Brattleboro rat. Peptides, 1985; 6(Suppl. 1): 69-76).

In humans, disorders such as ASD can have very early onset that may be detectable in children under two years old.

Little, however, is known about the affects of AVP deficiency on neonatal behavioral and brain development.


By day 10, di/di pups have an impaired adrenocorticotropin stress response to separation from their mother (Zelena D, Domokos A, Barna I, Mergl Z, Haller J, Makara GB. Control of the hypothalamo-pituitary-adrenal axis in the neonatal period: adrenocorticotropic and corticosterone stress responses dissociate in vasopressin-deficient Brattleboro rats. Endocrinology, 2008 Feb 14).

These results indicate that AVP deficiency is behaviorally detectable early in life and that these early developmental effects are related to abnormalities previously detected in adult di/di rats.

Does AVP deficiency in di/di pups have behaviorally detectable effects on locomotor and social development?
Figure 1. Locomotor neural development in rats has four stages. During the fetal stage, motor neurons become excitable, the CPG begins to function, and the first projections from the brainstem develop. During the immature stage, infant rats crawl, maturation of posture in locomotion proceeds cephalocaudally, the corticospinal tract begins to develop around day 7 after birth, and by day 10 (the end of the immature stage) coupled activity in groups can be detected. During the transitory period, the eyes and ears open and walking begins. By the beginning of the adult period, adult locomotion begins.

Metrics:

For individual trials, the following metrics were computed from the tip-of-snout and base-of-tail measurements for 5 second time intervals:

1) activity: change in position or orientation between each time interval;

2) Δ distance: if active, the distance moved over a time interval as measured from the tip-of-the snout;

3) Δ orientation: if active, the change in body orientation over a time interval as measured in degrees;

4) wall contact: body contact with a wall of the arena at each time interval; (5) corners: the number of corners visited by a pup during an experimental session;

6) inner cells: the arena was divided into an array of 6 × 9 cells of equal size with inner cells defined as the number of cells not adjacent to a wall that the tip-of-the-snout entered;
(7) outer cells: the total number of cells adjacent to a wall that the tip-of-the-snout entered;

(8) total cells: the proportion of the total number of cells that the tip-of-the-snout entered. For group trials, there was one additional metric,

(9) subgroups: the number of different contact groups that formed out of eight pups at each time interval, which ranged from 8 (no pups in contact with each other) to 1 (all pups in direct or indirect contact).

**Results**

**Table 1.** One-way ANOVAs for phenotypic and sex effects for each age group and across all metrics (see Table 2 for sex effects).

<table>
<thead>
<tr>
<th>Metric</th>
<th>Context</th>
<th>Day 7</th>
<th>Day 10</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>F</td>
<td>df</td>
</tr>
<tr>
<td>Activity</td>
<td>single</td>
<td>5.1</td>
<td>2, 138</td>
</tr>
<tr>
<td>ΔDistance</td>
<td>single</td>
<td>2.61</td>
<td>2, 138</td>
</tr>
<tr>
<td>ΔOrientation</td>
<td>single</td>
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<tr>
<td>Wall Contact</td>
<td>single</td>
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<td>single</td>
<td>2.01</td>
<td>2, 138</td>
</tr>
<tr>
<td>Inner Cells</td>
<td>single</td>
<td>4.68</td>
<td>2, 138</td>
</tr>
<tr>
<td>Outer Cells</td>
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<td>0.09</td>
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<td>group</td>
<td>1.32</td>
<td>2, 67</td>
</tr>
<tr>
<td>Activity</td>
<td>group</td>
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<td>2, 67</td>
</tr>
<tr>
<td>ΔDistance</td>
<td>group</td>
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<td>2, 67</td>
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<tr>
<td>Wall Contact</td>
<td>group</td>
<td>2.34</td>
<td>2, 67</td>
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</table>

* Significant under Benjamini-Hochberg correction procedure for \( \alpha = 0.05 \)
### Table 2. One-way ANOVAs for sex effects for each age group and across all metrics (see Table 1 for genotypic effects).

<table>
<thead>
<tr>
<th>Metric</th>
<th>Context</th>
<th>Day 7</th>
<th>Day 10</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Single/Group</td>
<td>F</td>
<td>df</td>
</tr>
<tr>
<td>Activity</td>
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<td>△Distance</td>
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<td>.867</td>
<td>1, 138</td>
</tr>
<tr>
<td>△Orientation</td>
<td>single</td>
<td>.096</td>
<td>1, 138</td>
</tr>
<tr>
<td>Wall Contact</td>
<td>single</td>
<td>.075</td>
<td>1, 138</td>
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<tr>
<td>Total Cells</td>
<td>single</td>
<td>1.44</td>
<td>1, 138</td>
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<tr>
<td>Inner Cells</td>
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<tr>
<td>Outer Cells</td>
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<td>0.001</td>
<td>1, 138</td>
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### Table 3. For day 7, planned t-test (2-tailed) for all pairwise combinations of genotypes for statistically significant metrics in Table 1.

<table>
<thead>
<tr>
<th>Metric</th>
<th>+/+ × +/-di</th>
<th>+/+ × di/di</th>
<th>+/-di × di/di</th>
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<tbody>
<tr>
<td></td>
<td>t</td>
<td>df</td>
<td>p</td>
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<tr>
<td>Activity</td>
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<td>.025</td>
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<tr>
<td>△Orientation</td>
<td>1.16</td>
<td>110</td>
<td>.25</td>
</tr>
<tr>
<td>Wall Contact</td>
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<td>110</td>
<td>.05</td>
</tr>
<tr>
<td>Inner Cells</td>
<td>2.92</td>
<td>110</td>
<td>.005*</td>
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</tbody>
</table>

* Significant under Benjamini-Hochberg correction procedure for α = 0.05
Table 4. For day 10, planned $t$-test (2-tailed) for all pairwise combinations of genotypes for statistically significant metrics in Table 1.

<table>
<thead>
<tr>
<th>Metric</th>
<th>Context</th>
<th>$+/+ \times +/di$</th>
<th>$+/+ \times di/di$</th>
<th>$+/di \times di/di$</th>
<th>$t$</th>
<th>$df$</th>
<th>$p$</th>
<th>$ES$</th>
<th>$t$</th>
<th>$df$</th>
<th>$p$</th>
<th>$ES$</th>
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<td>.42</td>
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<td>81</td>
<td>.002*</td>
<td>.77</td>
<td>4.91</td>
<td>78</td>
<td>.004*</td>
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<tr>
<td>$\Delta$Distance</td>
<td>single</td>
<td>1.71</td>
<td>107</td>
<td>.09</td>
<td>.33</td>
<td>4.45</td>
<td>81</td>
<td>.0001*</td>
<td>1.04</td>
<td>3.52</td>
<td>78</td>
<td>.0008*</td>
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<tr>
<td>$\Delta$Orientation</td>
<td>single</td>
<td>2.37</td>
<td>107</td>
<td>.02*</td>
<td>.45</td>
<td>2.38</td>
<td>81</td>
<td>.02*</td>
<td>.56</td>
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<td>78</td>
<td>.0002*</td>
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<tr>
<td>Wall</td>
<td>single</td>
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<td>107</td>
<td>.02*</td>
<td>.49</td>
<td>3.93</td>
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<td>.0002*</td>
<td>.92</td>
<td>1.36</td>
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<tr>
<td>Total Cells</td>
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<td>.80</td>
<td>.05</td>
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<td>81</td>
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<td>2.84</td>
<td>78</td>
<td>.006*</td>
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<tr>
<td>Outer Cells</td>
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<td>107</td>
<td>.56</td>
<td>.11</td>
<td>3.93</td>
<td>81</td>
<td>.0002*</td>
<td>.92</td>
<td>3.26</td>
<td>78</td>
<td>.002*</td>
</tr>
<tr>
<td>Subgroups</td>
<td>group</td>
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<td>.58</td>
<td>.17</td>
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<td>31</td>
<td>.002*</td>
<td>1.23</td>
<td>3.48</td>
<td>36</td>
<td>.002*</td>
</tr>
<tr>
<td>Activity</td>
<td>group</td>
<td>0.29</td>
<td>41</td>
<td>.77</td>
<td>.09</td>
<td>5.27</td>
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<td>.0001*</td>
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<td>.0001*</td>
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<tr>
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<td>41</td>
<td>.44</td>
<td>.24</td>
<td>7.99</td>
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<td>.0001*</td>
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<td>.0001*</td>
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<tr>
<td>$\Delta$Orientation</td>
<td>group</td>
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<td>41</td>
<td>.90</td>
<td>.04</td>
<td>6.76</td>
<td>31</td>
<td>.0001*</td>
<td>2.38</td>
<td>7.48</td>
<td>36</td>
<td>.0001*</td>
</tr>
</tbody>
</table>

* Significant under Benjamini-Hochberg correction procedure for $\alpha = 0.05$

Figure 2. Individual metrics for days 7 (grey bars) and 10 (white bars). Mean numbers of cells and 95% confidence intervals are illustrated for activity, $\Delta$ distance, $\Delta$ orientation, and wall contact.
Figure 3. Trajectory plots of 145 tip-of-snout points for a 12-minute session. The two plots on the left are from two di/di, 10-day-old pups that visited four corners. The two on the right are typical one-corner visits by +/+ (top right) and +/di pups (bottom right).
Figure 4. Group metrics for days 7 (grey bars) and 10 (white bars). Mean numbers of cells and 95% confidence intervals are illustrated for subgroups, activity, Δ distance, Δ orientation, and wall contact.
Figure 5. Three-dimensional bar-graph plots of the frequency distribution of activity and inactivity for the three genotypes (+/+, +/di, and +/di) as a function of the number of active and inactive pups each pup contacted for each time interval at day 10. Up to four active and inactive pups were observed to be in contact with each pup. The x-axis is the number of active pups each pup contacted and the y-axis is the number of inactive pups. For example, the frequency activity (or inactivity) when pups were not in contact with any other pups is at the origin of the x-y plane (back corner of each figure). The frequency of activity for +/+ and +/di pups was 65% and 66% respectively, and as illustrated in these figures, activity and inactivity were distributed almost identically for both genotypes. The distributions of activity and inactivity, however, differ markedly for di/di pups, which were active 91.5% of the time. Note that the inactivity panel on the right is not a strict inverse of the activity panel on the right even though the sum of the two distributions is 1.0 for each genotype. The reason is that activity and inactivity are not distributed as a strict inverse function of the number of active and inactive pups.
contacted.

**Figure 6.** Three-dimensional bar-graph plots of the distribution of transition frequency for activity at time $t$ to inactivity at time $t + 1$ (left side for all genotypes) and for inactivity at time $t$ to activity at time $t + 1$ (right side for all genotypes) as a function of the number of active ($n_A$) and inactive ($n_I$) each pup contacted. The distributions of transitions frequencies for both $+/+$ and $+/di$ pups at day 10 were nearly identical illustrating that the more inactive pups each $+/+$ or $+/di$ pup contacted, the more like it was to transition from activity to inactivity and the less likely it was to transition from inactivity to activity. For $di/di$ pups, these transition distributions are again markedly different. This is especially true for transitions from inactivity to activity. $di/di$ pups were much more likely to transition from inactivity to activity when in contact with inactive pups than were either $+/+$ or $+/di$ pups.
Figure 7. Three-dimensional bar-graph plots of the ratios of transition frequencies in Fig. 6 for di/di pups with +/+ and +/di pups. The +/+ and +/di transition frequency distributions for activity to inactivity and inactivity to activity were combined since they were nearly identical. The floating plane indicates ratios of 1.0. The transition frequencies from activity to inactivity for di/di pups were all less than for +/+ and +/di pups as indicated by all ratios less than 1.0, which means that in all contact combinations, di/di pups were less likely to make a transition from activity to inactivity than were +/+ and +/di pups. In contrast, all transitions frequencies from inactivity to activity were greater for di/di pups than for +/+ and +/di pups as indicated by all ratios greater than 1.0, which means that in all contact combinations, di/di pups were more likely to make a transition from inactivity to activity than were +/+ and +/di pups.

Possible Interpretations:

A particularly robust and strong result was the distance moved by 10-day-old di/di pups in both individual and group trials.

Ten-day-old di/di pups moved over twice the distance moved by either +/+ or +/di pups in group trials.

For individual trials, they visited more corners, entered more cells—especially those cells along walls.
Pups often repeatedly circled the arena, a behavior rarely observed in either +/+ and +/di pups.

Informal observations of 10-day-old di/di pups revealed that they were often able to lift their bodies off the surface of the arena and walk or run across the surface.

Walking or running on the surface of the arena was never observed for either day-10 +/+ or +/di pups.

**These results, taken as a whole, suggest that di/di pups have accelerated motor development**, which is consistent with advanced development of some morphological features such as early eye opening in di/di rats and with early studies that administered adrenocorticotropic hormone (ACTH) neonatally to rats resulting in accelerated motor development.

**If this interpretation is correct, di/di rats may be a source of insight into accelerated motor development sometimes seen in early ASD** (Johnson CP. Recognition of Autism Before Age 2 Years. Pediatr Rev, 2008; 29: 86-96).

**Another interesting behavior observed in 10-day-old di/di pups was a large degree of turning.**

For all day-7 and for day-10 +/+ and +/di pups, the average change in orientation was about 30° between 5-second observation intervals, but for day-10 di/di pups, it was 54°.

Informal time-lapse observations of di/di pups in groups confirmed this dramatic difference. Ten-day-old di/di pups appeared to swirl around each other in almost continuous motion for the 12-minute sessions.

This was in stark contrast to +/+ and +/di pups, which gradually coalesced into one or two groups that gradually became less active over the 12-minute sessions.
This continuous change in orientation could indicate the emergence of an early stereotypical behavior analogous to repetitive twirling or hand flapping sometimes in children ASD.

It could also be an indicator of a social deficit, since day-10, di/di pups did not aggregate as well as +/+ and +/-di pups.

Ten-day-old pups normally, aggregate into groups and exhibit synchronized quiescence.

Ten-day-old di/di pups exhibited very little decline in activity over a session and did not exhibit the normal contact-dependent changes in activity observed in pups of this age.

This suggests that the development of coupled activity, an indicator of the emergence of social behavior, is delayed or is a deficit in di/di rats. ---This result is consistent with previous findings that adult di/di rats have deficits in social recognition: a characteristic of ASD.

Issues:

1. There were no sex differences. Autism, however, occurs in males in about a 4:1 ratio. If Vasopressin is involved in autism, it is far from the whole story. Genetically, untangling the mechanisms of autism likely will involve understanding some rather complex genetic networks.

2. Autism is not associated with diabetes insipidus, but if vasopressin is somehow involved, it may be at the receptor level and/or at the gene regulatory level.
2. Multifactorial Methods (Quantitative Genetics)

A. Heritability

Heritability is an often misunderstood concept of quantitative genetics. It is a relative measure that cannot be used between populations for comparison.

Heritability is based on the statistical idea of variance. Recall that for natural selection to operate in a population, there must be variation in phenotypes. If we can measure phenotypes (e.g., the height of corn plants), then we can measure variation using the statistical concept of variance:

\[
V_p = \frac{\sum_{i=1}^{N} (x_i - E(x))^2}{N}
\]

Theoretically, phenotypic variance can be partitioned into a variety of components, and the main empirical problem is how to estimate each component. For example, the simplest partition is

\[
V_P = V_G + V_E
\]

where \(V_G\) is variation due to genetic factors and \(V_E\) is variation due to environmental factors.

Phenotypic variance decompositions can get complex, for example,

\[
V_P = V_A + V_D + V_I + V_E + V_{ExG}
\]

Heritability in the broad sense used in the IQ controversy is:

\[
h^2 = \frac{V_G}{V_P}
\]

If components of variance can be estimated for a population, then \(h^2\) heritability can be used to predict characters in the next generation. So if \(E(x)\) is the mean measure of the character for a population and \(x_{MP} = (x_i + x_j)/2\) is the mid parent value, then the offspring of these parents would be expected to have a mean value of
\[ x_O = E(x) + h^2(x_{MP} - E(x)) \]

For example, if \( E(x) = 100 \), \( x_{MP} = 120 \), \( h^2 = 0.5 \) then the expected value for the offspring is

\[ x_O = 100 + 0.5(120 - 100) = 110 \]

Thus, for example, if IQ is heritable in a population, then the IQ of offspring can be predicted from the IQ of parents.

**But, it is important to keep in mind that the notion of heritability is COMPLETELY relative to a population. It is meaningless as a comparison between populations as we will see later.**

Also, if heritability depends on maternal genes or genes of other individuals in a population, then heritability is much more complex and highly dependent on the genetic of a local population or even ecosystem.

**B. The IQ Controversy**

1. **What are Intelligent Tests?**

The first scale for measuring intelligence was introduced by Binet in Paris for the purpose of testing children in school. His test was based on the concept of mental age introduce by SE Chaillé in 1887.

The basic idea was to first find the average age at which most children could solve a problem, then second if say a 3 year old could solve problems solved by most 4 year olds, then his/her mental age would be 4 and his/her chronological age would be 3.

Putting these two concepts together we get the so-called intelligence quotient:

**IQ = MA/CA x 100**

Where MA is mental age and CA is chronological age.
It was crucial for the Binet IQ scale to determine exactly the average age at which a child is able to solve various problems, because as children age, they become more intelligent, so an IQ scale could be used as a measure of developmental progress.

There are at least 3 major Rules for constructing intelligent tests:
1) Items should not take too long to solve,
2) all items must have single correct answer, and
3) items should test problem solving and not knowledge.

**Tacit Assumptions:**
1) Intelligence can be measured by problems that do not take too long to solve.
2) Intelligence is “black or white.” There are not different perspectives one can take to a problem. Are real world problems “black and white”?
3) Problem solving can be separated from knowledge. This is assumption is important for those looking for race or group differences, because if problem solving is not separable from knowledge, then one must show no knowledge differences between groups.

![Distribution of IQ giving rough indication of the meaning of scores](image)

**Typical Group of Test Items**
1. Are Intelligence Tests Valid?

The validity of a test refers to whether the test really measures what it is intended to measure.

Thus, in assessing the validity of IQ tests we must ask what they are intended to measure.

Binet’s original intent was to be able to compare the development of intelligence in children. Notice that this is a very restricted notion, it is a comparison of a child with the average ability of children of different age classes. By itself, one can only say whether a given child does more or less well on a given set of problems than does the average child at a given chronological age class.

To say that such a comparison is relevant to intelligence, the problems contained in such a test must be a predictor of at least something we associate with intelligence, such as job performance.

1) It was assumed that this comparative measure could be straightforwardly transformed into an absolute measure of intelligence in adults. But, notice that as an absolute measure, all that IQ tests say is that there are differences in people’s ability to solve problems on IQ tests--a very
useless measure by itself. The problem of validity is ever more important.

2) IQ is heritable. This is a radical assumption, because it makes the huge leap that not only do IQ tests measure something we call intelligence but also it measures intelligence as an adaptive characteristic of humans.

There is not doubt that intelligence is an adaptive trait in humans and perhaps our most important adaptation. Thus, the validity of IQ tests used in this sense would have to show that it is measuring a trait or capacity of humans that is adaptive and is a major component of fitness.

There are several problems with establishing the validity of IQ tests in an adaptive sense.

1) The types of problems found on IQ tests do not match the kinds of problems faced for adaptive intelligence.

2) IQ tests have not been show to be good predictors of job performance let alone human capabilities that may be related to fitness.

3) Knowledge cannot be separated from problem solving so at best IQ tests can only be used to make comparisons among individuals that have exactly the same background knowledge. An notice this is even problematic.

Consider the sequence

3 8 12 15 17 Complete

I can think of several “correct” answers to this

3 8 12 15 17 18
3 8 12 15 17 18 18
3 8 12 15 17 cannot be completed, because the sequence is infinite if we assume negative numbers.
3 8 12 15 17 18 18 17...
4) The problem that knowledge cannot be separated from problem solving is a fundamental reason why IQ tests cannot be used to compare different groups, because they necessarily are confounded by difference in knowledge background.

2. **Is IQ Heritable?**

Some people such as J. Philippe Rushton claims that there are heritable differences in IQ among groups or races of people. In this video, he presents his statistical arguments. If one doesn’t have a basic understanding statistics and quantitative genetics, it is difficult to identify the nonsensical and racist use and understanding of statistics by people such as Rushton.

We have already seen that IQ tests are highly suspect as a measure of anything else but the ability to solve a specific set of problems relative to one’s background knowledge. This in itself should raise doubts about any claims that IQ is heritable. So, since such claims have been repeatedly made, one possibility is that such claims are based on error or misunderstanding of what heritability is.

Recall that the basic equation for partitioning phenotypic variance is

\[ V_P = V_G + V_E \]

\[ h^2 = \frac{V_G}{V_P} \]

There are two important things to notice about heritability

1) The \( V_E \) term for IQ tests will vary between groups if there are differences in background knowledge. If we assume, for example, that \( V_G \) is constant, then differences in \( V_E \) among groups will result in different heritabilities, when in fact genetically two groups have exactly the same \( V_G \). Cultural bias is one instance of this problem. Thus, different groups are not comparable with respect to heritability.

2) Point 1 implies that the comparison of heritability between groups or populations is meaningless. This can be illustrated by two examples raised by Richard Lewontin:
Take two completely inbred lines of corn. There is no genetic variation within lines because corn plants within lines are identical. Now if we plant them in different pots (one seed to a pot with ordinary potting soil) and a few weeks after they germinate, we will find variation in the height of the plants. The variation will be entirely environmental, thus

\[ V_P = 0 + V_E \]

\[ h^2 = \frac{V_G}{V_P} = 0 \]

But, there will be **genetic differences** between the two lines. Trying to estimate heritability across to populations is clearly meaningless in this case.

Now, suppose we take seeds from a sack containing seed of an open-pollinated variety containing lots of genetic variation. We grow each group of seeds in vermiculite watered with a nutrient called Knop’s solution, which is used by plant physiologists for controlled growth experiments. Suppose however that for one group we remove some of the nutrients from the Knop’s solution. The results we get would be this:

1) **Within** Knops solution conditions, \( V_E \) would be very close to 0 and thus \( h^2 = \frac{V_G}{V_P} = 1 \) within groups.

2) **Between** groups there would be systematic differences in height, but it would be entirely environmental. Thus, again to say that corn height is heritable across groups is meaningless. This is exactly analogous to the problem of different background knowledge across groups or populations.

**To sum things up about the IQ controversy**

1) It has never been shown that IQ tests measure real world success or adaptive intelligence.

2) Problem-solving ability is always confounded with background knowledge, so for comparisons where background knowledge is not controlled, it is impossible to separate problem-solving ability from background knowledge--this is essentially the problem of group or cultural bias.
3) The heritability of IQ is meaningless comparison across groups because it is only mathematically defined relative to a group and not between arbitrary groups.