

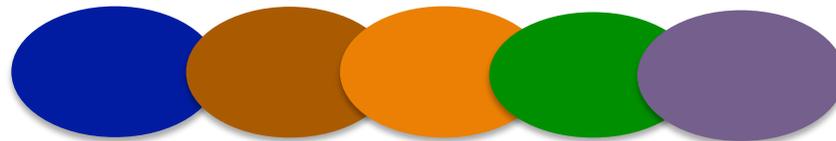
# Learnus



## Genetics and Education

Michael Thomas

The Inaugural Annual Learnus Lecture  
2015



# Genetics and education

The interest

The future is mechanism

What's surprising

Labelling

The science

Screening

What use is that to teachers?

Personalised learning

What is changeable?

What do we want from education?

# The interest

## Genetics and education

The future is mechanism

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MENU **TIME** Health

REFUGEE CRISIS IN EUROPE 2015 UNHCR

LATEST MAGAZINE VIDEOS

HEALTH RESEARCH

### In 2025, Everyone Will Get DNA Mapped At Birth

Alice Park @aliceparkny | June 30, 2014

Scientists have scoured trends in research grants, patents and more to come up with these 10 innovations that will be reality in 10 years (or so they think)

OWN TODAY... >

# Genetics and education

**What's surprising**

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# The Elephant in the Classroom

Helping Children Learn and Love Maths



$$5 \times 7 =$$

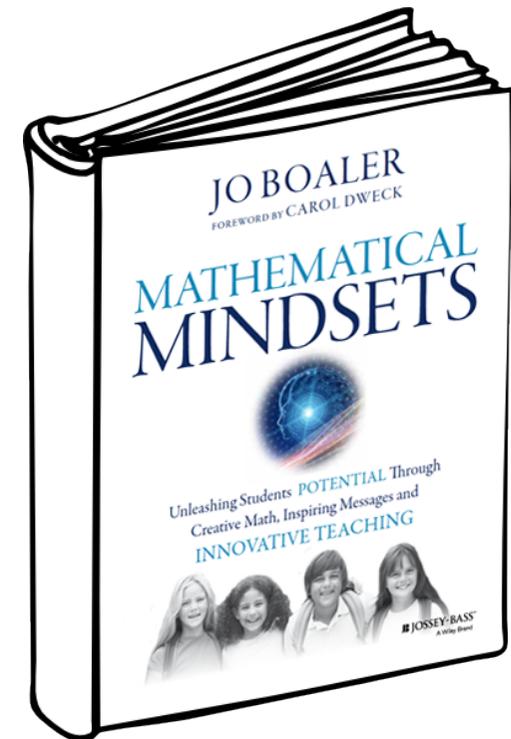
$$r = \sqrt{\frac{18.2}{4}}$$

$$A = 4\pi r^2$$

$$16 \div 4 =$$



JO BOALER



New findings about brain science, mindset and learning.



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The Brain Science that can Transform Math Classes

Anyone Can Learn to High Levels



Ideas of "Giftedness" Hurt Students



Mistakes Grow Your Brain



Speed and Time Pressure Block Working Memory



When You Believe In Yourself Your Brain Operates Differently



Visual Math Improves Math Performance



When You Believe In Your Students They Do Better



Parents' Beliefs about Math Change Their Children's Achievement



Aligning Assessment to Brain Science



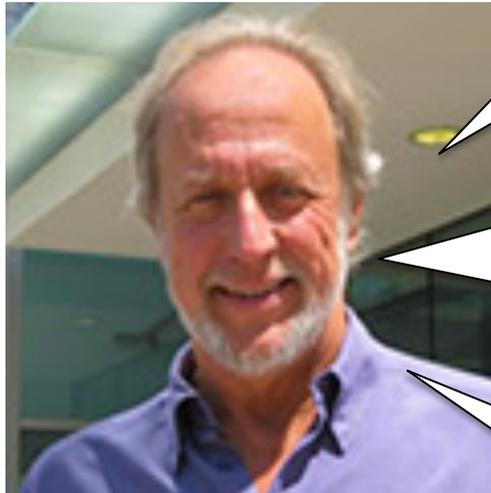
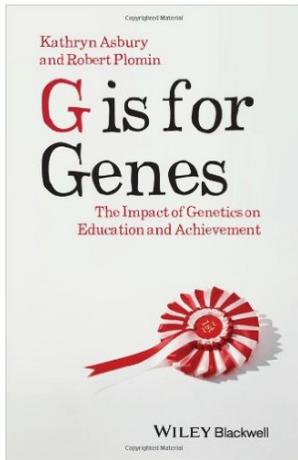
123456789

## Anyone Can I

Many people think that some  
with, but this idea has been  
grow and change within a re



Many people think that some students can work to high levels and some cannot because of the brains they are born with, but this idea has been resoundingly disproved. Study after study has shown the incredible capacity of brains to grow and change within a remarkably short period of time

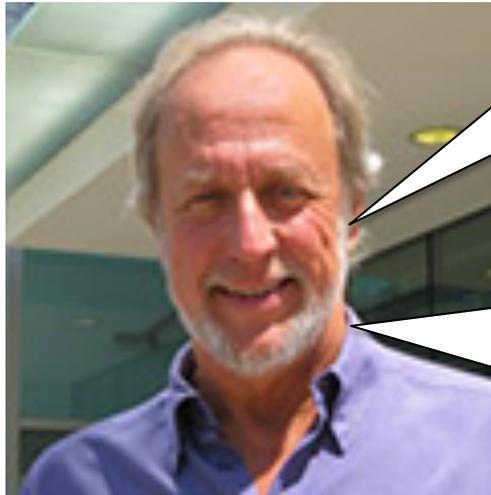
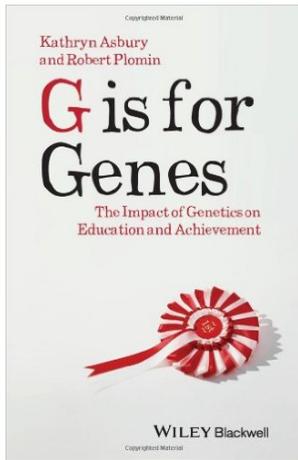


Professor Robert Plomin  
King's College London

Why are children so different in how well they do at school? ... We have assumed in education that this is all environmental

The bottom line is, genetics is incredibly important, it's so much more important than anyone ever thought... The differences between children are substantially due to DNA differences

You know, Michael Gove's Phonics Screening Check for 6-year-olds is one of the most heritable tests around. About 70% heritable



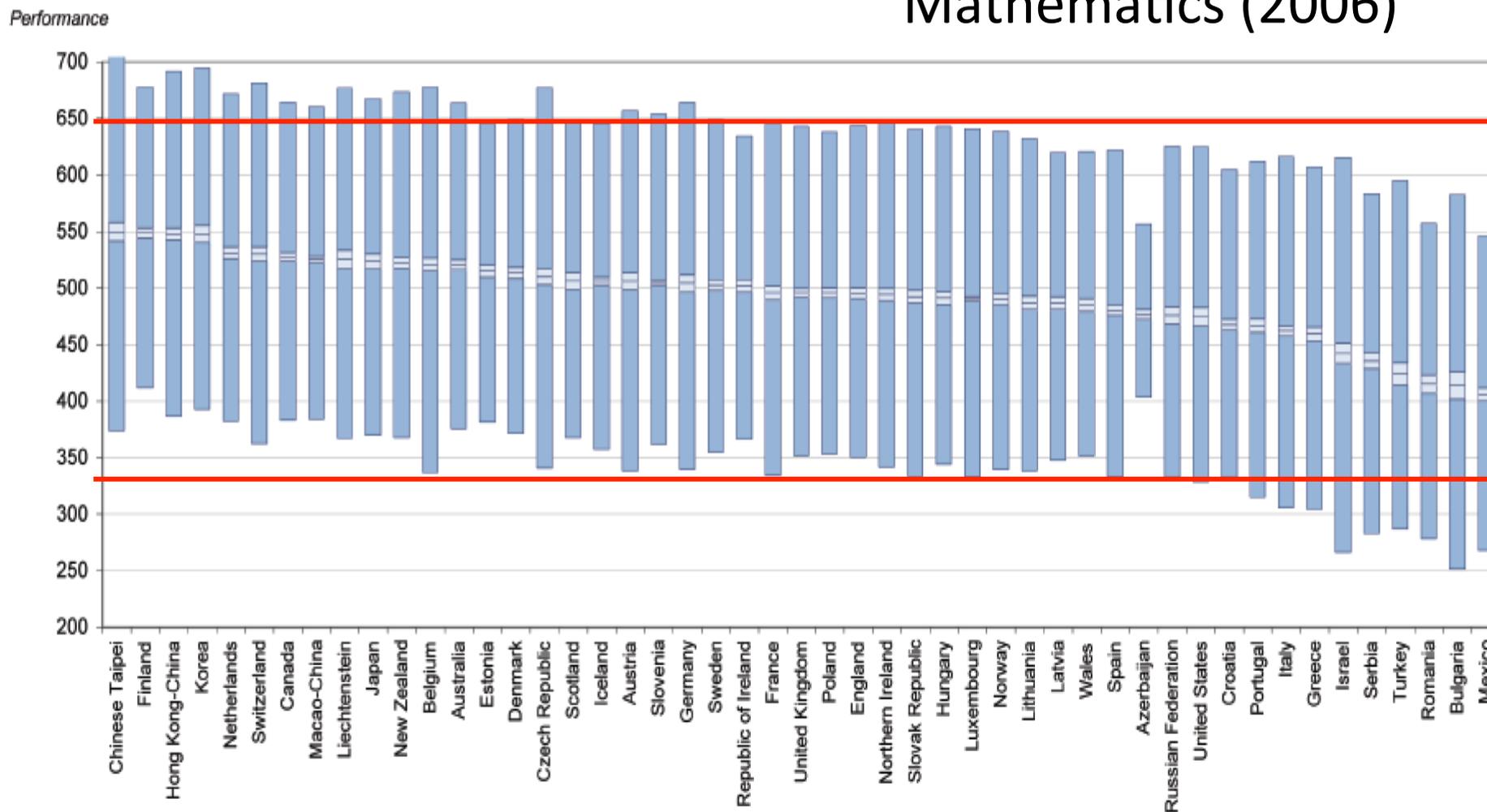
Most differences are not  
due to the environment

So blaming teachers,  
parents, schools for all  
differences between children  
is unwarranted

Professor Robert Plomin  
King's College London

### B.3 Distribution of student performance on the mathematics scale

# PISA results for Mathematics (2006)



Countries are ranked in descending order of mean score.

12 countries with scores below 430 omitted

-  Gradation bars extend from the 5th to the 95th percentiles
-  Mean score on the mathematics scale
-  95% confidence interval around the mean score

# Genetics and education

## The science

The interest

The future is  
mechanism

What's  
surprising

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What use is  
that to  
teachers?

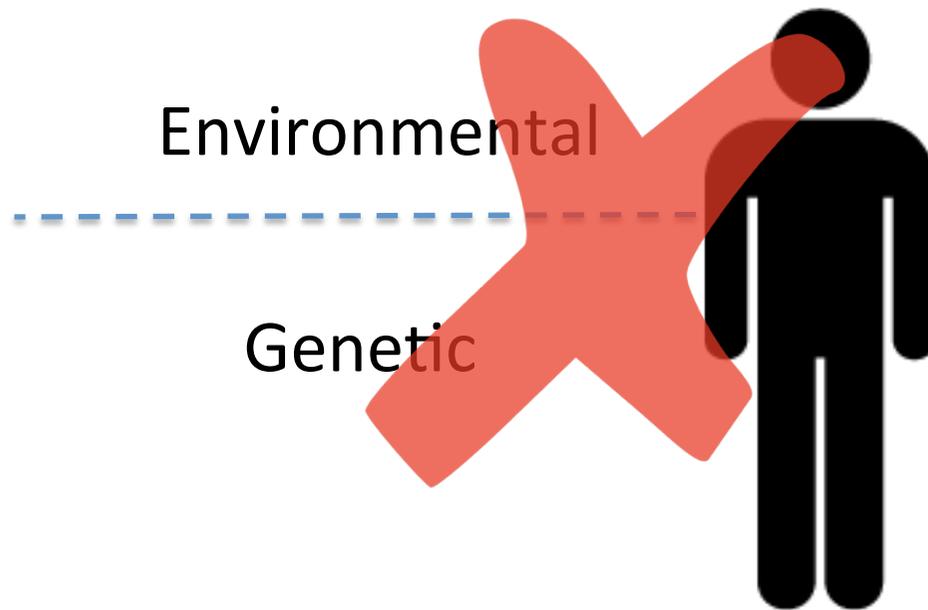
Personalised  
learning

What is  
changeable?

What do we  
want from  
education?

- What is heritability and how do you measure it?

# Heritability is not about individuals



Heritability is about differences between individuals in groups

- Heritability = % of variation in an ability that is explained by the genetic similarity between individuals

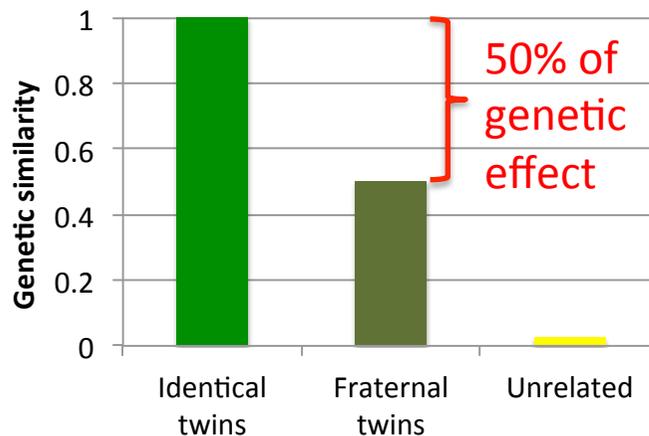
Identical twins	
Twin 1	Twin 2
56	50
34	32
21	25
83	78
..	..

Correlation = 0.9

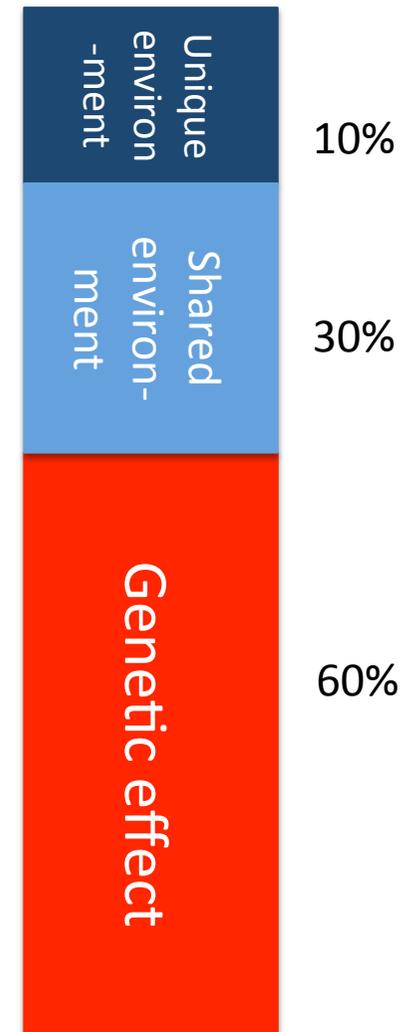
Non-identical twins	
Twin 1	Twin 2
25	40
52	92
35	33
43	38
..	..

Correlation = 0.6

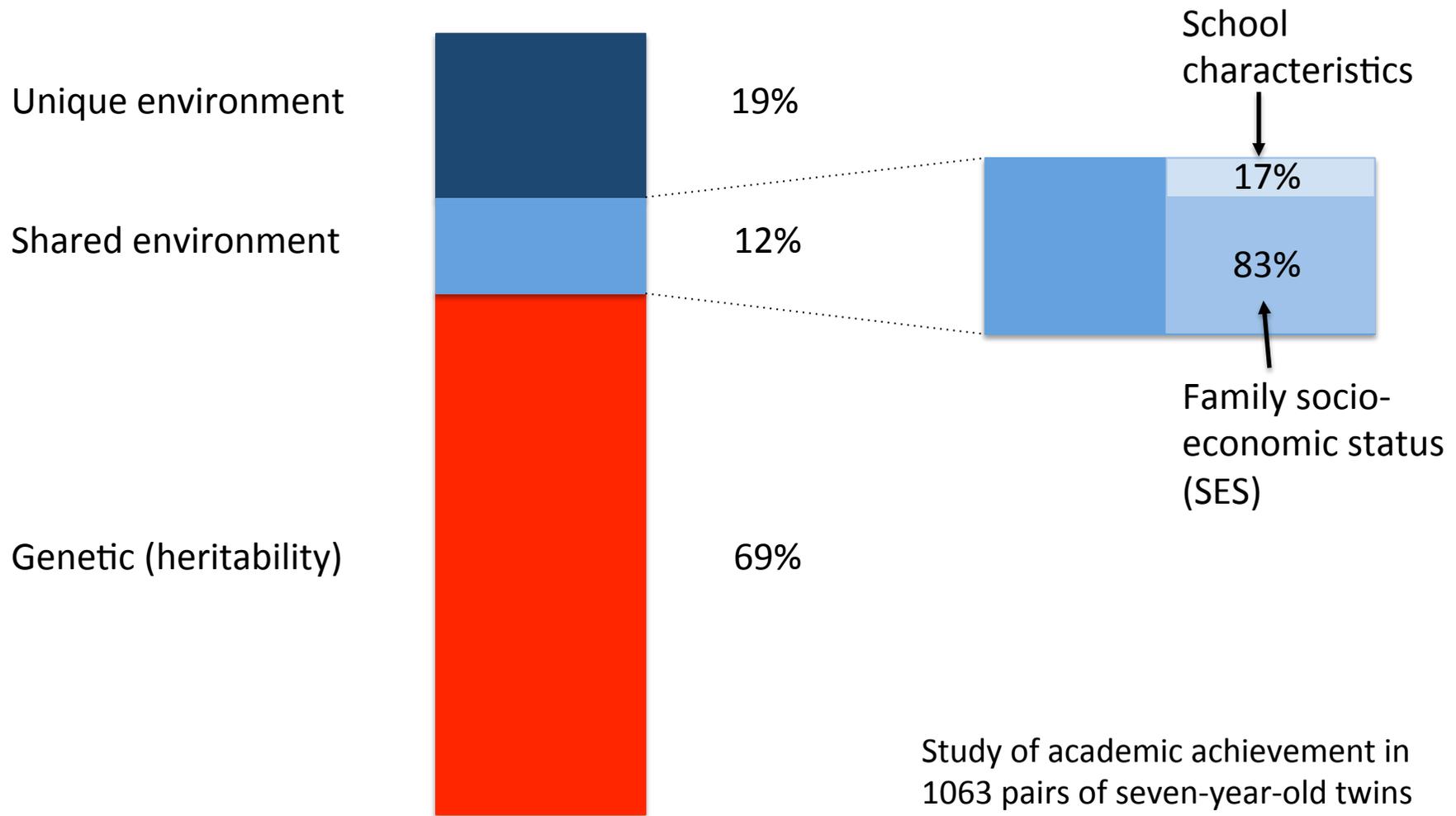
$$1 - 0.9 = 0.1$$



Difference =>  
 $0.9 - 0.6 = 0.3$   
 That's 50% of effect  
 Full effect =>  
 $0.3 \times 2 = 0.6$



School effects are 'shared environment' effects, making children in the same school more similar – how large are they?



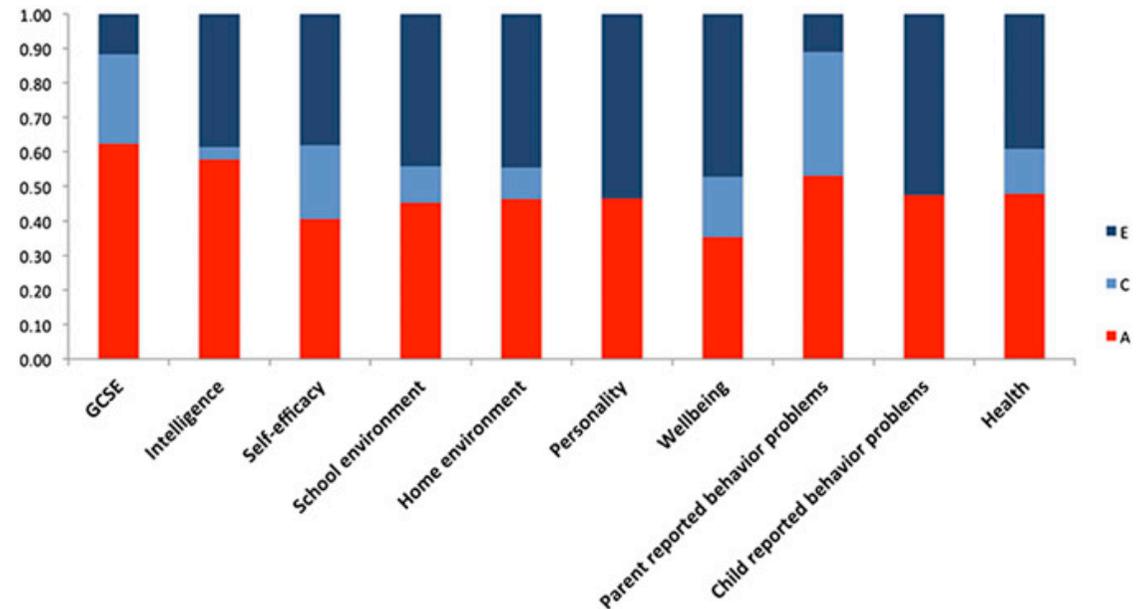
Study of academic achievement in 1063 pairs of seven-year-old twins (Walker, Petrill & Plomin, 2005)

# The high heritability of educational achievement reflects many genetically influenced traits, not just intelligence

Eva Krapohl<sup>a,1</sup>, Kaili Rimfeld<sup>a,1</sup>, Nicholas G. Shakeshaft<sup>a</sup>, Maciej Trzaskowski<sup>a</sup>, Andrew McMillan<sup>a</sup>, Jean-Baptiste Pingault<sup>a,b</sup>, Kathryn Asbury<sup>c</sup>, Nicole Harlaar<sup>d</sup>, Yulia Kovas<sup>a,e,f</sup>, Philip S. Dale<sup>g</sup>, and Robert Plomin<sup>a,2</sup>

Edited by [Name] S. Gazzaniga, University of California, Santa Barbara, CA, and approved September 10, 2014 (received for review May 13, 2014)

High heritability of achievement may also be due to many traits, such as personality, motivation, and psychopathology



**Fig. 1.** Model fitting results for additive genetic (A), shared environment (C), and nonshared environment (E) components of variance for GCSE and nine predictors.

# SCIENTIFIC REPORTS

OPEN

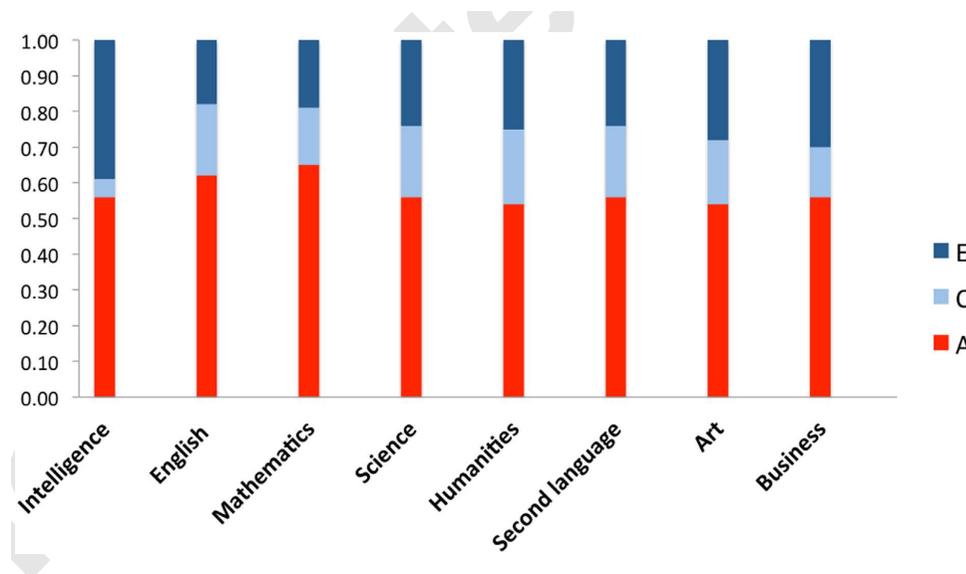
## Pleiotropy across academic subjects at the end of compulsory education

Received: 03 February 2015

Accepted: 03 June 2015

Kaili Rimfeld<sup>1</sup>, Yulia Kovas<sup>1,2,3</sup>, Philip S. Dale<sup>4</sup> & Robert Plomin<sup>1</sup>

Different academic subjects have similar high heritability. It appears to be largely a similar set of genes. And these are not just genes for general intelligence



**Figure 1. Univariate model-fitting results.** A = additive genetic, C = shared environmental, E = non-shared environmental components of variance for GCSE exam grades and intelligence.

OPEN

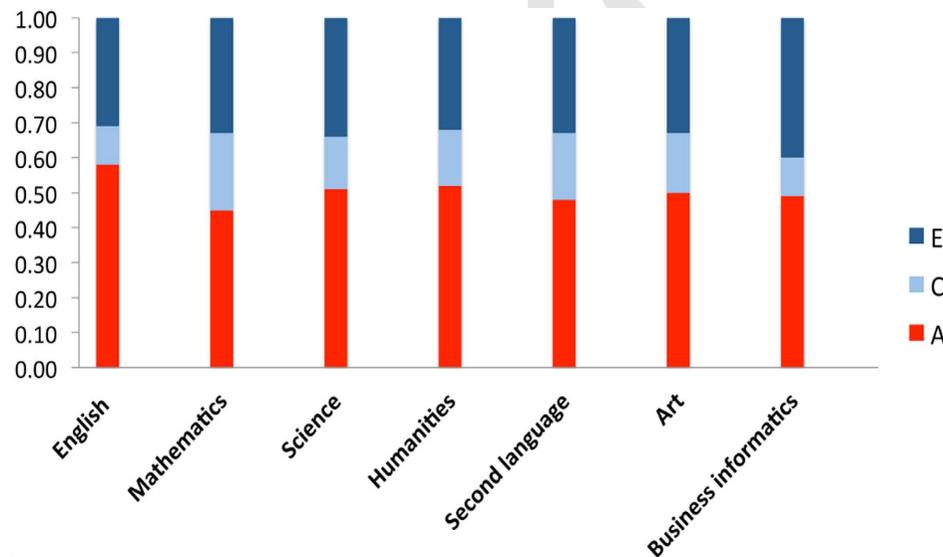
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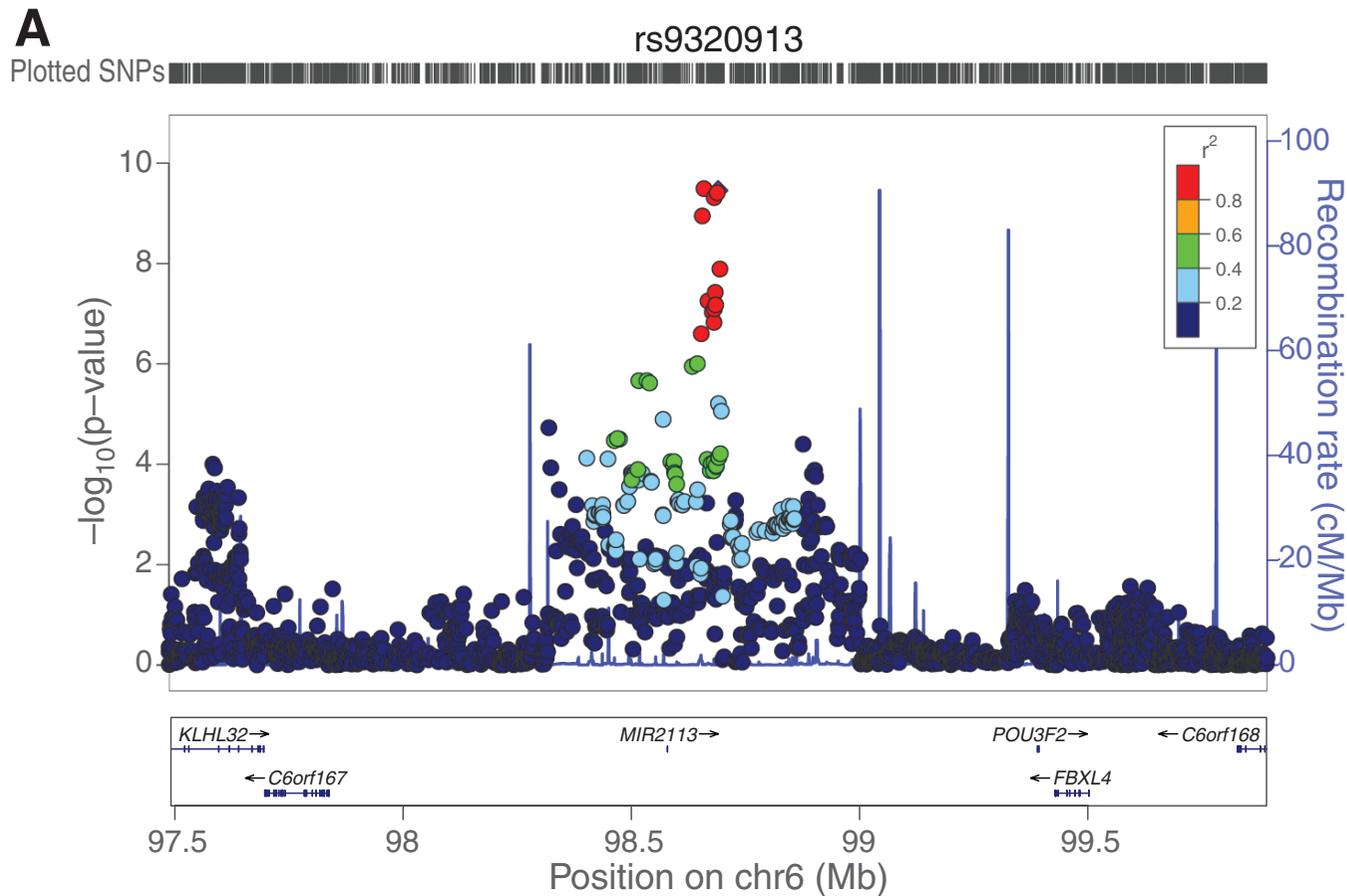


**Figure 2.** Univariate model-fitting results with GCSE exam grades corrected for intelligence. A = additive genetic, C = shared environmental, E = non-shared environmental components of variance.

# Heritability versus DNA

- Heritability is about traits that run in families
- It is a separate question what the *actual genes are*, in terms of DNA variation
- The exact genes for educational abilities have been hard to track down

# GWAS of 126,559 Individuals Identifies Genetic Variants Associated with Educational Attainment



Educational attainment = 40% heritable  
Identified DNA variation explains around 2%

- What would genes influencing education look like if we could properly find them?
- What sort of things would they do?

# GWAS of 126,559 Individuals Identifies Genetic Variants Associated with Educational Attainment

Terms directly related to neuronal or central nervous system function are marked with an asterisk \*

All authors with their affiliations appear at the end of this paper.

<i>GBX2</i>	*	1	nerve development	$1.4 \times 10^{-9}$	N
<i>GBX2</i>	*	1	neural tube development	$2.0 \times 10^{-9}$	Y
<i>GBX2</i>		1	regionalization	$2.5 \times 10^{-9}$	Y
<i>GBX2</i>	*	1	neuron fate commitment	$2.6 \times 10^{-9}$	N
<i>GBX2</i>		1	positive regulation of neuron differentiation	$4.6 \times 10^{-9}$	N
<i>GBX2</i>		1	pattern specification process	$5.0 \times 10^{-9}$	Y
<i>GBX2</i>	*	1	cranial nerve development	$6.0 \times 10^{-9}$	N
<i>GBX2</i>	*	1	neuron fate specification	$9.5 \times 10^{-9}$	N
<i>GBX2</i>		1	morphogenesis of embryonic epithelium	$2.3 \times 10^{-8}$	N
<i>GBX2</i>	*	1	negative regulation of glial cell differentiation	$2.5 \times 10^{-8}$	N
<i>GBX2</i>		1	cochlea morphogenesis	$4.6 \times 10^{-8}$	N
<i>GBX2</i>	*	1	parasympathetic nervous system development	$5.3 \times 10^{-8}$	N
<i>GBX2</i>	*	1	neuromuscular process	$5.8 \times 10^{-8}$	N
<i>GBX2</i>		1	cell fate specification	$5.9 \times 10^{-8}$	N
<i>GBX2</i>		5	Basal cell carcinoma	$9.3 \times 10^{-6}$	N
<i>GBX2</i>		2	Notch binding	$1.5 \times 10^{-5}$	N
<i>GBX2</i>		5	Renal cell carcinoma	$5.2 \times 10^{-5}$	N
<i>GBX2</i>		5	Notch signaling pathway	$8.2 \times 10^{-5}$	N
<i>GBX2</i>		5	Aldosterone-regulated sodium reabsorption	$3.2 \times 10^{-4}$	N
<i>GBX2</i>		5	Proximal tubule bicarbonate reclamation	$6.6 \times 10^{-4}$	N
<i>HIST1H family</i>		3	nucleosome	$3.5 \times 10^{-82}$	Y
<i>HIST1H family</i>		1	regulation of gene silencing	$2.5 \times 10^{-80}$	N
<i>HIST1H family</i>		1	nucleosome assembly	$8.3 \times 10^{-77}$	Y
<i>HIST1H family</i>		3	protein-DNA complex	$2.6 \times 10^{-75}$	Y
<i>HIST1H family</i>		1	chromatin assembly	$1.6 \times 10^{-74}$	Y
<i>HIST1H family</i>		1	nucleosome organization	$2.6 \times 10^{-73}$	Y
<i>HIST1H family</i>		1	protein-DNA complex assembly	$7.3 \times 10^{-73}$	Y
<i>HIST1H family</i>		4	Telomere Maintenance	$1.6 \times 10^{-26}$	Y
<i>HIST1H family</i>		4	Chromosome Maintenance	$4.7 \times 10^{-19}$	Y
<i>IP6K3</i>		1	skeletal muscle fiber development	$7.2 \times 10^{-7}$	N
<i>IP6K3</i>		3	acetylcholine-gated channel complex	$7.3 \times 10^{-7}$	N
<i>IP6K3</i>		3	Z disc	$8.2 \times 10^{-7}$	N
<i>IP6K3</i>		3	myosin filament	$9.7 \times 10^{-7}$	N
<i>IP6K3</i>		1	striated muscle cell differentiation	$1.4 \times 10^{-6}$	N
<i>IP6K3</i>		4	Acetylcholine Binding And Downstream Events	$1.6 \times 10^{-6}$	N
<i>IP6K3</i>	*	4	Activation of Nicotinic Acetylcholine Receptors	$1.6 \times 10^{-6}$	N
<i>IP6K3</i>		4	Postsynaptic nicotinic acetylcholine receptors	$1.6 \times 10^{-6}$	N
<i>IP6K3</i>		3	sarcoplasmic reticulum	$2.0 \times 10^{-6}$	N
<i>IP6K3</i>	*	4	Presynaptic nicotinic acetylcholine receptors	$2.8 \times 10^{-6}$	N
<i>RNF123</i>		1	hemoglobin metabolic process	$8.2 \times 10^{-15}$	N

## Brain

- Cognition
- Motivation

## But also:

- Health
- Immune system
- Fitness
- Metabolism
- Digestion
- Physical growth

# SES effects on education

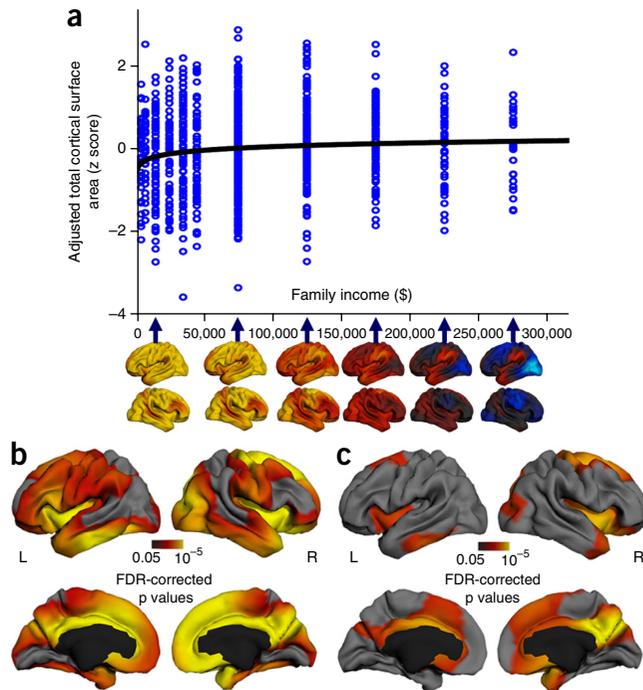


When you've made the SES effects go away,  
the remaining differences are genetic

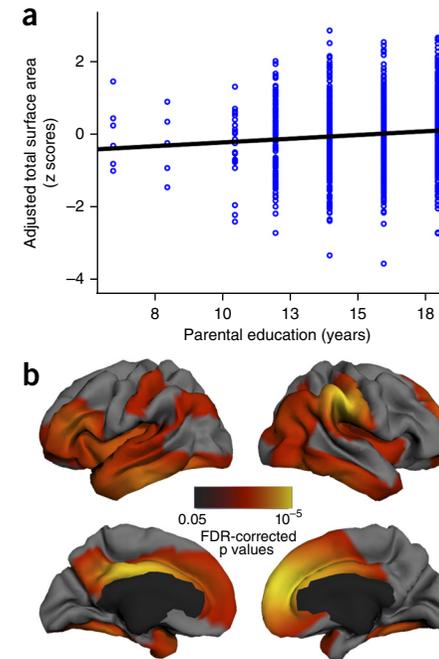
# Family income, parental education and brain structure in children and adolescents

Kimberly G Noble<sup>1,2,32</sup>, Suzanne M Houston<sup>3-5,32</sup>, Natalie H Brito<sup>6</sup>, Hauke Bartsch<sup>7</sup>, Eric Kan<sup>4,5</sup>, Joshua M Kuperman<sup>8-10</sup>, Natacha Akshoomoff<sup>10-12</sup>, David G Amaral<sup>10,13</sup>, Cinnamon S Bloss<sup>10,14</sup>, Ondrej Libiger<sup>15</sup>, Nicholas J Schork<sup>16</sup>, Sarah S Murray<sup>10,17</sup>, B J Casey<sup>10,18</sup>, Linda Chang<sup>10,19</sup>, Thomas M Ernst<sup>10,19</sup>, Jean A Frazier<sup>10,20</sup>, Jeffrey R Gruen<sup>10,21-23</sup>, David N Kennedy<sup>10,20</sup>, Peter Van Zijl<sup>10,24,25</sup>, Stewart Mostofsky<sup>10,25</sup>, Walter E Kaufmann<sup>10,26,27</sup>, Tal Kenet<sup>10,27,28</sup>, Anders M Dale<sup>8-10,29-31</sup>, Terry L Jernigan<sup>10,11,12,29</sup> & Elizabeth R Sowell<sup>4,5,10</sup>

Socioeconomic disparities are associated with differences in cognitive development. The extent to which this translates to disparities in brain structure is unclear. We investigated relationships between socioeconomic factors and brain morphometry, independently of genetic ancestry, among a cohort of 1,099 typically developing individuals between 3 and 20 years of age.



1-2% of variability



N=1099

# Developmental Science

Developmental Science (2015), pp 1–17

= planning, controlling, regulating behaviour

## PAPER “SES”

### Socioeconomic status and executive function: developmental trajectories and mediation

Daniel A. Hackman,<sup>1</sup> Robert Gallop,<sup>2</sup> ...  
Martha J. Farah<sup>1</sup>

1. Center for Cognitive Neuroscience, Center for Neuroscience and Society, Department of Psychology, University of Pennsylvania  
2. Department of Mathematics and Applied Statistics, West Virginia University, USA  
3. Departments of Design and Environmental Analysis and Human-Computer Interaction, Bronfenbrenner Center for Translational Research, Cornell University, USA

If schooling partly compensates for the effects of earlier deprivation, lower-SES children should 'catch up'

Early relation between SES and executive function persisted without narrowing or widening across early and middle childhood

**Table 3** Intercorrelation among potential mediators and measures of socioeconomic status

Measure	1	2	3	4	5	6	7	8	9	10	11
1. Birthweight	–										
2. Gestational age	.47***	–									
3. Maternal depression	–.02	.03	–								
4. Negative life events	.07*	.07*	.18***	–							
5. Parent stress	–.01	.07*	.50***	.10**	–						
6. Enrichment: Infant / Toddler	.10**	.01	–.23***	.02	–.10**	–					
7. Enrichment: Early Childhood	.05	–.02	–.24***	–.01	–.11**	.57***	–				
8. Maternal sensitivity: Infant / Toddler	.12***	–.02	–.24***	.01	–.12***	.48***	.46***	–			
9. Maternal sensitivity: Early childhood	.09**	–.05	–.21***	–.01	–.12***	.40***	.44***	.59***	–		
10. Early income-to-needs	.03	–.08*	–.24***	–.05	–.09**	.46***	.49***	.48***	.42***	–	
11. Maternal education	.07*	–.04	–.23***	–.03	–.06	.40***	.49***	.46***	.42***	.58***	–

\*  $p < .05$ ; \*\*  $p < .01$ ; \*\*\*  $p < .001$ .

NICHD Study of Early Childcare. N = 1009 children in US followed from birth to 8 years

The interest

What's surprising

The future is mechanism

Labelling

The science

Genetics and education

**What use is that to teachers?**

Screening

Personalised learning

What is changeable?

What do we want from education?

# But

- What if the genetics stuff, the high heritability of behaviour, wasn't a surprise?
  - Accept that some kids are brighter than others
- What if we moved straight on to the next question – what are we (parents, teachers, therapists, policymakers) supposed to make of the genetic results?

# You may think

- Leave the genetic bit, you can't change that. Focus on the things you can change, the environmental bit
- You'd be wrong in two ways
  - The genetic influences aren't inevitable
  - And the genetic effects can tell you how best to change the environment

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Sarah

Reading: C  
Maths: A\*

Sarah's parents  
are both  
mathematicians



Dominik

Reading: B  
Maths: B



Amy

Reading: B  
Maths: C



Jack

Reading: D  
Maths: E

Jack's  
parents are  
unemployed  
and the  
household is  
chaotic



Ffion

Reading: A\*  
Maths: A\*

Ffion's  
parents  
want to  
transfer her  
to a private  
school

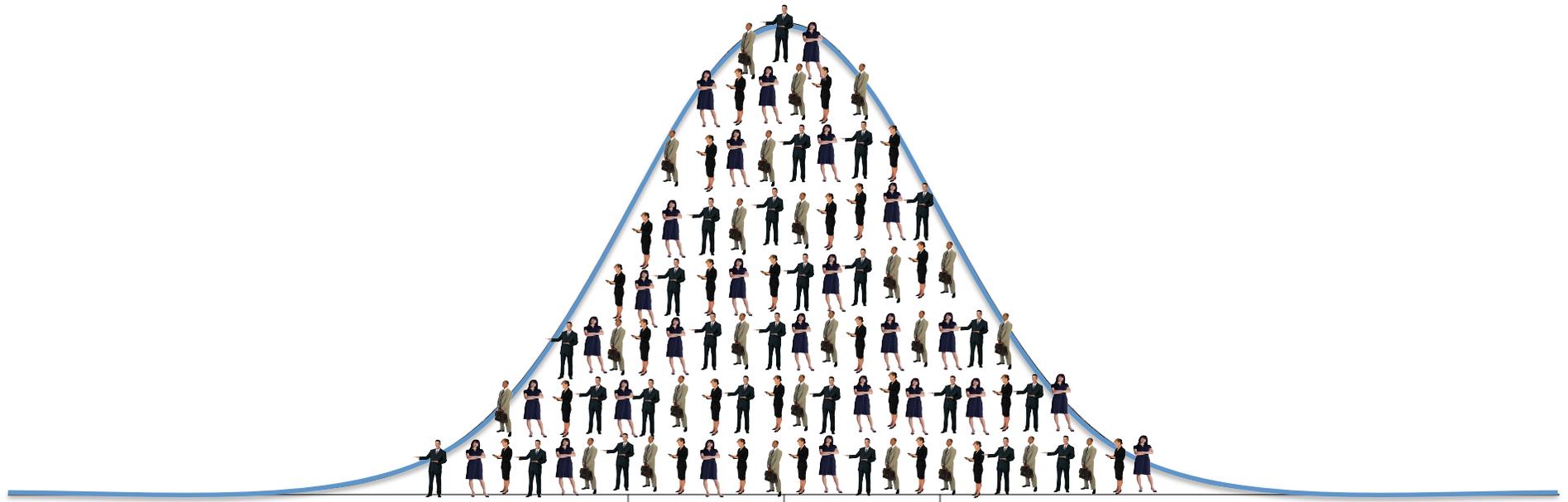


Billy

Reading: F  
Maths: B

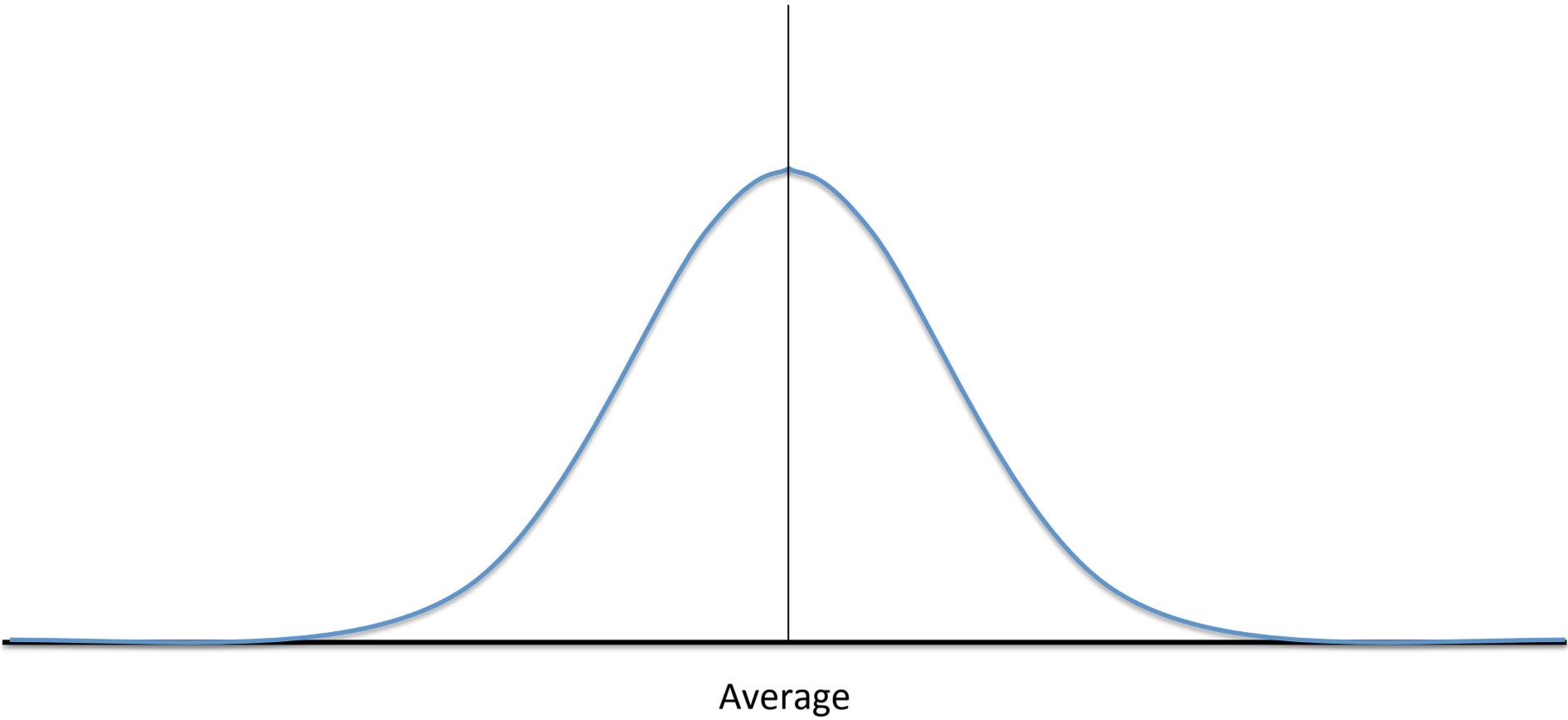
Billy really  
struggles  
with reading

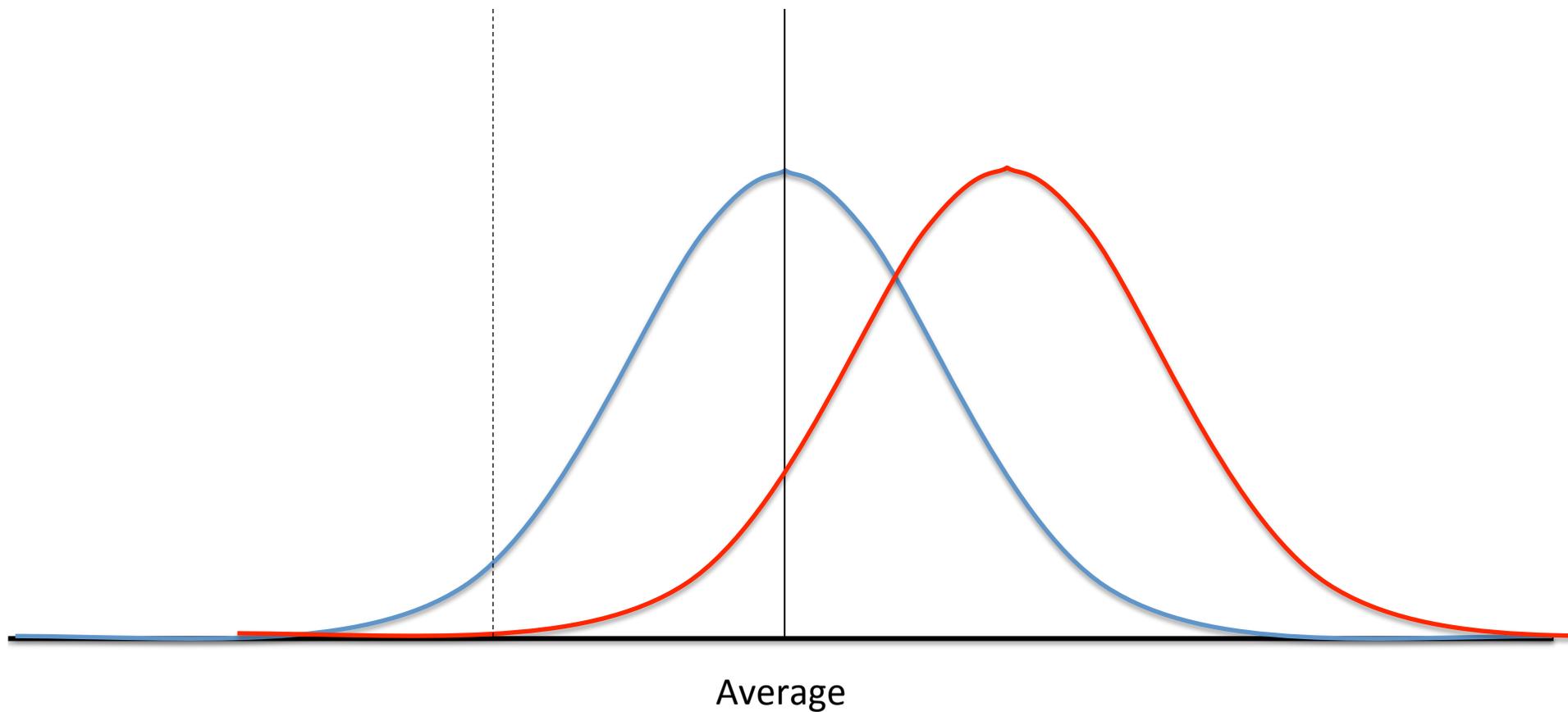
- “No child left behind”
- “Educate the best, forget the rest”
- “Too much too soon”
- “Every child should realise their potential”
- “The Finnish model” – minimum levels of literacy and numeracy in our society



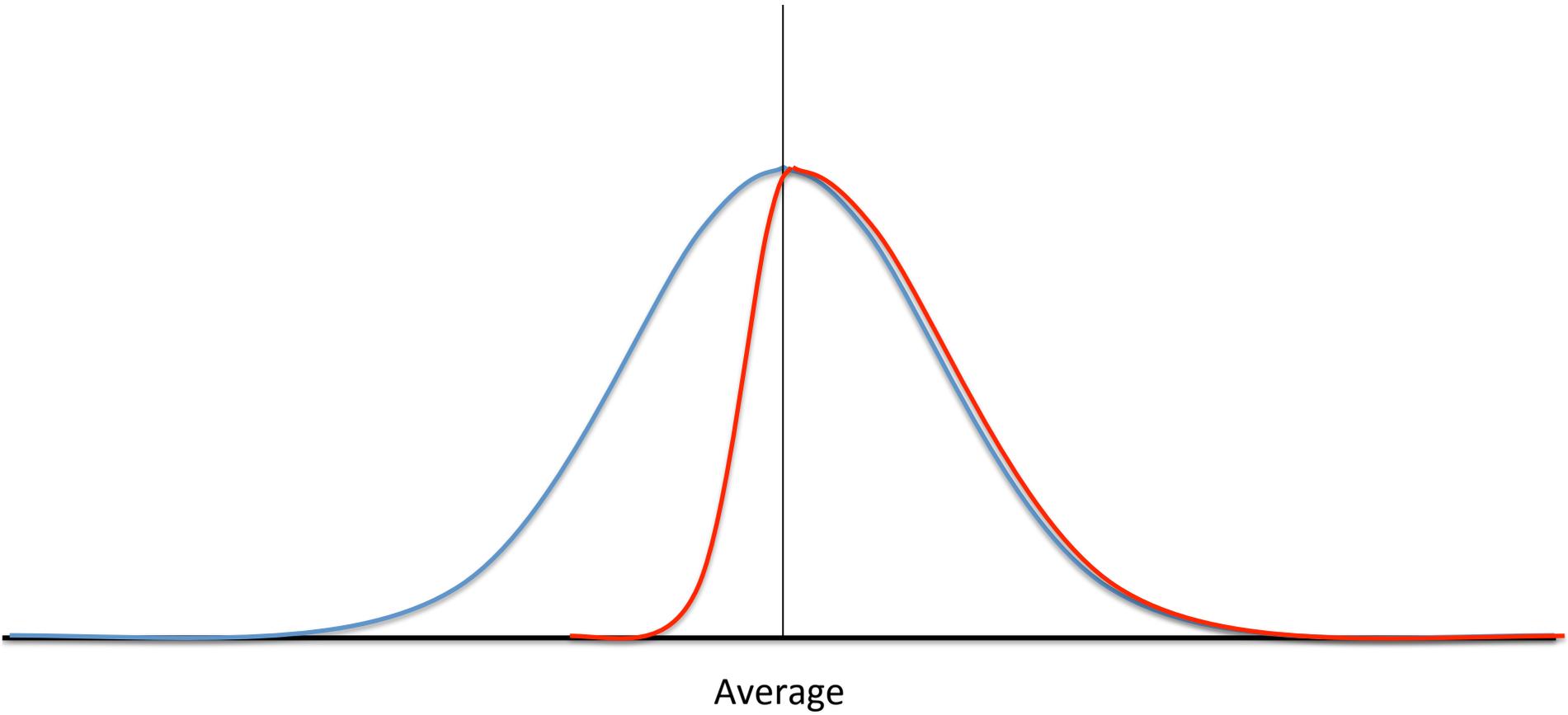
Average

Normal Distribution

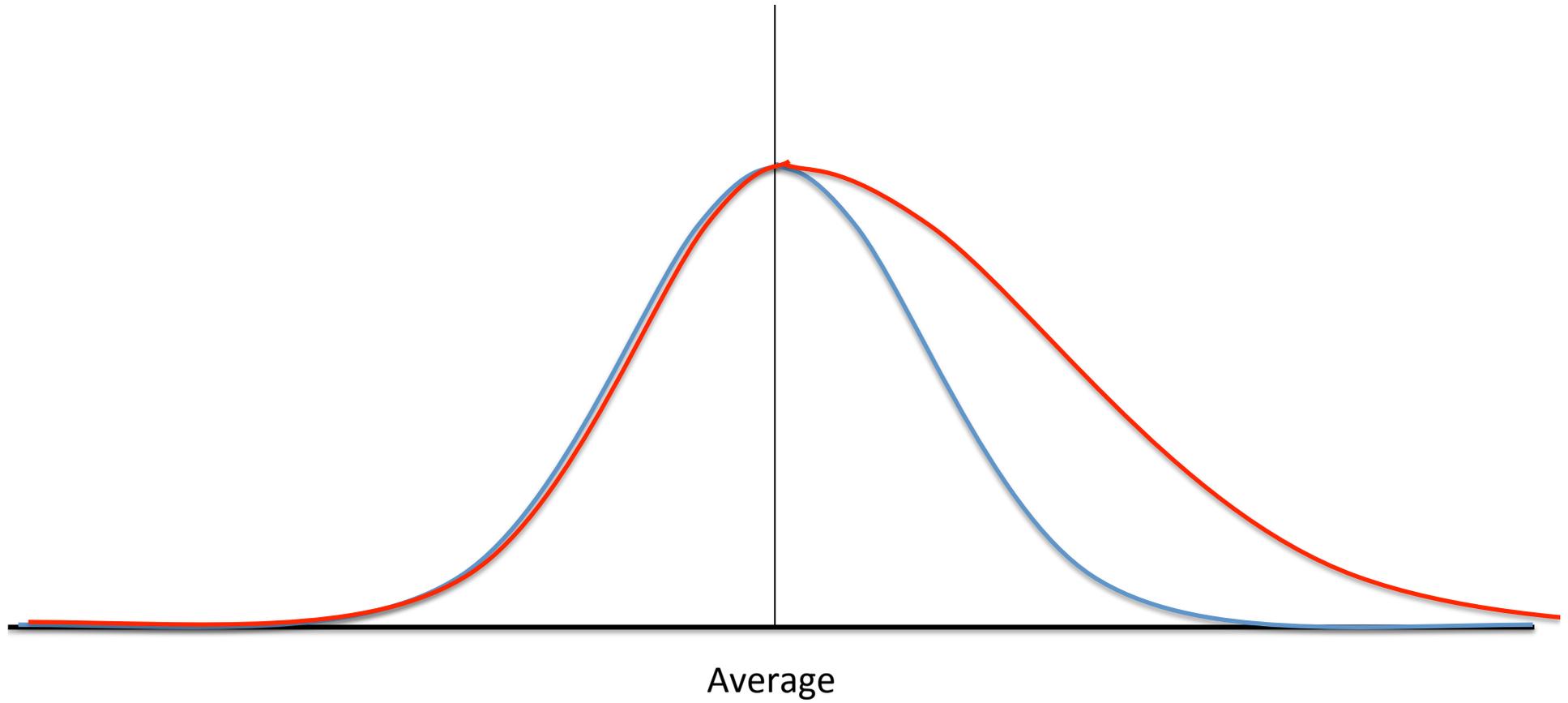




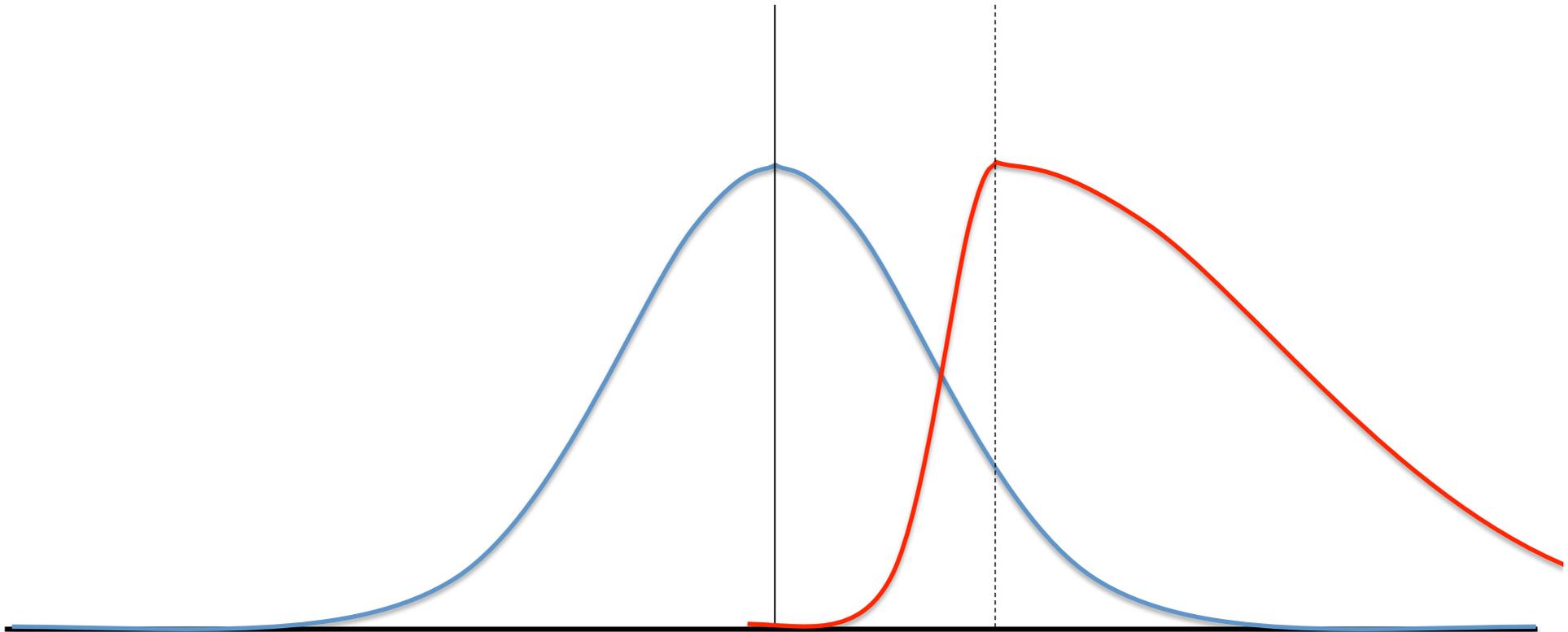
Finnish model – minimum levels of literacy and numeracy in society



No child left behind

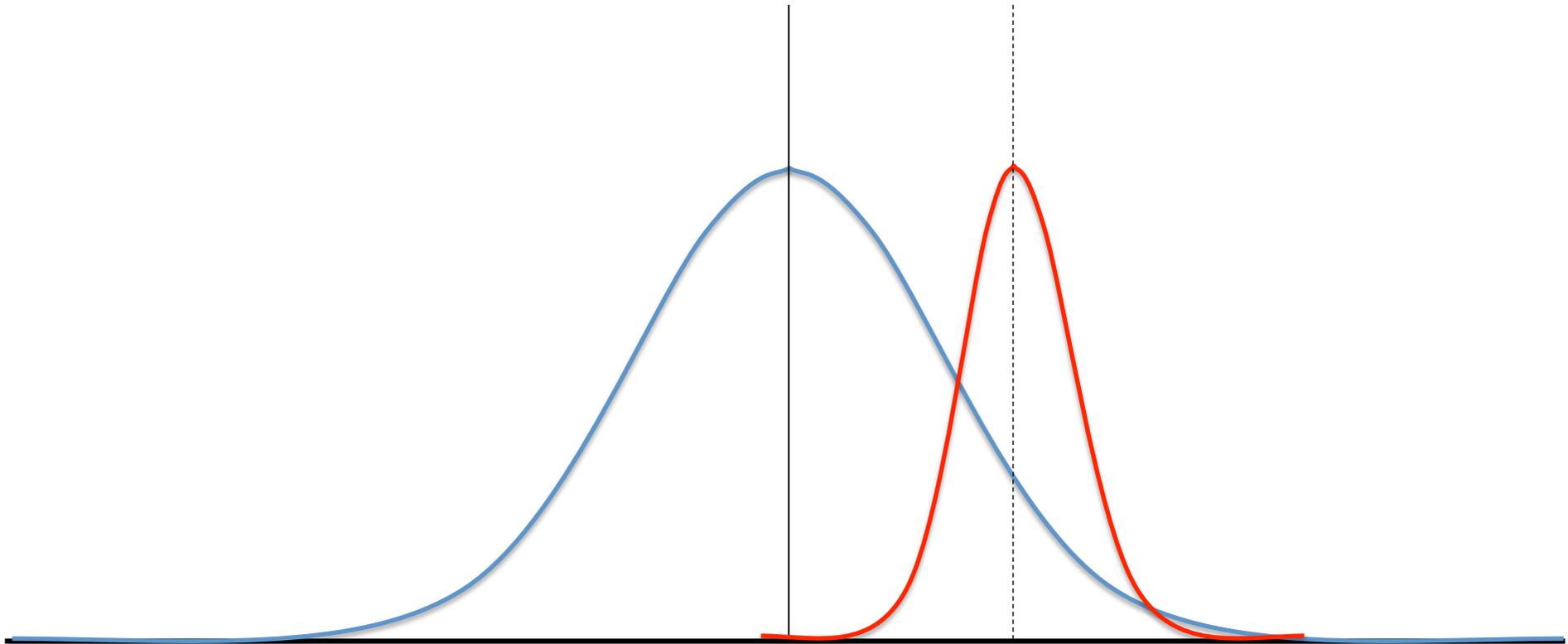


Educate the best forget the rest



Average

Panacea...?



Average

Panacea...?

- The relationship between the population average and individual differences is a tricky thing

5 October 2014 Last updated at 18:57

## Height differences 'could be caused by DNA changes'



Thousands of genes could be involved in height, according to the study

**Subtle changes in our genetic make-up could help to explain why some people are taller than others, the largest ever study of height has suggested.**

About 400 genome regions have been identified that may be responsible for the extra inches, according to research involving more than 250,000 people.

Scientists say this could pave the way for a simple test to reassure parents with fears about their child's growth.

It may also shed more light on cancer, where cell growth is out of control.

Studies suggest up to 80% of what determines height lies in our genetic code.

But the exact genes and other bits of DNA involved are only just being explored.

The first height gene to be identified was discovered in 2007.

But this report, in Nature Genetics, suggests many thousands of genes and other regions of DNA could all play a part.

Scientists from 300 institutions examined the DNA of more than a quarter of a million people across Europe.

### Related Stories

[Men's height 'up 11cm since 1870s'](#)

“

**This... could have real impact in the treatment of diseases that can be influenced by height such as osteoporosis or cancer”**

Prof Frayling  
 University of Exeter

# The heritability of height is 80-90% (perhaps 1000 genes)

## Men's average height 'up 11cm since 1870s'

COMMENTS (326)

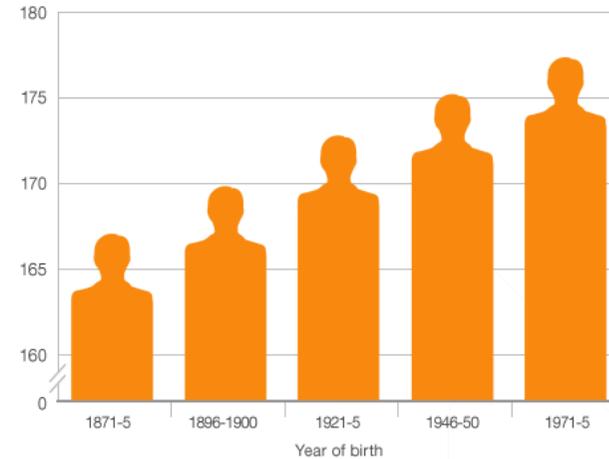
By Caroline Parkinson

Health editor, BBC News website

### A century of growth

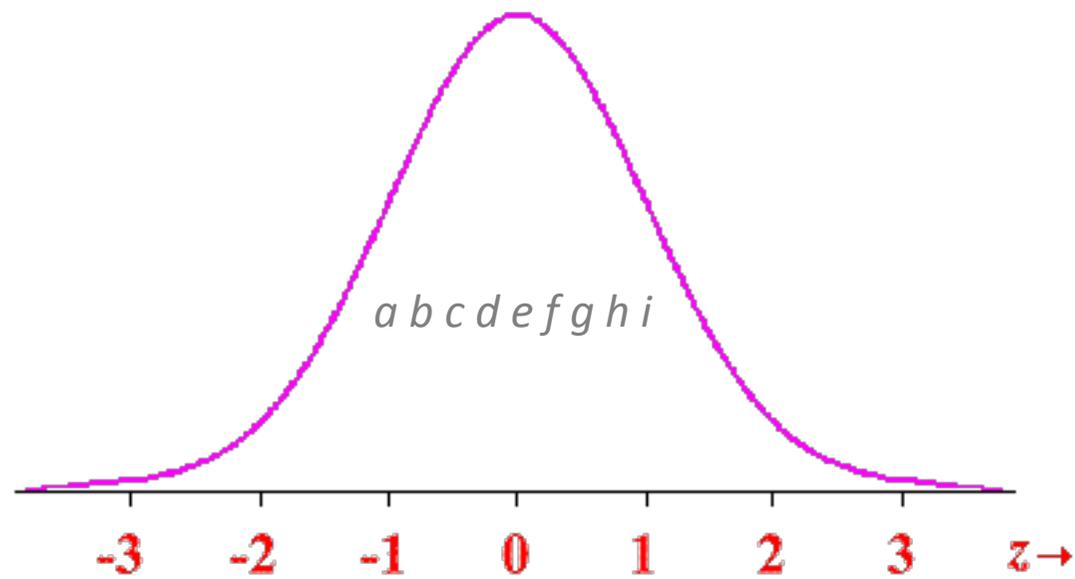
British males: Average height at age 21

Height cm

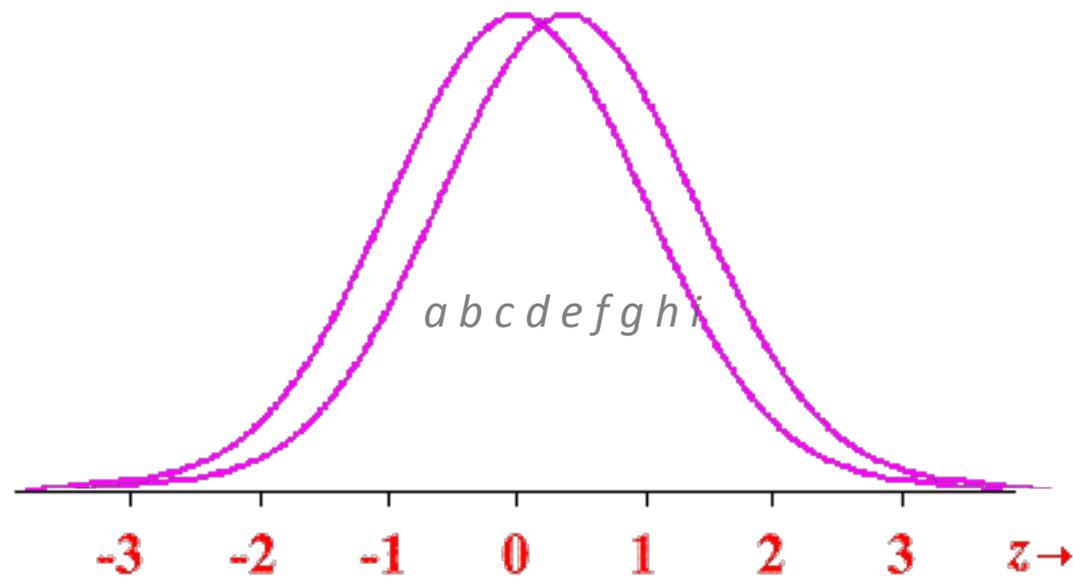


Source: Prof Tim Hatton et al, Oxford Economic Papers

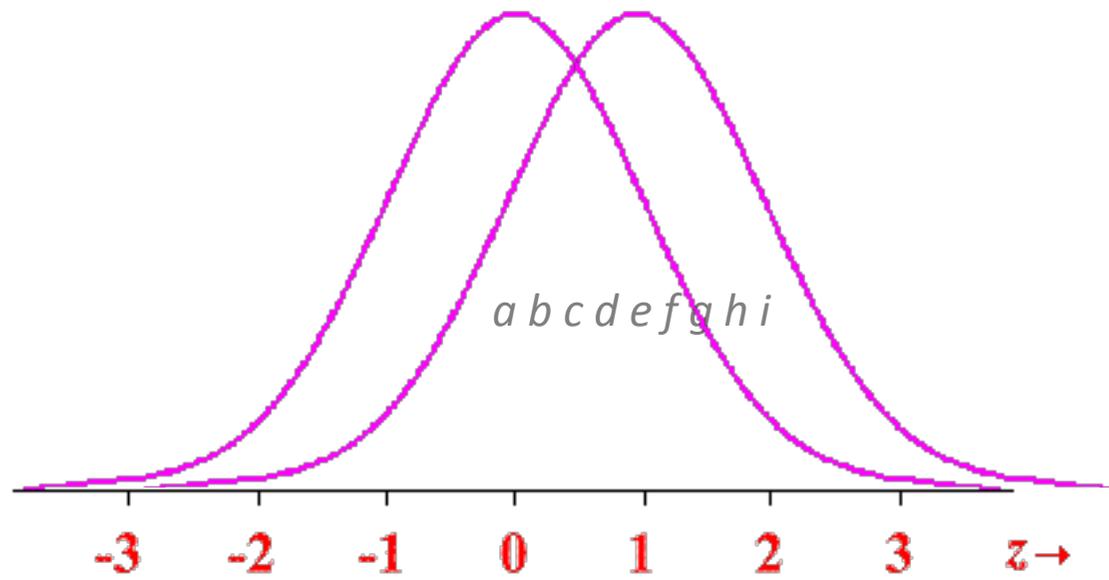
# Relative vs absolute levels



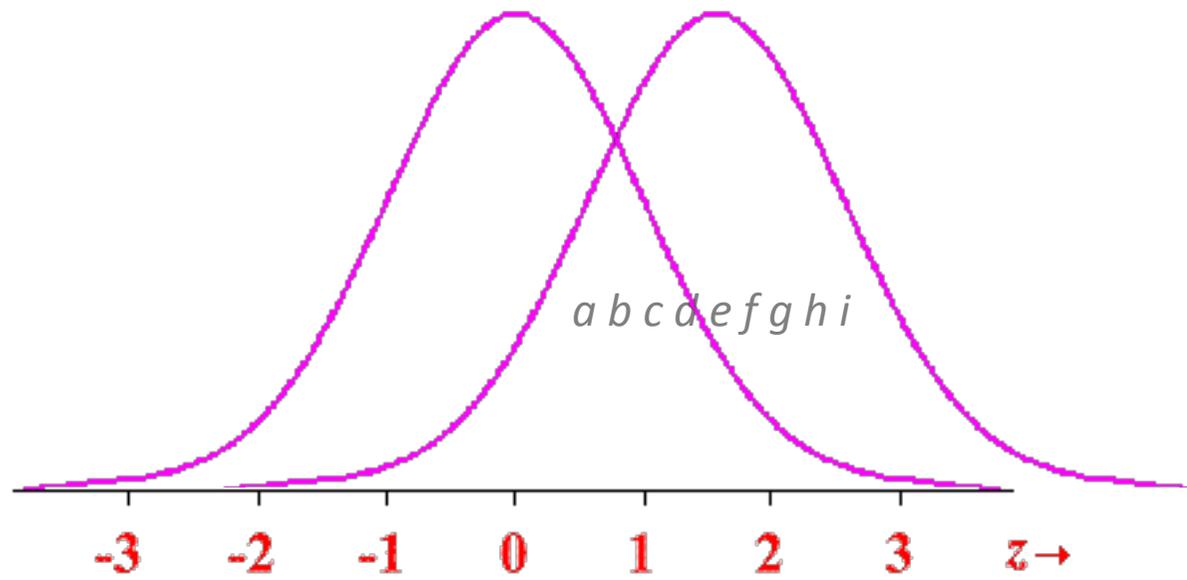
# Relative vs absolute levels



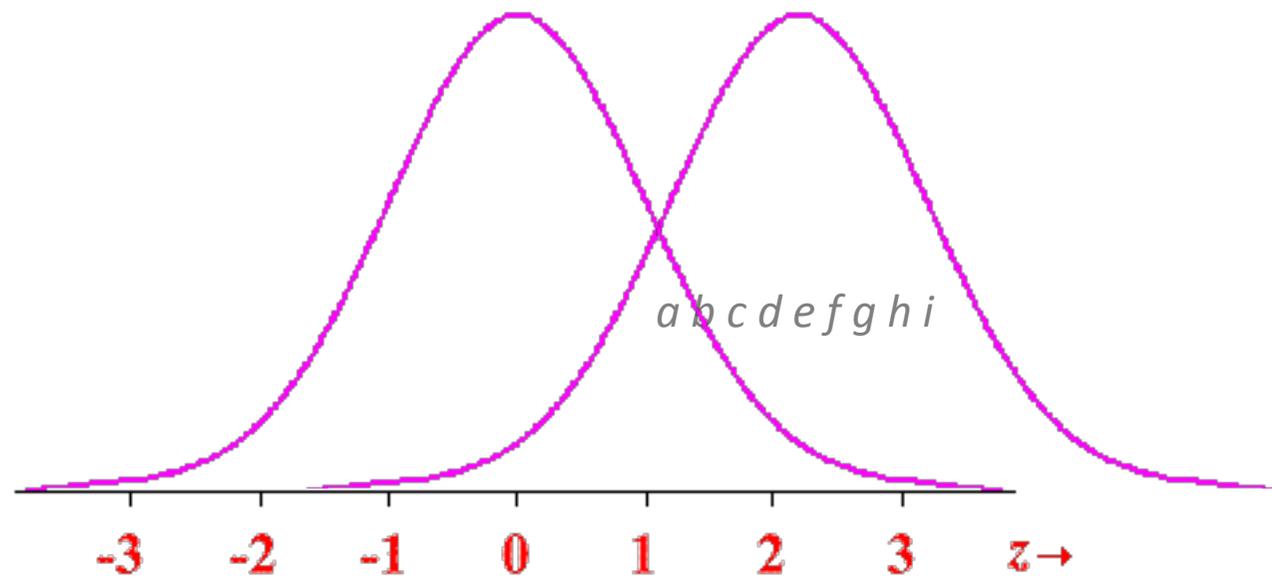
# Relative vs absolute levels



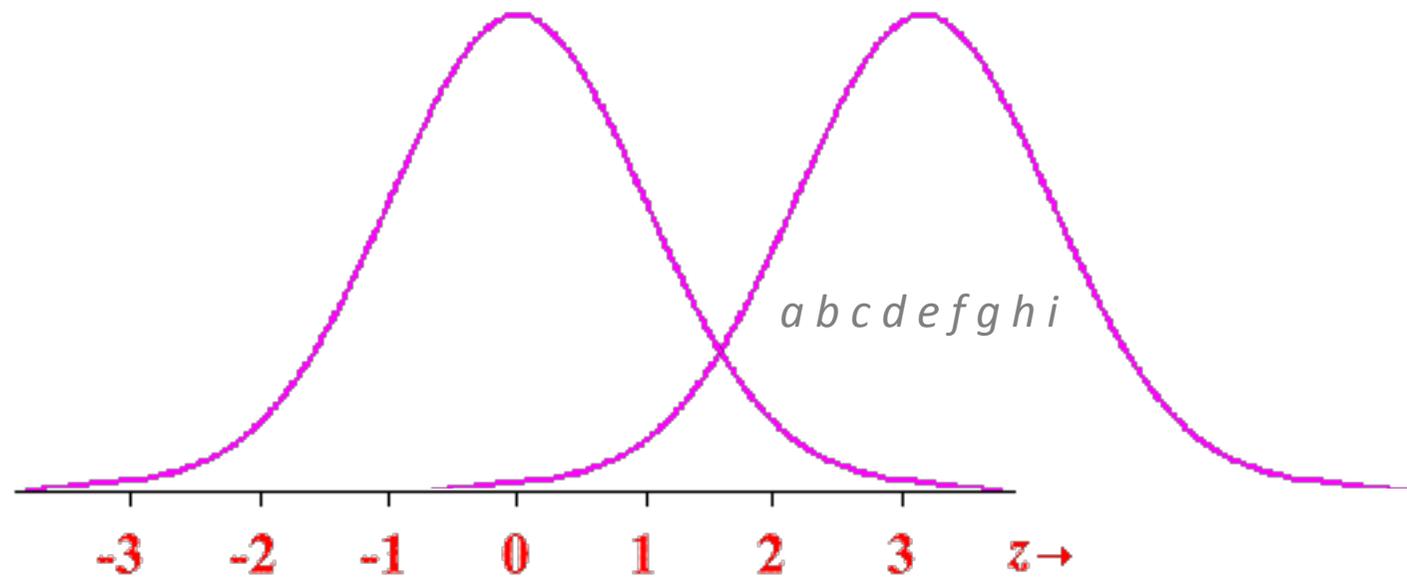
# Relative vs absolute levels



# Relative vs absolute levels



# Relative vs absolute levels





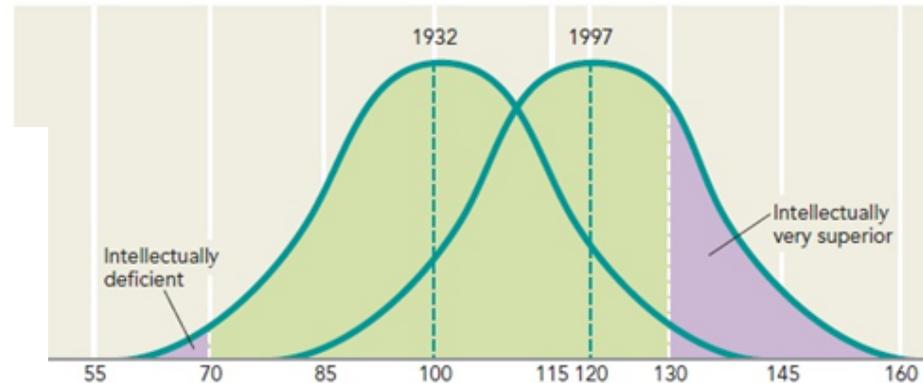
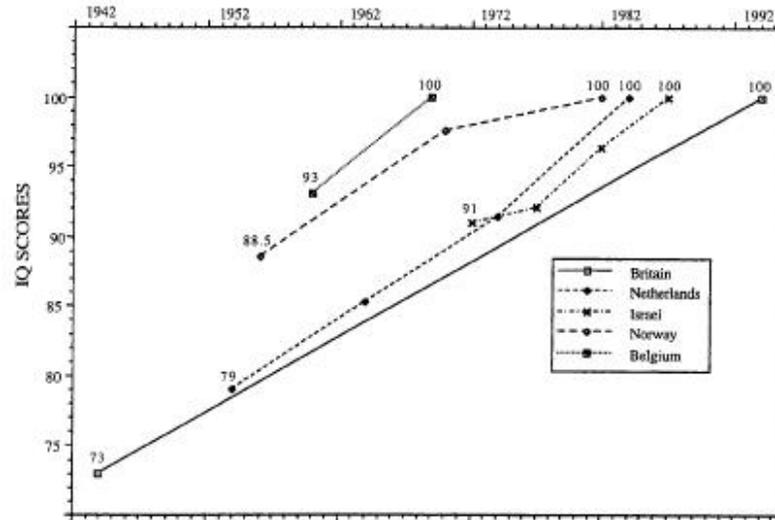
# Are We Getting SMARTER?

Rising IQ in the Twenty-First Century

James R. Flynn



## The Flynn Effect



Yet intelligence is  
60-70% heritable!

# The Phonics test

- Because scores are highly heritable does not mean we can't improve performance for everyone ('shift the distribution')
- National education policy is often about shifting the distribution

# Genetics and education

The interest

The future is mechanism

What's surprising

Labelling

The science

Screening

What use is that to teachers?

Personalised learning

What is changeable?

What do we want from education?

# Genetic effects are not deterministic

- Environmental interventions can alter genetic effects
- Phenylketonuria (PKU)
- Treatment:
  - Newborn screening
  - Diet low in phenylalanine + protein supplements



## Teacher Quality Moderates the Genetic Effects on Early Reading

J. Taylor,<sup>1\*</sup> A. D. Roehrig,<sup>2</sup> B. Soden Henler,<sup>1</sup> C. M. Connor,<sup>1,3</sup> C. Schatschneider<sup>1,3</sup>

Children's reading achievement is influenced by genetics as well as by family and school environments. The importance of teacher quality as a specific school environmental influence on reading achievement is unknown. We studied first- and second-grade students in Florida from schools representing diverse environments. Comparison of monozygotic and dizygotic twins, differentiating genetic similarities of 100% and 50%, provided an estimate of genetic variance in reading achievement. Teacher quality was measured by how much reading gain the non-twin classmates achieved. The magnitude of genetic variance associated with twins' oral reading fluency increased as the quality of their teacher increased. In circumstances where the teachers are all excellent, the variability in student reading achievement may appear to be largely due to genetics. However, poor teaching impedes the ability of children to reach their potential.

The ability to read proficiently is a critical skill, and children who fail in that skill are more likely to be retained a grade, drop out of school, and enter the juvenile criminal justice system (*1*)—all at substantial cost to society. Hence, we look to educators to ensure

that children achieve proficient literacy skills; yet, a large proportion of the variability in children's reading skills is associated with nonmalleable factors like genes (*2*). Small differences in heritability (estimate of genetic influence) from twins that do versus do not share a teacher raise doubts about the effect of teachers on students' reading development (*3*). At the same time, accumulating evidence from samples of unrelated children shows that teachers do affect children's reading skill gains (*4, 5*).

The dilemma is that research examining unrelated children cannot address whether effects are associated with genes or with the shared

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## Use patients' DNA to tailor treatment, doctors urged

Chris Smyth Health Editor

Patients should routinely have their whole genetic code read to decide on drug doses, one of the world's leading experts on personalised medicine says.

Common medicines such as statins, painkillers and blood thinners can have radically different effects that could be predicted by analysing a patient's DNA, said Gianrico Farrugia, chief executive of the Mayo Clinic in Florida.

Medicine is on the verge of a "seismic shift" where sequencing a patient's whole genome becomes a routine starting point for treatment, Dr Farrugia said. Babies could have their DNA read at birth to help doctors treat them over the course of their lives, he suggested.

Doctors are increasingly excited about the potential of tailoring treatment to a patient's genetic code rather than just their symptoms, with many of the latest cancer drugs targeting key mutations that drive the disease.

Trials are under way into deciding treatment based on the DNA profile of patients and their tumour, rather than where in the body it occurs, but genetic analysis is yet to become routine.

In an interview on a visit to Britain this week, Dr Farrugia urged doctors to "stop treating personalised medicine as special". He added: "That's a profound

...the surgery outweigh  
...it's a pretty easy decision,  
...to operate. She deserves treat-

Among the prime ministers of her  
lifetime, she recalled she "quite liked  
Churchill — he did his job well and it

shift that needs to happen in this country if we really want to democratise individual medicine. Otherwise it will remain the domain of the few."

Mayo patients are now routinely offered genetic analysis as emerging research finds it can help administration even of basic drugs, a process known as pharmacogenomics.

"There are some patients who tell you they take pain medication and it doesn't work, and some say half a dose knocks them out," Dr Farrugia said.

He said that the difference was down to genetic variations. About a quarter of patients had genes that mean they process drugs such as codeine very quickly, while others cannot break it down "so it's like giving them candy", he said.

With millions of patients urged to take cholesterol-lowering statins to cut their heart-attack risks, concern has centred on the side effects. Dr Farrugia said which individuals would get the most severe muscle pain was "totally predictable" using genetic analysis.

Currently gene sequencing costs more than £1,000, but Dr Farrugia said that prescribing based on genes was likely to cut costs by reducing side effects and the number of wasted doses.

"We want to get it down to \$100. At \$100 we think it becomes standard," he said.

# Precision medicine

## Deconstructed, parsed, and diagnosed.

A hypothetical example illustrates how precision medicine might deconstruct traditional symptom-based categories. Patients with a range of mood disorders are studied across several analytical platforms to parse current heterogeneous syndromes into homogeneous clusters.

### Symptom-based categories

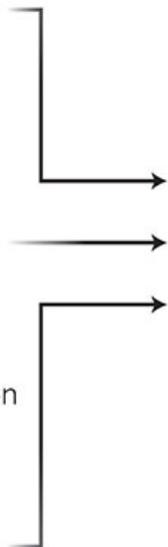
Major depressive disorder



Mild depression (dysthymia)



Bipolar depression



### Integrated data

**Genetic risk**  
polygenic risk score

**Brain activity**  
insula cortex

**Physiology**  
inflammatory markers

**Behavioral process**  
affective bias

**Life experience**  
social, cultural, and environmental factors



### Data-driven categories

Cluster 1



Cluster 2



Cluster 3



Cluster 4



Prospective replication and stratified clinical trials

- Your chairs have been fitted with DNA detectors

- See what we do. We change the environment.
- The question is which environment. And how.

# Genetics and education

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**Personalised learning**

What is changeable?

What do we want from education?

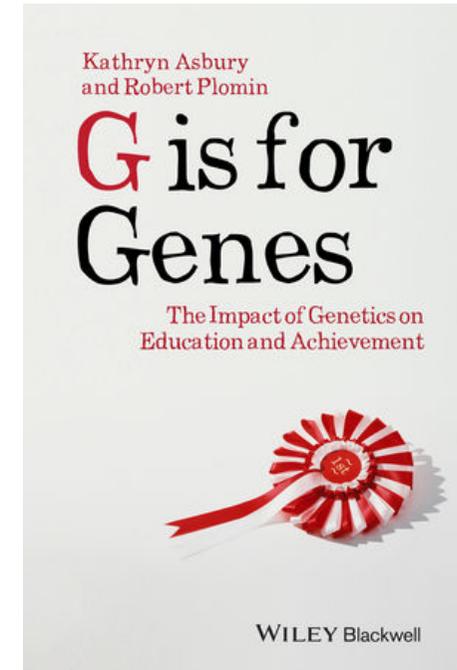
# Personalised learning

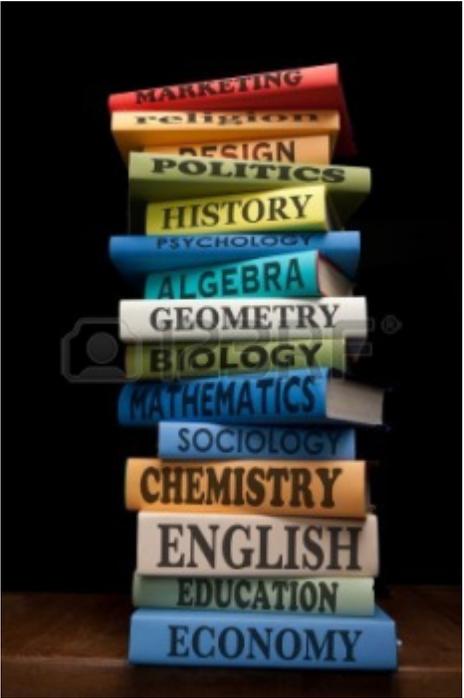


I think a genetic view suggests an active model of education. In genetics, we call this a gene-environment correlation. It's the idea that children create and modify and select environments that are correlated with their genetic propensities.

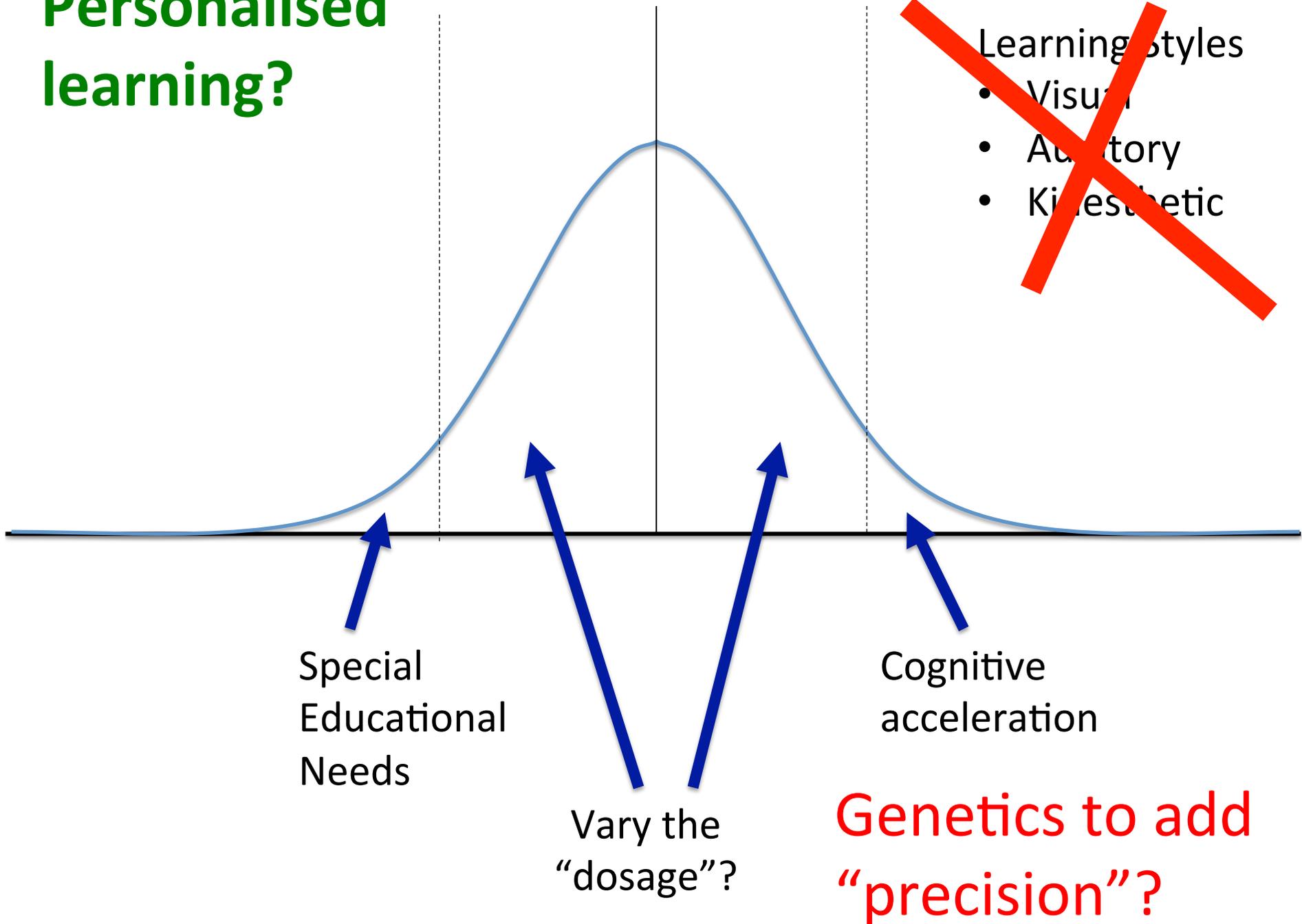


Professor Robert Plomin  
King's College London





# Personalised learning?



# Adaptive learning

An educational method which uses computers as interactive teaching devices, to orchestrate the allocation of human and mediated resources according to the unique needs of each learner



# More subtle possibilities

- Different methods will work for different kids
  - e.g., interventions for behavioural difficulties
  - e.g., training working memory



*Emotional and Behavioural Difficulties*, 2013  
<http://dx.doi.org/10.1080/13632752.2012.757097>



### Can developmental cognitive neuroscience inform intervention for social, emotional and behavioural difficulties (SEBD)?

Norah Frederickson<sup>a\*</sup>, Alice P. Jones<sup>b</sup>, Laura Warren<sup>c</sup>, Tara Deakes<sup>d</sup> and Geoff Allen<sup>e</sup>

<sup>a</sup>*Health Psychology, UCL, London, UK;* <sup>b</sup>*Department of Psychology, UCL, London, UK;* <sup>c</sup>*Educational Psychologist;* <sup>d</sup>*Head of School Psychology, UCL, London, UK;* <sup>e</sup>*Head of School Psychology, UCL, London, UK*

Designing an intervention to address neuro-conduct problems was undertaken in this study. Unemotional traits, a novel intervention was used in a school for children with social, emotional and behavioural difficulties. A mixed methods design was used to investigate the effectiveness of the intervention. Changes in the change process, alongside changes in behaviour. Both qualitative and quantitative methods were used to investigate externalising behaviour and improvements in cognitive and affective processes. While different genotypes, associated changes in underlying cognitive and affective processes. While different genotypes, associated changes in underlying cognitive and affective processes. While different genotypes, associated changes in underlying cognitive and affective processes.

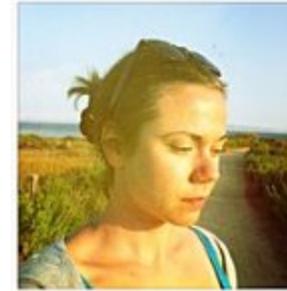
unemotional traits; SEBD; intervention; evaluation

Remove sanctions and emphasise a reward-focus

Table 4. Correlations between change scores for externalizing behaviour, executive functions and CU traits.

	Change in Externalising Behaviour score		
	Total Sample N = 29	High CU N = 14	Low CU N = 15
Change in CU trait score	.56**	.62*	.50
Change in Executive Function score	.55**	.44	.82**

\*p < .05, \*\*p < .01.



### Polymorphisms in the Dopamine Receptor 2 Gene Region Influence Improvements during Working Memory Training in Children and Adolescents

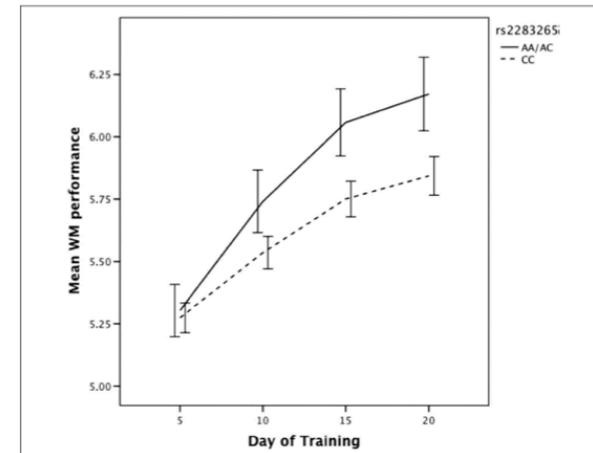
Stina Söderqvist, Hans Matsson, Myriam Peyrard-Janvid, Juha Kere, and Torkel Klingberg

#### Abstract

Studying the effects of cognitive training on children and adolescents is important for better treatments, but it can also be a topic of interest for brain plasticity and cognitive skills. In this study, we investigated how polymorphisms (SNPs) and ratings of intelligence associated to interindividual differences in working memory training. The study included 7–19 years who were genotyped for near eight candidate genes previously identified in genome-wide association studies: COMT, SLC6A3 (DAT1), DRD4, DRD2, PMAOA, LMX1A, and BDNF. Ratings on the Wechsler Intelligence Scale were also available for 156 of these participants performed at least 20 sessions of working memory training, and performance during the training was used as the outcome variable. We found that two

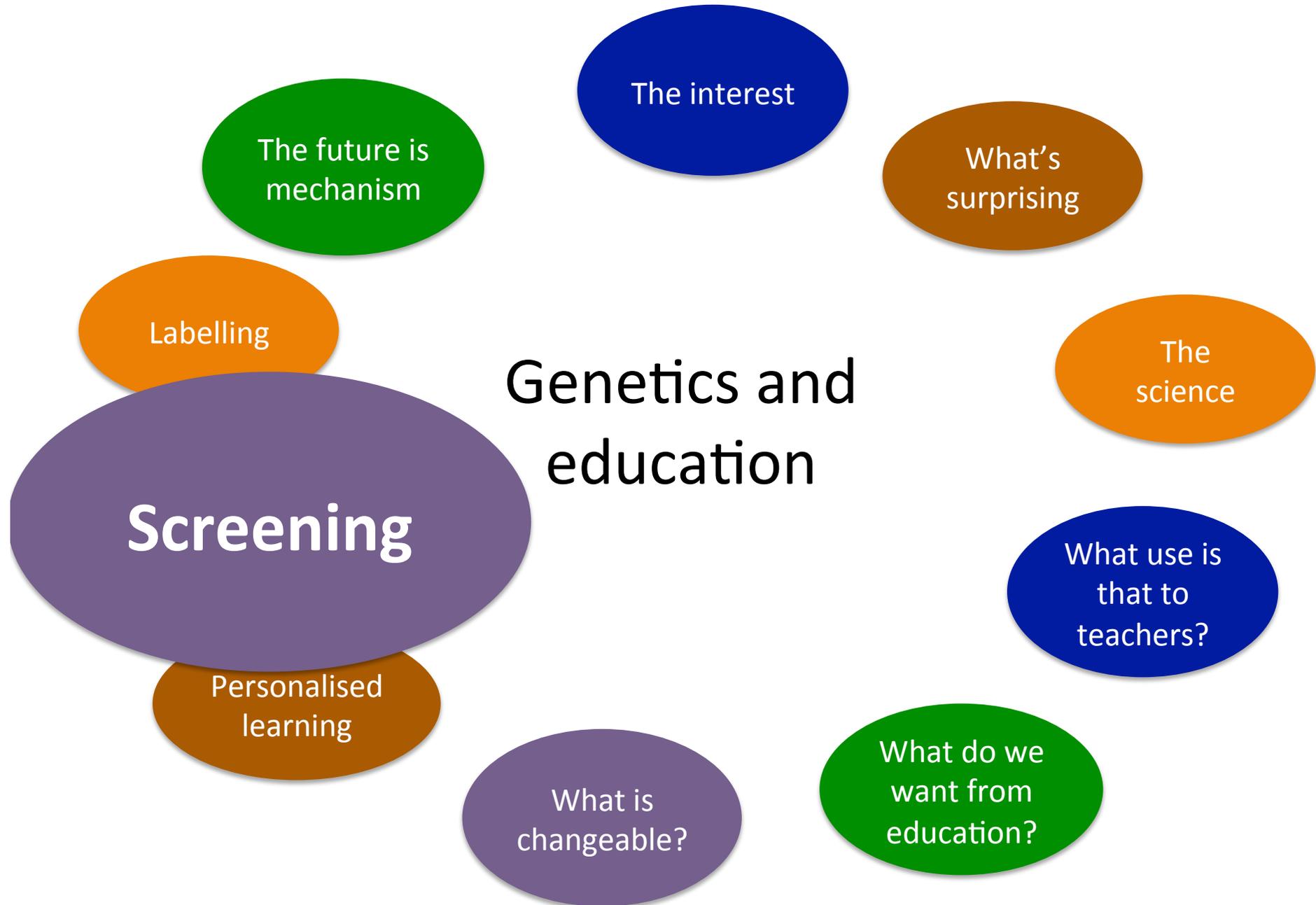
Some individuals respond better to working memory training

Figure 3. Performance on a backward digit span and visuospatial grid task during the training period, according to rs2283265 genotypes. Error bars show ±1 SEM.



## Which environment to change?

- Won't necessarily all be pedagogical or behavioural
- Could be health, diet, fitness, sleep, timing
- The potential drawback is that so many genes are involved (and so many environments)



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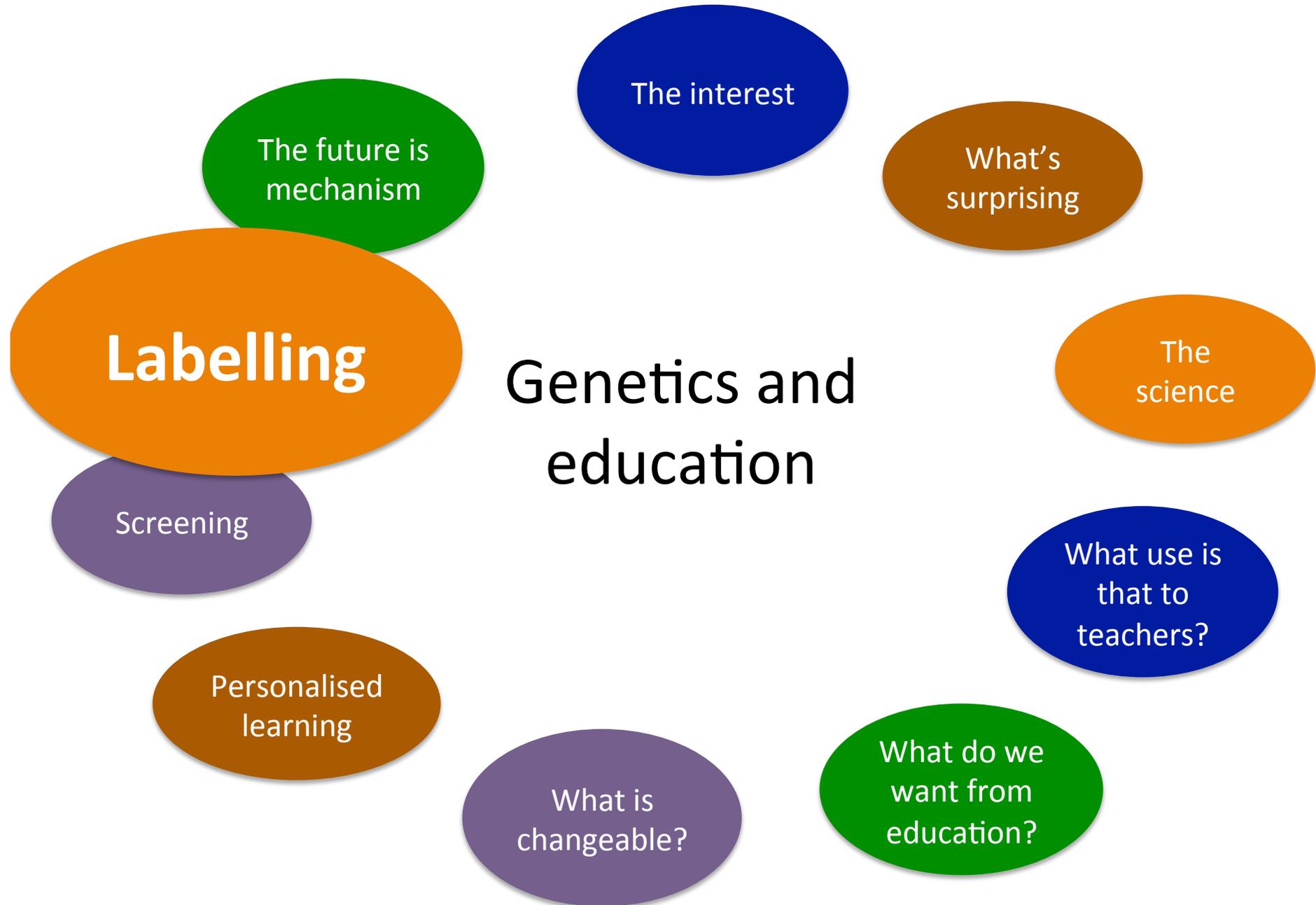
What do we  
want from  
education?

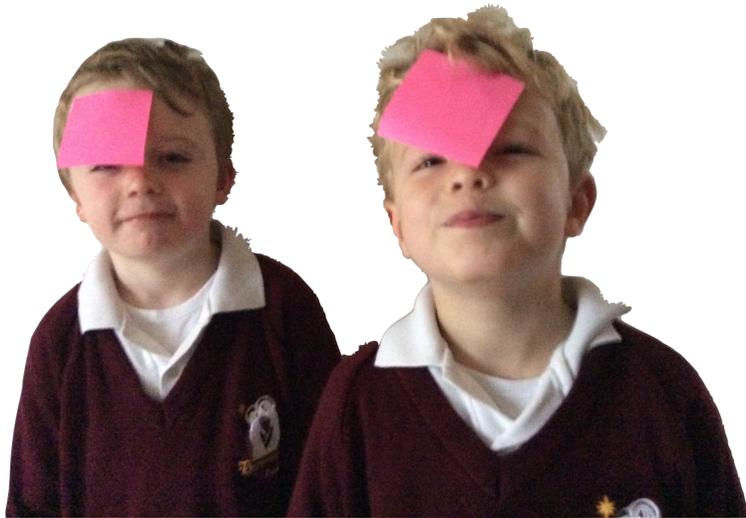
What is  
changeable?

# Does genetics point inevitably to screening?



- Early (pre-school)
- Independent of SES
- Better than 'averaging the parents'?





### Labelling theory and the self-fulfilling prophecy

- Labelling means attaching a 'tag' to pupils e.g. 'bright', 'lazy', 'dumb' etc
- **self-fulfilling prophecy** = 'what teachers believe about pupils, pupils achieve'
- Teachers labels kid bright → pupil internalises label → pupil becomes more enthusiastic, tries harder, ends up succeeding
- On other hand labelling as 'thick' can lead to underachievement

- Would genetic screening be just another form of labelling?
- How do we translate (ethically, practically) from population risk to the individual?

**The future is  
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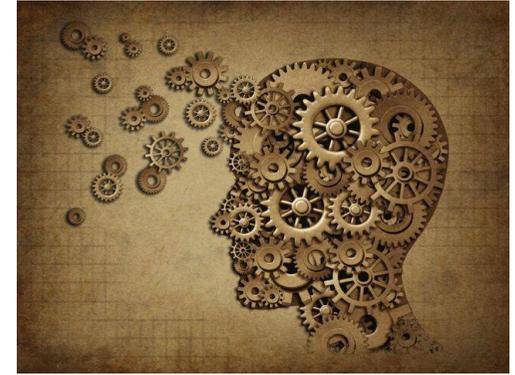
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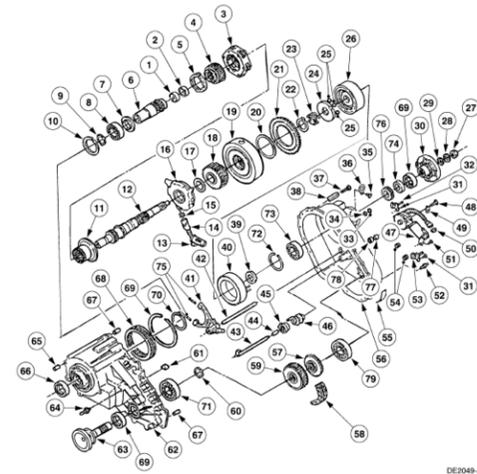
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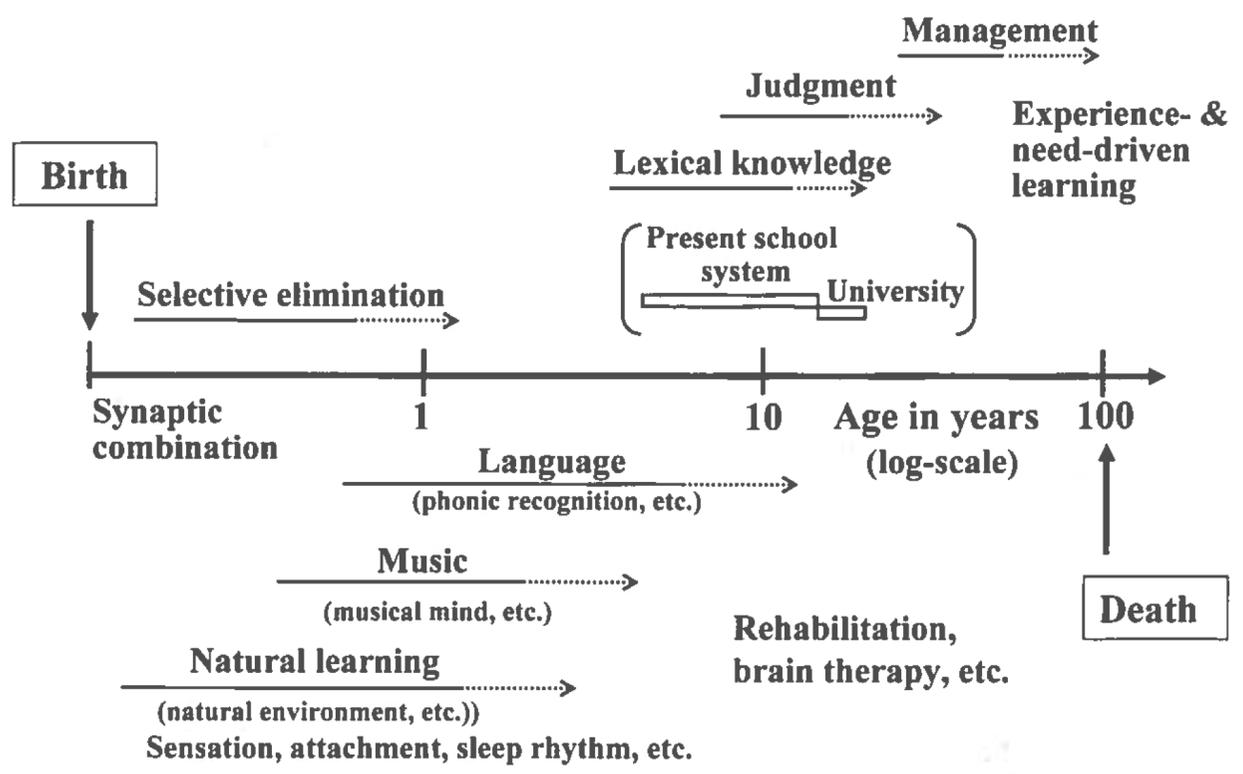
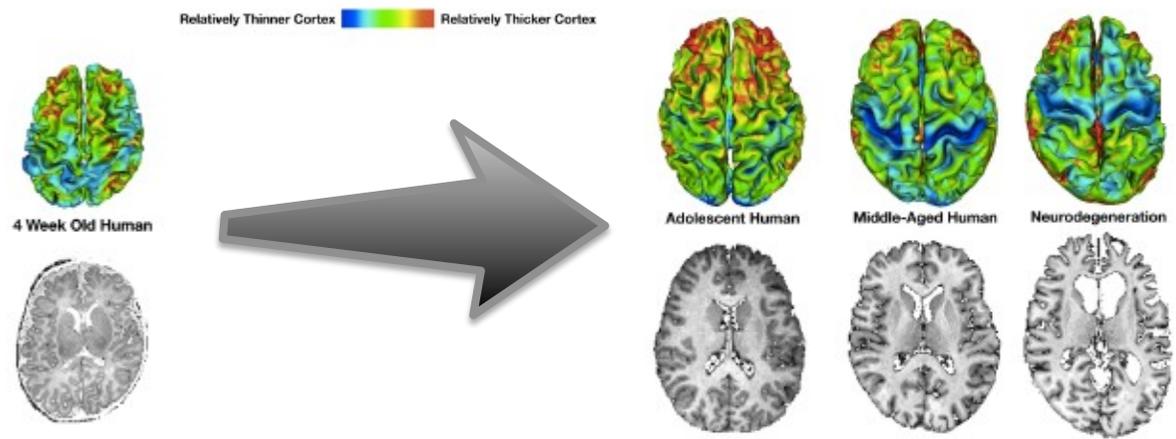
# Educational neuroscience



- Genetics can't just be about correlations, we have to understand biological and cognitive mechanisms
- Mechanisms that influence
  - learning,
  - willingness to learn
  - fitness to learn
  - opportunity to learn
  - persistence and retention of learning



DE2049-A



Source: Koizumi H., *Seizon and Life Sci.* (1998)

- What might genetic variation relevant to education influence?
  - Brain plasticity, brain power, neurotransmitter balance, development of low-level sensory and motor abilities, placing the right number of neurons in the right places and right wiring early in brain development
  - ... but also maybe limbic system function (anxiety, approach-avoidance, exploit-explore in reward-based learning)
  - ... maybe also immune response, oxygen transfer, energy consumption, resilience to stress
- We don't yet know, but likely that answer will be wider than a focus on cognitive abilities alone



Genetics and education: Is there an example of a hereditary trait or feature that has an impact on education or teaching? Knowing that height is mainly inherited doesn't seem to have an effect on the teaching techniques in high jump. So why are genetics of any interest to the average educator?



So why are genetics of any interest to the average educator?

Not all differences in educational achievement are environmental

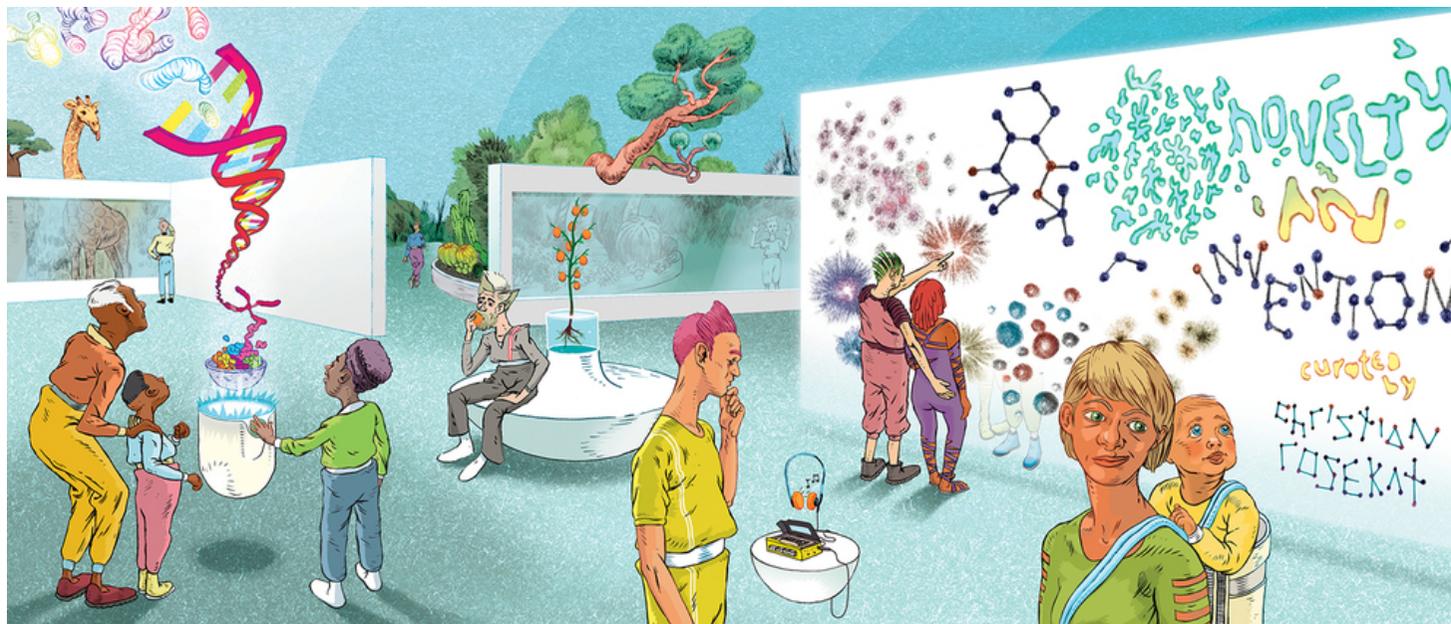
Society must determine the importance of overall population level vs. individual differences in education

Understanding of mechanism will tell us which environments to change: pedagogical but also health / fitness / timing?

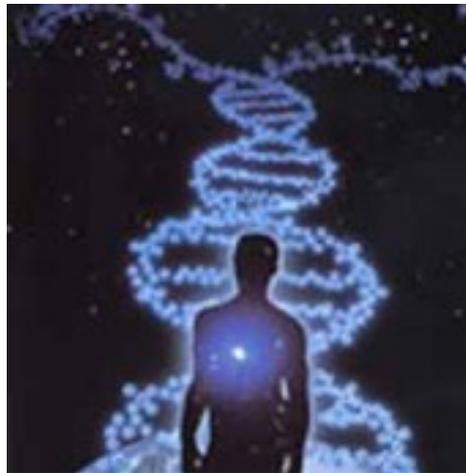
Genetic influences can reduce or increase in different environments: personalised learning

# Genes are not chains

- There are activities that humans haven't yet thought of doing that, if we all did them tomorrow, differences between us would be heritable



# The future is not fixed!



Thank you for your attention

