

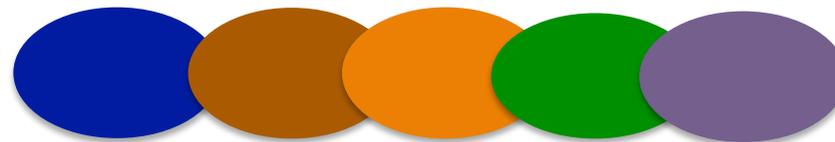
Learnus



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

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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... >

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What use is that to teachers?

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The Elephant in the Classroom

Helping Children Learn and Love Maths



$$5 \times 7 =$$

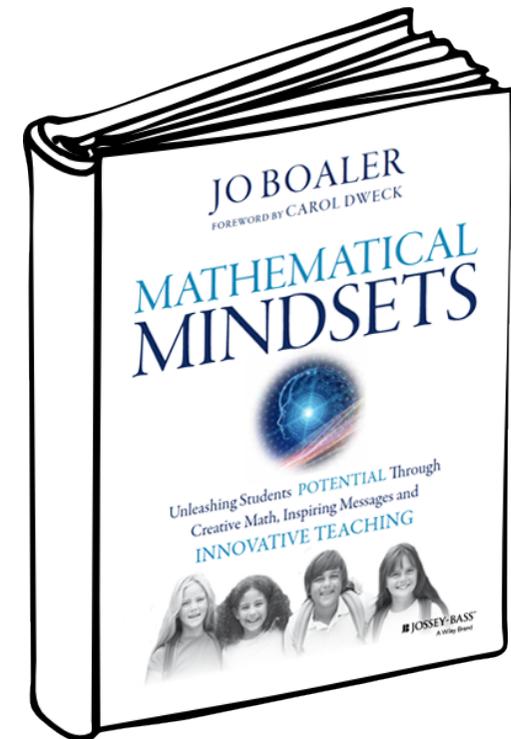
$$r = \sqrt{\frac{18.2}{4?}}$$

$$A = 4?r^2$$

$$16 \div 4 =$$



JO BOALER



New findings about brain science, mindset and learning.



Think It Up™



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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



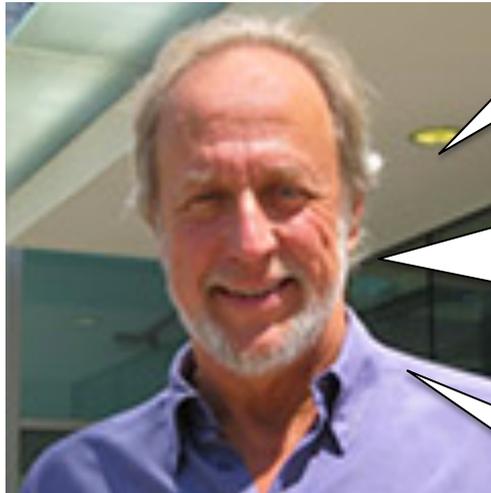
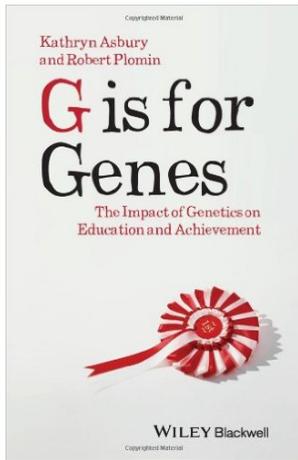
123456789

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

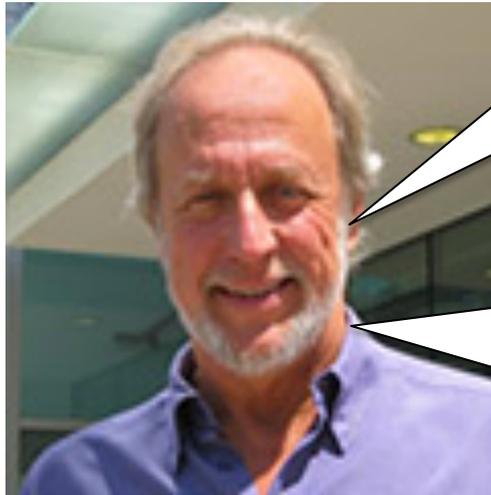
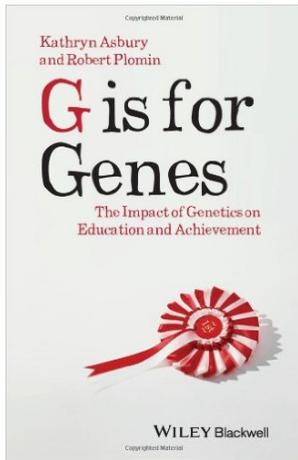


Professor Robert Plomin
King's College London

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



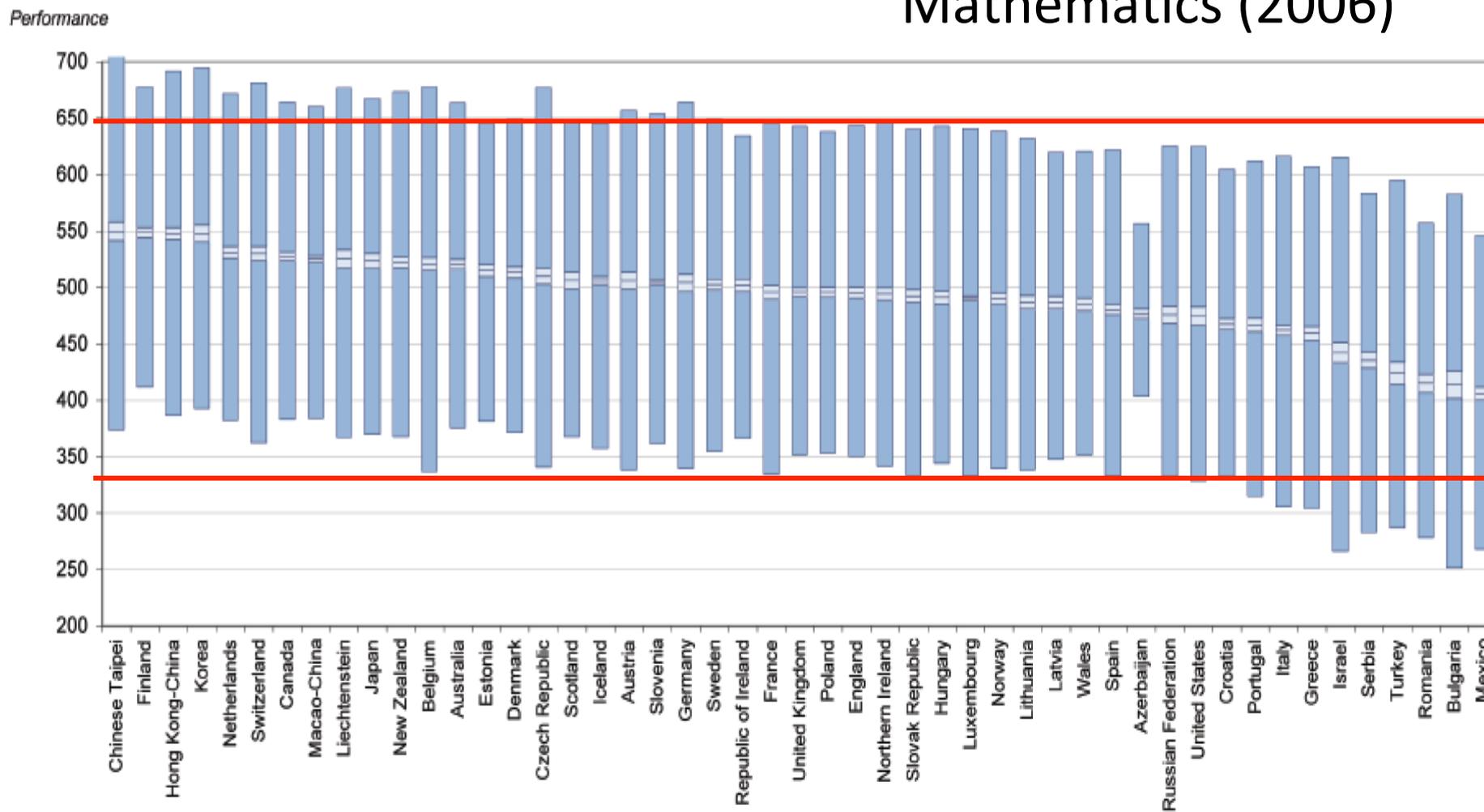
Most differences are not
due to the environment

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

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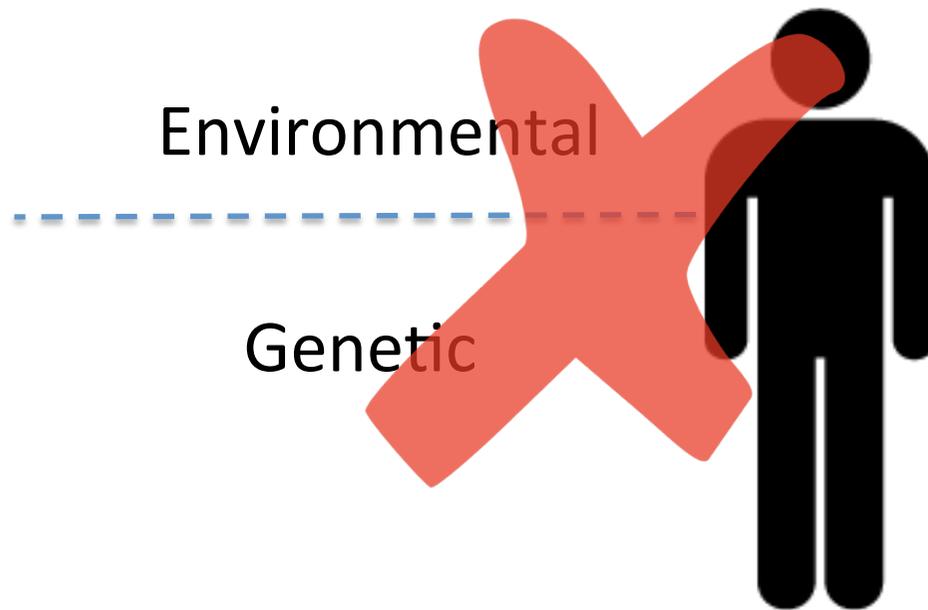
Personalised
learning

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

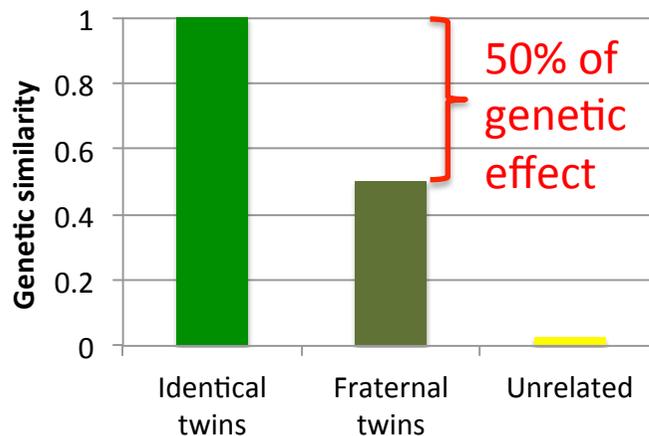
Identical twins	
Twin 1	Twin 2
56	50
34	32
21	25
83	78
..	..

Correlation = 0.9

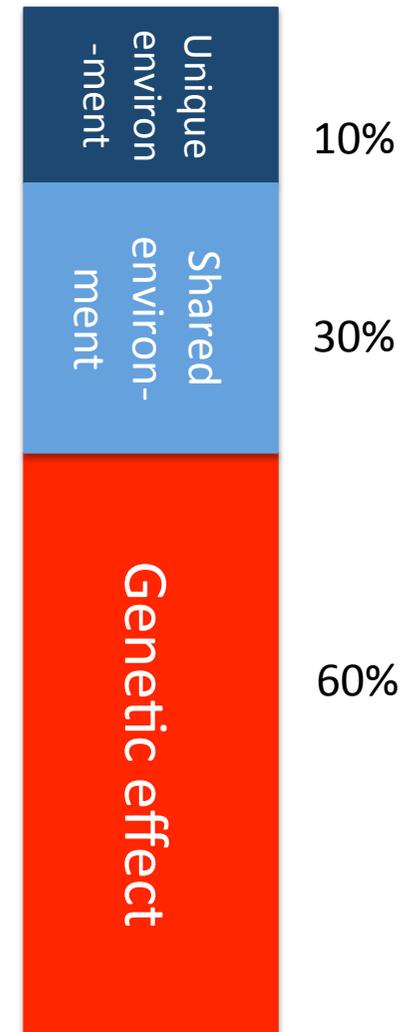
Non-identical twins	
Twin 1	Twin 2
25	40
52	92
35	33
43	38
..	..

Correlation = 0.6

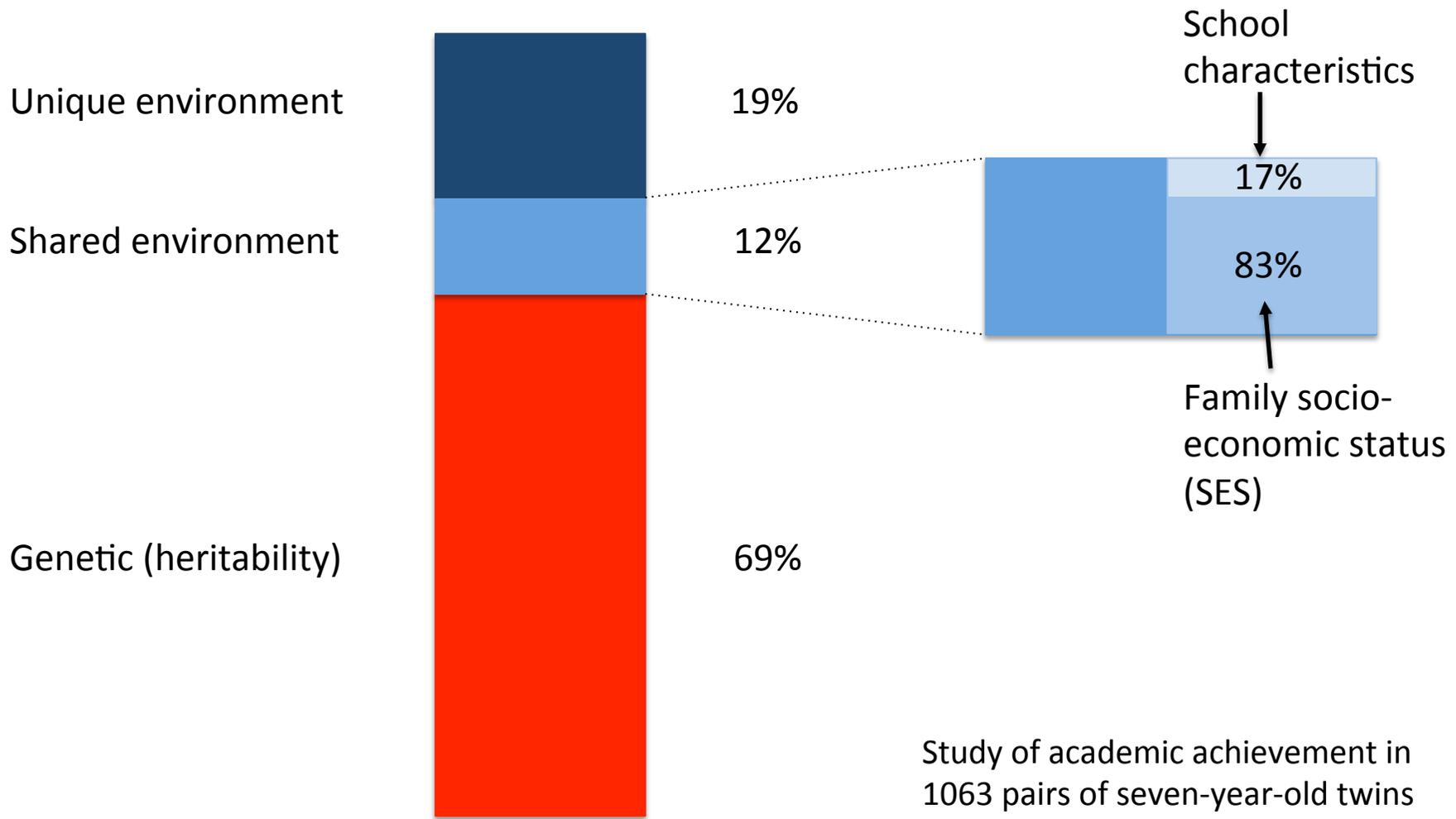
$$1 - 0.9 = 0.1$$



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

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

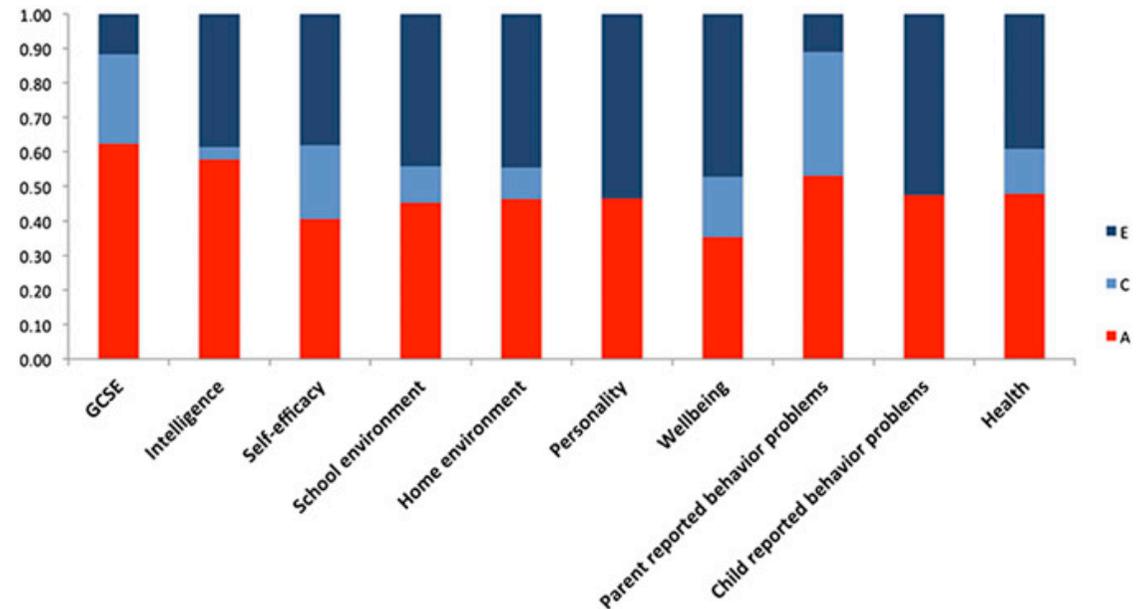


Fig. 1. Model fitting results for additive genetic (A), shared environment (C), and nonshared environment (E) components of variance for GCSE and nine predictors.

SCIENTIFIC REPORTS

OPEN

Pleiotropy across academic subjects at the end of compulsory education

Received: 03 February 2015

Accepted: 03 June 2015

Kaili Rimfeld¹, Yulia Kovas^{1,2,3}, Philip S. Dale⁴ & Robert Plomin¹

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

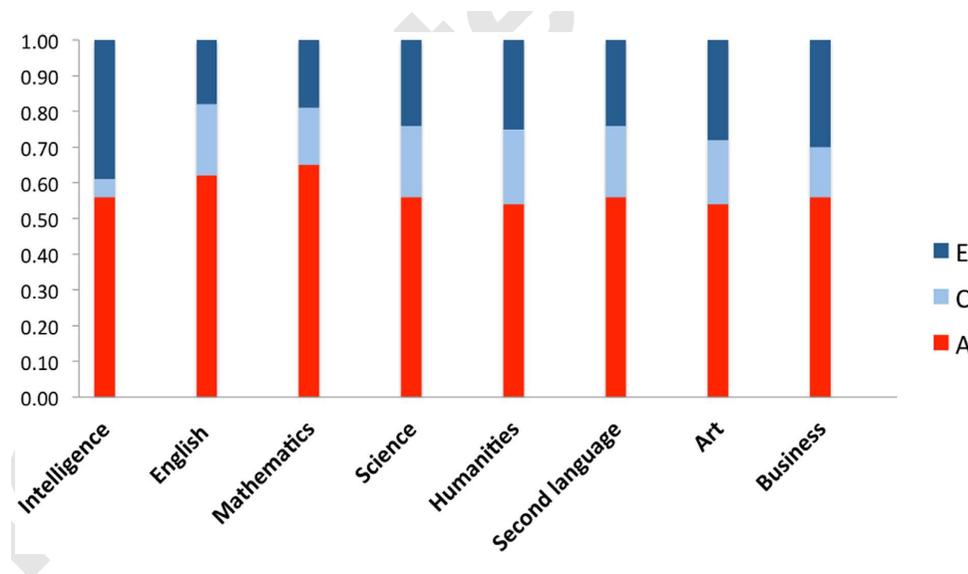


Figure 1. Univariate model-fitting results. A = additive genetic, C = shared environmental, E = non-shared environmental components of variance for GCSE exam grades and intelligence.

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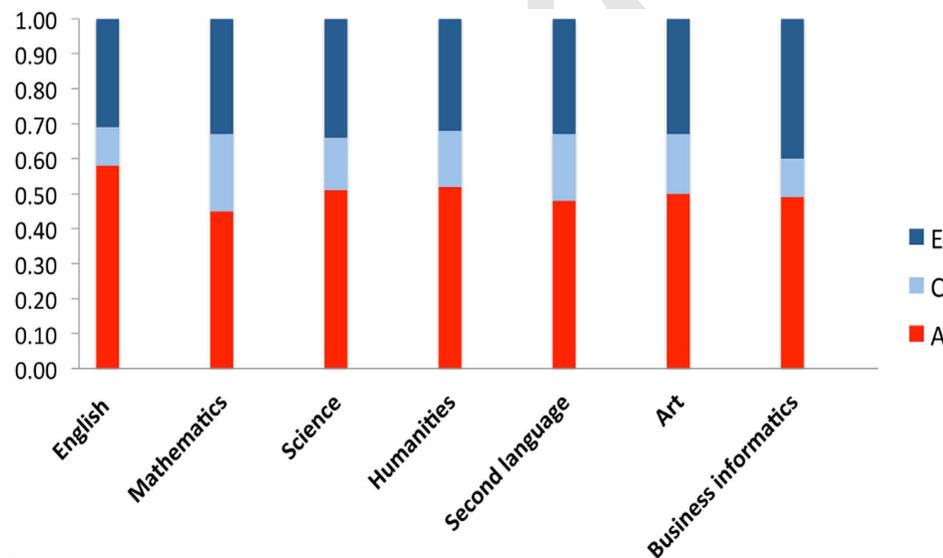
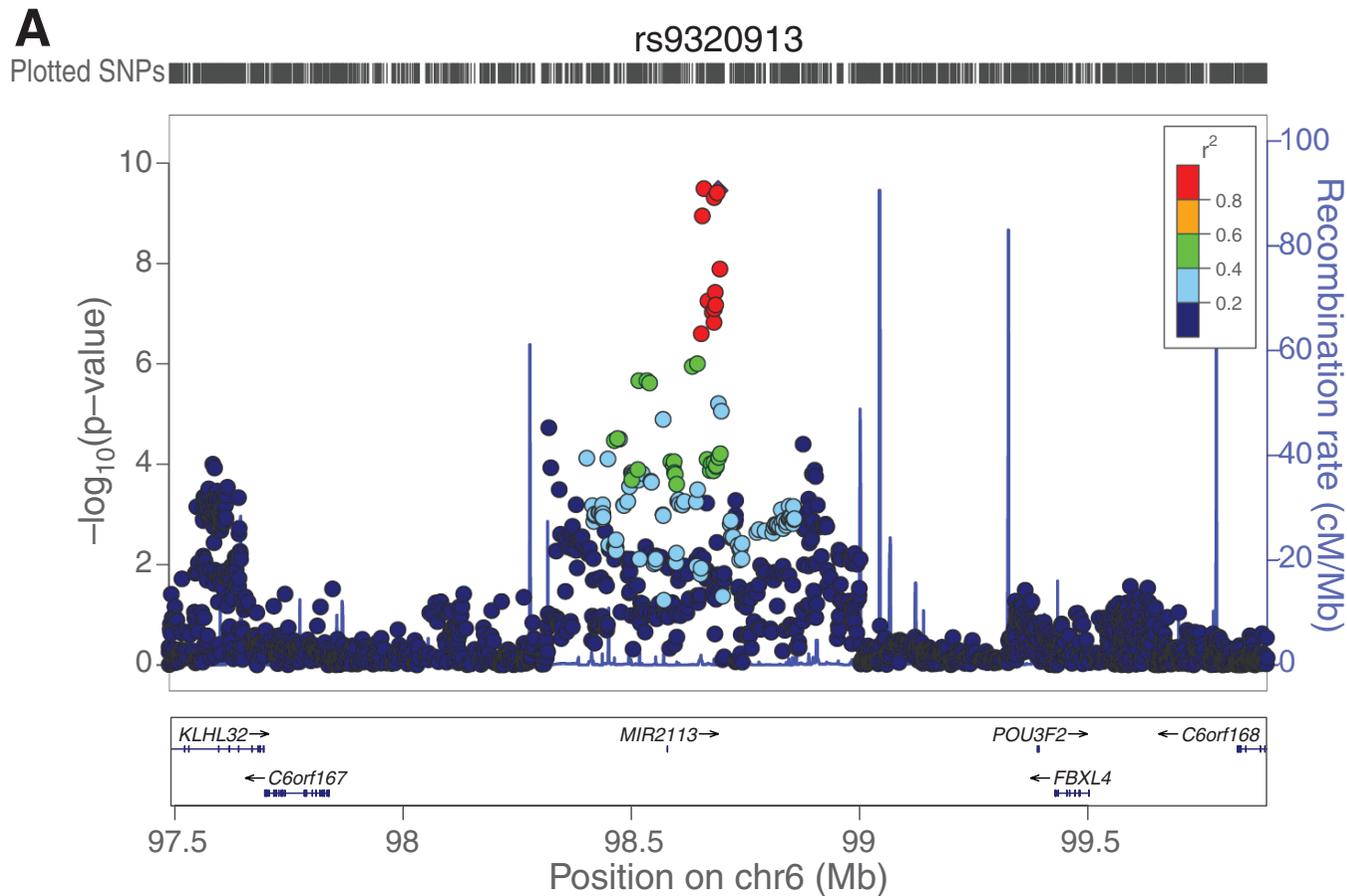


Figure 2. Univariate model-fitting results with GCSE exam grades corrected for intelligence. A = additive genetic, C = shared environmental, E = non-shared environmental components of variance.

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

Brain

- Cognition
- Motivation

But also:

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

SES effects on education

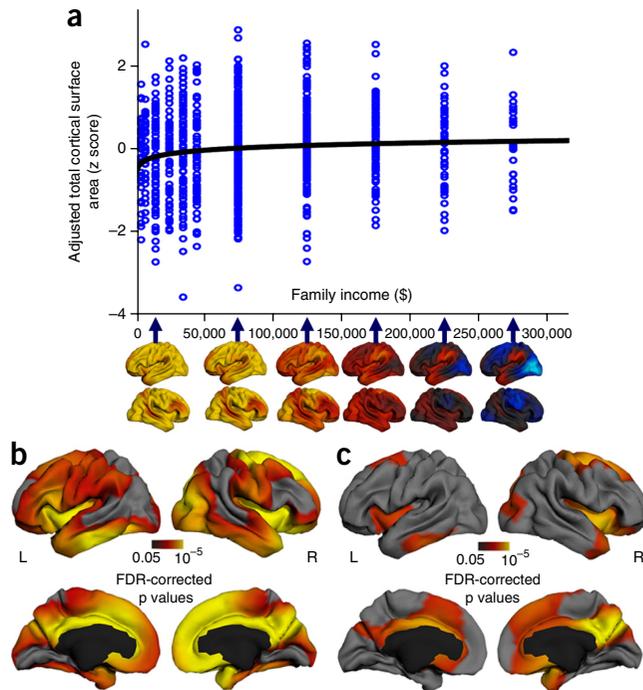


When you've made the SES effects go away,
the remaining differences are genetic

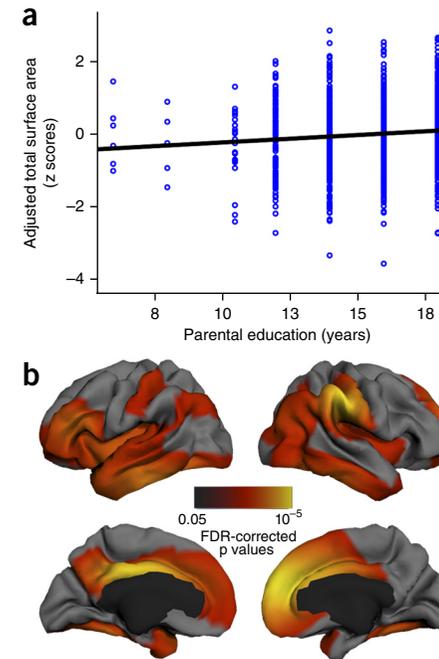
Family income, parental education and brain structure in children and adolescents

Kimberly G Noble^{1,2,32}, Suzanne M Houston^{3-5,32}, Natalie H Brito⁶, Hauke Bartsch⁷, Eric Kan^{4,5}, Joshua M Kuperman⁸⁻¹⁰, Natacha Akshoomoff¹⁰⁻¹², David G Amaral^{10,13}, Cinnamon S Bloss^{10,14}, Ondrej Libiger¹⁵, Nicholas J Schork¹⁶, Sarah S Murray^{10,17}, B J Casey^{10,18}, Linda Chang^{10,19}, Thomas M Ernst^{10,19}, Jean A Frazier^{10,20}, Jeffrey R Gruen^{10,21-23}, David N Kennedy^{10,20}, Peter Van Zijl^{10,24,25}, Stewart Mostofsky^{10,25}, Walter E Kaufmann^{10,26,27}, Tal Kenet^{10,27,28}, Anders M Dale^{8-10,29-31}, Terry L Jernigan^{10,11,12,29} & Elizabeth R Sowell^{4,5,10}

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,¹ Robert Gallop,² ...
Martha J. Farah¹

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

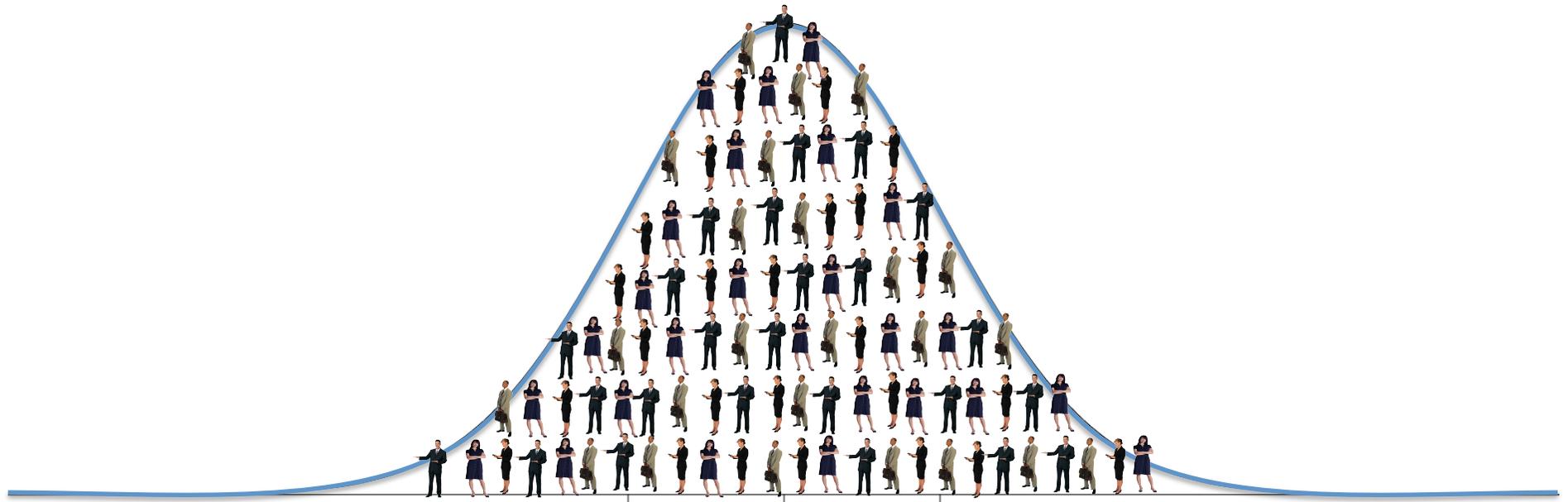


Billy

Reading: F
Maths: B

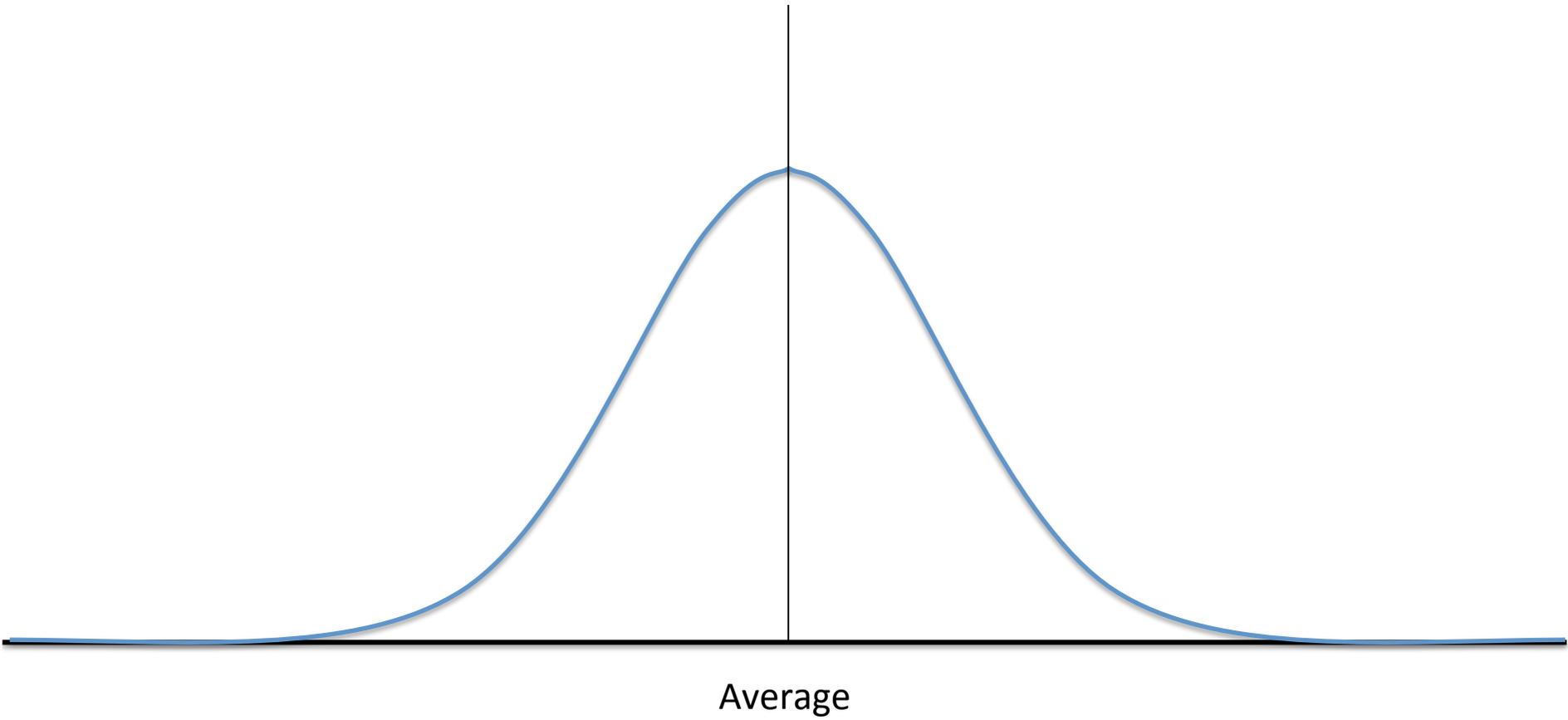
Billy really
struggles
with reading

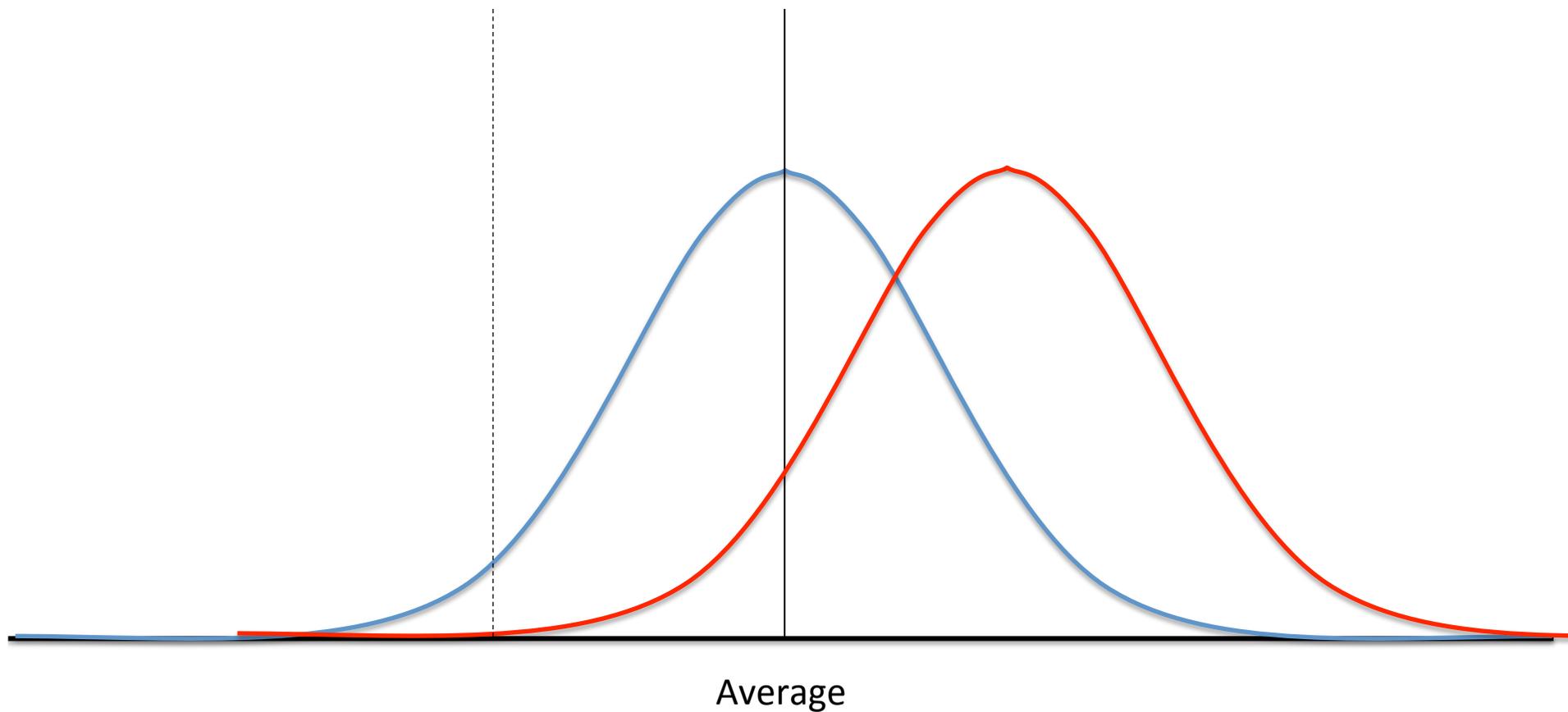
- “No child left behind”
- “Educate the best, forget the rest”
- “Too much too soon”
- “Every child should realise their potential”
- “The Finnish model” – minimum levels of literacy and numeracy in our society



Average

Normal Distribution

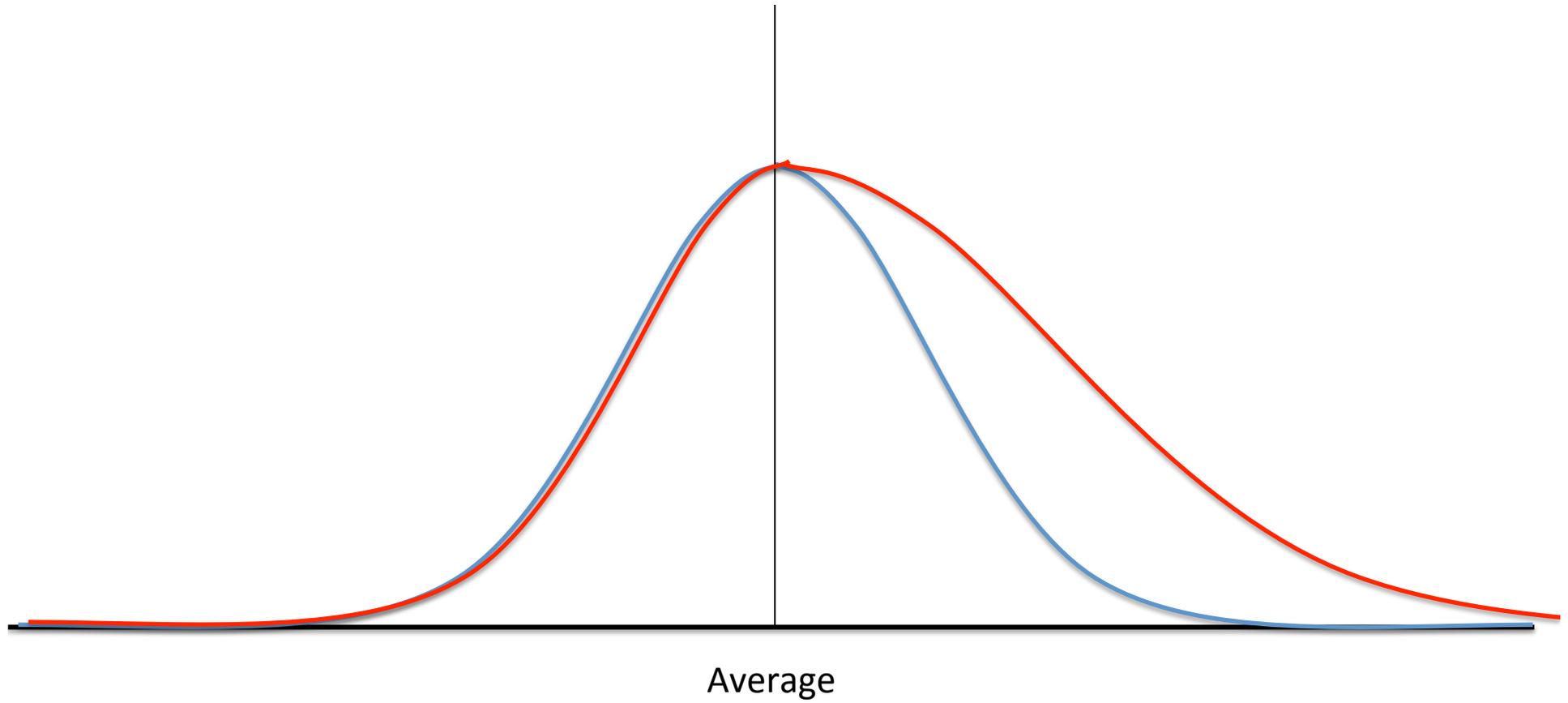




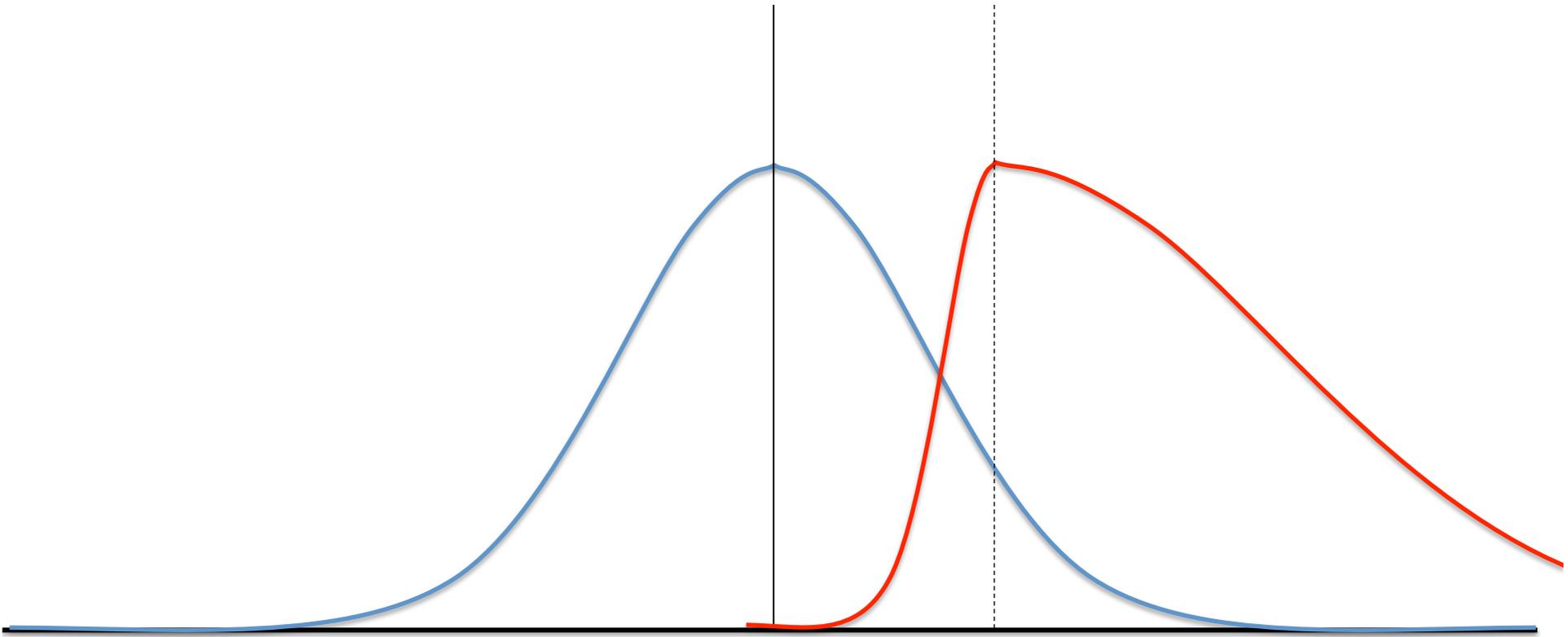
Finnish model – minimum levels of literacy and numeracy in society



No child left behind

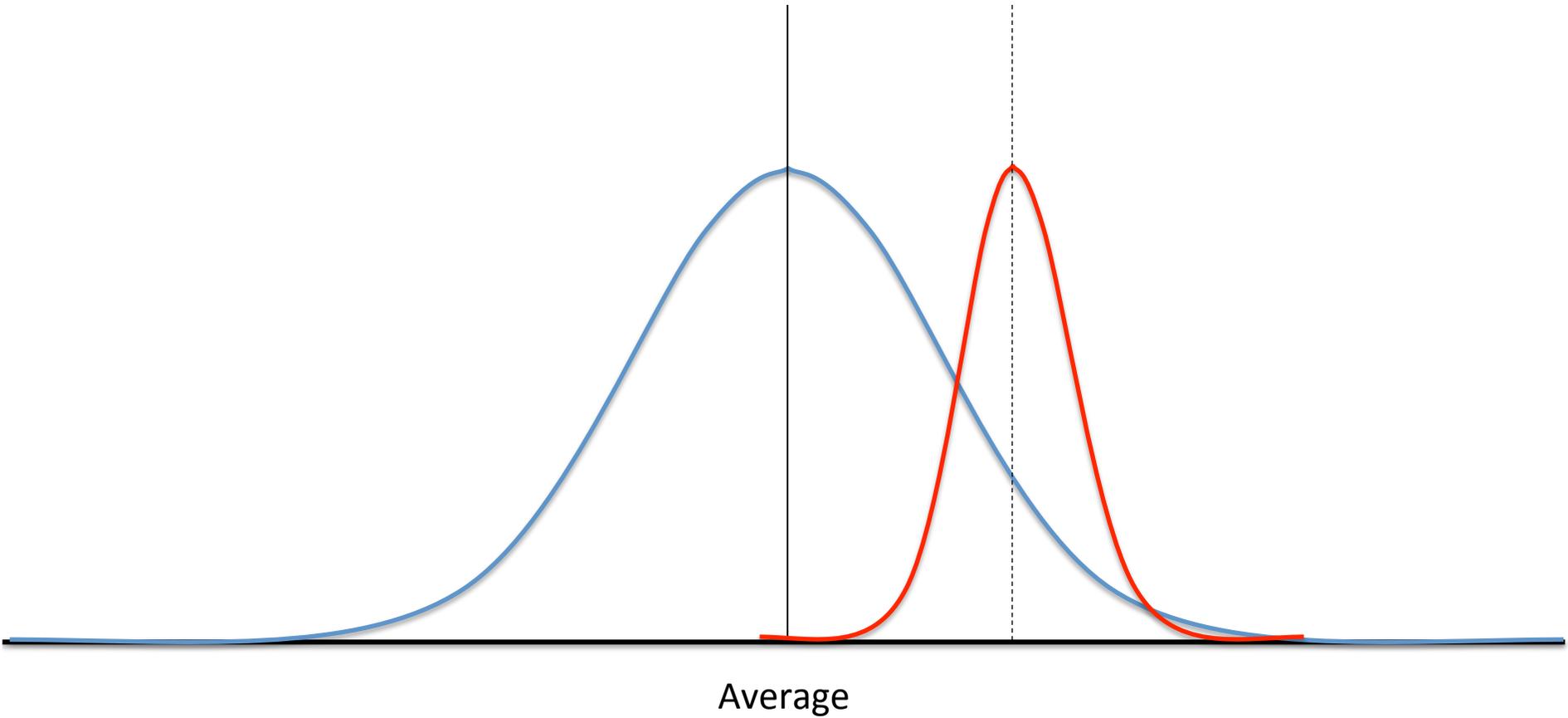


Educate the best forget the rest



Average

Panacea...?



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.

Share f t e

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)

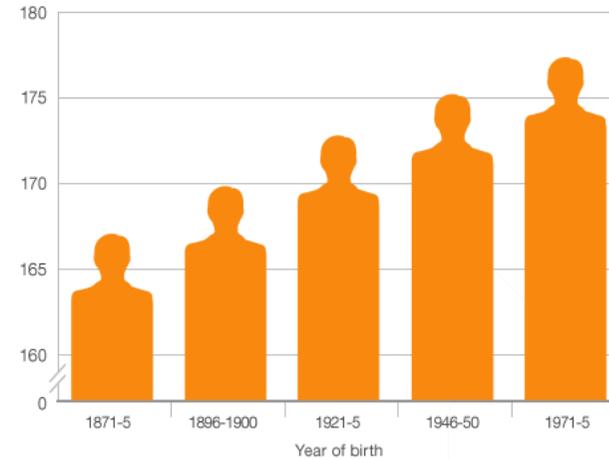
By Caroline Parkinson

Health editor, BBC News website

A century of growth

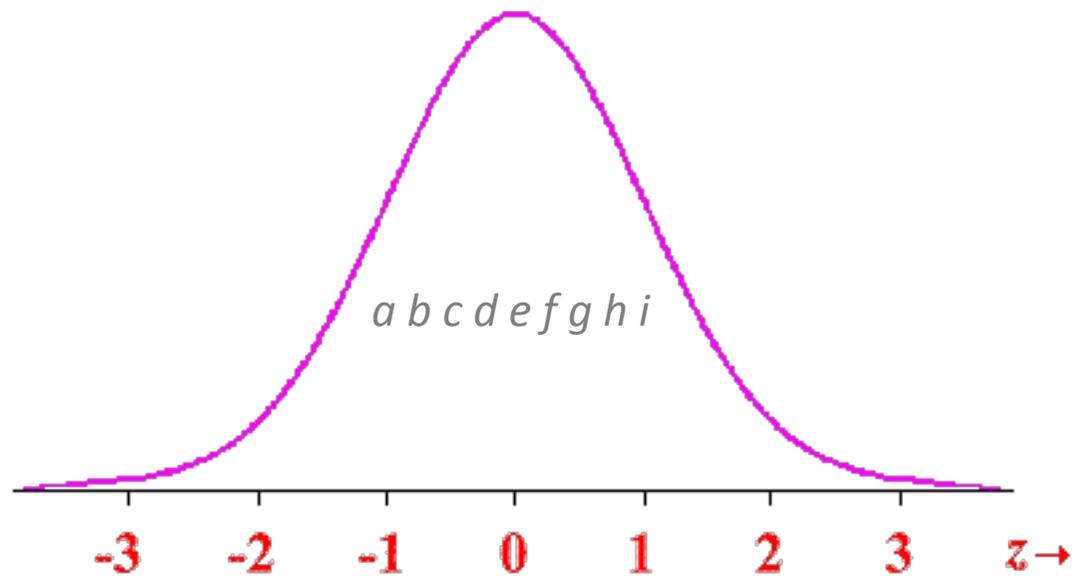
British males: Average height at age 21

Height cm

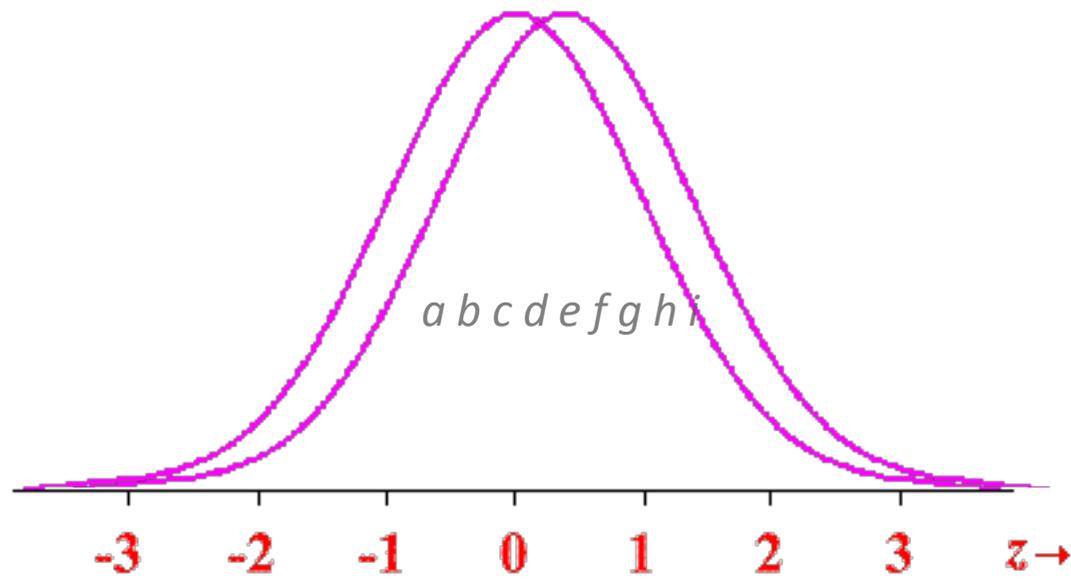


Source: Prof Tim Hatton et al, Oxford Economic Papers

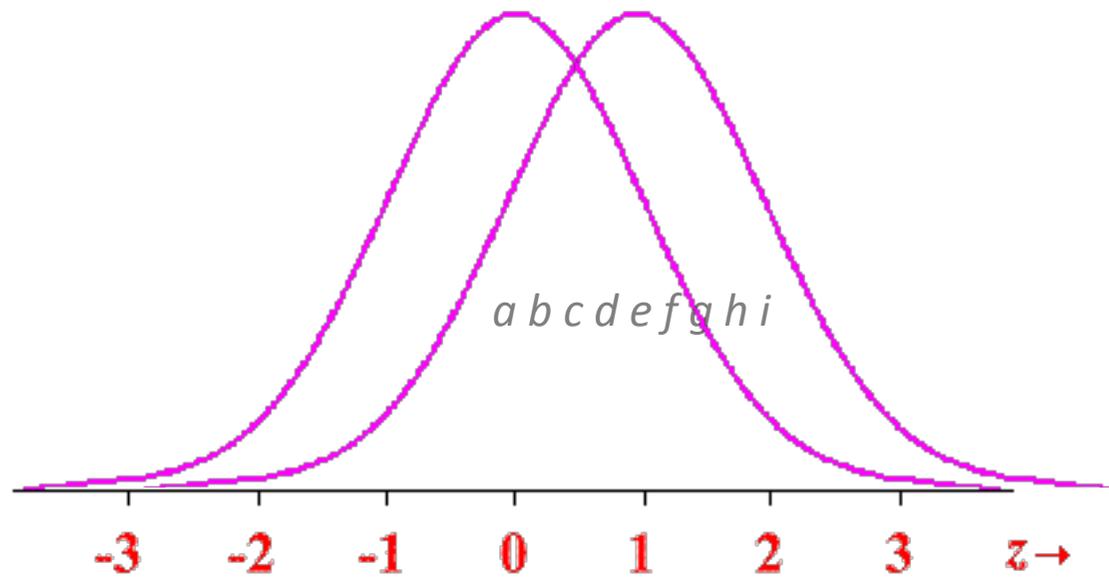
Relative vs absolute levels



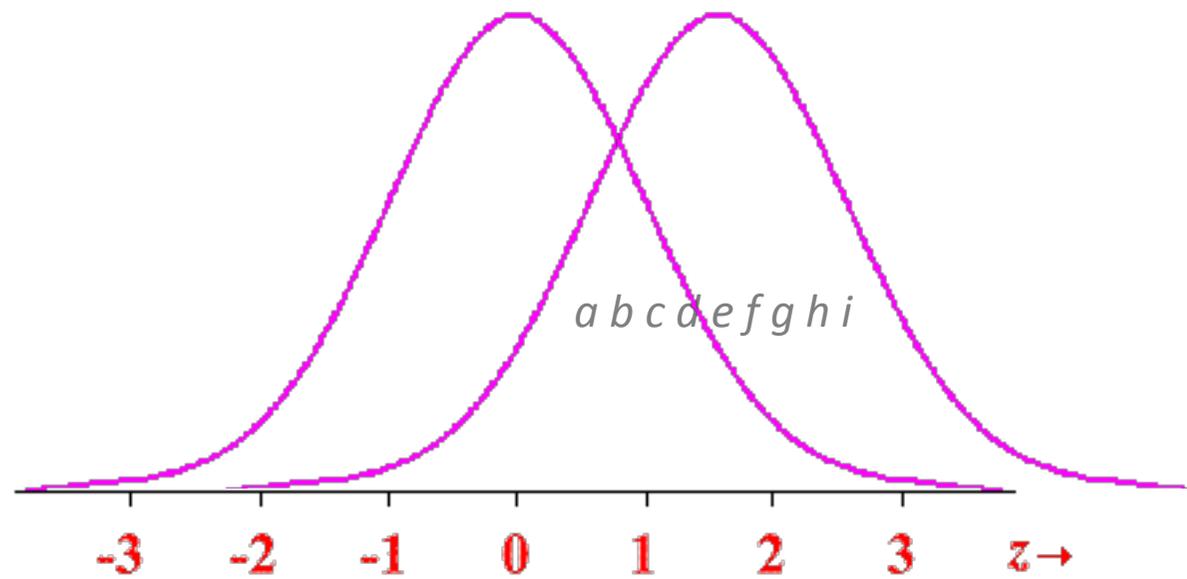
Relative vs absolute levels



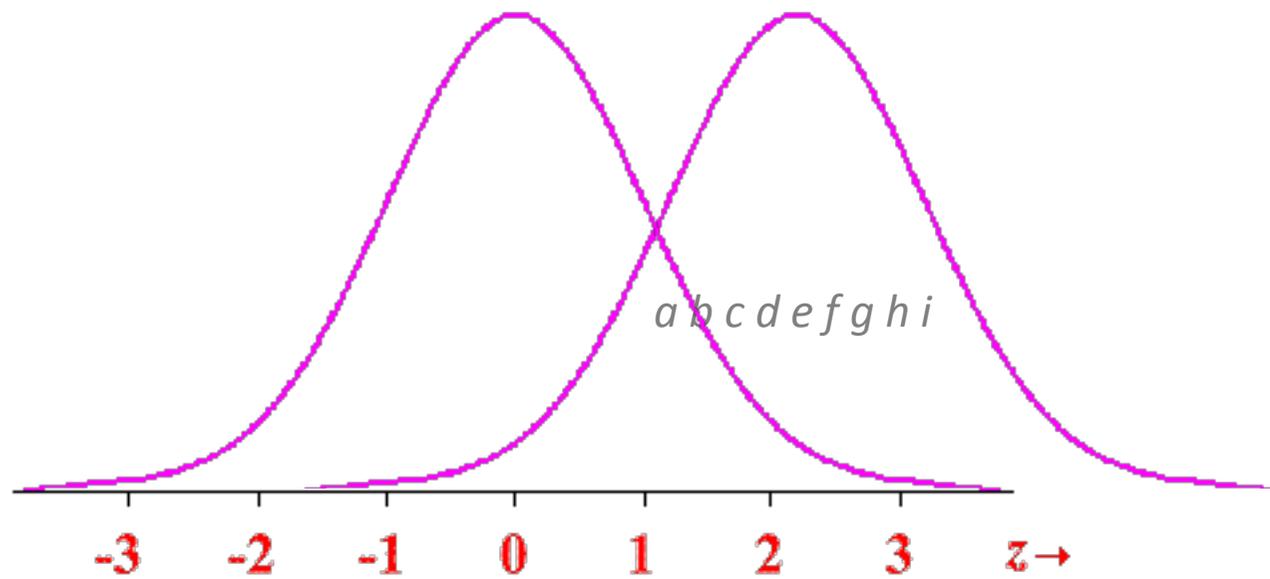
Relative vs absolute levels



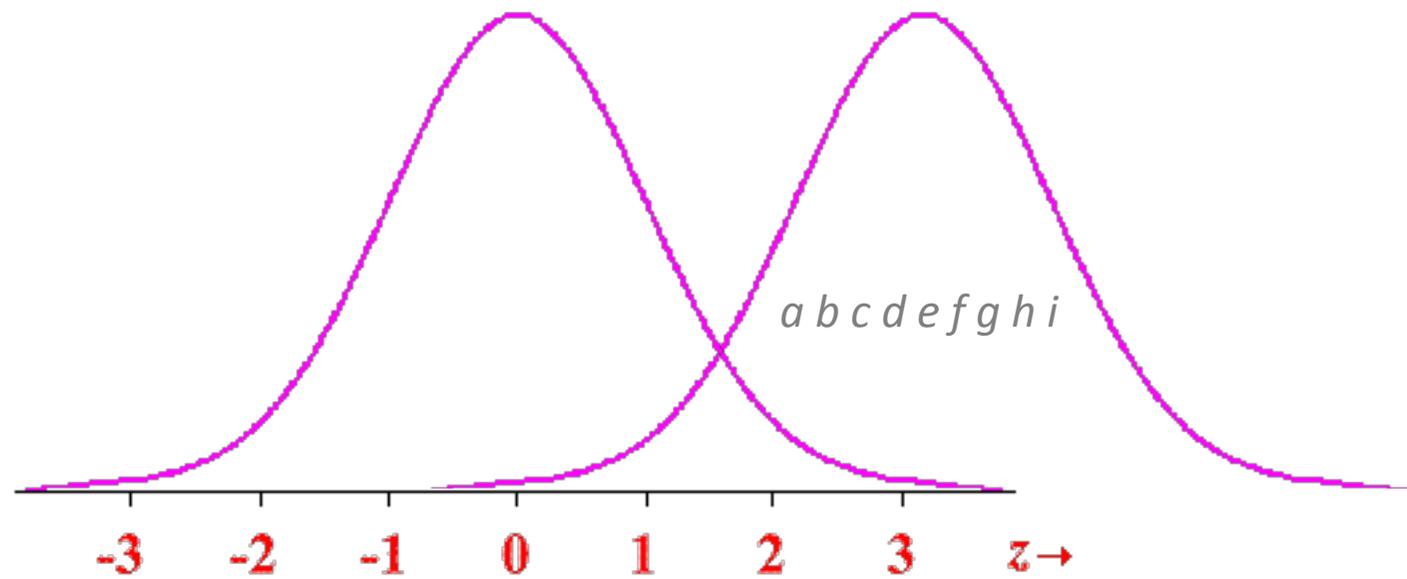
Relative vs absolute levels



Relative vs absolute levels



Relative vs absolute levels





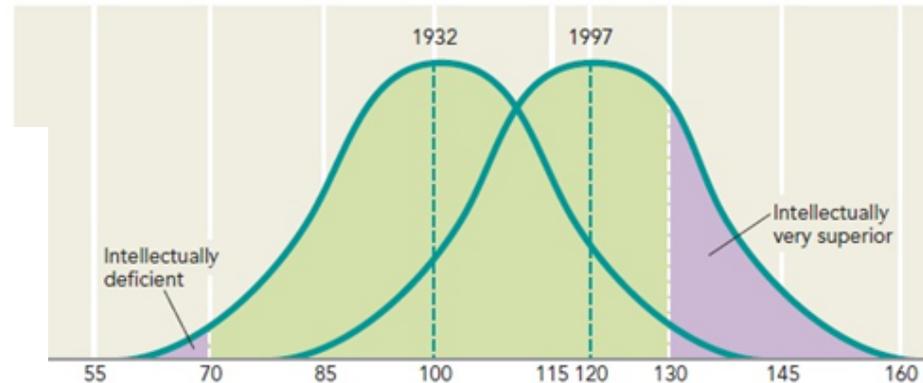
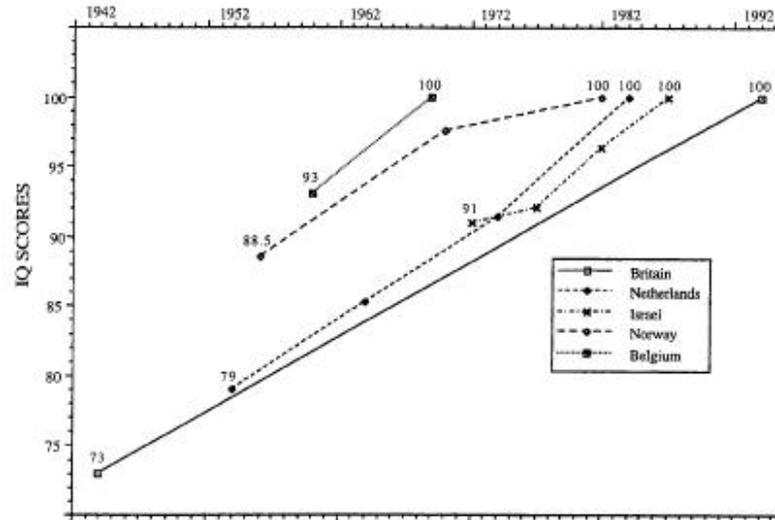
Are We Getting SMARTER?

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

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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,^{1*} A. D. Roehrig,² B. Soden Henler,¹ C. M. Connor,^{1,3} C. Schatschneider^{1,3}

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

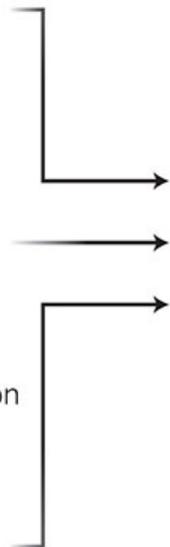
Major depressive disorder



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

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?

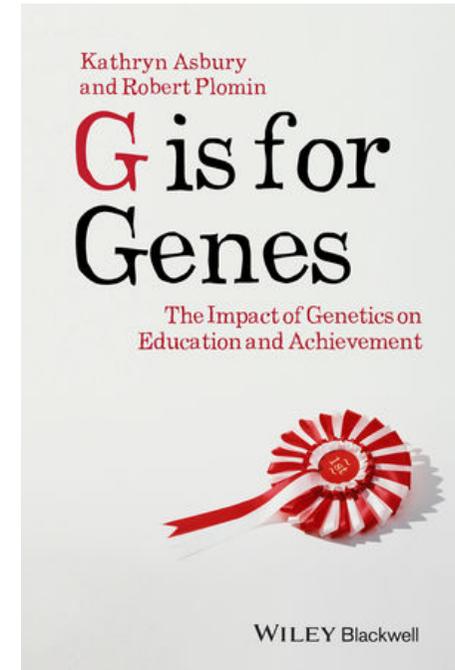
Personalised learning

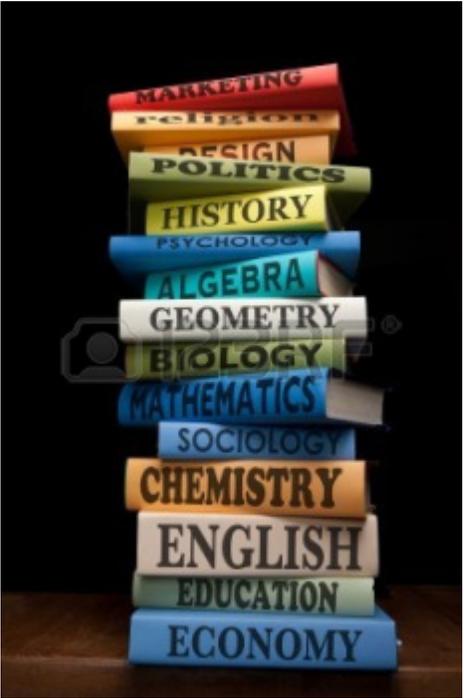


I think a genetic view suggests an active model of education. In genetics, we call this a gene-environment correlation. It's the idea that children create and modify and select environments that are correlated with their genetic propensities.

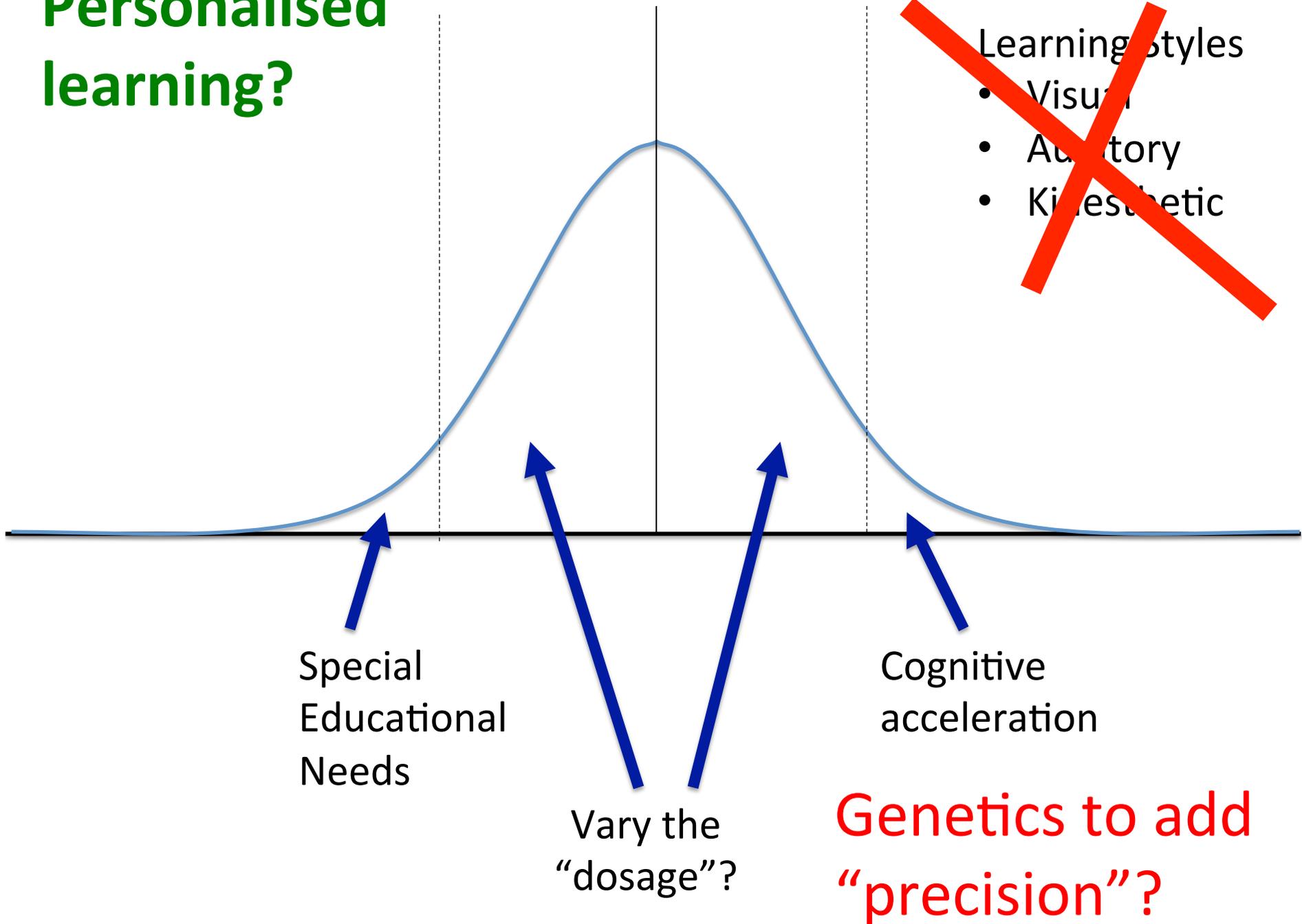


Professor Robert Plomin
King's College London





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)?

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Designing an intervention to address neuro-conduct problems was undertaken in this unemotional traits, a novel intervention and in a school for children with social methods design was used to investigate changes in the change process, alongside change and behaviour. Both qualitative externalising behaviour and improvement cognitive and affective processes. While types, associated changes in underlying potential value of neuroscience-informed

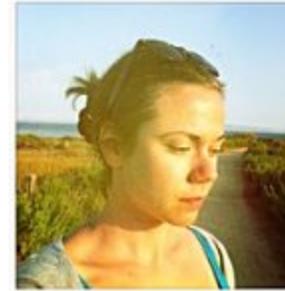
unemotional traits; SEBD; intervention; evaluation

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

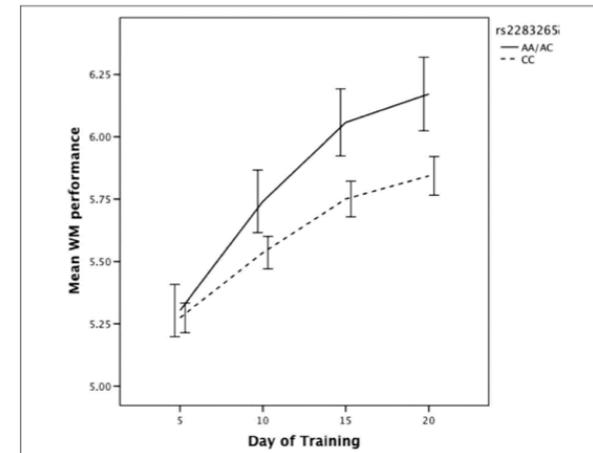
Stina Söderqvist, Hans Matsson, Myriam Peyrard-Janvid, Juha Kere, and Torkel Klingberg

Abstract

Studying the effects of cognitive training on better treatments, but it can also be a to factors important for brain plasticity and skills. In this study, we investigated how polymorphisms (SNPs) and ratings of intr associated to interindividual differences in working memory training. The study aged 7–19 years who were genotyped near eight candidate genes previously in COMT, SLC6A3 (DAT1), DRD4, DRD2, MAOA, LMX1A, and BDNF. Ratings on the inventory 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

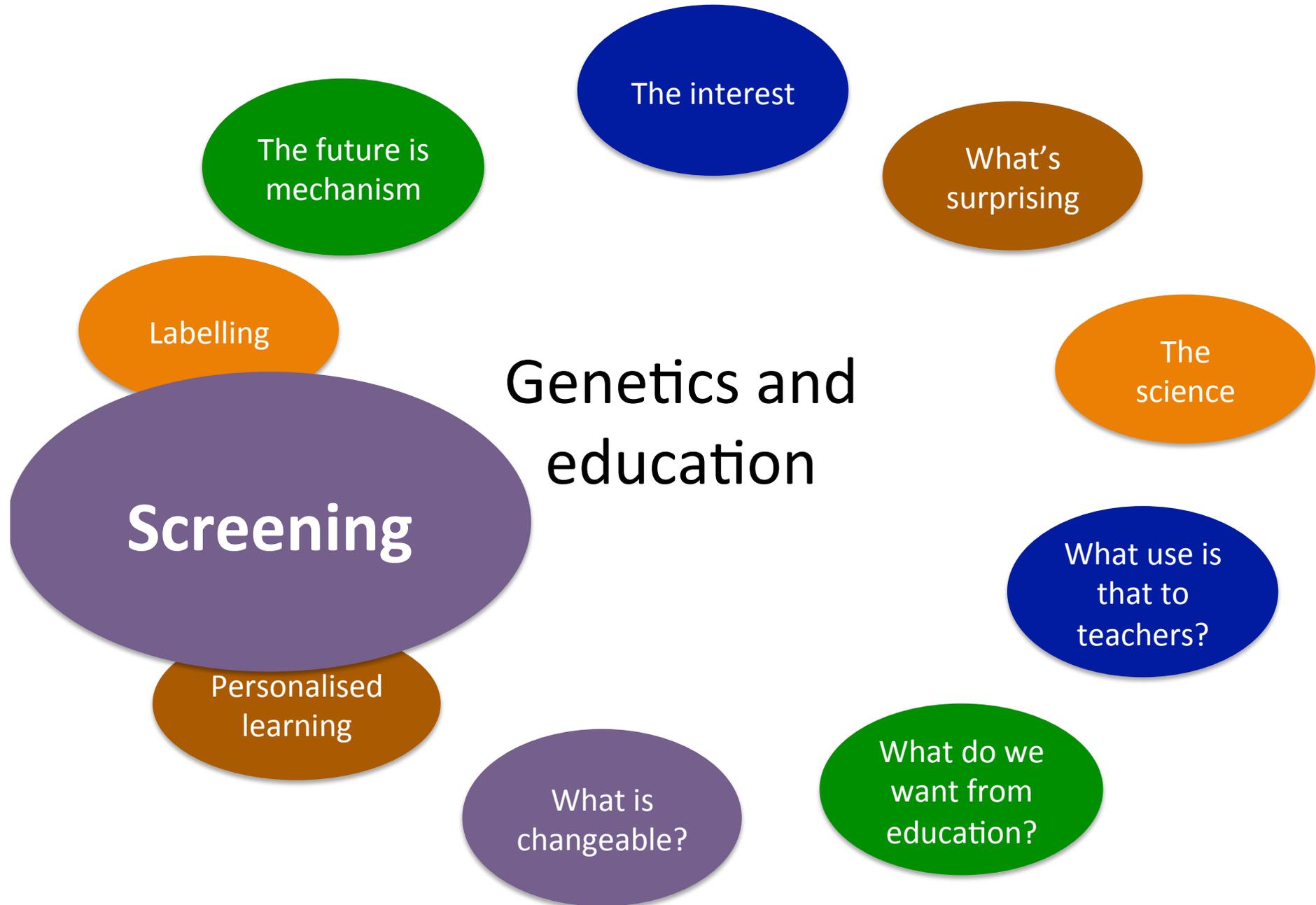
Some individuals respond better to working memory training

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?

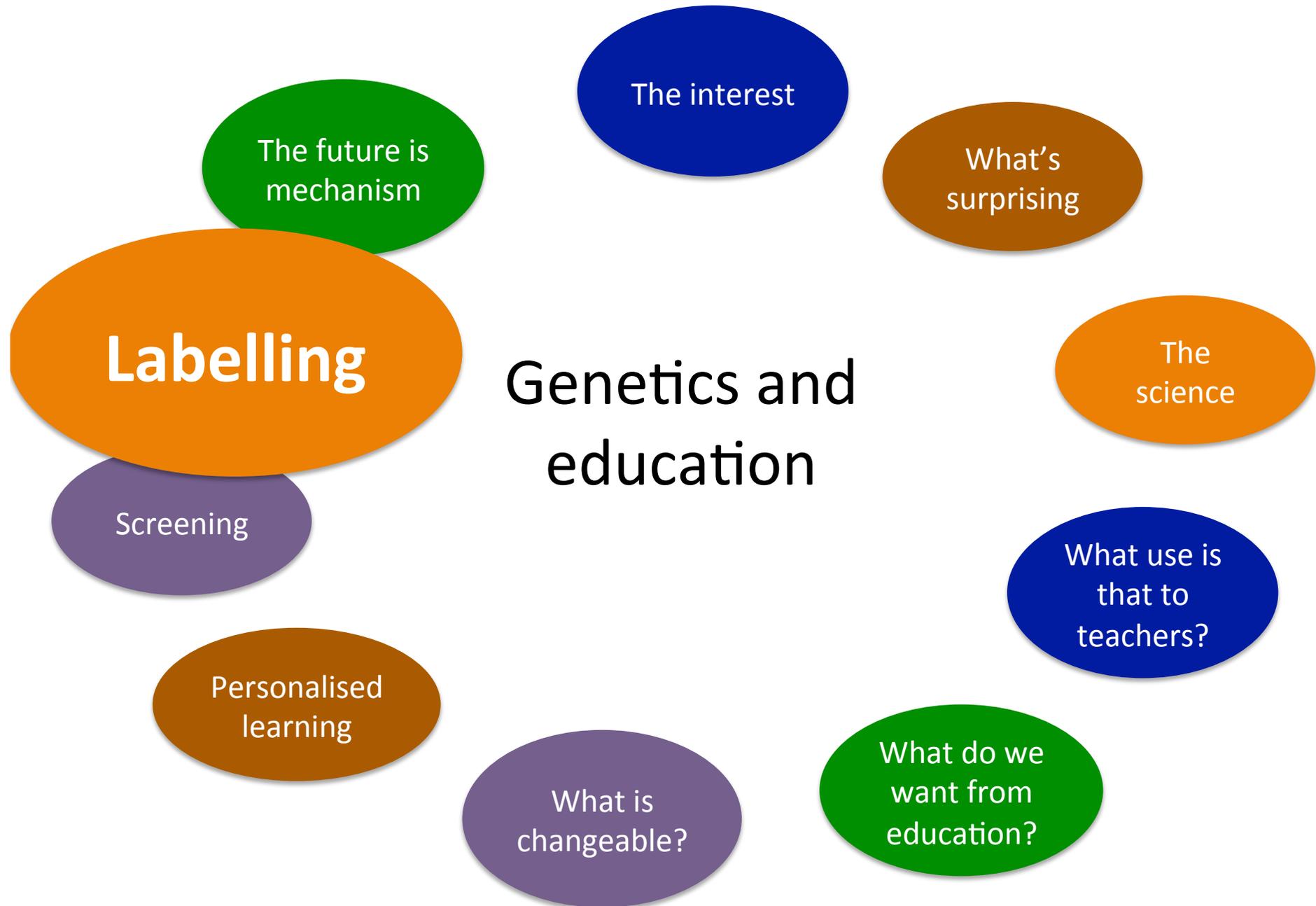
- Won't necessarily all be pedagogical or behavioural
- Could be health, diet, fitness, sleep, timing
- The potential drawback is that so many genes are involved (and so many environments)

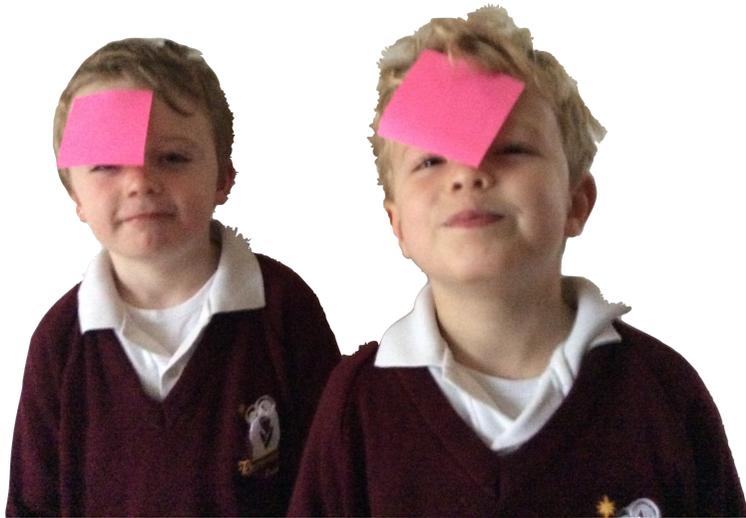


Does genetics point inevitably to screening?



- 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
mechanism**

interest

What's
surprising

Labelling

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education

The
science

Screening

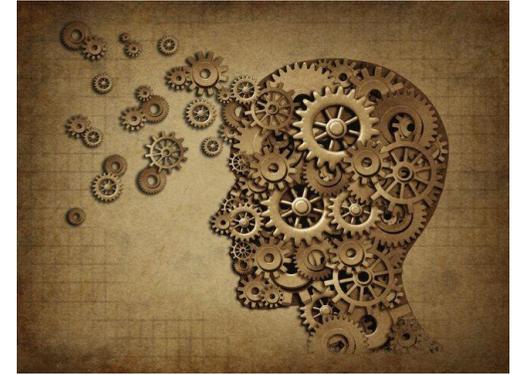
What use is
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Personalised
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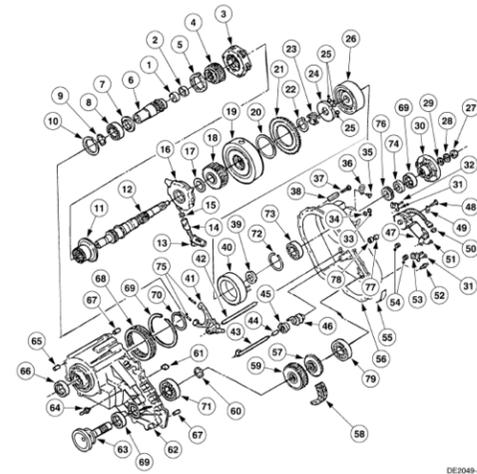
What is
changeable?

What do we
want from
education?

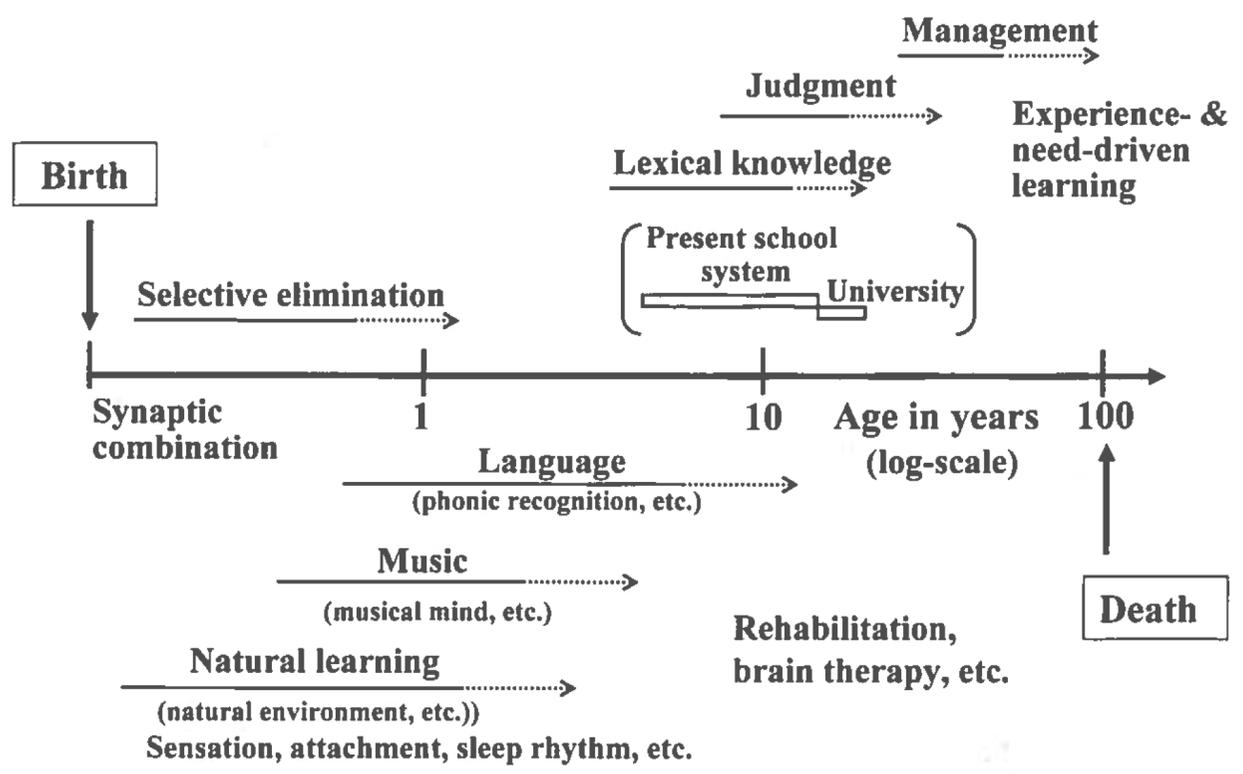
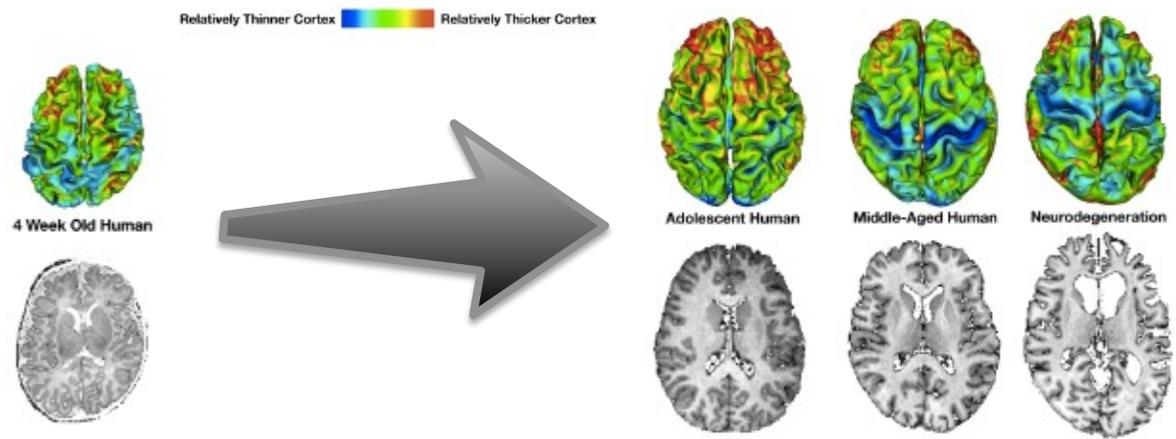
Educational neuroscience



- 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



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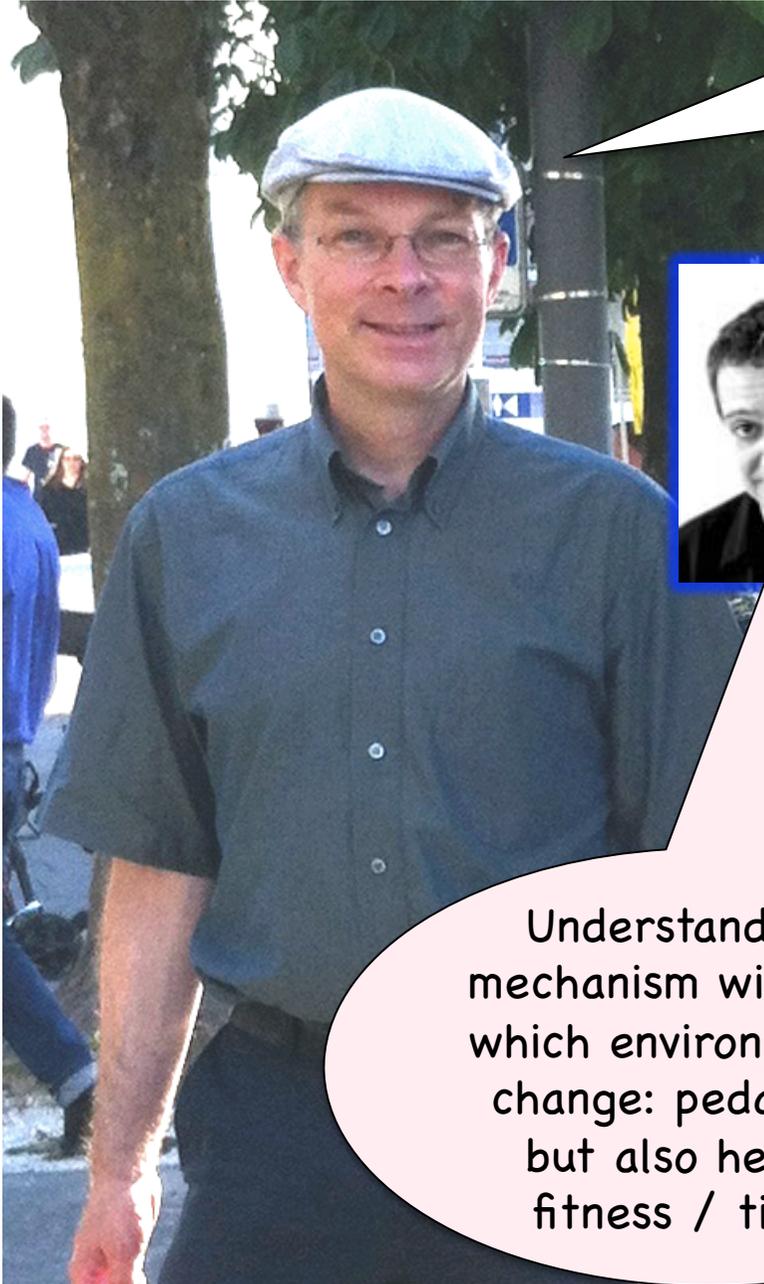


Source: Koizumi H., *Seizon and Life Sci.* (1998)

- 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

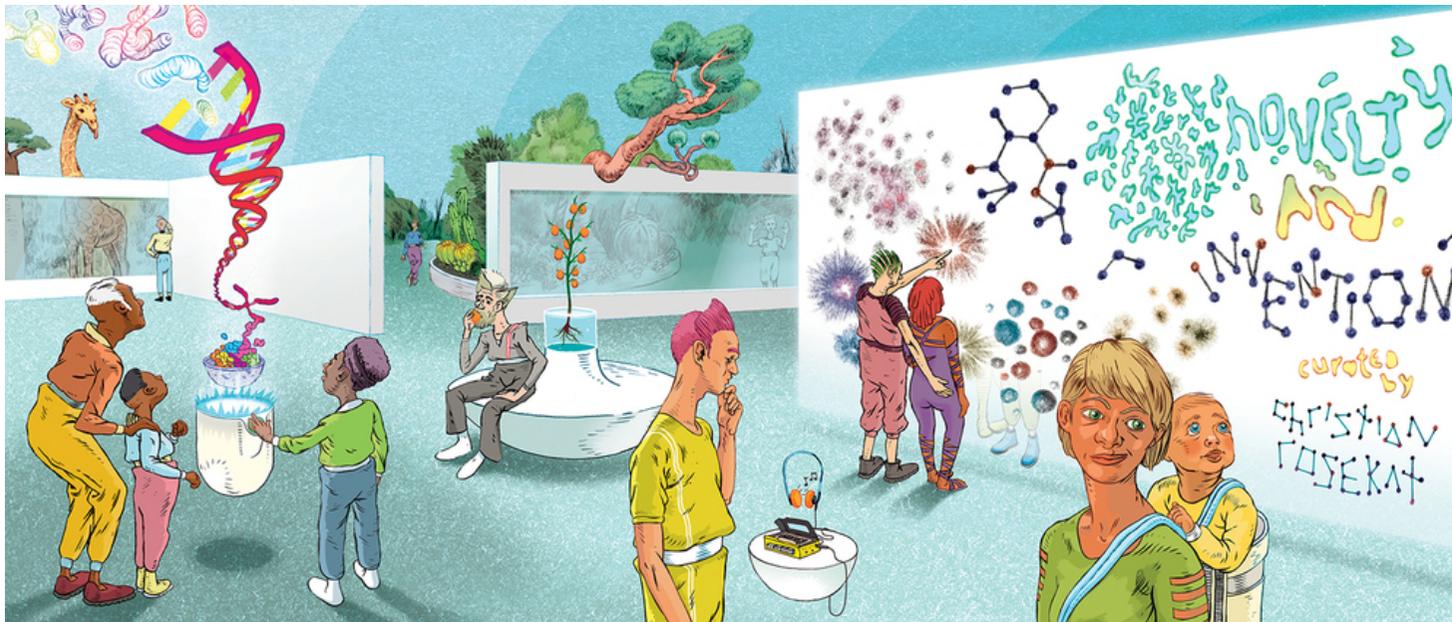
Society must determine the importance of overall population level vs. individual differences in education

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

