جامعة نيويورك أبوظبي

NYU | ABU DHABI

PSYCH-UH 2218: Language Science

Class 20: The critical period (and developmental disorders?)

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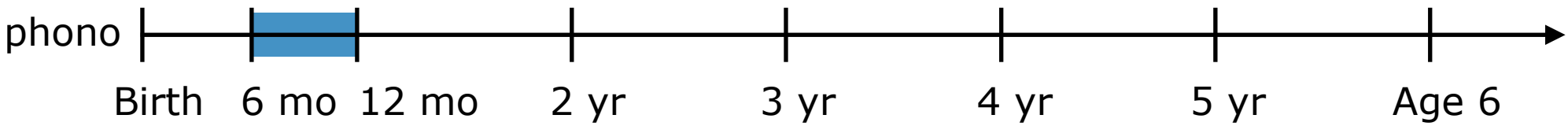
Psychology

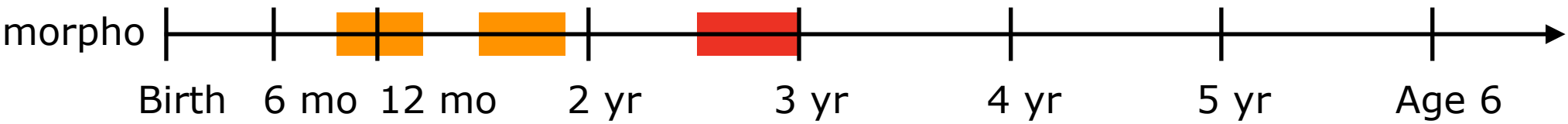
Quick review of the typical trajectory of native language acquisition

The timeline for typical first language acquisition

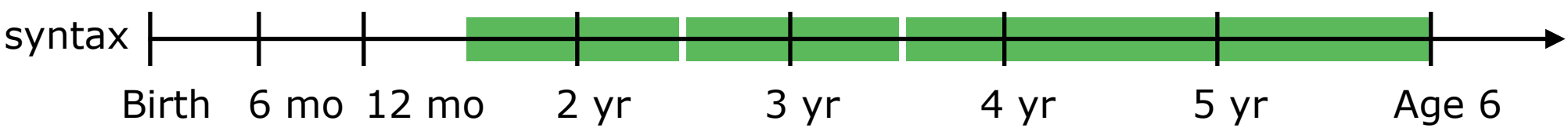
A timeline for first language acquisition:



Babbling begins at 6mo, becomes variable and language-specific by 12 mo.



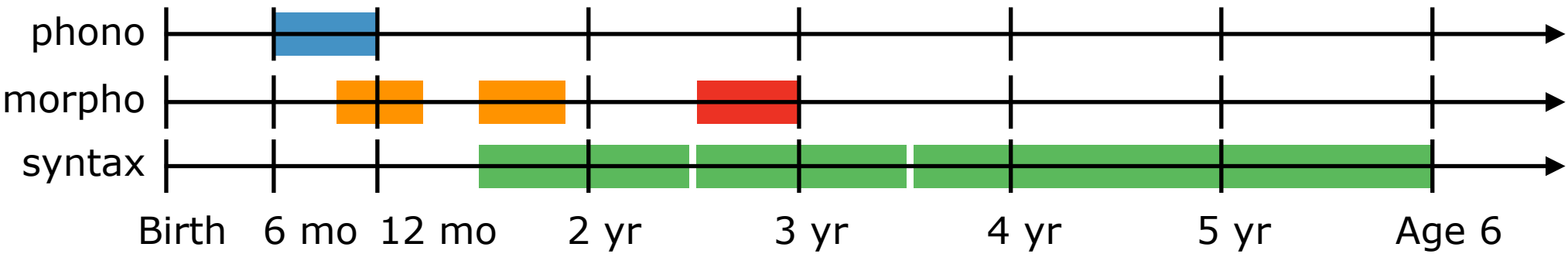
First words are produced between 10-15 mo. For many children, word learning accelerates dramatically around 18 mo. This is called the **vocabulary explosion**. Complex morphology appears 2.5-3 years.



Two word utterances at 1.5-2.5, function words at 2.5-3.5, and complex syntax after that.

Quick note: stages, not ages.

People like to map developmental milestones to specific ages. This is useful for doctors to help identify when there may be a developmental delay. But notice that many milestones tend to happen over a range of ages. There is variation in the specific age when children do specific things.

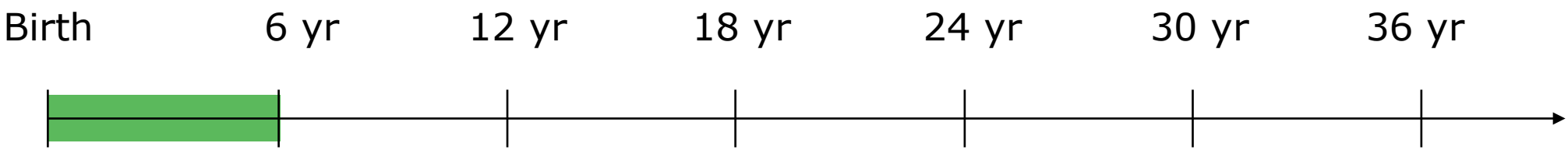


So, while the age ranges are useful, it is not typically useful for parents or doctors to worry if something isn't occurring by a specific age. Instead, we tend to focus on **stages** of language acquisition. Children should progress through each of these stages in this order. There is no variation (or very very limited variation) in the order of the stages. If they don't reach a stage at all, that is cause for concern. But simply hitting it later in the range is not necessarily a problem - that is what we expect with milestones that appear with some amount of variation.

But are there limits to when native languages can be acquired?

A look at a longer timeline

So far we've been looking at typical first language acquisition, which tends to happen between birth and approximately age 6.



But what happens if language acquisition is delayed? Or, what happens if somebody tries to learn a language later in life?

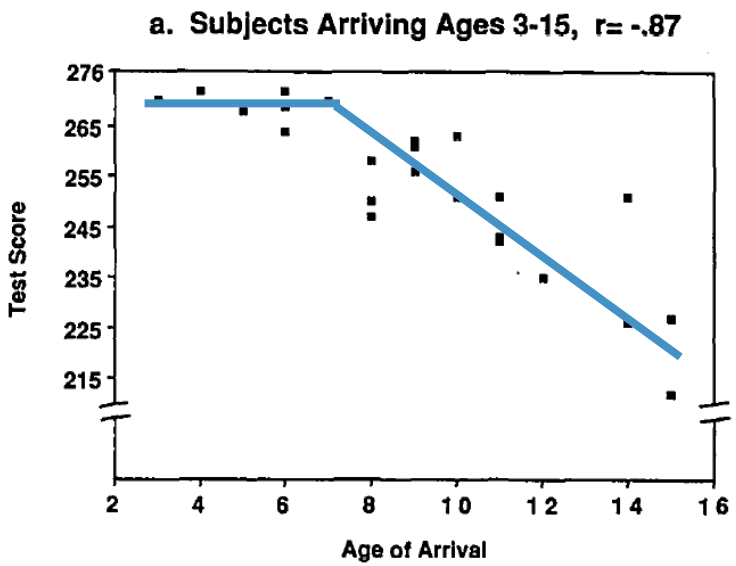
It turns out that there appears to be a change in the way that language is acquired later in life. People appear to be less successful at language acquisition after puberty.

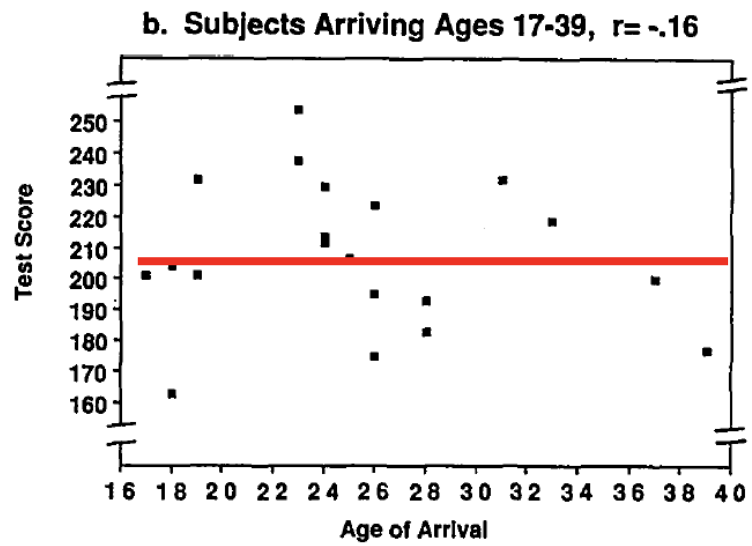
This suggests that there is a **critical period** for language acquisition (up to puberty). After the critical period is over, language acquisition changes.

Evidence from second language acquisition

One major piece of evidence for the critical period comes from studies of language acquisition later in life, such as the language acquisition of immigrants.

Johnson and Newport (1989) studied the English ability of US immigrants that arrived in the country at different ages. They made sure that every person in the study had the same amount of exposure to English, the same motivation to learn English, and the same amount of instruction in English. So the only thing that varied was how old they were when they first started learning English.

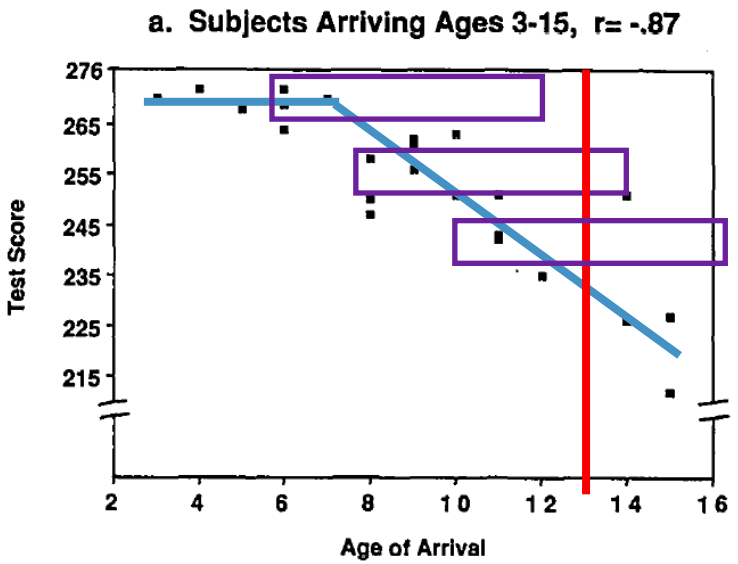




They found a steady decrease in success starting just before puberty, followed by no effect of age after age 16. This looks like a critical period!

Evidence from second language acquisition

The gradual decline from age 8 to 16 is compatible either with a true gradual decline, or a harder cutoff coupled with a process that unfolds over several years. So, that is still an open question:



To see this, imagine that native language acquisition takes 6 years. If someone starts at least 6 years before the window closes, they could reach complete nativity. If they start within 6 years, they may end up with partial success. And if they begin after the window closes, they may not reach native proficiency at all.

In general, discussions about a critical period tend to be agnostic about the nature of the cutoff. It could be a hard cutoff, or a gradual change. And, in any case, it is probably variable (just like the onset of puberty is variable). But the major finding is that there is a quantifiable shift in language proficiency when learning begins after puberty.

Evidence from language deprivation

Another major piece of evidence for the critical period comes from individuals who have been (tragically) deprived of language input.

Genie - discovered in LA in 1970 at age 13.

TLC documentary about her (it is an hour long):

https://www.youtube.com/watch?v=DD-pZ7LwL4A

She was kept in isolation until she was discovered at age 13. She had absolutely no language input.

After being discovered, various psychologists and linguists (and other doctors) worked with her to try to help her learn everything she missed.

By age 17 (four years later), she had a 5 year old's vocabulary, and could combine words to form (ungrammatical) sentences. But, as far as we know, she never progressed past this. She never acquired language completely:

Mama wash hair in sink.
Like go ride yellow school bus.

Man motorcycle have.
Father hit Genie cry long time.

Evidence from language deprivation

We can compare cases like Genie, where the child was discovered after the critical period began to close, to cases where the child was discovered in the middle of the critical period.

Isabelle - discovered in Ohio in 1938 at age 6.

Isabelle's mother was deaf, and because her parents (and society) did not understand deafness, her mother was uneducated, and generally hidden from society.

When Isabelle was born, her grandparents locked both her and her mother in a darkened room with no interaction with anyone else. Isabelle lived like this for 6.5 years. Finally her mother managed to escape, taking Isabelle with her. When they were discovered, Isabelle had no language.

Unlike Genie, Isabelle made remarkable progress. By age 9, she had learned an extensive vocabulary, had acquired complex syntactic constructions, and was scoring in the normal range on IQ tests.

The difference between Genie and Isabelle appears to be a difference in age of first exposure to language: Genie was after the critical period began to end, and Isabelle was in the middle of the critical period.

Pigeons and Creoles:
a consequence of the critical period!

What happens when two adults who don't speak the same language try to communicate?

From 1850 until 1946, thousands of laborers immigrated to Hawai'i to work on the sugarcane plantations.

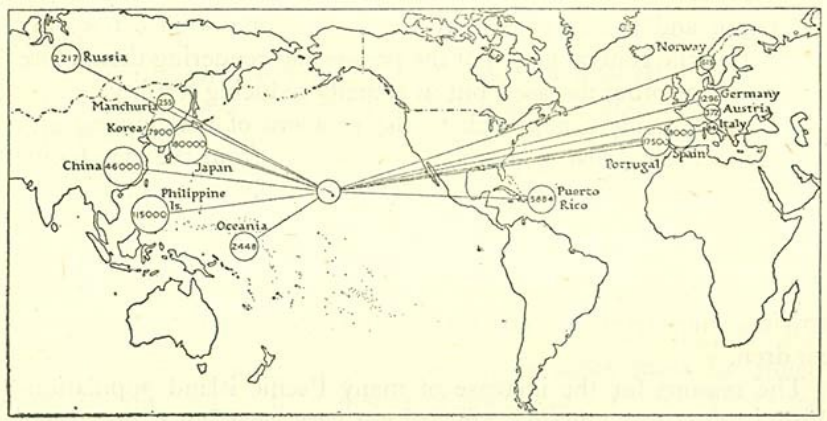


Fig. 98. Sources of immigrants into the Hawaiian islands, 1853–1933
Immigration from the U.S.A. and from the U.K. are not shown. The polyglot character of the population is clearly brought out. Based on A. W. Lind, *An Island Community : Ecological Succession in Hawaii*, p. 194 (Chicago, 1938).

These immigrants came from China, Japan, the Philippines, Korea, Germany, Norway, and Portugal (among many others).

The map on the left is from 1938, showing the relative sizes of the immigration waves.

These immigrants not only needed to find a way to communicate with each other, but they also needed to communicate with the English-speaking plantation owners and bosses, and the Hawai'ian-speaking native population.

c. 1900, Japanese immigrants

What happens when two adults who don't speak the same language try to communicate?

The immigrants that came to work on the plantations were adults. They were well past the critical period for language acquisition. So they were NOT able to learn each other's languages easily.

What they did instead was come up with a system for communication. This system had the following properties:

1. One dominate language contributed most of the words, although the other languages also contributed some words. We can call this language the **lexifier**. In Hawai'i, the lexifier was English.

2. The grammar of the communication system was a compromise mix of grammatical properties of the contributing languages. The result is a system that is grammatically simpler than any of the contributing languages.

3. The system had a lot of variation from speaker to speaker in terms of both word choice and grammar. Over time, the system becomes more and more rigid, but maintains some amount of individual variation.

This system was called **Hawai'ian Pidgin English**.

What is a Pidgin?

The system that the Hawai'ian immigrants created is just one specific example of a very general process that happens whenever large communities of adults who speak different languages are put into close contact.

Linguists call these communication systems **pidgins**. All pidgins share the same properties as Hawai'ian Pidgin English:

1. The lexifier is the language that contributes most of the vocabulary.

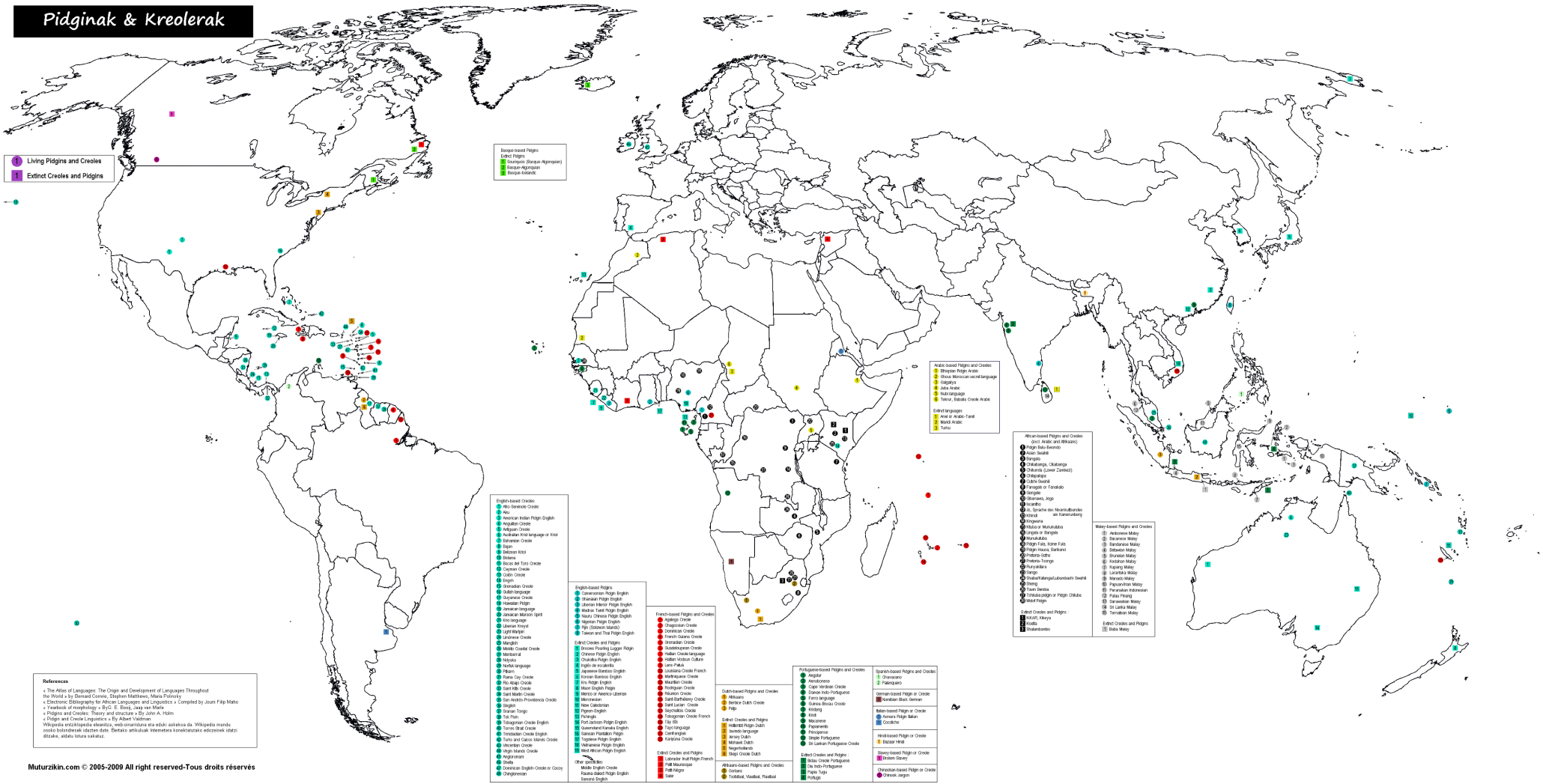
2. The grammar of the pidgin is a simplified compromise mix of grammatical properties of the contributing languages.

3. The pidgin shows variation from speaker to speaker in terms of both word choice and grammar. Over time, the pidgin becomes more and more rigid, but maintains some amount of individual variation.

4. There are no native speakers of pidgins. Pidgins are only spoken by adults who speak other languages natively. Pidgins are learned by adults who are too old to learn a new language natively.

Pidgins are very common

Pidgins arise anytime two communities of adult speakers need to communicate. This map (http://www.muturzikin.com/cartepidgin.htm) documents both living and dead pidgins across the world.



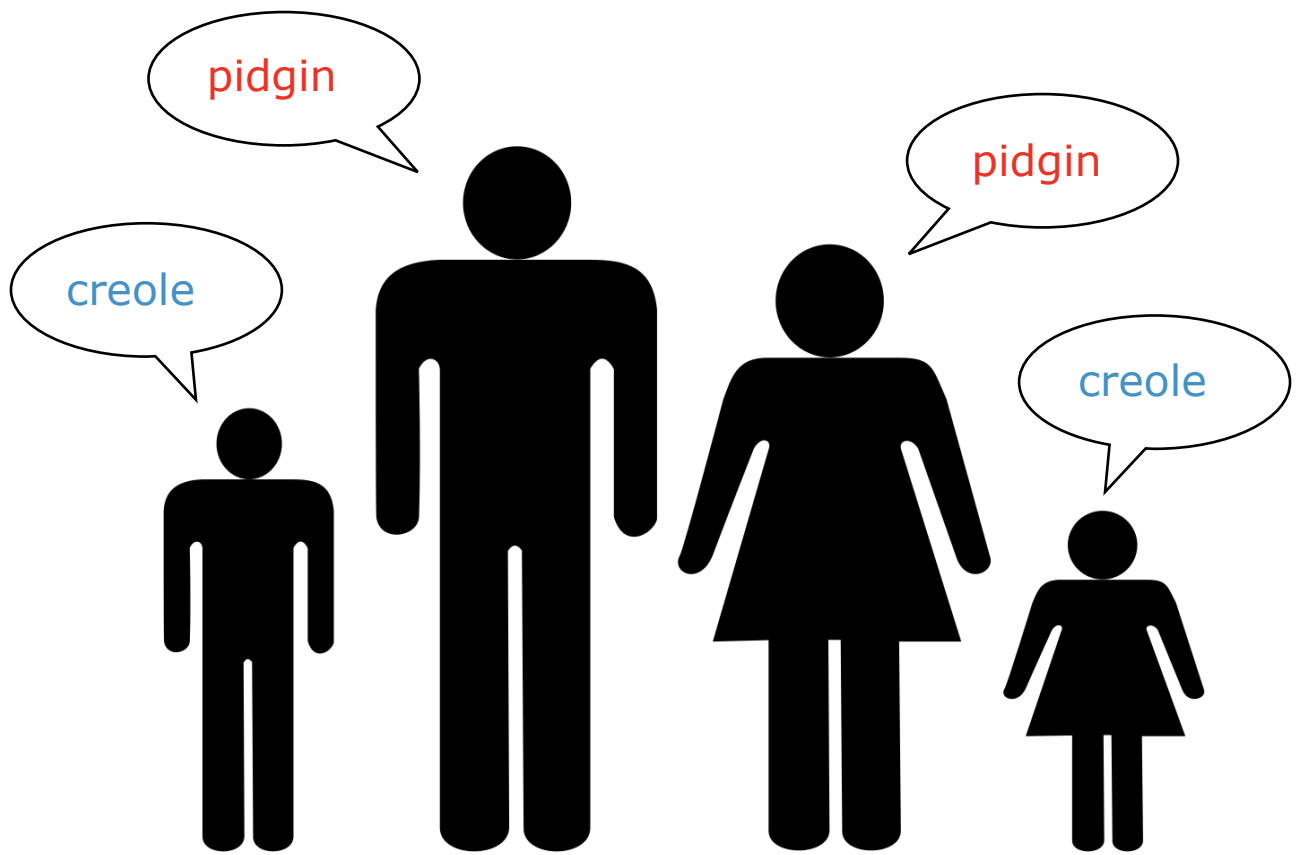
What happens when children are exposed to a pidgin as their first language?

Remember, one of the critical properties of a pidgin is that there are no native speakers of pidgins. Pidgins are created and spoken by adults, who are past the critical period for language acquisition.

So what happens when children are exposed to a pidgin during their critical period for language acquisition? They acquire, and in fact create, a creole!

Properties of creoles:

1. Larger vocabulary
2. Regular rules for phonology, morphology and syntax.
3. Very little variation between speakers of the creole.
4. Is the native language of the speakers.



Hawai'ian Creole English

By the early 1900s, the large immigrant communities in Hawai'i had raised a generation of children who were exposed to Hawai'ian Pidgin during their critical period for language acquisition. Those children created Hawai'ian Creole English.

HCE is still spoken by approximately 600,000 people in Hawai'i today. Speakers of the language often call it Hawai'ian Pidgin English because that was its name when it was a pidgin. But, technically, it is now a creole:

https://www.youtube.com/watch?v=O7X9AAeDCr4

Here are some properties of HCE:

Phonology:

The "th" sound in General American English is replaced by a "t" or "d" (depending on whether it is voiced or voiceless).

think → tink
this → dis

The "r" sound after vowels is deleted.

better → beta

Hawai’ian Creole English

Lexical items and syntactic rules:

The past tense is indicated by adding the word ‘wen’ (from ‘went’) before the verb:

General American English: They painted his skin.

Hawai’ian Creole English: Dey wen pein hiz skin.

The future tense is indicated by adding the word ‘go’, ‘gon’, or ‘gonna’ before the verb:

GAE: You will turn in your paper late.

HCE: Yu gon trn in yaw pepa leit.

Where GAE would use the word ‘to’ after a verb, HCE uses the word ‘fo’:

GAE: Everybody comes to see that house.

HCE: Eribadi kam fo si daet haus

Hawai'ian Creole English is a full language, not a sub-standard form of English

Because HCE sounds so similar to General American English (GAE), many people, including native speakers of HCE, incorrectly believe it is a sub-standard form of English.

But as we just saw, it is a rule-based language, just like GAE. It has a set of phonemes that are similar but different from GAE, and it has a set of lexical items and syntactic rules that are similar to but different from GAE.

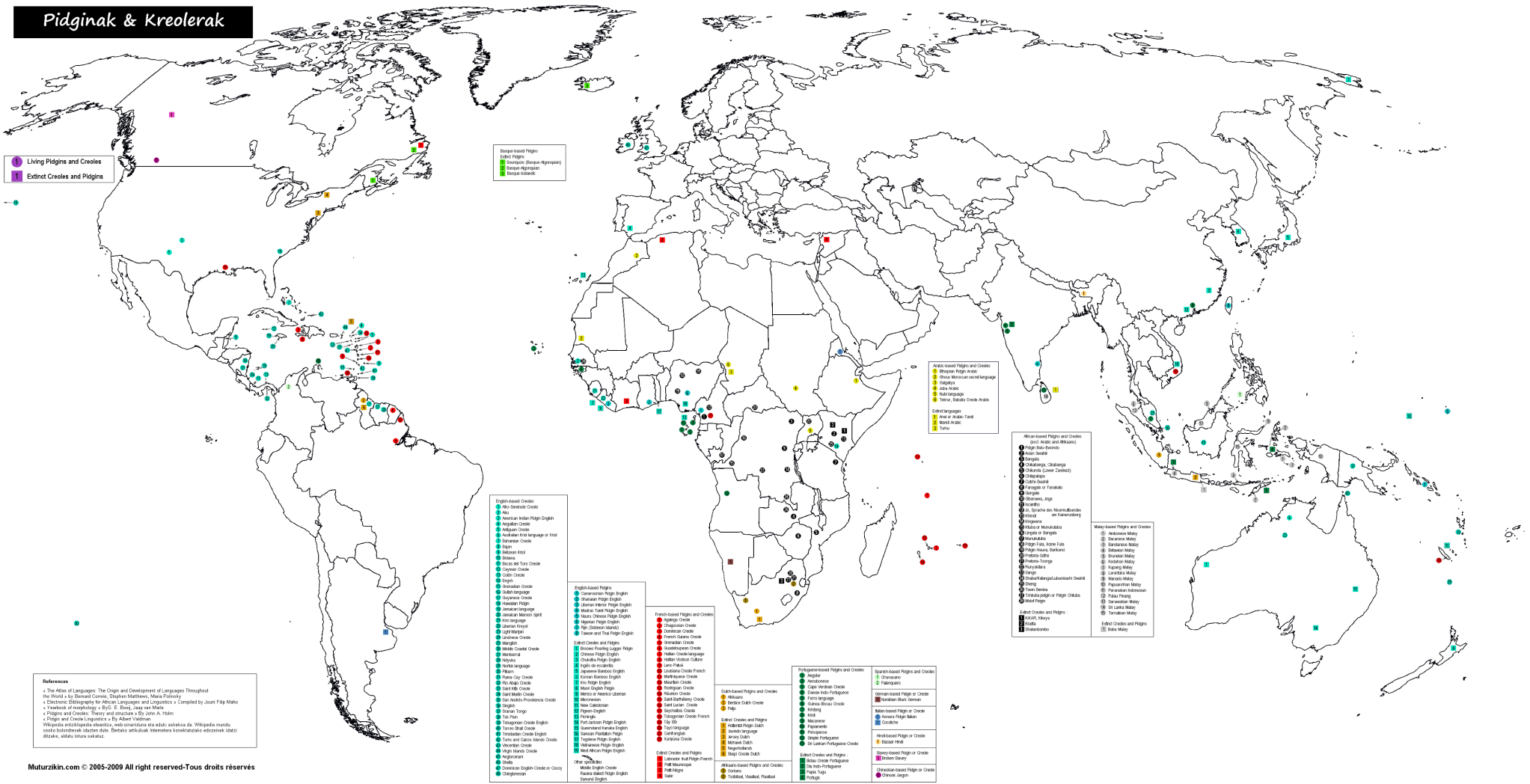
It is important for everyone (speakers of HCE, and speakers of GAE) to realize that HCE is a full language, just like GAE, so as not to perpetuate any negative stereotypes about the speakers. This is especially important for people who only speak HCE. Otherwise, we are telling them that the only language they speak is inferior to other languages.

Here is a link to an an online article about a poet who writes in HCE:

http://jamarattigan.com/2012/05/25/friday-feast-hawaiis-pidgin-guerrilla/

Creoles are also very common!

Creoles arise anytime children are exposed to pidgins during their critical period for language acquisition. This map (http://www.muturzikin.com/cartepidgin.htm) also documents both living and dead creoles across the world.



Where do the properties of creole grammar come from?

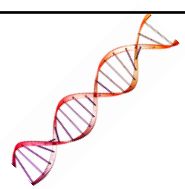
One big question for us is this:

> Where does the complexity and regularity of creole grammars come from?

Remember, all the children are hearing around them is the pidgin. The pidgin has less complexity than a full-fledged language, and also shows much more variation.

This means that the complexity and regularity of the creole cannot come from the pidgin itself. In other words, it can't come from evidence!

The genetic hypothesis



It looks like these properties come from children's minds. If the genetic hypothesis/nativism is correct, they have innate knowledge about how languages should work. They appear to take the simple and variable input from the pidgin, and turn it into a complex, regular, full-fledged language!

And why is it only children who can create a creole (not adults, who instead create pidgins)?

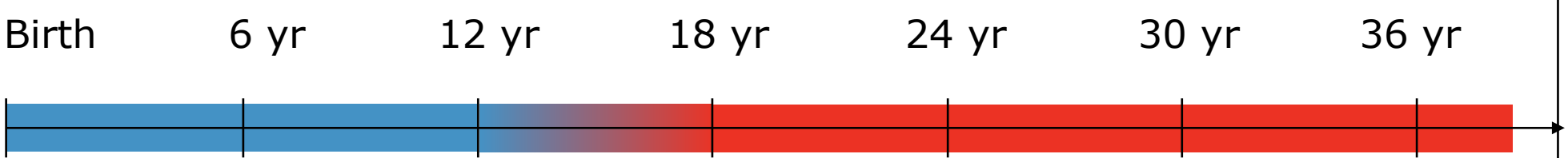
A second big question for us is this:

> Why is it that adults can't use their innate knowledge to create a creole out of a pidgin?

Remember, the adults that speak the pidgin are unable to create a creole. They are also unable to learn the native languages of the other immigrants.

It seems like these two facts might be related: the reason that adults can't learn other native languages is the same reason that they cannot turn a pidgin into a full language (a creole):

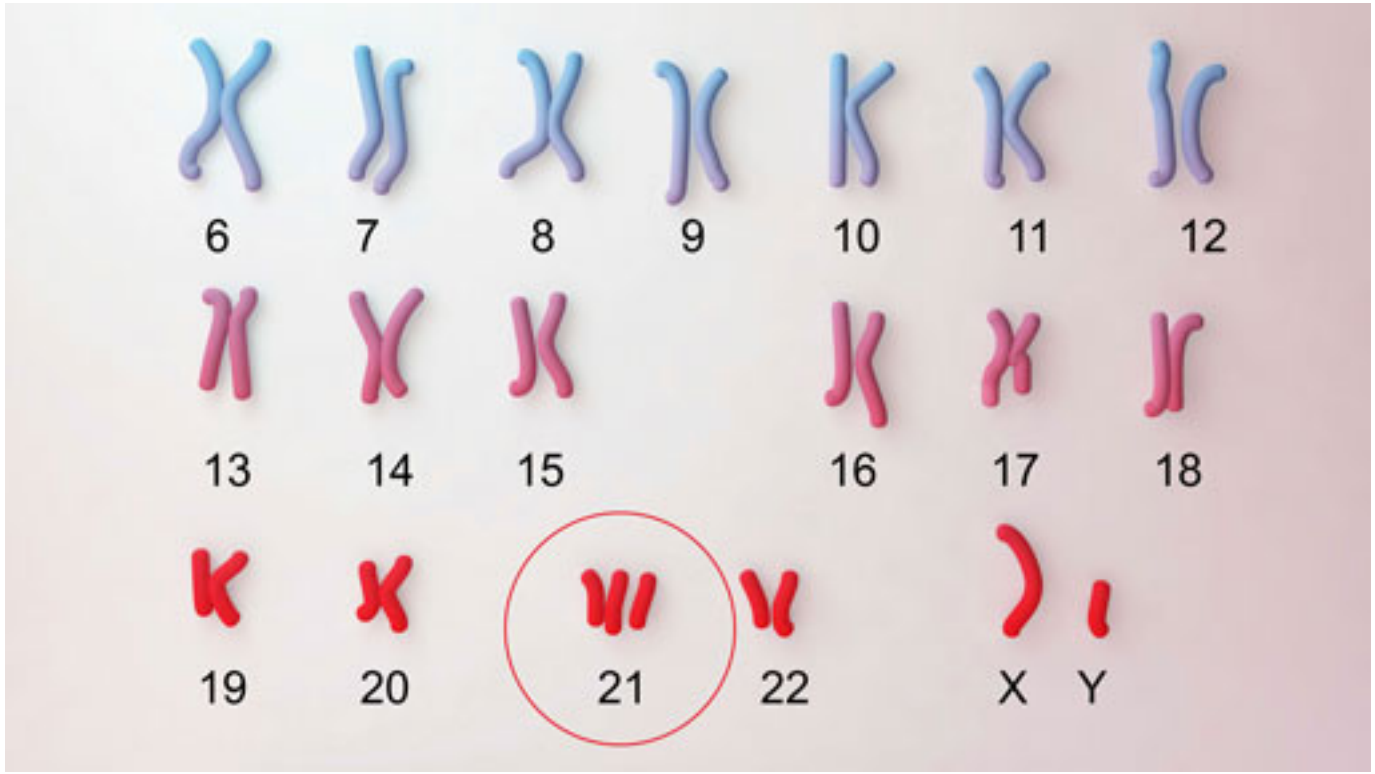
The Critical Period: Humans only have access to their innate language learning mechanisms for a short period after birth (approximately from birth until puberty). After that critical period is over, language acquisition no longer proceeds the same way.



Developmental disorders and the dissociation between language and intelligence

Down Syndrome/Trisomy 21

Down Syndrome is a genetic disorder caused when abnormal cell division results in an extra full or partial copy of chromosome 21. [Mayo Clinic]

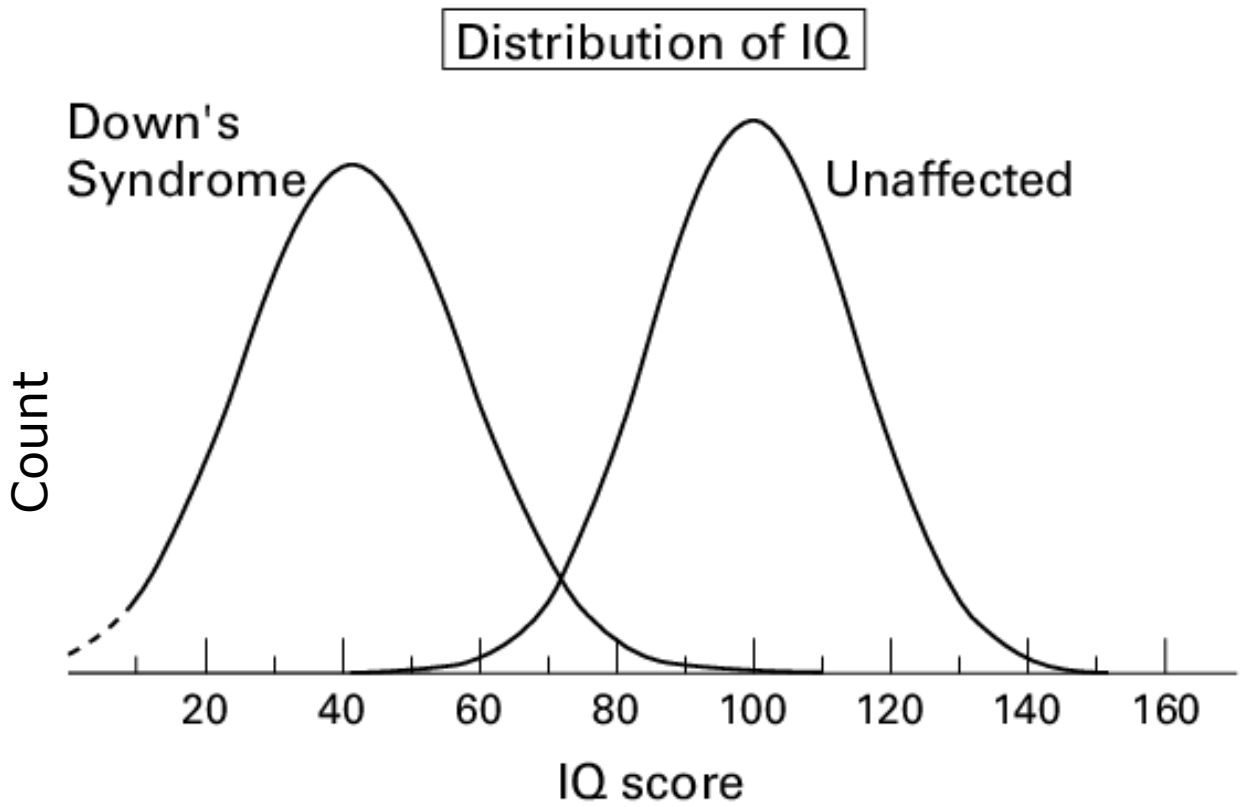


Cognitive Effects: Intelligence

Down Syndrome leads to a profound deficit in general intelligence as measured through standardized intelligence tests like the **Wechsler Adult Intelligence Scale**.

Intelligence scales are standardized such that 100 is mean for the population, with a standard deviation of 15.

This means that about 5% of typically developing adults will have an IQ lower than 70. It also means that about 5% of typically developing adults will have an IQ above 130.



Language effects

Down Syndrome often results in noticeable effects on language ability. Here you can see the responses of two children asked to describe a page from a picture book:

(M. Mayer, "Frog Where are You")

DNS age 13

There you are. Little frog. There another little frog. They in that... water thing. That's it. Frog right there.

DNS age 18

Thy're hiding; see the frogs... the baby frogs. Uh, the boy, and, and the dog saw the frogs. The frog's got babies. The boy saw the... no, the boy say good bye.

(Reilly, Klima & Bellugi, 1990)

An important double dissociation

Williams Syndrome and Specific Language Impairment form a double dissociation between intelligence and language ability. This suggests that language ability is independent of general of intelligence.

Williams Syndrome

Affects general intelligence

Has no (or few) effects on language

Specific Language Impairment

Has no effects on general intelligence

Affects language acquisition

What is a double dissociation?

Double dissociation is just a way of saying that two abilities can vary independently of each other.

When we say that language ability and intelligence are doubly dissociated, what we are really saying is that all four combinations of ability are possible:

	Intelligence	**Language**
option 1:	unaffected	unaffected
option 2:	affected	affected
option 3:	unaffected	affected
option 4:	affected	unaffected

Under a double dissociation, all four options are possible!

Double dissociations are important tools in cognitive science because they show us that the two abilities are independent of each other. There is no necessary relationship between them.

Williams Syndrome - the genetic cause

Williams Syndrome is caused by a deletion of about **20 genes** on **chromosome 7** - the specific location is called 7q11.23.

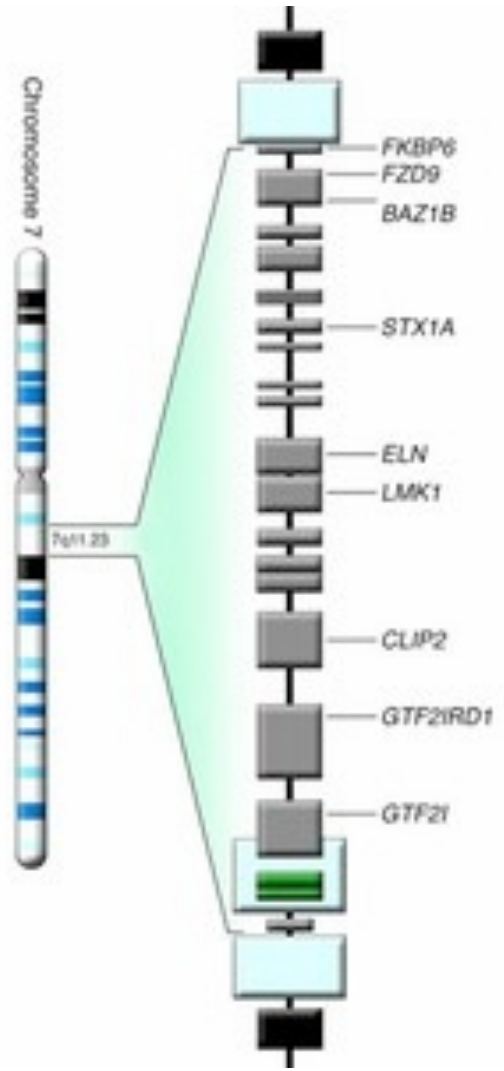
The number 7 refers to the chromosome.

The letter q refers to the long arm of the chromosome (the shorter arm is p).

The number 11 refers to a specific band that is visible on the chromosome when it is stained.

The number 23 refers to a sub-band of that primary band.

The exact role of many of these genes is still a matter for research. However **ELN** is the gene responsible for the protein **elastin** (plasticity in human organs), and **LMK1** may be related to visual-spatial cognition.

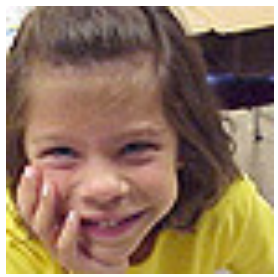


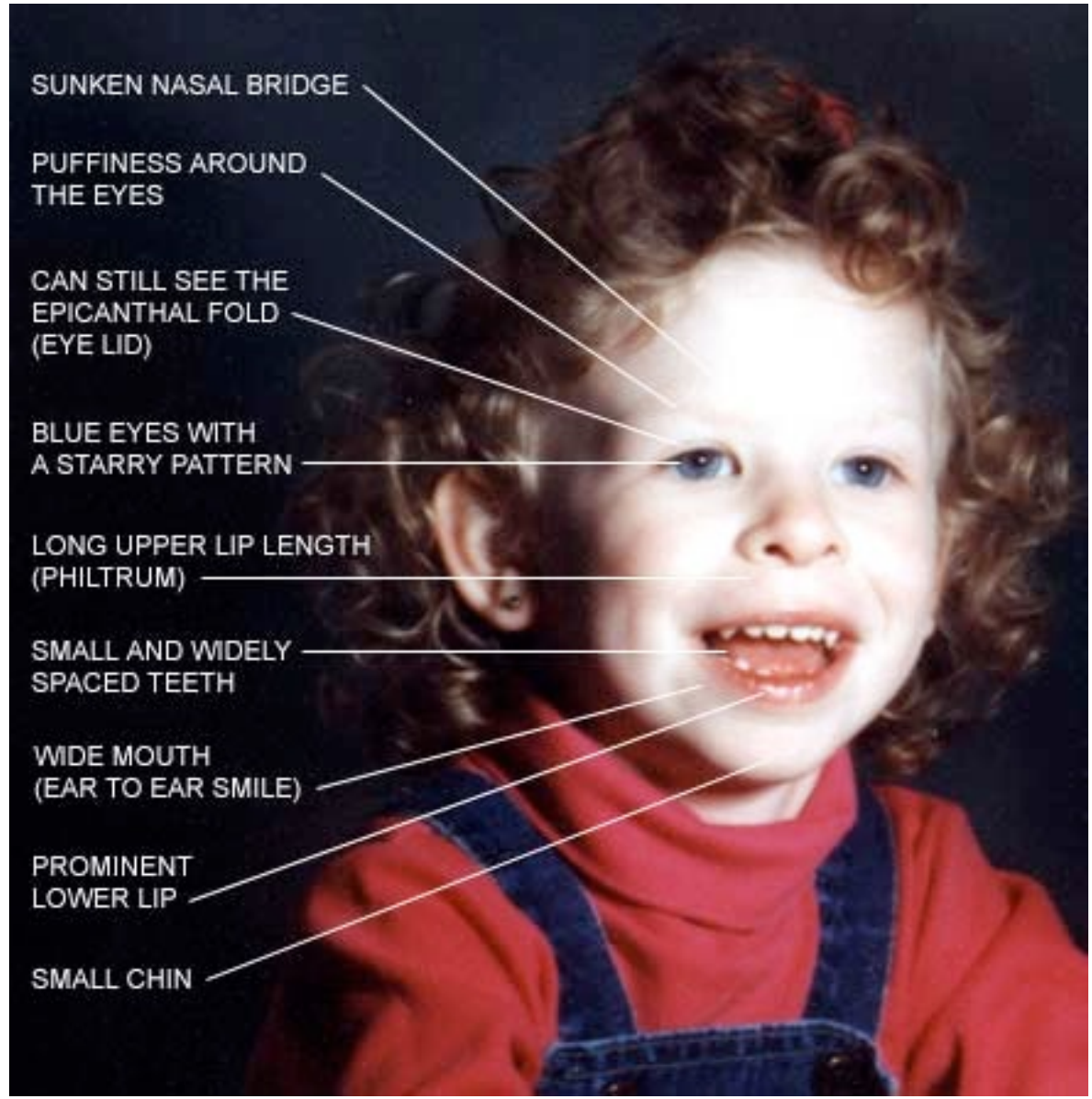
Williams Syndrome - physiological effects

Williams Syndrome leads to characteristic changes to facial features.

The constellation of features is often described as youthful, even for adults.



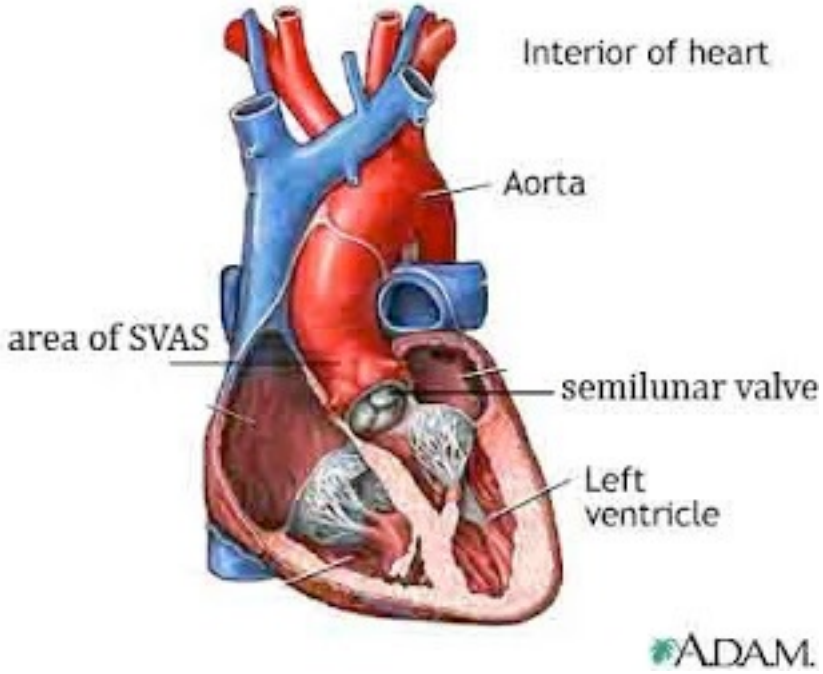


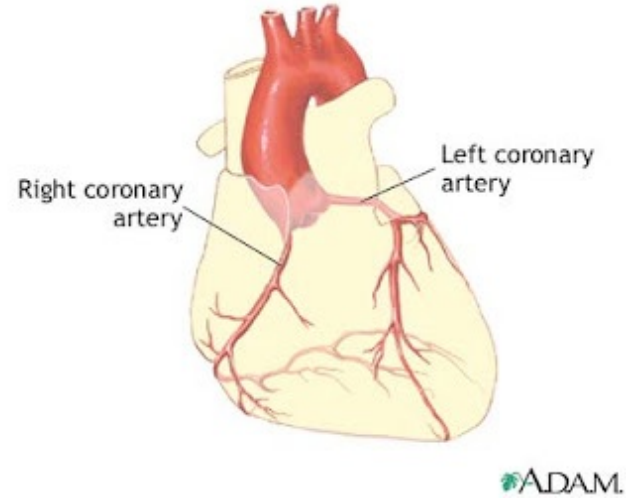



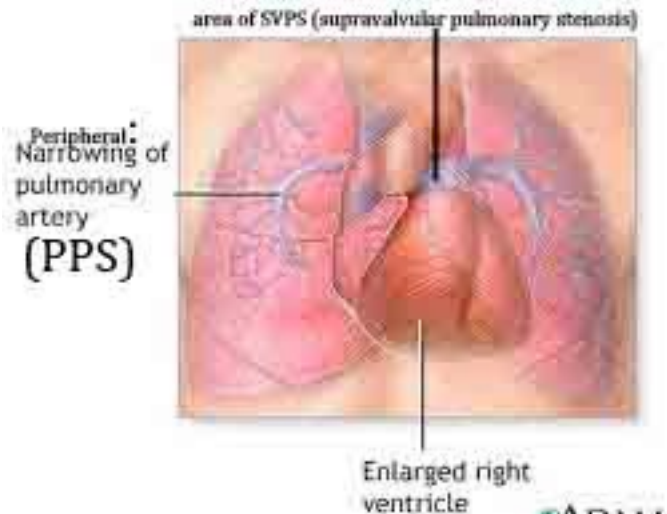
Williams Syndrome - physiological effects

The **elastin deficiency** caused by WS leads to a narrowing of the blood vessels (**stenosis**) throughout the body, most dangerously in the heart, lungs, and kidneys.

supravalvular aortic stenosis

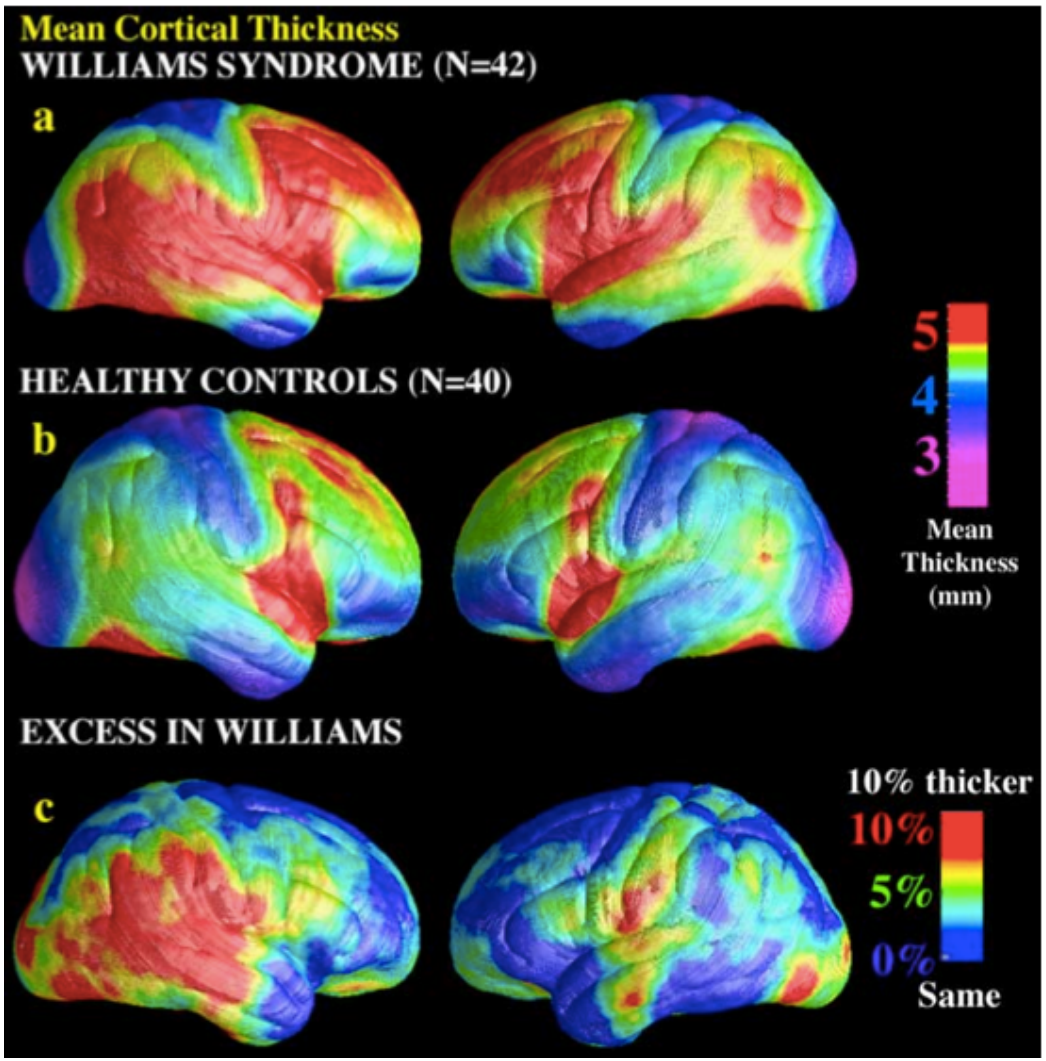


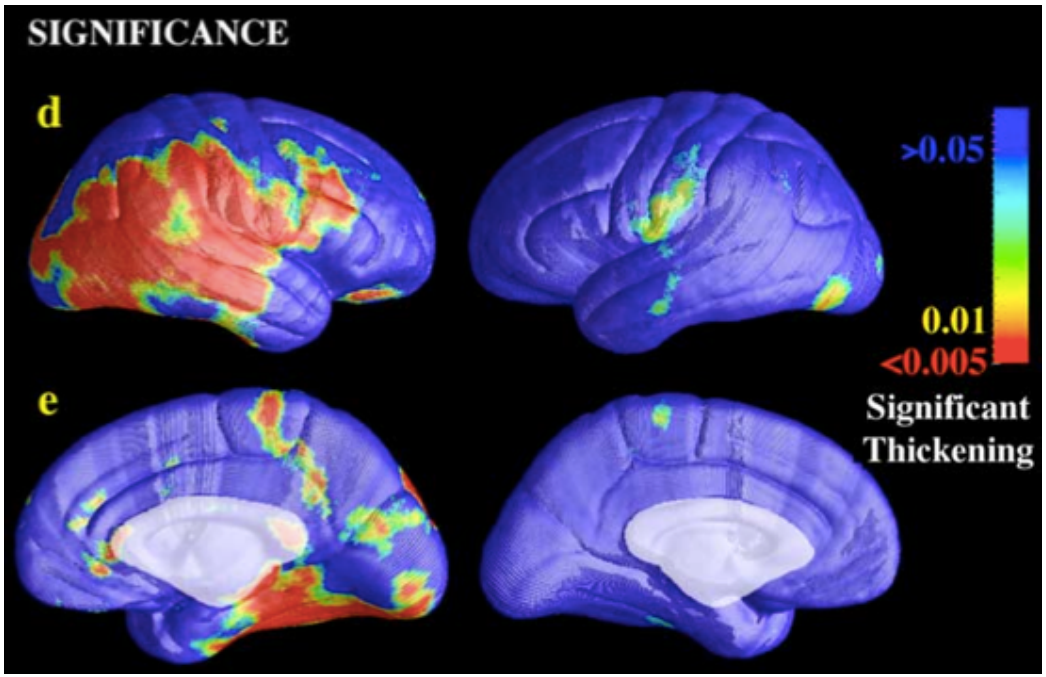




Williams Syndrome - physiological effects

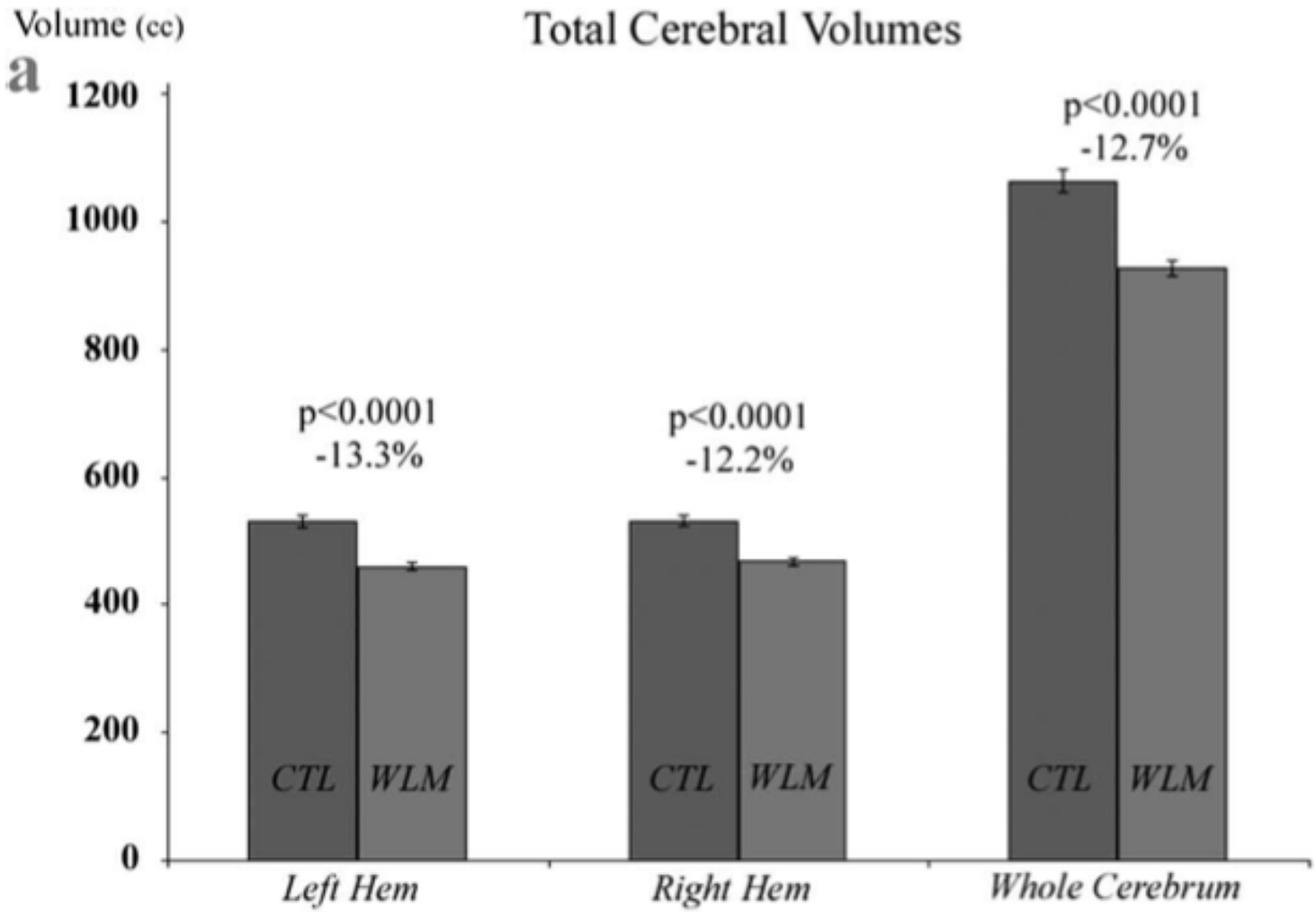
Williams Syndrome leads to a thickening of the cortex of the right hemisphere. But we have no idea how this would affect cognition.





Williams Syndrome - physiological effects

Williams Syndrome also leads to an overall decrease in cortical volume. The decrease occurs in all lobes, and in both gray and white matter. However, the majority of the reduction is in white matter.



Williams Syndrome - cognitive effects

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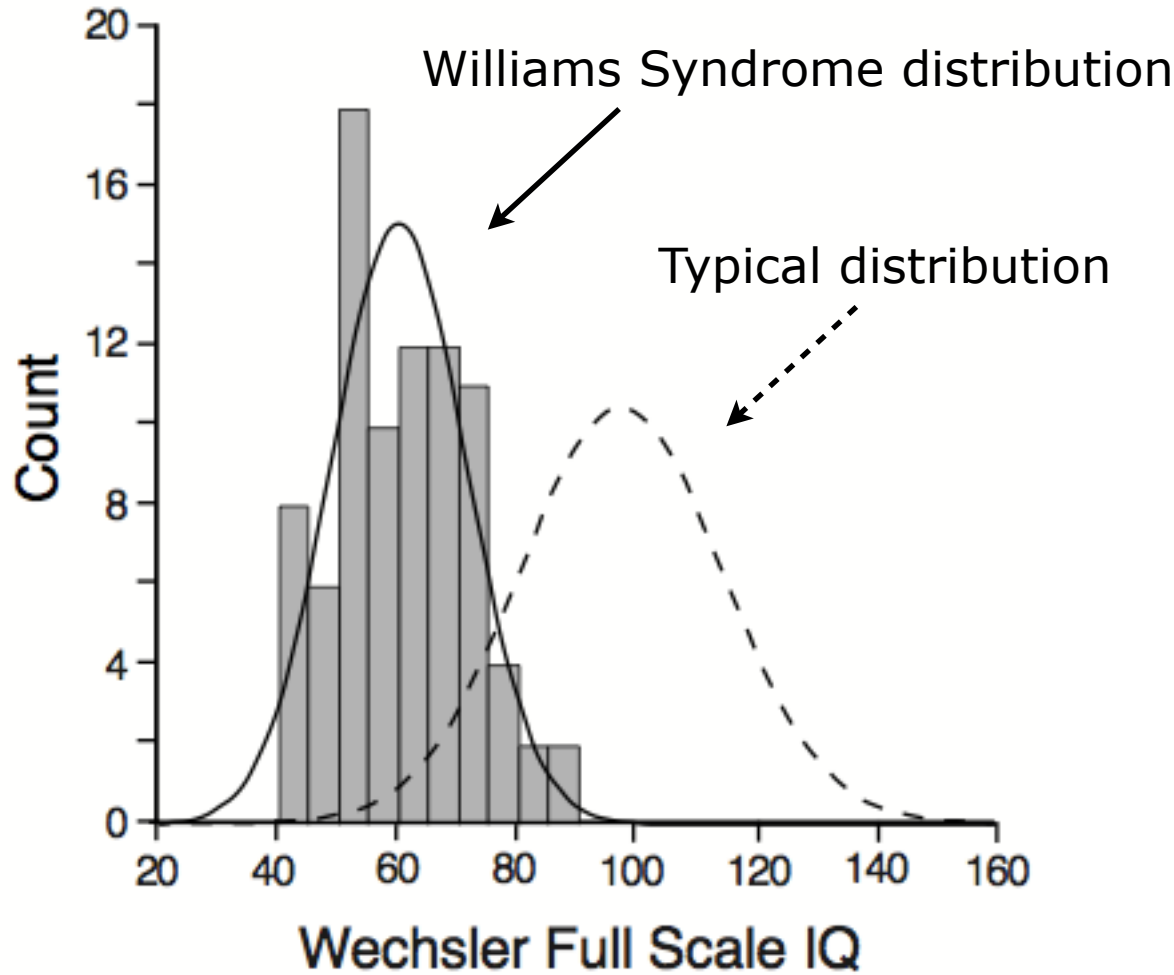
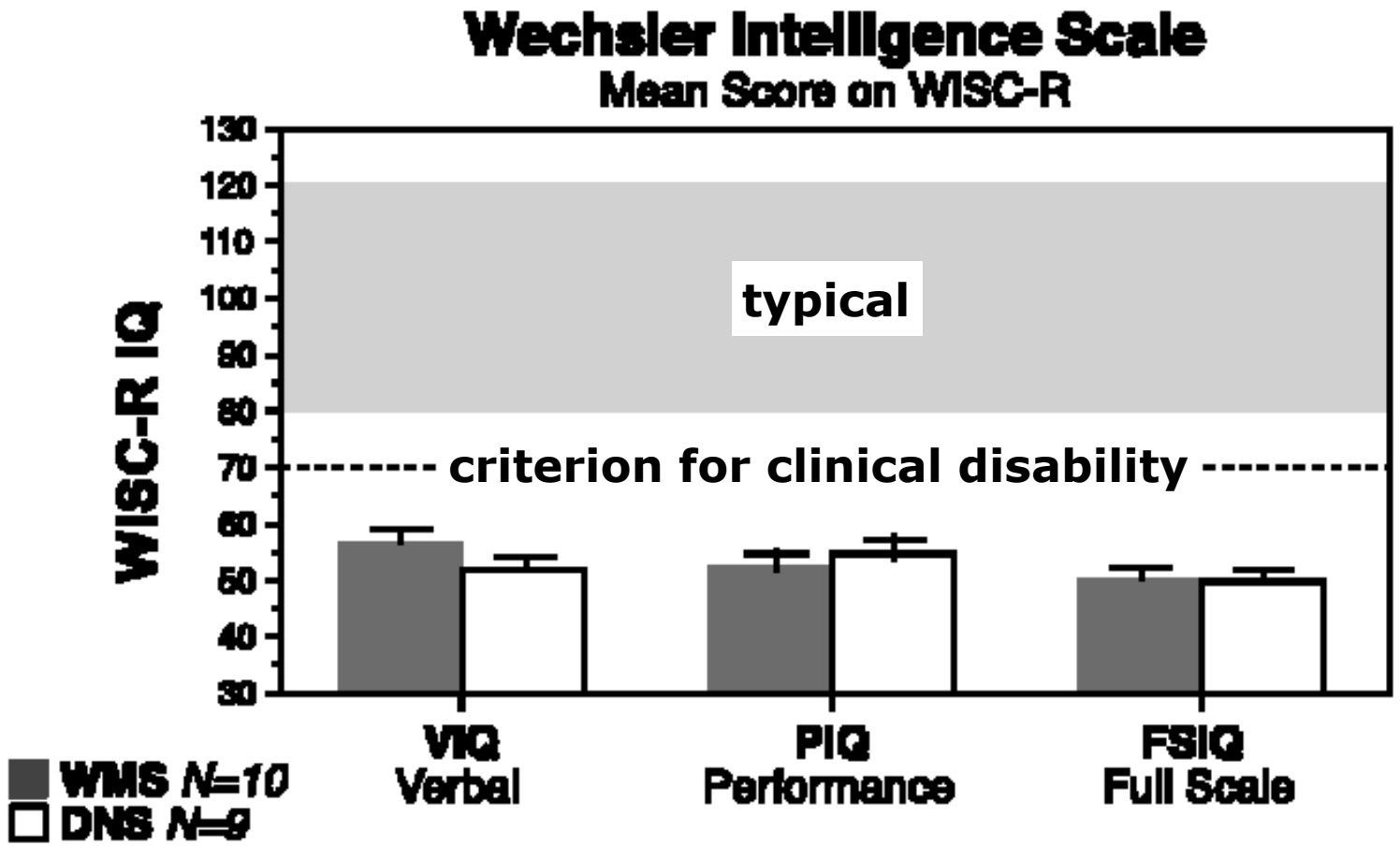


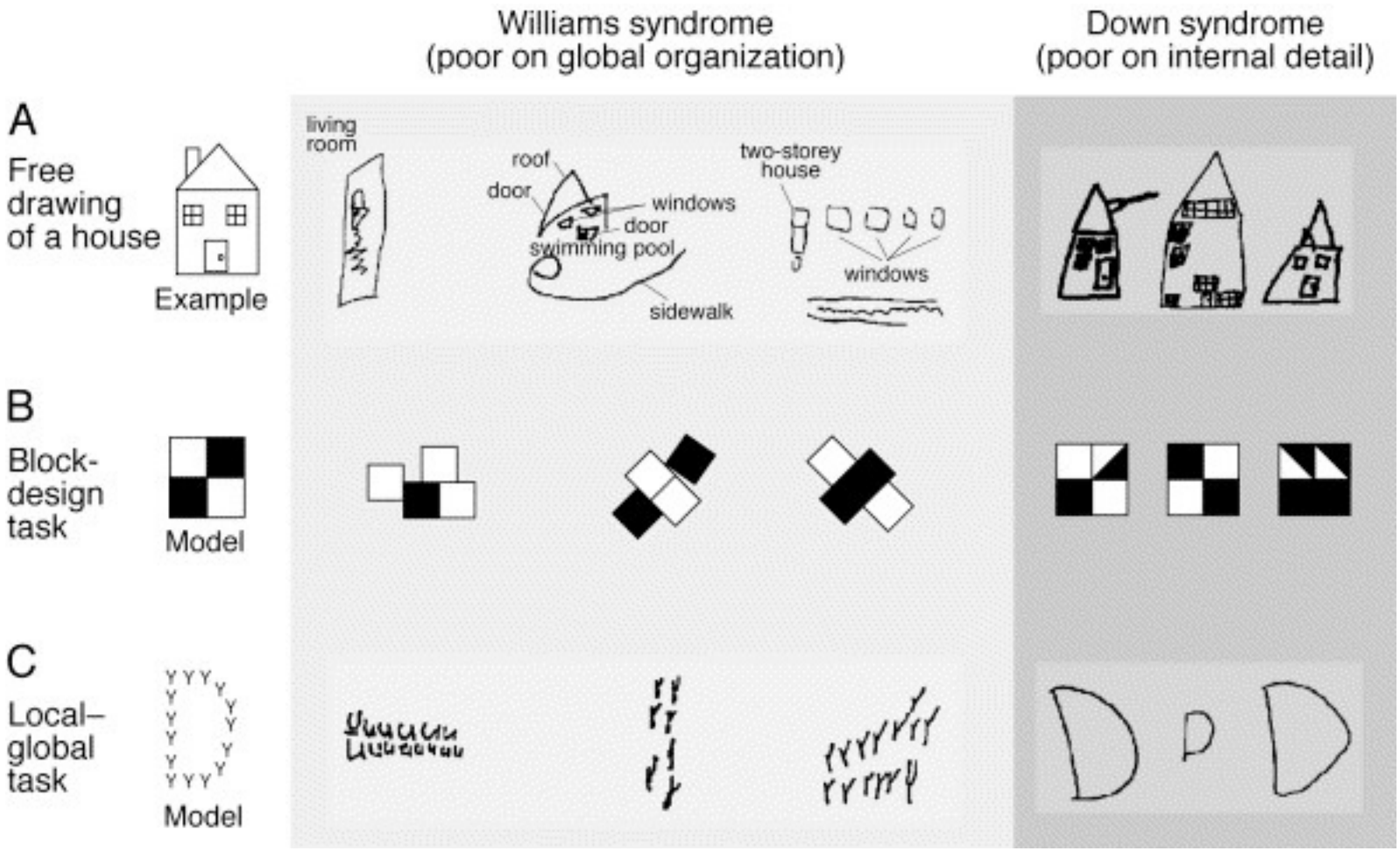
Comparing effects on intelligence in Down Syndrome and Williams Syndrome

The two disorders appear to have very similar effects on general intelligence. Keep this in mind — it means we should expect to see the same level of effect on language (if there were a link between them). But we won't!



Comparing effects on visual-spatial processing in DS and WS

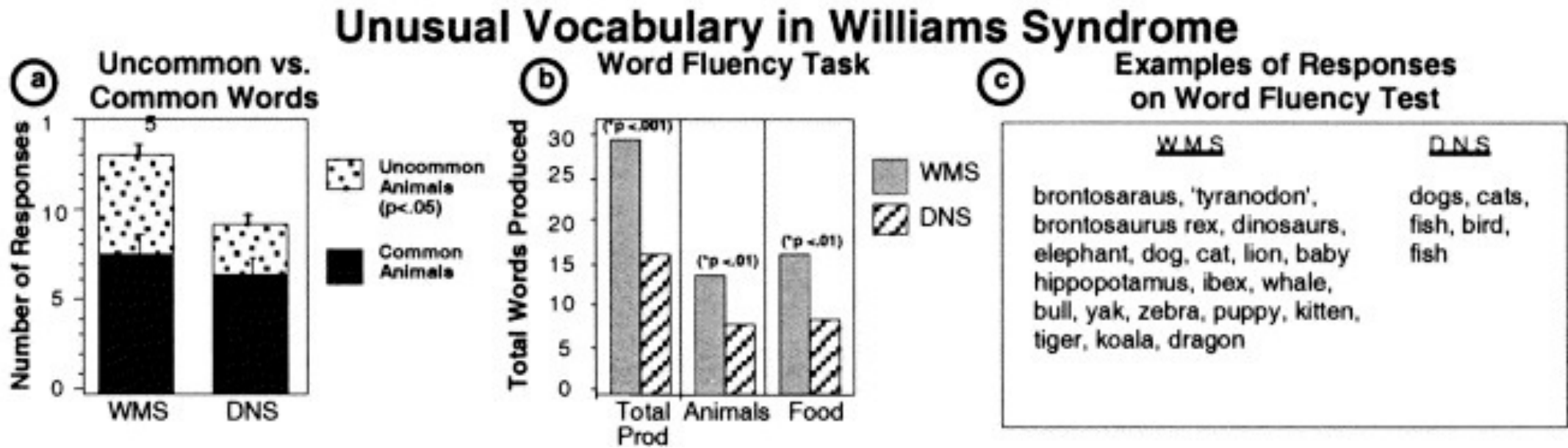
Both WS and DS lead to visual-spatial deficits. However, they are distinct deficits: WS seems to preserve internal details, but loses global organization; DS seems to lose internal details, but preserves global organization



Comparing effects on language in DS and WS

If one were to look at DS alone, one might conclude that general intelligence deficits and language deficits go hand-in-hand. But a comparison with WS shows that general intelligence deficits can occur **without** language deficits.

Patients with Williams Syndrome tend to have larger **productive vocabularies** (I am not sure about receptive vocabulary differences — the studies that I have seen have focused on productive vocabulary).



Comparing effects on language in DS and WS

When asked to describe a picture, patients with WS will produce a longer, more coherent narrative, with far fewer grammatical mistakes.

Qualitative Examples of Increased Linguistic Evaluation in Adolescents with Williams Syndrome

(M. Mayer, "Frog Where are You")

WMS age 13

And he was looking for the frog. What do you know? The frog family! Two lovers. And they were looking. And then he was happy 'cause they had a big family. And said "good bye" and so did the frog. "Ribbit."

DNS age 13

There you are. Little frog. There another little frog. They in that... water thing. That's it. Frog right there.

WMS age 17

Suddenly when they found the frogs... There was a whole family of frogs... And ah he was amazed! He looked... and he said "Wow, look at these... a female and a male frog and also lots of baby frogs". Then he take one of the little frogs home. So when the frog grow up, it will be his frog... The boy said "Good bye, Mrs. Frog... good bye many frogs. I might see you again if I come arounmd again". "Thank you Mr. Frog and Mrs. Frog for letting me have one of your baby frogs to remember him".

DNS age 18

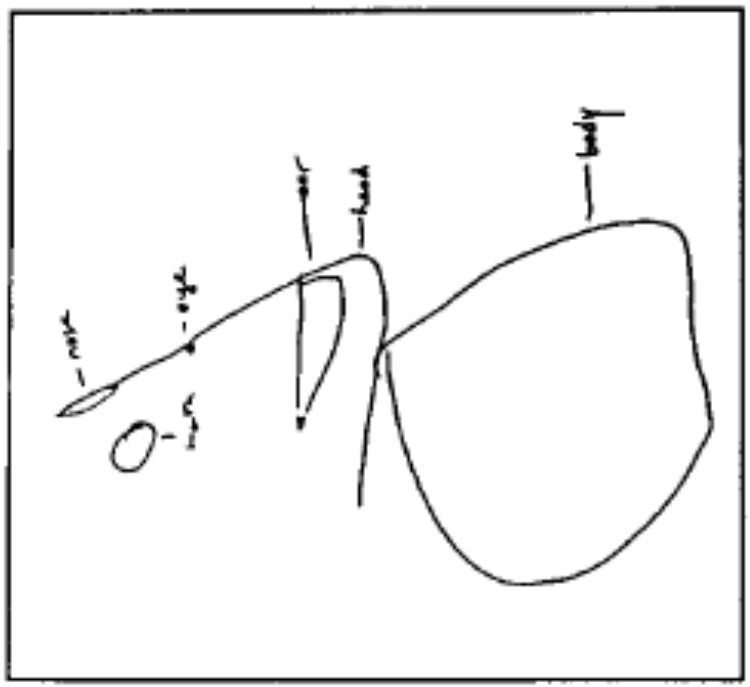
Thy're hiding; see the frogs... the baby frogs. Uh, the boy, and, and the dog saw the frogs. The frog's got babies. The boy saw the... no, the boy say good bye.

(Reilly, Klima & Bellugi, 1990)

WS: comparing visual-spatial abilities and language abilities

The contrast between visual-spatial and language abilities in WS is particularly striking when patients are asked to describe the pictures that they draw:

a.



b.

And what an elephant is, it is one of the animals. And what the elephant does, it lives in the jungle. It can also live in the zoo. And what it has, it has long gray ears, fan ears, ears that can blow in the wind. It has a long trunk that can pick up grass, or pick up hay....If they're in a bad mood it can be terrible...If the elephant gets mad it could stomp; It could charge, like a bull can charge. They have long big tusks. They can damage a car...It could be dangerous. When they're in a pinch, when they're in a bad mood it can be terrible. You don't want an elephant as a pet. You want a cat or a dog or a bird...

FIG. 2.6. Contrast between visuospatial and language abilities in WS. (a) Drawing of an elephant by an 18-year-old WS woman, whose IQ is 49. (b) Her verbal description of an elephant.

This is one half of our dissociation

Williams Syndrome shows that general intelligence can be affected while language ability is (mostly) unaffected. (This also shows that the correlation seen in Down Syndrome is specific to Down Syndrome.)

Williams Syndrome

Affects general intelligence

Has no (or few) effects on language

Specific Language Impairment/ Delayed Language Disorder

Has no effects on general intelligence

Affects language acquisition

What is Specific Language Impairment?

Specific Language Impairment (SLI), also called Delayed Language Disorder (DLD), is a developmental disorder that specifically affects language, without any other disorder that can explain it (hearing, general cognitive development, etc).

General clinical symptoms:

Production delay in first words

Deviant production of speech sounds

Simplified grammatical productions (omission of tense markers, etc)

Restricted vocabulary in both production and comprehension

Trouble repeating words or sentences (perhaps due to short term memory deficits)

Comprehension difficulty with complex sentences and/or rapid speech

How is SLI/DLD diagnosed?

By definition, SLI is a deficit in language development without any other accompanying cognitive or sensory deficits that could explain it. This means that a diagnosis of SLI requires the elimination of any other possible causes.

General Diagnostic criteria:

Language production and/or comprehension in lowest 10% for age on standardized test

Nonverbal IQ and other cognitive abilities fall within normal limits for age

No hearing loss, physical abnormality of the speech organs, or brain damage

No deprivation of language input in the environment

The rate of SLI in kindergarten-aged children has been estimated to be **as high as 7%**; however, such estimates are likely inflated, as large scale studies have not combined both inclusionary criteria (language impairment) and exclusionary criteria (nonverbal cognitive abilities).

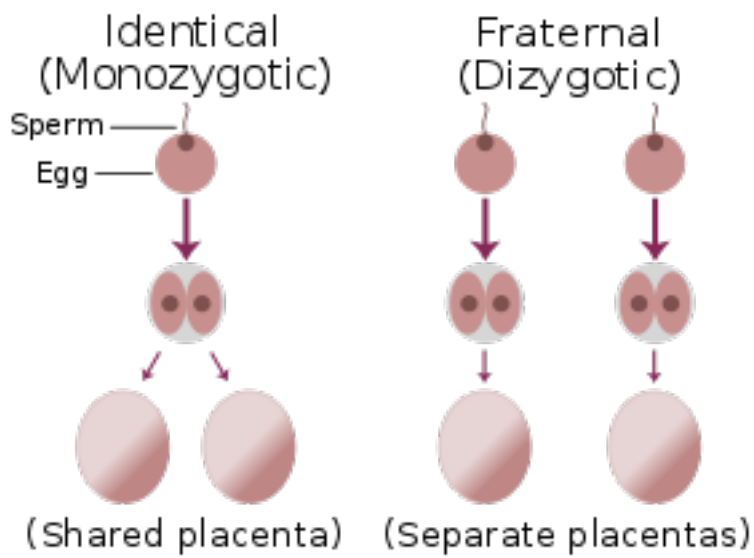
What causes SLI/DLD?

The cause of **Specific Language Impairment** (SLI) is likely genetic; however, unlike Williams Syndrome, the genetic cause has not been identified.

So how do we know it is genetic?

The primary evidence comes from the rate of incidence between different types of twins.

The idea is that siblings are generally exposed to the **same environmental factors** (parenting, education, nutrition, etc) but can vary in genetic relatedness.



The proportion of pairs of monozygotic twins (one egg - identical twins) in which **both have SLI** is much higher than the proportion of pairs of dizygotic twins (two eggs - fraternal twins) in which both have SLI. **This suggests genes are a stronger cause than environment.**

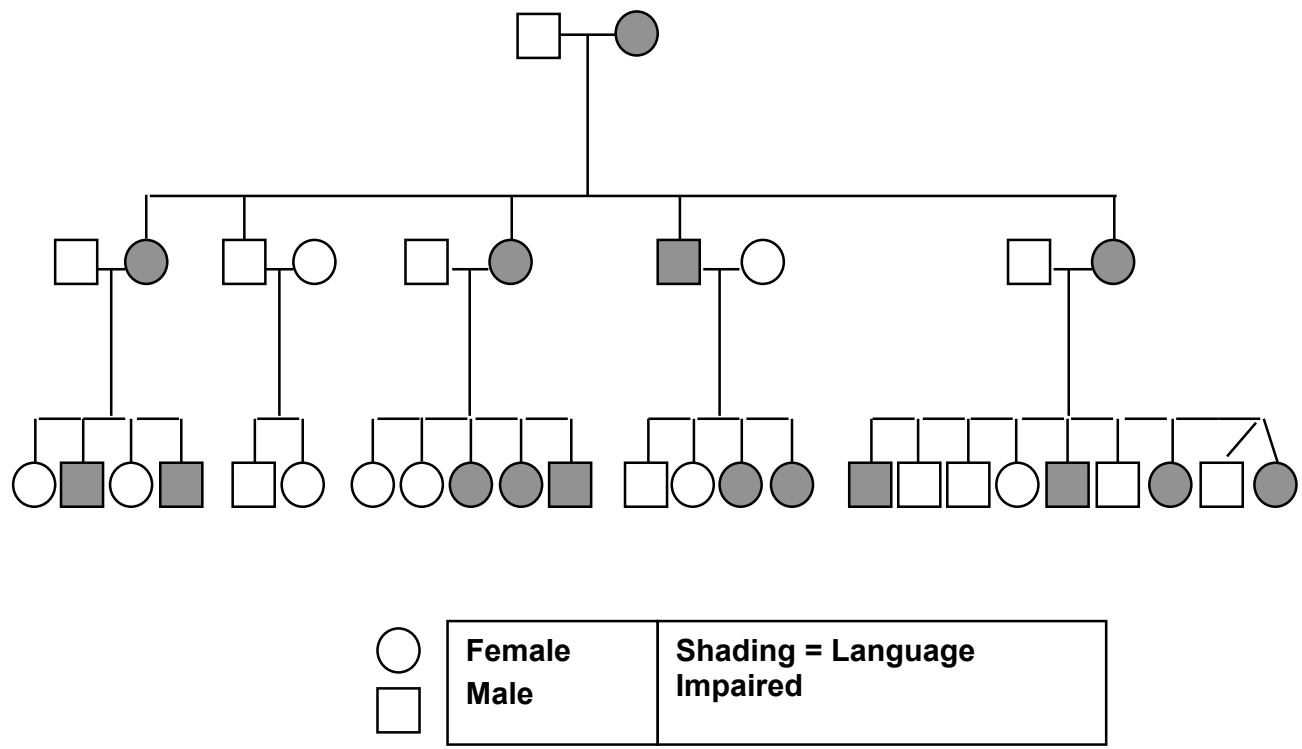
Furthermore, in cases where only one member of monozygotic twins has SLI, the other tends to show some language impairment, though perhaps not severe enough to meet the diagnostic criteria for SLI.

A specific type of SLI/DLD: the KE family

There is a family in London that exhibits a particularly severe form of SLI.

What is particularly relevant about this family is that the deficit has appeared in nearly half of the family members, across at least three generations. This has allowed researchers to investigate both the behavioral deficits and any genetic differences between family members.

Genetic tests have revealed a mutation in the **FOXP2** gene, which is located on chromosome 7, specifically at 7q31



A specific type of SLI: the KE family

Comparisons of unaffected and affected family members on a wide battery of tests reveals that the deficits in the KE family are much broader than the deficits reported in the general SLI population:

Affected members show the typical SLI/DLD language deficits:

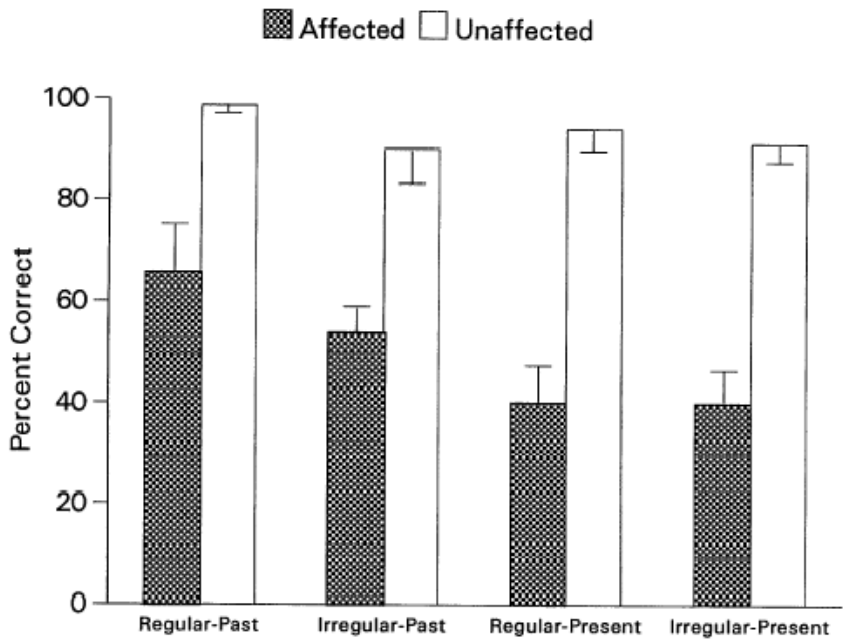


FIG. 2. Production of tenses. Scores are means ± standard errors. See Table 2 for examples of test items.

But they also show deficits in broader oral-facial abilities:

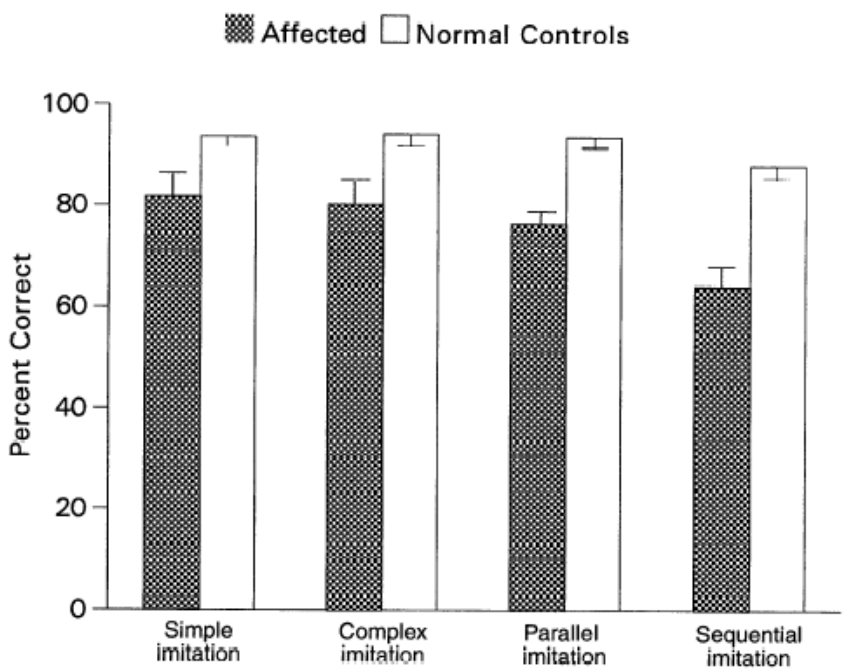


FIG. 3. Imitation of oral and facial movements. Scores are means ± standard errors.

Some conclusions: Double Dissociation

Williams Syndrome and Specific Language Impairment form a double dissociation between intelligence and language ability. This suggests that language ability is independent of general of intelligence.

Williams Syndrome	**Specific Language Impairment**
Affects general intelligence	Has no effects on general intelligence
Has no (or few) effects on language	Affects language acquisition

In short, there appears to be a **biological basis** for investigating the language faculty as a distinct system from general intelligence.