Answers4Seekers, Session #4

Topics Covered: Intersex, Dysphoria, & Orientation Genes

A) What is Intersex (and what it isn't)?
B) What is Rapid Onset Gender Dysphoria?
C) Is Sexual Orientation fixed by Our Genes?
D) Dysphoria Persistence, Suicide Rates, and the Nuclear Family
E) Exhibits

Session's Bonus Video Links:

- **Dr. Leonard Sax on "How Today's Trans Activism Actually Emboldens Gender Stereotypes"** (link: **https://www.youtube.com/watch?v=Qk1hT_Uuuko)** 8min.
- **Dr. Debra Soh How Parents Can Find a Rational Therapist on Gender Identity and Sexuality"** Link: **https://www.youtube.com/watch?v=19jhYKEKZ00** 13min.
- **Dr. Leonard Sax on "Truth About Gender Differences, Danger of "Affirm Only" Care** (link: **https://www.youtube.com/watch?v=gmTX2T44UDs**) 1hr:37min

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A) What is Intersex (and what it isn't)?

a. Why Discuss Intersex First?

Before we can embark properly on the topic of gender-dysphoria, it is helpful to first address the topic of what is "Intersex" (and what it isn't), since some organizations are confused and will use sexual birth disorders to promote transgenderism.

b. Like other human birth defects and disorders, "intersex" does occur, but is rare (about 1 in 10000 births). Jesus appears to be mentioning this rare birth disorder in Matthew 19:12 when He said, ***"eunuchs [those without functioning testes, ovaries, or genitals], who were <u>born</u> so from their mother's womb,"*** but Jesus also reconfirmed that "… ***from the beginning of creation, God made them male and female …"*** (Matthew 19:4). Yet, birth defects do occur in a fallen world.

c. From a biblical perspective, even in our fallen world (which experiences birth disorders), each baby and person was planned from the foundation of the world (*Ephesians 1:4–5),* and was created for an eternal purpose *(Jerimiah 29:11, Revelation 21:4),* starting in this world and then throughout eternity, where every wrong thing will be made right eternally. *(Daniel 7:13-14, Revelation 13:8)*

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d. What is the clinical definition of "Intersex"?

i. The clinical definition of Intersex is a birth disorder of sex development that would include one of these conditions:

1. (a) the phenotype (physical characteristics) of individual's genitals is not classifiable as either male or female, or

2. (b) individual's chromosomal sex (XX or XY) is inconsistent with their phenotypic sex (physical characteristics).

ii. Clinical Intersex births occur about 1 in 5500, and are pretty rare; compared to the frequencies of other birth defects or disorders (see below). The prevalence of more severe intersex conditions occurs at a rate less than 1 in 10000 (see table below), yet all of these can live full lives and many are able to perform and enjoy sexually in their phenotype, but are almost always infertile.

iii. Gonads defined: Gonads are human reproductive sex glands that produce sex (gametes) cells: Male gonads are Testes (whether descended or undescended) and produce sperm cells, and Female gonads are Ovaries which produce ovum egg cells. Possessing functional ovaries means you are a female, possessing functional testes means you are male.

iv. The SRY Gene: Sex-determining Region Y, is the primary gene producing the **male sex** of the child and their phenotype and is normally located on the Y-Chromosome. The SRY gene is what codes for the development of testicles (male gonads) and through the testicles the production of testosterone which produces the masculinization of the brain and genitals (penis) and at puberty the secondary male features: muscle mass, height, hair, lower voice, growth in penis and testicles, and gamete production (sperm). Without the presence of the SRY gene the development of the embryo typically maintains the female phenotype.

v. In general, when there is a conflict between a person's "karyotype sex chromosomes" and their phenotype, their biological sex is resolved by whether or

not they possess the SRY Gene and do not carry defective "Androgen Insensitivity Gene." The presence of the SRY Gene without the Androgen Insensitivity Gene means they are Male, and the absence of the SRY Gene OR the presence of the Androgen Insensitivity Gene (for CAIS) means they are Female. **More simply, if they possess functional, sperm producing testicles they are Male, if they possess functional, ovum producing Ovaries, they are Female. This covers about 99.99% of all people.**

vi. Masculinization of the Brain: The SRY gene initiates testes development in embryos, leading to testosterone production that shapes neural development during critical prenatal windows. Testosterone (and its conversion to estradiol in the brain) promotes masculinization, influencing structures and connectivity patterns. Research using diffusion tensor imaging suggests that, on average, male brains show stronger intra-lobar (within-hemisphere) connectivity, supporting localized, modular processing often associated with spatial and motor problem-solving. Female brains, by contrast, tend to show more inter-lobar and

interhemispheric connectivity, facilitating integration across regions, which is linked to language fluency and multitasking. Both sexes are capable of high performance in language and complex problem-solving.

vii. No documented cases exist of a human having both fully functional ovaries and fully functional testes, let alone where they produce both viable eggs and sperm at the same time (therefore no true Hermaphrodites exists).

e. For Comparison and Perspective: Sampling of Non-Sex Development Birth Disorders that occur at a higher rate than "clinical intersex":

i. Clubfoot (a birth defect where one or both feet are rotated inward and downward): **Occurs 1 in every 593 births**

ii. Down syndrome, Trisomy 21 (Down syndrome) babies have an extra copy of chromosome #21 is a genetic condition where this person has three copies of chromosome 21 instead of the usual two and so instead of 46 chromosomes total

(23 pairs), this person with Trisomy 21 has 47 chromosomes total (an extra chromosome): **Occurs 1 in every 707 births**

iii. Atrioventricular septal defect (a heart defect affecting the valves between the heart's upper and lower chambers): **Occurs 1 in every 1,859 births**

iv. Cleft lip without cleft palate, A birth defect that occurs when a baby's lip or mouth do not form properly: **Occurs 1 in every 2,807 births**

v. Spina bifida, A condition that affects the spine and is a type of neural tube defect. **Occurs 1 in every 2,857 births**.

vi. Anencephaly, Where a baby is born without parts of the brain and skull; nearly always fatal: **Occurs 1 in every 4,762 births**

f. Individuals born with completely missing or undefinable gonads are extremely rare:

i. **Ovotestes**: Is where the gonadal tissue is mixture of ovarian tissue [female] and testicular tissue [male], typically called "Streak Gonads," and is typically non-functional, and therefore sterile (they are unable to reproduce). Ovotestes is caused by gene mis-regulation, occurring within some "XX,Males with SRY" and "XX/XY mosaicism," among other rare conditions**.** While their genitals are typically ambiguous, their bodies typically favor the female phenotype including a clitoris and are able to receive sexual pleasure as adults, so most choose to live as females. **These persons are true intersex**. Ovotestes has a rare occurrence of around "**1 in 150,000 births**."

ii. **CAIS & PAIS (Complete and Partial Androgen Insensitivity Syndrome):** Occurs when the body is insensitive to androgens (male hormones like testosterone), caused by a defective X-chromosome in the "Xq11–q12" gene section. For CAIS and Extreme-PAIS they typically appear female because their body can't respond to male hormones (testosterone) due to Androgen Insensitivity. Being XY they cannot

develop a uterus or ovaries, but Mild PAIS follows closely to the normal Male pathway for development and many can father children.

1. **Complete "CAIS"** typically will follow the female phenotype pathway and develop a Vagina and clitoris, maintain their default feminine brain, have undescended and unfunctional testes, but typically can receive sexual pleasure and orgasm as an adult. Maintaining a female anatomy, including a Vagina and Clitoris, and typically receive breasts at puberty, but have no uterus or ovaries. These "XY" CAIS qualify as sterile females. **Occurs 1 in 60,000**. **These persons are females.**

2. **Partial "Extreme PAIS"** typically will follow the female phenotype pathway way and develop a partial or ambiguous genital structure (clitoris may be enlarged -- clitoromegaly] and labia may be sealed. Surgery may be helpful depending on the degree of genital ambiguity, but with some prenatal exposure to testosterone, partial masculinization of the default feminine brain may have occurred. Having undescended and unfunctional testes they are

sterile, but erectile tissue is typically in place to allow sexual orgasm as an adult. These "XY" Extreme-PAIS qualify as sterile Intersex, but most are live as females. **Occurs 1 in 250,000. These persons are Intersex.**

3. **Partial "Mild PAIS"** typically will follow the male phenotype pathway and develop functional testes, and penis, and through testosterone receive a masculinized brain, at puberty receive secondary male sex traits, can experience sexual orgasm as an adult, and many produce viable sperm and can father children. **Occurs 1 in 55,000**. **These persons are Males.**

4. **See Exhibit-A (below).**

iii. **Swyer Syndrome** (Complete Gonadal Dysgenesis): Is where a "XY-46 chromosome baby" lacked the SRY (male) gene on their Y-Chromosome, and therefore lack the genes that code for testes and testosterone, and therefore could never developed testes or a penis. By not developing through the XX Karyotype female pathway, they cannot develop ovaries, but they do develop a vagina, clitoris, and uterus,

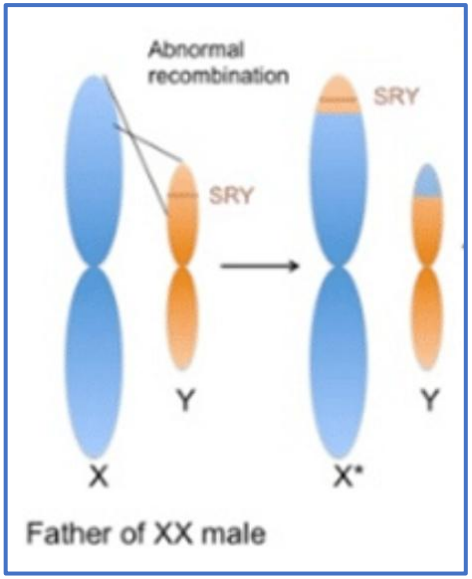
since that is the default pathway when the SRY gene is absent. They are born with the female phenotype and keep the normal feminized brain, and after puberty they typically experience full female sexual function, yet are completely sterile having no ovaries or eggs. **This person is female.** This occurrence has a frequency of **1 in 80000** human births. [see image below]

1. Swyer syndrome occurs when the Father's sperm lost its SRY Gene section the Y-Chromosome (typically through translocation), and through coitus joins with the Female egg to initiate conception.

g. XX, MALE: Is an XX individual, where one of the X's contains the SRY gene that was abnormally translocated from the Y chromosome onto one of the X chromosomes during paternal meiosis; this condition is often called XX male syndrome. In most cases, the presence of SRY initiates testes development, leading to production of testosterone and anti-Müllerian hormone, so affected individuals typically develop male external genitalia (penis) and a masculinized brain. However, their testes are usually small and nonfunctional (no sperm production), and some individuals may have ambiguous

genitalia depending on how fully downstream genes are activated. They do have the SRY gene, but lack other gene parts that would be contributed by the full Y-chromosome. Thus, SRY-positive XX individuals are phenotypically male, but with varying degrees of masculinization, but they are infertile due to the absence of other critical Y-Chromosome genes needed for normal spermatogenesis.

This person is male. **Occurs 1 in 20,000**. [see image below]



h. Chromosome Mosaics: Chromosomal mosaicism is a condition where a single individual has two or more genetically different cell lines that originated from the same fertilized egg (zygote). A chromosome mosaic is when not all cells in the body have the same chromosomes. Some chromosome mosaics examples: (45,X/46,XX), or (46,XY/46,XX), or (46,XY/47,XXY). Most have reduced or absent reproductive capacity. Overall, fertility is uncommon. Again, biological sex can be determined if there is the presence of functional testes for males, or the presence of functional ovaries for females. **Occurs about 1 out of every 10,000.**

i. For clarification, the following types of sex-development disorders do not meet the clinical definition of "Intersex":

   i. Classic & Non-Classic Congenital Adrenal Hyperplasia [CAH]: A group of genetic disorders that affect the adrenal glands, and not a sex develop disorder.

      1. **Non-Classic CAH** is milder and more common. It may not be identified until childhood or early adulthood. Phenotype: female with excess facial/body hair,

acne, oily skin, menstrual irregularities, sometimes infertile, and sometimes mild clitoral enlargement. Effects females, but they still have ovaries and can become pregnant, typically is noticed after childhood. **Non-Classic occurs in 1 in 200 in the USA (1 in 1000 worldwide).**

2. **Classic CAH**: Effects females, but they still have ovaries, clitoromegaly (enlarge clitoris), possible sealed labia, but many can become pregnant, but surgery may be required. **Occurs ~1 in 12,500.**

3. **CAH in XY Males:** They essentially follow the typical male development pathway.

4. **In CAH, XX persons are Female, XY persons are Male**
   a. https://chatgpt.com/c/69e678bc-6ed0-8331-9645-5f09e5eae926
   b. https://chatgpt.com/c/69e70f3b-d930-832f-b46c-5e5f28f138d2
   c. See Exhibit B.

ii. Vaginal Agenesis: A rare disorder in which the vagina doesn't fully develop, and the womb (uterus) may only develop partially or not at all; The genitals look like a typical female, and surgery can help with some of these issues. The ovaries typically are fully developed and functional. **Occurs in 1 in 5000. This person is female.**

iii. Turner's Syndrome (45,X): A condition that affects only females, results when one of the sex X-chromosomes is missing or partially missing. Turner syndrome can cause a variety of medical and developmental problems, including short height, failure of the ovaries to fully develop and heart defects. Turner's Syndrome and 45,X refer to the same genetic condition, often used interchangeably. Turner's Syndrome is the clinical diagnosis, while 45,X (or 45,XO) is the specific chromosomal karyotype where an individual is missing all or part of one X chromosome. It affects approximately **1 in 2,000**. **This person is female.**

iv. 4) Klinefelter's Syndrome: A genetic condition that results when a boy is born with an extra copy of the X chromosome. Klinefelter syndrome is a genetic condition

affecting males, affects testicular growth, reduced muscle mass, reduced body and facial hair, and enlarged breast tissue, but assisted reproductive procedures may make it possible for some men with Klinefelter syndrome to father children. Having the Y chromosome, the SRY gene, testes, testosterone, a masculinized brain, and able to produce sperm, **this person is male. Occurs in 1 in 650 males**.

v. Sex Chromosome Aneuploidies: Sex chromosome aneuploidies occurs where an individual has an atypical number of sex chromosomes. Some examples: **47,XXY** (Klinefelter syndrome) occurs in approximately **1 in 650 males**, while **47,XYY** (Jacobs syndrome) occurs in roughly **1 in 1,000 males**. Both conditions often remain underdiagnosed and are caused by random errors in cell division rather than inheritance. **Anytime a person possesses a sex-chromosome set that contains the SRY Gene (normally on the Y-Chromosome), their phenotype is male, they developed testes, produce testosterone, their brain has been masculinized, and are true males**.

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j. The birth of a clinical intersex child, far from being "a fairly common phenomenon," is a rare event, occurring in less than **1 out of every 5500** births (or for more severe intersex, **less than 1 in 10000**). The available data supports the conclusion that human sexuality is a dichotomy, not a continuum, since more than 99.99% of all humans can be clearly identified as being either male or female.

k. Also, based on the above data, the birth of a child with complete or significant sexual gonad ambiguity or disfunction (i.e., Swyer, Extreme-PAIS, or Ovotestes) is **less than 1 in 43000**.

l. Also, the term "Intersex" can never be used to describe people who have matching chromosomes, gonads, and genitalia. Therefore, a transgender person who has sex matching chromosomes, gonads, and genitalia can never claim to be "Intersex."

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General Biological Sex Development Table 4-30-26rd

Main Chromosomal Karyotype	XY	XYY w/ SRY gene (Aneuploidies)	XXY w/ SRY gene (Aneuploidies)	XY (MAIS)	XX (XX,Male)	MOSIAC (w/ SRY early bipotential gonadal cells, XX with XY)	XY (PAIS)	XY (CAIS)	XX
Possesses a functional SRY Gene expressed in early bipotential gonadal cells	YES	YES	YES	YES	YES	YES, but in Mosaism	YES	YES	N/A
DOES NOT HAVE functional SRY Gene expressed in early bipotential gonadal cells	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	YES
Possesses a Y-Chromosome	YES	YES	YES	YES	NO	YES	YES	YES	NO
Possesses Complete AIS (CAIS)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	YES	N/A
Possesses Partial AIS (PAIS)	N/A	N/A	N/A	N/A	N/A	N/A	YES	N/A	N/A
Possesses Mild AIS (MAIS)	N/A	N/A	N/A	YES	N/A	N/A	N/A	N/A	N/A
Testes Present & Functional	YES	High, 80% Functional	Low, 6% Functional	YES	Reduced Functionality	10% of the time	Greatly Reduced Function	Weak Function	NO
Present & Functional Ovaries	N/A	N/A	N/A	N/A	N/A	7% of the time	N/A	N/A	Yes

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Main Chromosomal Karyotype	XY	XYY w/ SRY gene (Aneuploidies)	XXY w/ SRY gene (Aneuploidies)	XY (MAIS)	XX (XX,Male)	MOSIAC (bipotential gonadal cells, XX with XY)	XY (PAIS)	XY (CAIS)	XX
Genitalia	Male	Male	Male	Male	Male	Range: Male, Intersex, Female	Range: Male (50%), Intersex (30%), Female (20%)	Female (blind)	Female
Brain Masculinized or Feminine	Masculinized	Partially masculinized	Masculinized	Masculinized	Partially masculinized	Partially masculinized	Partially masculinized	Female	Female
Fertility Level	Fully Fertile	80% Fertile	5% Fertile	Typically Fertile	Sterile	15% fertile (5% Male, 10% female)	Essentially Sterile	Sterile	Fully Fertile
Prevalence of Occurrence (for males or females)	99.8% of All Males (998 of 1,000)	0.100% of Males (1 of 1000)	0.100% of Males (1 of 1000)	0.010% of Males (1 of 10,000)	0.010% of Males (1 of 10,000)	~0.001% of Population (1 of 100,000)	~0.001% of Population (1 of 100,000)	0.005% of Females (1 of 20,000)	99.99% of all Females (9,999 of 10,000
THIS PERSON's SEX IS:	**Male (Typical)**	**Male**	**Male**	**Male**	**Male**	**Range:** Male (45%), **Intersex (30%),** Female (25%)	**Intersex**	**Female**	**Female (Typical)**

- **Male sexual development: When the Functional SRY gene is expressed in early bipotential gonadal cells at the correct time, initiating testis formation and male differentiation, regardless of chromosomal location (Y [typically] or X [atypically]).**
- **In rare CAIS cases and "extreme PAIS" cases, the testes may form, but "androgen signaling" fails, preventing normal male pathway development. Rare CAIS usually results in a sterile female (1 in 10,000], and rare "extreme PAIS" usually results in an "intersex" person (1 in 100,000]. AIS = Androgen Insensitivity Syndrome, C = Complete, P = Partial, M = Mild.**
- **Comment: In early embryos, the gonad is initially "bipotential" — meaning it can become: Testes (if SRY → SOX9 pathway is strongly activated) or Ovaries (if SRY is absent).**
- **Biological Sex Determination Hierarchy (Male/Female): 1) Functional Gonads, 2) Genital Phenotype, 3) Brain (Feminized or Masculinized), Chromosome Karyotype.**

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m. Comparison of Swyer, CAIS, 46,XX Male, and Ovotesticular DSD

Feature	Swyer syndrome (46,XY gonadal dysgenesis)	CAIS (Complete Androgen Insensitivity Syndrome)	46,XX male (De la Chapelle syndrome)	Ovotesticular DSD (Ovotestes)
Prevalence (live births)	~1 in **80,000** (PMC)	~1 in **50,000** (median of 1:20k–1:99k) (NCBI)	~1 in **20,000** males (NCBI)	Extremely rare (~5% of DSD; overall <<1 in 100,000 births)
Karyotype	46,XY	46,XY	46,XX	46,XX (most), or mosaic XX/XY
Gonads (type)	**Streak gonads** (nonfunctional) (NCBI)	**Testes (undescended)**	**Testes (small, dysgenetic)**	**Both ovarian + testicular tissue**
Functional ovaries (viable eggs)	❌ None	❌ None	❌ None	✅ Sometimes (ovarian tissue often functional; pregnancies reported) (PMC)
Functional testes (viable sperm)	❌ None	❌ No (infertile)	❌ No (azoospermia) (NCBI)	⚠️ Extremely rare (only isolated documented cases) (PMC)
Typical fertility	Infertile (may carry pregnancy with donor egg)	Infertile (no uterus)	Infertile	Rare fertility (mostly as female)
Primary cause	Failure of testis development (e.g., SRY or downstream gene defects)	Androgen receptor mutation → body cannot respond to testosterone	Usually, **SRY gene translocation** to X chromosome (NCBI)	Mixed/variable (SRY anomalies, mosaicism, SOX9, etc.) (NCBI)

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Feature	Swyer syndrome (46,XY gonadal dysgenesis)	CAIS (Complete Androgen Insensitivity Syndrome)	46,XX male (De la Chapelle syndrome)	Ovotesticular DSD (Ovotestes)
Hormonal environment (prenatal)	**Low testosterone**	**Normal testosterone, but ineffective**	**Testosterone present**	Variable (mixed ovarian/testicular output)
Brain masculinization (prenatal testosterone effect)	❌ No (no significant androgen exposure)	❌ No (androgen resistance prevents effect)	✅ Yes (testosterone active)	⚠️ Variable (depends on androgen exposure)
External genital phenotype	Female	Female	Male (usually typical)	Ambiguous or male/female spectrum
Internal reproductive structures	Female (uterus present)	No uterus (AMH from testes suppresses Müllerian structures)	Male internal structures	Mixed/variable
Suitability for intercourse	Yes (typical female anatomy)	Yes (short/blind vagina common but functional)	Yes (typical male anatomy)	Variable (depends on phenotype)
Orgasmic function	Yes (normal female neuroanatomy)	Yes (clitoral/vaginal sensation present)	Yes (male sexual function present, though fertility absent)	Variable but often present
Puberty (without treatment)	Absent (needs hormones)	Spontaneous breast development (from aromatized androgens)	Male puberty (often incomplete)	Variable

B) What is Rapid Onset Gender Dysphoria?

1) In on-line forums, parents have reported that their children seemed to experience a sudden or rapid onset of gender dysphoria, appearing for the first time during puberty or even after its completion. Parents describe that the onset of gender dysphoria seemed to occur in the context of belonging to a peer group where one, multiple, or even all of the friends have become gender dysphoric and transgender-identified during the same timeframe.

   Parents also report that their children exhibited an increase in social media/internet use prior to disclosure of a transgender identity. Recently, clinicians have reported that post-puberty presentations of gender dysphoria in natal females that appear to be rapid in onset is a phenomenon that they are seeing more and more in their clinic.

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Academics have raised questions about the role of social media in the development of gender dysphoria. (Dr. Lisa Littman, 2018, Signs of a Rapid Onset of Gender Dysphoria)

2) View the Video Links listed on the first page of this Session #4 for more information.

3) What is Gender Dysphoria (per American Psychiatric Association, APA's DSM-5):

The APA says Gender dysphoria refers to the distress that may accompany the incongruence between one's experienced or expressed gender and one's assigned gender (biological sex) …. previous DSM-IV term [was] "gender identity disorder." (DSM-5, page 452)

4) Prevalence of Gender Dysphoria among Adults (frequency of occurrence):

For natal [born] adult males, prevalence ranges from 0.005% to 0.014%, and for natal [born] adult females, from 0.002% to 0.003%. (DSM-5, page 454). Therefore, it appears once a person reaches adulthood, the occurrence of gender dysphoria is very rare.

5) So, the prevalence of gender-dysphoria occurring in adult individuals is less than 1 in 7100 for adult males and less than 1 in 33000 for adult females.

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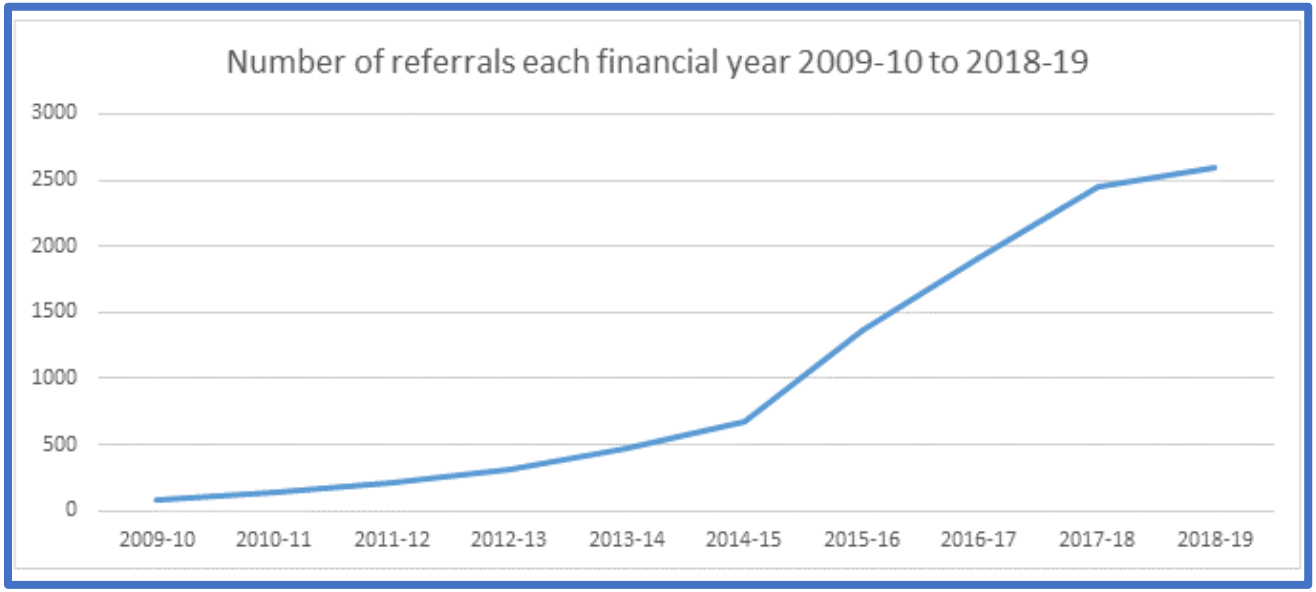
6) **Now Let's compare the rise of Gender Dysphoria among youths to the recent rise in the usage of smartphones and Social-Media among youths:**

A) Gender Dysphoria consultations increased 50X in Britain between 2010 to 2019:

Britain's "Tavistock" Gender Clinic



https://answers4seekers.org/wp-content/uploads/2024/08/2019-tavistock-referrals-to-the-gender-identity-development-service-gids-level-off-in-2018-19.pdf

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News Article: Britain's "Tavistock" Gender Clinic May Be Sued By Parents (8-11-22)

Daily Mail .com

Home | U.K. | News | Sports | U.S. Showbiz | Australia | Femail |

Tavistock transgender clinic is facing mass legal action 'from 1,000 families' | Daily Mail Online

Tavistock transgender clinic could face mass legal action 'from 1,000 families of children who claim they were rushed into taking life-altering puberty blockers' weeks after NHS shut it down in wake of damning report

- **Thousands of young people were treated by Tavistock centre in north London**
- **Many of them were prescribed powerful drugs to delay onset of adolescence**
- **But now the NHS has ordered it to be shut down in the wake of a damning report**

By MARTIN BECKFORD, POLICY EDITOR FOR THE DAILY MAIL
PUBLISHED: 04:26 EST, 11 August 2022 | **UPDATED:** 04:53 EST, 11 August 2022

uks-tavistock-transgender-clinic-is-facing-mass-legal-action-8-11-22

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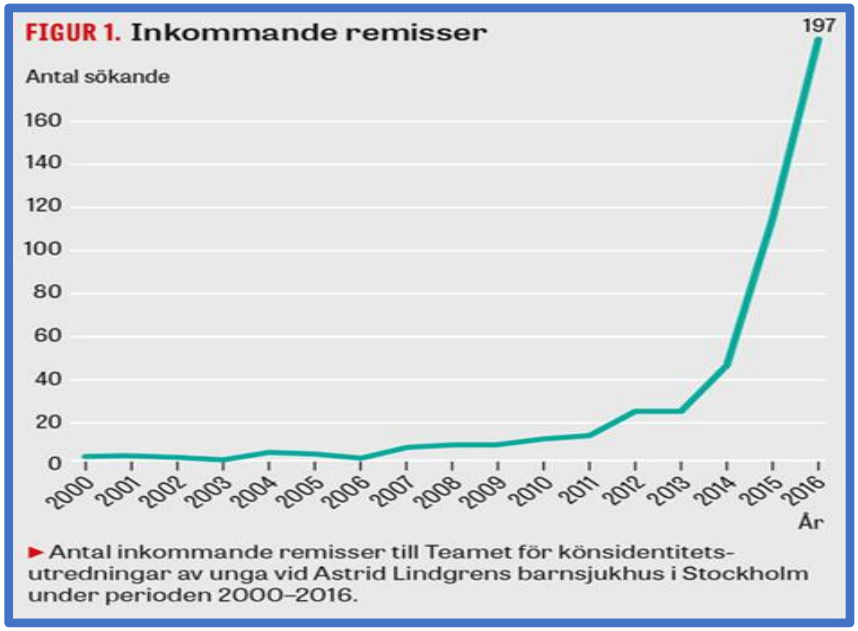
B) **Sweden's Astrid Lindgren's Children's Hospital, incoming referrals regarding gender dysphoria increased 65X from 2000 to 2016:**



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C) **In the US, transgender identification has increased 20X among young adults (Gen-Z) when compared to older adults (Baby Boomers) living in the US today.** (Gallup, 2021):

GALLUP, 2021	Transgender %
Generation	
Generation Z	2.1
Millennials	1.0
Generation X	0.6
Baby boomers	0.1

https://news.gallup.com/poll/389792/lgbt-identification-ticks-up.aspx

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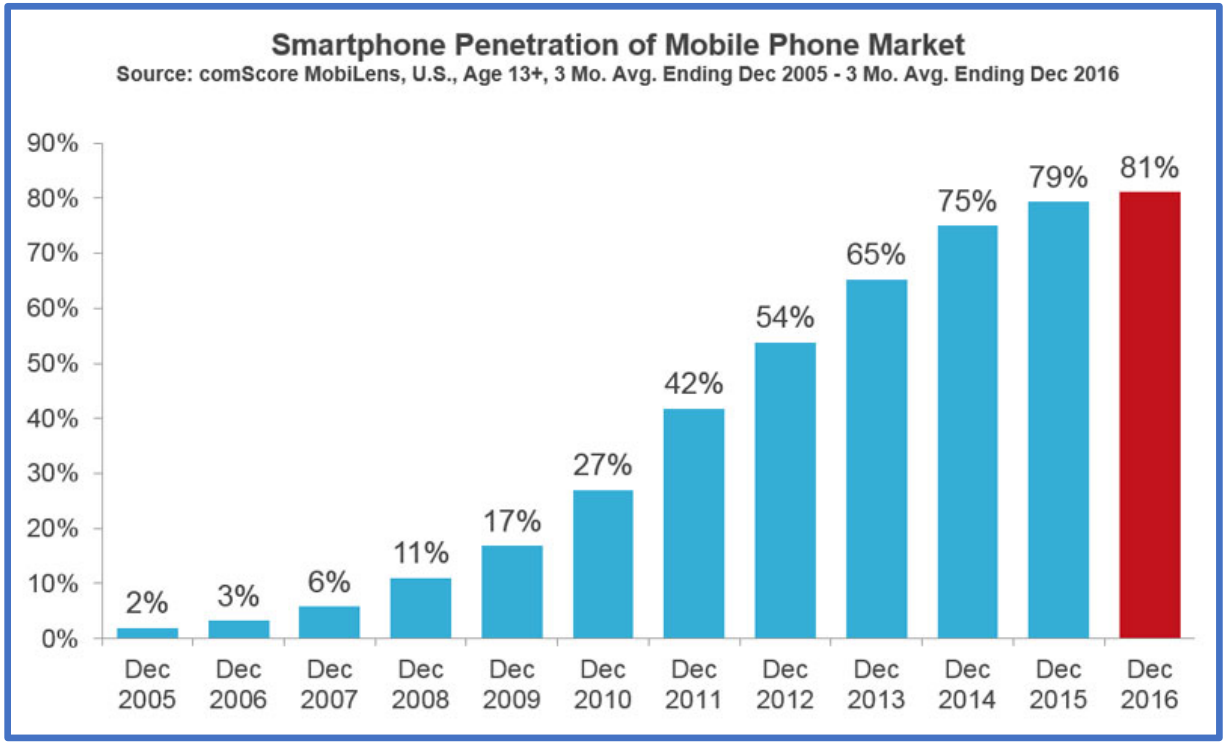
D) Smartphone Usage Increased from 2% in 2005 to 81% in 2016



https://www.comscore.com/Insights/Blog/US-Smartphone-Penetration-Surpassed-80-Percent-in-2016

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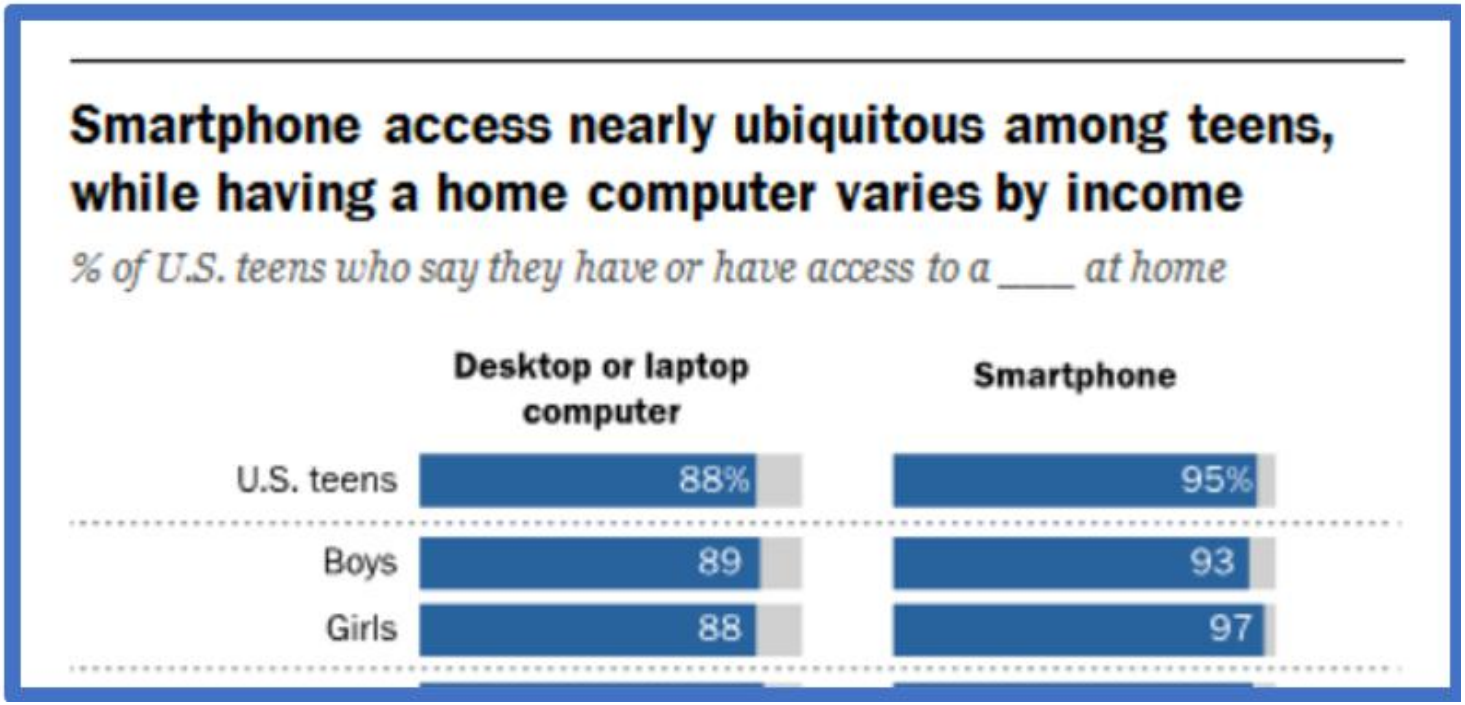
E) Smartphone Usage Among Teens is 95% in 2018



https://www.pewresearch.org/internet/2018/05/31/teens-social-media-technology-2018/

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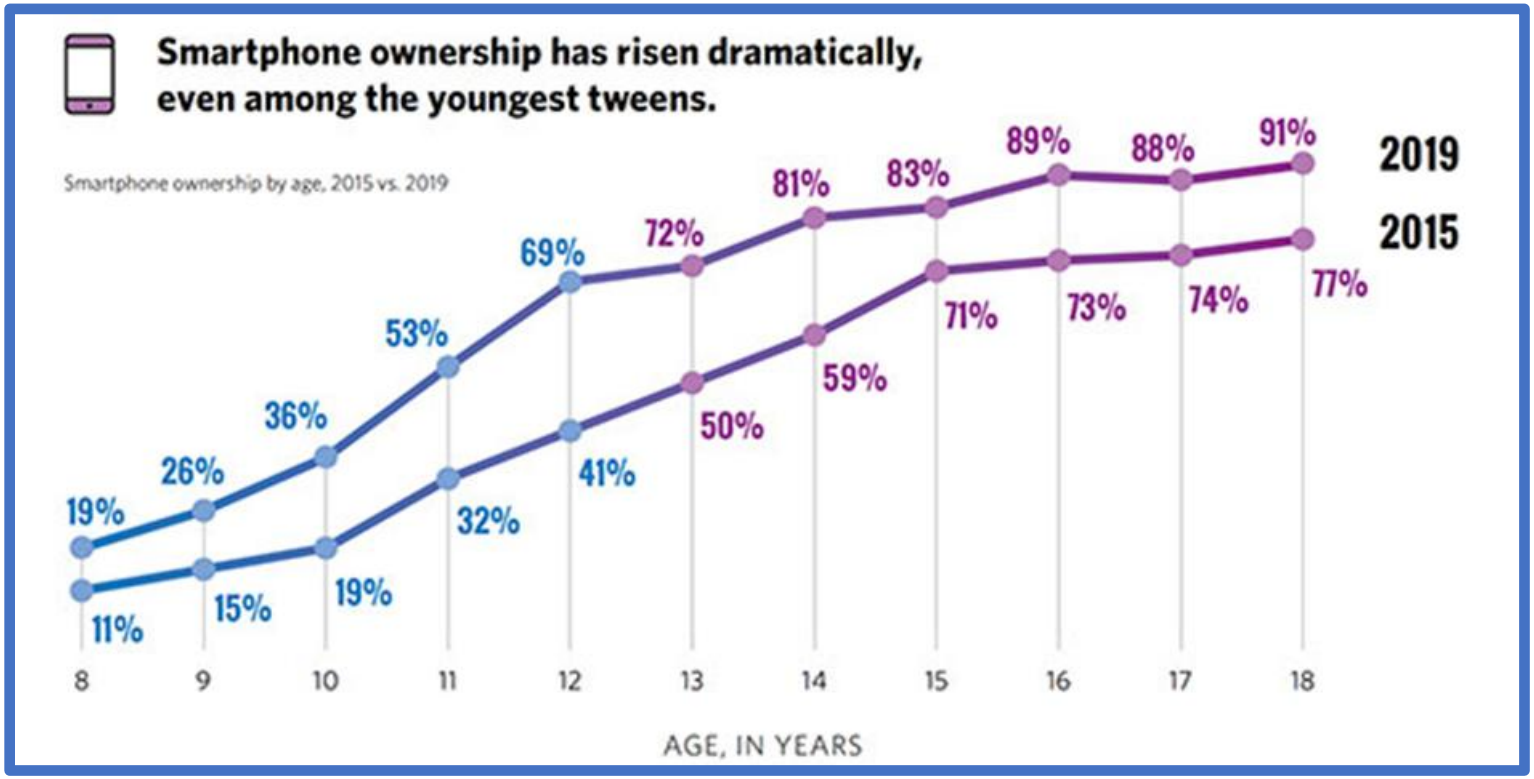
F) Smartphone Usage Among 12 year-olds is 69% in 2019



https://thejournal.com/articles/2019/10/30/how-teens-consume-digital-media.aspx

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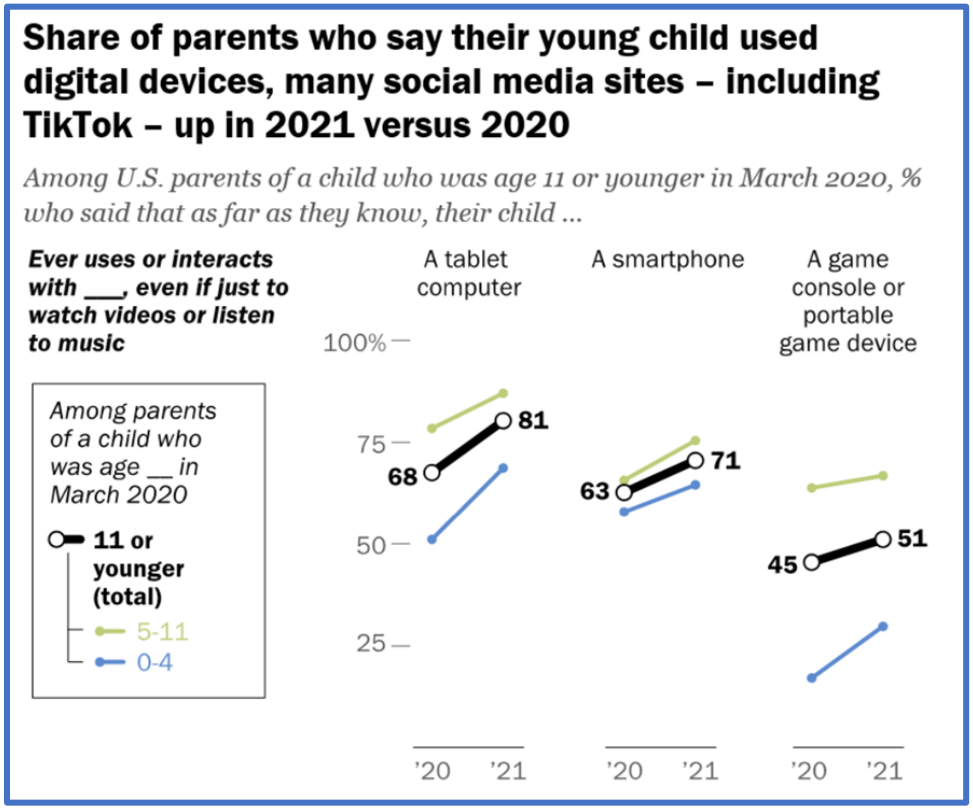
G) Smartphone Usage Among Children 1-4 years-old in 2021: 64%



https://www.pewresearch.org/fact-tank/2022/04/28/how-parents-views-of-their-kids-screen-time-social-media-use-changed-during-covid-19/

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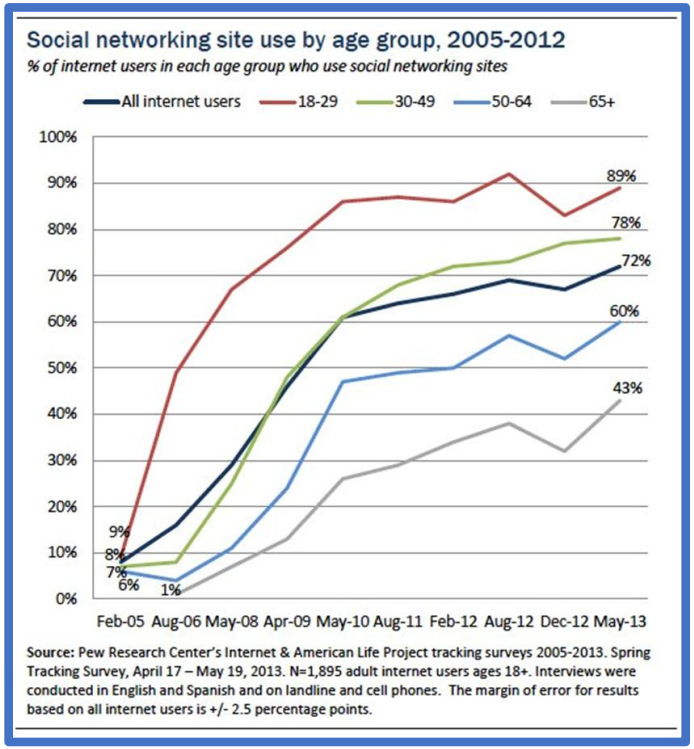
H) Social Media Use Grew from 8% in 2005 to 89% in 2013 (for 18+)



https://kamber.com.au/thinking/2013/11/social-media-trends-2014-part-two

C) Is Sexual Orientation fixed by Our Genes?

1) What are Identical Twins?

Identical twinning is officially described as monozygotic. Monozygotic twins form from a single (mono) fertilized egg (zygote). The zygote splits into two parts after conception, resulting in the development of two individual embryos. Because the two embryos are the result of a single egg/sperm combination, they have the same genetic origins and thus the same DNA.

The odds of having identical twins are about 1 in 333, whereas the birth rate for all twins (identical and fraternal) is about 1 in 30.

2) Identical TWIN's Study on Homosexuality

(**Reference**: My Genes Made Me Do It, Chapt-10, Whitehead, 2020)

Studies of twins have provided some of the strongest numerical evidence that "Our genes do not make us do it." In a nutshell, if you take pairs of identical twins in which one twin is homosexual, the identical co-twin (a monozygotic [MZ] twin) is usually not homosexual.

Jones and Yarhouse, examining the important Australian Bailey et al. (2000) SSA twin study paper, finds that for self-declared lesbians and gays the pairwise concordance is 14% and 11% respectively.

This means that for every nine (9) sets of male identical (MZ) twins (where one of the twins is a homosexual), one (1) twin-set will have both as homosexuals, while the other eight (8) twin-sets will be a mix of one homosexual and one heterosexual. Based on that study, a male identical MZ twin-set where both are homosexuals had an occurrence of only 11%, which is not very much.

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Homosexuality is not genetically inevitable. If it were, identical twins would show 100% concordance for SSA (Same-Sex Attraction), not 11%. This is a long way from genetic determinism.

That means, given that identical twins are always genetically identical, homosexuality cannot be genetically dictated.

That said, this does not mean that Same-Sex Attraction is never felt, it just means it is not determined by their genes; and since sexual orientation and attraction has evidence of fluidity, there exists the possibility for change, when desired.

Felt sexual attractions can also exist between heterosexual singles or heterosexual married persons, but these don't require an action. For example, a heterosexual sexual attraction of a single person or a married person towards another person (who is not their spouse) can be experienced, but to honor that person or their spouse (and if that person is a Christian, to honor Christ), that person can choose not to act on that sexual attraction.

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Regarding SSA changes in adults:

About half of those with exclusive SSA move towards heterosexuality over a lifetime. Put another way, 3% of the practicing heterosexual population (both men and women) claim to have once been either bisexual or homosexual.

- These changes are not therapeutically induced, but happen "naturally" in life, some very quickly.

- Most changes in sexual orientation are towards exclusive heterosexuality.

- The Numbers of people who have changed towards exclusive OSA (Opposite Sex Attraction) are greater than current numbers of bisexuals and exclusive SSA people combined. In other words, "Ex-gays outnumber actual gays."

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• Exclusive OSA is 17 times as stable as exclusive SSA for men, and 30 times as stable as exclusive SSA for women. (Women move about more in their sexual orientation than men.)

Regarding SSA changes in adolescence:

• Most teenagers will change from SSA. In fact, in the 16 to 17 year age group, 98% will move from homosexuality and bisexuality towards heterosexuality.

• 16-year-olds saying they are SSA or Bi-attracted are 25 times more likely to say they are opposite sex attracted at the age of 17 than those with a heterosexual orientation are likely to identify themselves as bi-sexual or homosexual.

• 16-year-olds who claim they are opposite sex attracted will overwhelmingly remain that way.

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D) Dysphoria Persistence, Suicide Rate, and Nuclear Family

- **Only 2.5% to 20% of all cases of GID [Gender Dysphoria] in childhood and adolescence result in a persistent gender identity disorder. (Korte, A; Goecker, D et al, 2008). 80%+ of gender identity disorders resolve in individuals after adolescence.**

- **According to APA's DSM-5 (pg 454), once a person reaches adulthood, the prevalence (occurrence within the population under review) of gender dysphoria averages only 1 in 5,892 adults (4 Males for every 1 Female).**

- **Wikipedia lists a total of 11 Transgender Youths who died by suicide since 1870 (past 154 years) in the United States.** Every suicide is tragic, but when compared to the total of 57,876 (calculated estimate) for all youth-aged suicides (15-24 for year-olds) in the US during past 13 years (2009-2023), it hides the bigger problem – Why are so many youths committing suicide?

- **Please see the charts below the inculcated that as the nuclear family dissolved youth suicides doubled**.
  - The historic annual Suicide rates among youth (15-24) from 1900 thru 1960 (pg 225) averaged **4.5/100K**.
  - Annual Suicide rates among youth (15-24) from 1970 thru 2016 averaged **10.6/100K**.
    - That is a 135% increase from the stable past.

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- **The start of the decline of the nuclear family in the United States can be estimated to be around 1965; it is interesting to note that youth suicide rates quickly doubled starting from the 1970's and have stayed doubled thru today.**
- **Adult Transgender people's attempted-suicide rate is 8.9 times more than that of the average US population (lifetime rate, 40.4% vs 4.6%).** (Why, as adults, are their suicide attempt rates still 9X of the US population? Is gender transitioning not the solution that they are really needing?)
- **The Transgender Suicide-attempt rate remains very high throughout their whole adult life, especially from age 18 thru age 64 (please see page 10).**

See chart below

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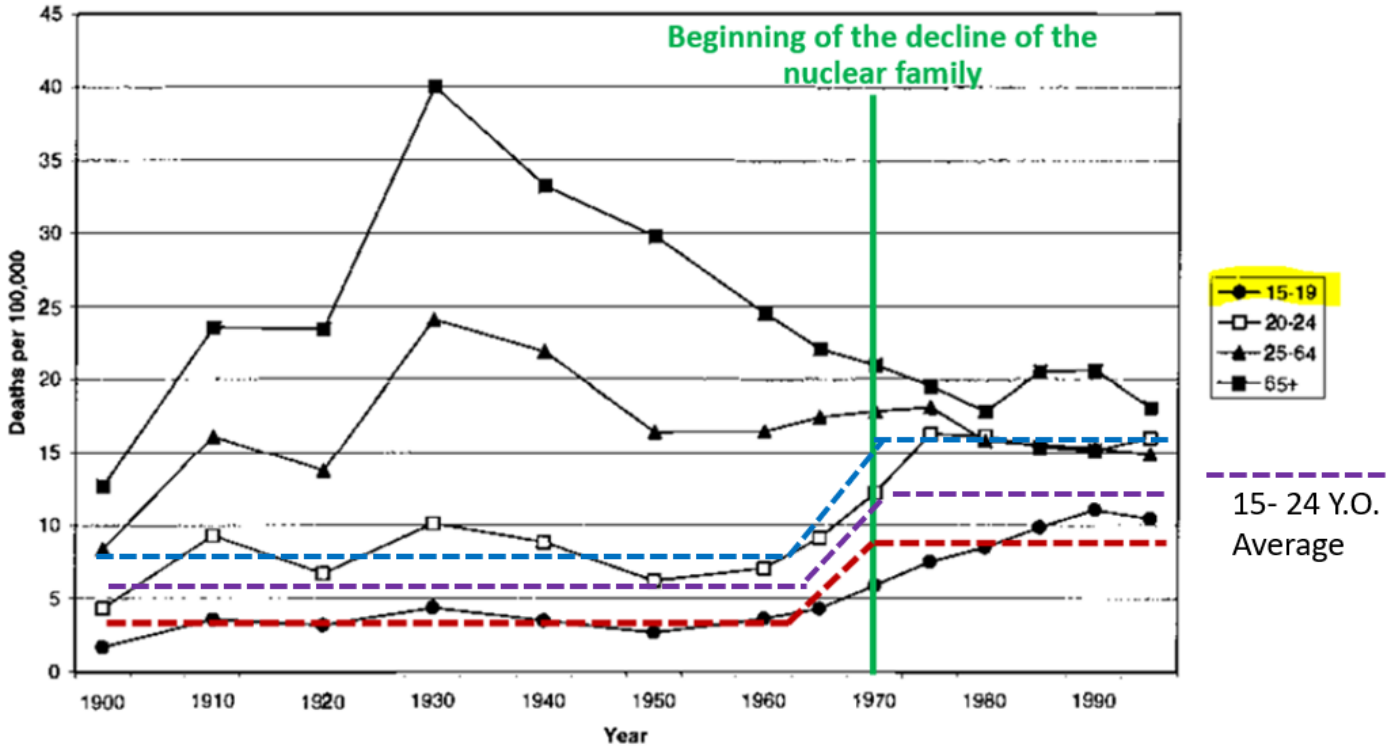

Fig. 5.2 Suicide rates by age over time

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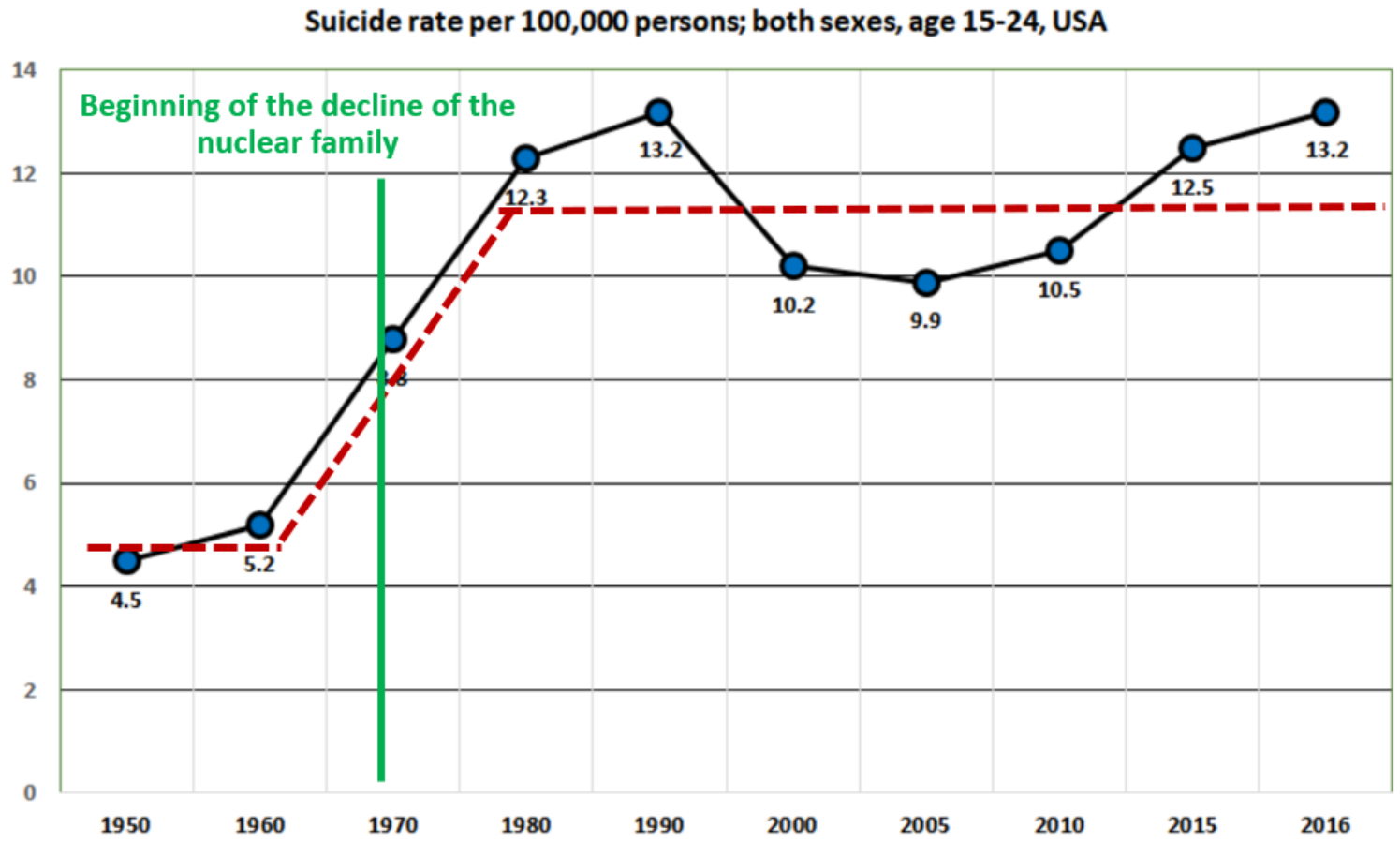

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E) EXHIBITS:

a. Exhibit A:

Comparison of Complete and Partial Androgen Insensitivity Syndrome.

Occurs when body is insensitive to androgens (male hormones like testosterone). Because the body can't respond to male hormones: External appearance is typically fully female. They develop breasts at puberty (from estrogen converted from testosterone). Being XY, they do not develop a uterus or ovaries.

Feature	CAIS	Extreme (Severe) PAIS	Very Mild PAIS
Sex chromosomes	XY	XY	XY
androgen receptor gene location	X chromosome (Xq11–q12)	X chromosome (Xq11–q12)	X chromosome (Xq11–q12)
AR gene function	Nonfunctional	Severely reduced function	Mildly reduced function
Prevalence (approx.)	~1 in 60,000	~1 in 250,000	~1 in 55,000
SRY (Testes) gene present?	Yes	Yes	Yes
Testes formed?	Yes	Yes	Yes
Testes location	Undescended (abdomen/groin)	Often undescended	Usually descended

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Viable sperm	No	Usually no	Sometimes possible (rare)
Brain androgen effect (testosterone)	Very minimal → more feminized pattern	Intermediate (partial masculinization)	Near typical male pattern
Brain overall pattern	Feminized	Mixed	Masculinized
Phenotype at birth	Female-typical external genitalia	Ambiguous or under-virilized genitalia	Male-typical genitalia
Phenotype after puberty	Breast development; no virilization	Variable: some virilization, gynecomastia possible	Mostly male puberty; may have gynecomastia or reduced hair
External genitalia	Vagina + clitoris present	Variable: small penis / enlarged clitoris, hypospadias	Penis, usually functional
Vagina present?	Yes (blind-ending)	Often partial or ambiguous structure	No
Clitoris present?	Yes	Often enlarged phallic/clitoral structure	No
Orgasm possible?	Yes	Yes (variable)	Yes
Typical gender identity / social sex	Female	**Often female or ambiguous assignment**	Male
Biological Sex Type	Female	**(This Person Is Intersex)**	Male

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b. Exhibit B:

Congenital Adrenal Hyperplasia (CAH) Comparison Table

Congenital Adrenal Hyperplasia (CAH) is not a sex development defect, but it is an **adrenal steroid pathway disorde**r: Low cortisol, High androgen precursors. Resulting in excess androgen exposure in fetuses.

Feature	Mild / Non-classic CAH (46,XX)	Classic / Severe CAH (46,XX)	Mild / Non-classic CAH (46,XY)	Classic / Severe CAH (46,XY)
Chromosome Karyotype	**XX Female**	**XX Female**	**XY Male**	**XY Male**
CAH Comparison Table (Severe and Mild)	~40% of CAH	3% (of CAH)	~40% of CAH	3% (of CAH)
Defective Gene Location	Chromosome 6p21.3 CYP21A2 gene	Chromosome 6p21.3 CYP21A2 gene	Chromosome 6p21.3 CYP21A2 gene	Chromosome 6p21.3 CYP21A2 gene
Prevalence	~1:1000 (varies by population, XX&XY)	~1:14,000 births (XX & XY)	~1:1000 (varies by population, XX&XY)	~1:14,000 births (XX & XY)
SRY gene (Develop testes)	N/A	N/A	Present	Present

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Brain androgen influence (typical trend)	remains mostly feminine	Higher prenatal androgen exposure in XX → partial masculinization	masculinized	masculinized
Functional gonads	Ovaries	Ovaries	Testes	Testes (may be undescended and not fully functional)
Fertility (sperm/eggs)	Usually preserved	Reduced fertility risk but possible	Usually preserved	Often yes, but can be impaired (e.g., testicular adrenal rest tumors)
External genitalia at birth	normal female	Often Clitoromegaly (Often ambiguous genitalia, with enlarged clitoris)	Normal male	Normal male
Internal reproductive organs	Uterus, ovaries present	Uterus, ovaries present	Normal male ducts	Normal male ducts
Able to get pregnant and carry and deliver baby	Often normal fertility	**Yes, but reduced fertility** (surgery and hormones may be needed)	N/A	N/A
Surgery typically needed to optimize genitalia	No	**Yes**	No	No

Orgasm capability	Yes	Yes	Yes	Yes
Typical sex of rearing	Female	Female	Male	Male
Biological Sex Type	**Female**	**Female**	**Male**	**Male**