



**UNITED STATES DISTRICT COURT  
MIDDLE DISTRICT OF ALABAMA  
NORTHERN DIVISION**

REV. PAUL A. EKNES-TUCKER,	)	
<i>et al.</i> ,	)	
	)	
<i>Plaintiffs</i> ,	)	
	)	
v.	)	No. 2:22-cv-00184-LCB-SRW
	)	
KAY IVEY, in her official capacity	)	
as Governor of the State of Alabama,	)	
<i>et al.</i> ,	)	
	)	
<i>Defendants.</i>	)	

**DECLARATION OF QUENTIN L. VAN METER, M.D.**

My name is Quentin L. Van Meter. I am over the age of 19, I am qualified to give this declaration, and I have personal knowledge of the matters set forth herein.

My CV is attached to this declaration. My recent publications in the *Journal of Clinical Endocrinology and Metabolism* are listed on my CV.

In the past four years, I have provided expert testimony in state legislative committee hearings in Alabama, Pennsylvania, Missouri, Iowa, and California, and I have been deposed as an expert witness in Virginia, Ohio, Missouri, and Georgia:

- 2018: Court of the Queens Bench Ontario, court file 1808-00144, deposed
- 2018: Sieffert v Hamilton Co Ohio, court testimony
- 2019: Gavin Grimm v Gloucester Co Virginia School Board, deposed
- 2019: Multiple Plaintiffs v State of Ohio Bureau of Records, deposed
- 2020: Loughman v Loughman, Harris County, Texas, deposed
- 2021: Spahr v Spahr, St Louis County, MO, court testimony
- 2021: Laura Cauthen v James Cauthen, Cobb County GA, court testimony

I am compensated at the rate of \$350.00 per hour for record review and document preparation and \$450.00 per hour for deposition or court testimony on this matter. My compensation is not dependent upon the substance of my opinions or the outcome of the case.

### **Qualifications**

I have been retained by counsel for Defendants as an expert in connection with the above-captioned litigation. I have actual knowledge of the matters stated in this declaration. My professional background, experience, and publications are detailed in my curriculum vitae. A true and accurate copy is attached as Exhibit A to this declaration. I received my B.A. in Science at the College of William and Mary and my M.D. from the Medical College of Virginia, Virginia Commonwealth University. I am currently a pediatric endocrinologist in private practice in Atlanta, Georgia. I am the President of Van Meter Pediatric Endocrinology, P.C. I am on the clinical faculties of Emory University School of Medicine and Morehouse College of Medicine, in the role of adjunct Associate Professor of Pediatrics. I am board certified in Pediatrics and Pediatric Endocrinology. I have been licensed to practice medicine in Georgia since 1991. I have been previously licensed to practice medicine in California, Louisiana, and Maryland.

I did my Pediatric Endocrine fellowship at Johns Hopkins Hospital from 1978-1980. The faculty present at that time had carried on the tradition of excellence established by Lawson Wilkins, M.D. Because of the reputation of the endocrine program as a center for exceptional care for children with disorders of sexual differentiation, I had well-above average exposure to such patients. As a Pediatric Fellow, I was also exposed to adults with Gender Identity Disorder, then called Trans-Sexuality, and received training from John Money, Ph.D., in his Psychohormonal Division.

### **Differentiation in the Fetus**

From the moment of conception, a fetus is determined to be either a male (XY), female (XX), or in rare cases, to have a combination of sex-determining chromosomes, many of which are not compatible with life, and some of which are the cause of identifiable clinical syndromes.

The presence of a Y chromosome in the developing fetus directs the developing gonadal tissue to develop as a testicle. The absence of a functional Y chromosome allows the gonadal tissue to develop as an ovary. Under the influence of the mother's placental hormones, the testicle will produce testosterone which directs the genital tissue to form a penis and a scrotum.

Simultaneously, the testicle produces anti-Müllerian Hormone (AMH) which regresses development of the tissue that would otherwise develop into the uterus, fallopian tubes, and upper third of the vagina. This combination of actions in early fetal development is responsible for what we subsequently see on fetal sonograms, and what we observe at birth as male or female genitalia. It is only when the genital structures are ambiguous in appearance that sex assignment is withheld until a thorough expert team evaluation has occurred.

For reasons most often occurring as random events, there are malfunctions of the normal differentiation. These aberrations of normal development are responsible for what we classify as Disorders of Sexual Differentiation (DSD), and they represent a very small fraction of the human population. The incidence of such circumstances occurs in 1:4500 to 1:5500 births.<sup>1</sup> Sex is binary, male or female, and is determined by chromosomal complement and corresponding reproductive role. The exceedingly rare DSDs are all medically identifiable deviations from this sexual binary norm. The 2006 consensus statement of the Intersex Society of North America and the 2015 revision of the Statement do not endorse DSD as a third sex.<sup>2</sup> DSD outcomes range from appearance of female external genitalia in an XY male (complete androgen insensitivity syndrome) to appearance of male external genitalia in an XX female (severe congenital adrenal hyperplasia).

As one would expect, there are variations of the degree of hormonally driven changes that create ambiguous genital development that prevent assigning of a specific classification as

either male or female at birth. DSD patients are not “transgender”; they have an objective, physical, medically verifiable, physiologic condition. Transgender people generally do not have intersex conditions or any other verifiable physical anomaly. People who identify as “feeling like the opposite sex” or “somewhere in between” do not comprise a third sex. They remain biological men or biological women.

In some DSDs there exist more than one set of chromosomes. When there is a divergence of the appearance of the external genitalia from the chromosomally determined sex due to the presence of both an ovarian and testicular cell lines in a patient simultaneously, the patient is classified as having ovo-testicular DSD (formerly termed a true hermaphrodite). When there is a disruption in the development of genital structures but there is solely testicular tissue present in the chromosomal male or solely ovarian tissue in the chromosomal female, the term 46 XY DSD or 46 XX DSD is used instead respectively (formerly termed male pseudohermaphrodite or female pseudohermaphrodite).

The decision to assign a sex of rearing is complex and is specific to the diagnosis. Patients with complete androgen insensitivity (CAIS) are XY DSD but are never reared as a male. Because testosterone never influences development, they become happy, functional female adults with infertility. Females with severe congenital adrenal hyperplasia (CAH) are XX DSD but are not reared as males despite the male appearance of the genitalia at birth. Although these girls may show a tendency for male play behaviors as children, they generally assume a female sexual identity. Therapeutic interventions in the DSD individuals from infancy onward are aimed at what function can be expected from their disordered sexual anatomy in terms of function and fertility. Most often, the chromosomal sex aligns with the sex of rearing.

## **Gender Identity**

“Gender” is a term that refers to the psychological and cultural characteristics associated with biological sex. It is a psychological concept and sociological term, not a biological one. The term gender possessed solely a linguistic meaning prior to the 1950s. This changed when sexologists of the 1950s and 1960s co-opted the term to conceptualize cross-dressing and transsexualism in their psychological practice. “Gender identity” is a term coined by my former endocrine faculty member John Money in the 1970s and has come to refer to an individual’s mental and emotional sense of being male or female. The norm is for individuals to have a gender identity that aligns with one's biological sex.

Gender discordance (formerly Gender Identity Disorder) is used to describe a psychological condition in which a person experiences marked incongruence between his experienced gender and the gender associated with his biological sex. He will often express the belief that he is the opposite sex. Gender discordance occurs in 0.001% of biological females and in 0.0033% of biological males.<sup>3</sup> Exact numbers are hard to document since reporting is often anecdotal. Gender discordance is not considered a normal developmental variation.

“Gender Dysphoria” is a diagnostic term to describe the emotional distress caused by gender incongruity.<sup>4</sup> John Money played a prominent role in the early development of gender theory and transgenderism. He understood gender to be “the social performance indicative of an internal sexed identity.”<sup>5</sup> He joined the Johns Hopkins faculty in 1951 specifically to have access to children diagnosed with DSD, hoping to prove his theory that gender was arbitrary and fluid. Money experimented with DSD infants by assigning them to the opposite biological sex through surgical revision, counseling, and hormonal manipulation during puberty. His mode of operation was to have a theory and then experiment with patients to see how his theory worked. This kind

of endeavor does not anticipate or prevent adverse outcomes and is the antithesis of ethical science. Money never submitted his research proposals for review; today, Institutional Review Boards (IRBs) serve to rigorously review proposed clinical research protocols to prevent all potential and real harm to patients.

Because of his experience with infants, Money initially garnered support from endocrine colleagues and surgical colleagues, and Johns Hopkins became a renowned center for care of patients with DSD in the 1970s, garnering referrals from around the world. Follow-up studies on these infants later showed, however, that altering their natal sexual identity via social intervention could lead to severe psychological harm. Clinical case reports of children with DSD have revealed that gender identity is indeed not immune to environmental input.<sup>6</sup>

Meanwhile, Money had expanded into the field of adult patients with persistent gender identity disorder. This very small group of patients chose voluntarily, as adults, to enter a very precise protocol which began with living socially as the opposite sex for a year, eventually receiving hormonal therapy to change their physical appearance to some extent. The final step was surgical revision of the body structures that would otherwise be at odds with their desired gender. This small group of patients was followed for a number of years past their final surgical procedures and required continuous counseling. These patients expressed some degree of subjective satisfaction but showed no objective improvement in overall wellbeing.<sup>7</sup> The legacy of John Money fell into disrepute and the transsexual treatment program at Johns Hopkin was closed in the 1980s based on the lack of evidence that this protocol produced an effective cure.

### **Etiology of Gender Disorders**

Transgender affirming professionals claim transgender individuals have a "feminized brain" trapped in a male body at birth and vice versa based upon various brain studies. Diffusion-

weighted MRI scans have demonstrated that the pubertal testosterone surge in boys increases white matter volume. A study by Rametti and colleagues found that the white matter microstructure of the brains of female-to-male (FtM) transsexual adults, who had not begun testosterone treatment, more closely resembled that of men than that of women.<sup>8</sup> Other diffusion-weighted MRI studies have concluded that the white matter microstructure in both FtM and male-to-female (MtF) transsexuals falls halfway between that of genetic females and males.<sup>9</sup> These studies, however, are of limited clinical significance due to the small number of subjects and failure to account for neuroplasticity.

Neuroplasticity is the well-established phenomenon in which long-term behavior alters brain microstructure. For example, the MRI scans of experienced cab drivers in London are distinctly different from those of non-cab drivers, and the changes noted are dependent on the years of experience.<sup>10</sup> There is no evidence that people are born with brain microstructures that are forever unalterable, but there is significant evidence that experience changes brain microstructure.<sup>11,12</sup> Therefore, any transgender brain differences would more likely be the result of transgender behavior than its cause.

Furthermore, infants' brains are imprinted prenatally by their own endogenous sex hormones, which are secreted from their gonads beginning at approximately eight weeks' gestation.<sup>13,14,15</sup> There are no published studies documenting MRI-verified differences in the brains of gender-disordered children or adolescents. The DSD guidelines also specifically state that current MRI technology cannot be used to identify those patients who should be raised as males or raised as females.<sup>16</sup> Behavior geneticists have known for decades that while genes and hormones influence behavior, they do not hard-wire a person to think, feel, or behave in a particular way. The science of epigenetics has established that genes are not analogous to rigid

“blueprints” for behavior. Rather, humans “develop traits through the dynamic process of gene-environment interaction. ... [genes alone] don't determine who we are.”<sup>17</sup>

Regarding transgenderism, twin studies of adults prove definitively that prenatal genetic and hormone influence is minimal. The largest twin study of transgender adults found that only 20 percent of identical twins were both transgender-identified.<sup>18</sup> Since identical twins contain 100 percent of the same DNA from conception and develop in exactly the same prenatal environment exposed to the same prenatal hormones, if genes and/or prenatal hormones contributed to a significant degree to transgenderism, the concordance rates would be close to 100 percent. Instead, 80 percent of identical twin pairs were discordant. This difference would indicate that at least 80 percent of what contributes to transgenderism as an adult in one co-twin consists of one or more non-shared post-natal experiences including but not limited to non-shared family experiences. These findings also mean that persistent GD is due predominately to the impact of nonshared environmental influences. These studies provide compelling evidence that discordant gender is not hard-wired genetically.

### **Gender Dysphoria vs. Gender Identity Disorder**

Up until the recent revision of the DMS-IV criteria, the American Psychological Association (APA) held that Gender Identity Disorder (GID) was the mental disorder described as a discordance between the natal sex and the gender identity of the patient. Dr. Kenneth Zucker, who is a highly respected clinician and researcher from Toronto, carried on evaluation and treatment of GID patients for forty years. His works, widely published, found that the vast majority of boys and girls with GID identify with their biological sex by the time they emerge from puberty to adulthood, through either watchful waiting or family and individual counseling.<sup>19</sup> His results were mirrored in studies from Europe.<sup>20,21</sup>



When the DMS-V revision of the diagnosis of GID was proposed by the APA committee responsible for revision, Dr. Zucker strongly opposed the change to the term Gender Dysphoria, which purposefully removed gender discordance as a mental disorder apart from the presence of significant emotional distress. With this revision, Gender Dysphoria describes the mental anguish which is experienced by the gender discordant patient. The theory that societal rejection is the root cause of Gender Dysphoria was validly questioned by a study from Sweden which showed that the dysphoria was not eliminated by hormones and sex reassignment surgery even with widespread societal acceptance.<sup>22</sup>

### **Treatment of Gender Dysphoria**

The treatment of children and adolescents with gender discordance and accompanying gender dysphoria should include an in-depth evaluation of the child and family dynamics. This evaluation provides a basis on which to proceed with psychologic therapy. The entire biologic and social family should be involved in psychological therapy designed to assist the patient, if at all possible, to align gender identity with natal sex. Psychological support by competent counselors with an intent of resolving the gender conflict should be provided as long as the patient continues to suffer emotionally. Given the high degree of eventual desistance of gender discordance/dysphoria by the end of puberty, it would be ethical and logical to counsel the patient and family to rear the child in conformity with natal sex.

There should be no interruption of natural puberty. Natural pubertal maturation in accordance with one's natal sex is not a disease. It is designed to carry malleable, immature children forward to be healthy adults capable of conceiving their own progeny. Puberty affects physical changes, some of them painful, unique to the natal sex to reflect the laws of nature. Interruption of puberty has been reserved for children who begin puberty at an age much

younger than normal in an effort to preserve final height potential and avoid the social consequences of precocious maturation.

There are a number of physical changes that are a consequence of normally timed puberty that could be classified as disadvantageous: changes in body proportions can alter success with dance and gymnastics; acne can be severe and disfiguring; a boy soprano can suddenly hardly carry a tune. It has not been the ethical standard of care to stop puberty so that these changes can be circumvented. Erikson described the stage of adolescence as "Identity versus Role Confusion" during which the teen works at developing a sense of self by testing roles then integrating them into a single identity.<sup>23</sup> This process is often unpleasant regardless of the presence or absence of gender identity conflicts. The major benefit of enduring puberty in a GD patient is that it provides a strong likelihood of alignment of his gender identity with his natal sex. There is no doubt that these patients need compassionate care to get them through their innate pubertal changes.

The light at the end of the tunnel is the proven scientific evidence that 80%- 95% of pre-pubertal children with GD will come to identify with their biological sex by late adolescence. Some will require lifelong supportive counseling while others will not.<sup>24</sup> Intervention at a young age with gonadotropin releasing hormone analogs (often referred to as puberty blockers) to either stop puberty early on or prevent it from starting before it naturally occurs is suggested by guidelines developed by WPATH without scientific basis. There is evidence that bone mineral density is irreversibly decreased if puberty blockers are used during the years of adolescence.<sup>25</sup> To treat puberty as a pathologic state of health that should be avoided by using puberty blockers (GnRH analogs) is to interrupt a major necessary physiologic transformation at a critical age when such changes can effectively happen. We have definite evidence of the need for estrogen in

females to store calcium in their skeleton in their teen years. That physiologic event can't be put off successfully to a later date. It is very difficult to imagine ethical controlled clinical trials that could elucidate the effects of delaying puberty until the age of consent.

The use of cross-sex hormones during this same time frame has no basis of safety and efficacy. The use of such treatment in adults raises scientifically valid concerns that were amply expressed in the 2009 Endocrine Society Guidelines on Transgender treatment. The next step in WPATH-recommended intervention is to use cross-sex hormone therapy during the time when the patient would naturally be experiencing endogenous pubertal changes. This too is not based on scientifically proven theories. The use of cross-sex hormones can cause permanent infertility.<sup>26</sup>

The final recommended step is so-called "sex reassignment surgery," which can include surgical removal of the breasts in natal females, or removal of the penis and scrotum in natal males. Each of these steps has adverse outcomes, some reversible and others not. Mastectomies leave scars, and there is great difficulty in creating a functional vaginal-like orifice, and certainly no success in creating an innervated erectile penis where none existed previously. Sex reassignment surgery is, by nature, permanent.

### **Recurrent Themes in the Plaintiff Declarations**

Puberty blockers are stated to be completely reversible in their effects on the adolescent who has entered puberty based on clinical studies in young children with precocious puberty who have been treated with these drugs. This is comparing apples to oranges. Precocious puberty, by definition, is defined as puberty which starts before the 8<sup>th</sup> birthday for a female child or the before the 9<sup>th</sup> birthday in a male child. The end of treatment is carefully timed so that resumption of puberty occurs at the average age for females (10.5 years) and males (11.5 years). This allows

the necessary functions of puberty to prepare the body for reproduction and affects the bones, gonads, and brain, among other body systems. On the other hand, blocking puberty at the age of normal puberty prevents the needed accretion of calcium into the skeleton and prevents the maturation of the gonads. There is no long-term data that compares bone, gonad, and brain health in pubertal-aged patients who have had puberty interrupted and those who have not, as was noted as a concern in the Endocrine Society Guidelines. There are no such ongoing studies completed that guarantee the full reversibility of blocking puberty in this age group, but there is evidence that normal bone density can't be fully reestablished. Without any verifiable safety data, using the puberty blockers for interrupting normal puberty is not a sanctionable off-label use of these drugs and is therefore to be considered uncontrolled, non-consentable experimentation on children.

It has been stated that the plaintiffs are only asking that established standards of care be followed. There are no standards of care for transgender health. Standards of care established by broad consensus are reached by inclusion of the whole spectrum of opinions, clinical experience and published science in the formation thereof. The guidelines published by WPATH,<sup>27</sup> the Endocrine Society,<sup>26,28</sup> the American Academy of Pediatrics,<sup>29</sup> and the Pediatric Endocrine Society<sup>30</sup> are solely the opinions of like-minded practitioners who excluded any contrary opinion. The Endocrine Society Guidelines, as mentioned before, clearly stated that they are not to be considered standards of care. Before true consensus-driven standards of care are established for the treatment of transgender patients of all ages, following the current guidelines is risky experimentation.

The plaintiff declarations repeatedly refer to the established increased risk of suicide if any of the affirmation strategies are not followed to completion. There are only two total

population studies in the peer-reviewed medical literature.<sup>22,31,32</sup> They show that when every recorded case in the population of Sweden was analyzed, neither medical affirmation or medical affirmation followed by surgical affirmation improved the mental health of the patients in the long run.

Finally, I am curious about the clear lack of documentation of references in the plaintiffs' declarations. They are merely stating their personal opinions without supporting evidence and relying on anecdotal case reports.

Pursuant to 28 U.S.C. § 1746, I declare under penalty of perjury that the foregoing is true and correct. Executed on 1 May, 2022.

  
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Quentin L. Van Meter, M.D.

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**updated 29 April, 2022**

**PERSONAL**

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**EDUCATION:**

Undergraduate: College of William & Mary, 1969  
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Medical School: Medical College of Virginia, 1973  
M.D. – 1973

**CLINICAL TRAINING:**

Institution: The University of California, San Francisco  
Hospital: Naval Regional Medical Center, Oakland  
Position: Pediatric Intern – 1973 – 1974  
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Institution: Johns Hopkins University  
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Position: Fellow, Pediatric Endocrinology 1978 – 1980  
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**PROFESSIONAL CERTIFICATION & SOCIETIES:**

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Fellow: American Academy of Pediatrics, Georgia Chapter 1975 -present  
President, Uniformed Services West Chapter, 1987 – 1990  
District VIII member, AAP Committee on Awards for  
Excellence in Research, 1990-1994  
Editor, The Georgia Pediatrician, 1994 – 1998  
  
Chairman, Georgia Chapter Legislative Committee, 1996 – 2006

Fellow: The American College of Pediatricians, 2007 – present  
Member of the Board of Directors, 2008- present  
President, 2018-present

Member: Pediatric Endocrine Society, 1989 – present

Member: American Diabetes Association Professional Section, 1988 – present

Member: Endocrine Society, 1994-present

Member: Southern Pediatric Endocrine Society, 1992 – Present

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Position: Assistant Clinical Professor, Pediatrics, 1980 – 1986

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#### MILITARY SERVICE:

Commission: Medical Corps, United States Navy, August 1971  
Rank: Captain, retired  
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Intern and Resident, Pediatrics, Naval Regional Medical Center,  
Oakland, 1973 – 1976  
  
Staff Pediatrician, Naval Regional Medical Center,  
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PUBLICATIONS: (Articles in Peer Reviewed Journals)

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Laidlaw, MK Von Mol A, Van Meter Q, and Hansen JE, Letter to the Editor from Laidlaw et al: “erythrocytosis in a large cohort of transgender Men using testosterone: a long-term follow-up study on prevalence, determinants, and exposure years” J Clin Endocrinol Metab, 2021 December 2021, e5275-35276 <https://doi/10.1210/clinem/dg ab514>

ABSTRACTS/LETTERS:

Van Meter, Q L, & Lee, PA: “Evaluation of Puberty in Male and Female Patients with Noonan Syndrome,” Pediatric Research 14:485, 1980.

Van Meter, QL, et al: "Characterization of Pituitary Function in Double Bolus GnRH Infusion as a Diagnostic Tool," Pediatric Research 32:111, 1984.

Van Meter, QL, Felix, SD, Lin, FL: "Evaluation of the Pituitary-Adrenal Axis in Patients Treated with nasal Beclomethasone," (Presented at the 1991 Annual Meeting of the Endocrine Society and the 6<sup>th</sup> Annual Naval Academic Research Competition, Bethesda, MD, 17 May, 1991).

Rogol AD Midyett LK Van Meter Q, Frane J, Baily J, and Bright GM, Recombinant Human IGF-1 for Children with Primary IGF-1 Deficiency (IGFD): Safety Data from Ongoing Clinical Trials (presented at the PAS 2007, Toronto).

Van Meter Q, Midyett LK, Deeb L et al, Prevalence of primary IGFD among untreated children with short stature in a prospective, multicenter study (Poster POO715) ICE Rio de Janeiro, Brazil 2008.

G.M. Bright<sup>1</sup>, W.V.Moore<sup>2</sup>, J.Nguyen<sup>3</sup>, G. Kletter<sup>4</sup>, B. S. Miller<sup>5</sup>, Q. L. Van Meter<sup>6</sup>, E. Humphriss<sup>1</sup>, J.A. Moore<sup>7</sup> and J.L. Cleland<sup>1</sup> Results of a Phase 1b Study of a new long-acting human growth hormone (VRS-317) in pediatric growth hormone deficiency (PGHD). PAS 2014 May 2014

Van Meter Q, Welstead B and Low J, Characteristics of a Population of Obese Children and Adolescents: Suggesting a New Paradigm, presented at ESPE meeting, Dublin 2014.

Wayne V. Moore<sup>1</sup>, Patricia Y. Fechner<sup>2</sup>, Huong Jil Nguyen<sup>3</sup>, Quentin L. Van Meter<sup>4</sup>, John S. Fuqua<sup>5</sup>, Bradley S. Miller<sup>6</sup>, David Ng<sup>7</sup>, Eric Humphriss<sup>8</sup>, R. W. Charlton<sup>8</sup>, George M. Bright<sup>8</sup>: Safety and Efficacy of Somavaratan (VRS-317), a Long-Acting rhGH, in Children with Growth Hormone Deficiency (GHD): 3-Year Update of the VERTICAL & VISTA Trials, presented at the 2017 Endocrine Society meeting in Orlando FL

Bradley S. Miller<sup>1</sup>, Wayne V. Moore<sup>2</sup>, Patricia Y. Fechner<sup>3</sup>, Huong Jil Nguyen<sup>4</sup>, Quentin L. Van Meter<sup>5</sup>, John S. Fuqua<sup>6</sup>, David Ng<sup>7</sup>, Eric Humphriss<sup>8</sup>, R. W. Charlton<sup>8</sup>, George M. Bright<sup>8</sup>, 3-Year Update of the Phase 2a and Long-term Safety Studies (VERTICAL and VISTA) of Somavaratan (VRS-317), a Long-acting rhGH for the Treatment of Pediatric Growth Hormone Deficiency, presented at the 2017 IMPE meeting in Washington D.C.

ADDITIONAL PRESENTATIONS/LECTURES:

Pediatrics Update, CME Associates, San Diego – Orlando Annual Conferences: Lectures on Pediatric Endocrine Subjects – 1986 – 2001. Course Moderator, 1997, 1998, 1999, 2000, 2001

Endocrine and Gastroenterology Update, CME Associates, Maui HI Nov 2001, Lecturer and Course Moderator

Lecture on Panhypopituitarism, Pharmacia Conference, Nashville TN April 2002.

Family Medicine Review Course, Orlando, FL, 1992 – 2001

Pediatric Grand Rounds, Tanner Medical Center, October 1997

Pediatric Grand Rounds, Hughes Spaulding Children’s Hospital, September, 2003

Pediatrics in the Park, Fall CME meeting for the Georgia Chapter of the American Academy of Pediatrics, November 2003

Pediatric Grand Rounds, Columbus Regional Medical Center, January 2004

Frontiers in Pediatrics CME Course, sponsored by the Atlanta Children’s Health Network, Atlanta, March 2004.

Pediatric Grand Rounds, Eggleston Children’s Hospital, May 2004.

Sue Schley Matthews Pediatric Conference, Columbus Regional Medical Center, September 2004

56<sup>th</sup> Annual Scientific Assembly and Exhibition of the Georgia Academy of Family Physicians, Nov 2004

Program Co-Chairman: Southern Pediatric Endocrine Society Annual meeting, Nov 2004, November 2014

Presentations on Diabetes, Growth Failure, and Thyroid Disease to the Postgraduate Pediatric Nurse Practitioner Program, Georgia State University, Nov 2005, June 2006, May 2007

Issues in Medicine, US Medical Congress Conference and Exhibition, Las Vegas, meeting planner and speaker, June, 2006

CME Presentations for the Georgia Chapter of the American Academy of Pediatrics Spring and Fall Meetings 2004-present

Pediatric Grand Rounds, Columbus Regional Medical Center, Columbus, GA, 2011-present

Human Growth Foundation Regional CME Conference, Atlanta GA  
March 2013, February 2014 Columbus Georgia

International Federation of Therapeutic Counseling Choice: Transgender Medicine, IFTCC Launch, October 15, 2018 London, Third International Congress, October 25 2018 Budapest.

Southern Pediatric Endocrine Society, Orlando FL, Feb 2019

Matthew Bulfin Conference, Indianapolis IN April 2019

CMDA annual conference, Ridgecrest NC, May 2019

Support 4 Family conference, London, UK June 2019

Audio Digest Pediatrics - ① v. 41, no. 4; ② v. 41, no. 20; ③ v. 43, no. 17

Audio Digest Family Practice - ① v. 42, no. 5; ② v. 44, no. 11; ③ v. 44, no. 44; ④ v. 45, no 15

Audio Digest Otolaryngology - ① v. 32, no. 14

#### CURRENT HOSPITAL APPOINTMENTS:

Eggleston/Scottish Rite Children's Hospitals, active  
staff, Pediatric Endocrinology

#### PAST AND CURRENT CLINICAL RESEARCH:

2006	Sanofi-Aventis HMR1964D/3001	study completed 2007
2006	Tercica MS301-	study completed 2008
2007	Tercica MS310-	study completed 2008
2007	Tercica MS306-	study completed 2010
2007	Tercica MS316-	study completed 2012
2008	EMD Serono 28358	study completed 2009
2012	Versartis 12VR2	study completed 2014
2012	Debiopharm 8206-CPP-301	study started July 2012
2013	Versartis 13 VR3	study started Dec 2013
2014	Novo-Nordisk Elipse	study started 2014
2015	Versartis 14 VR4	study completed 2017
2017	Mannkind MKC-TI-155	study completed 2019
2018	Abbvie M16-904	study started 2018
2019	Novo-Nordisk Real-4	study started 2019
2019	Lilly 18B-MC-ITSB	study started 2019
2021	Pfizer PROGRES	study started 2021



2021	Lumos Oragrowth210	study started July 2021
2022	Novo-Nordisk Real-8	study starts July 2022

LEGAL EXPERT WITNESS:

2017 North Carolina Legislature- transgender bathroom bill  
2018 Jessica Siefert transgender case, Cincinnati, OH  
2018 Alberta, Canada school system transgender case  
2018 Decatur GA School Board transgender case  
2019 British Columbia transgender case  
2019 Gavin Grimm transgender case, Gloucester County, VA  
2019 Rowe vs Isle of Wight School Board, UK  
2019 Younger transgender case, Dallas, TX  
2020 Alabama State House and Senate committee hearings  
2020 Pennsylvania State House Health Subcommittee hearings  
2020 Iowa State House committee hearing  
2020 California State House committee hearing  
2020 Harris Count TX custody case  
2021 Missouri State House committee hearing  
2021 NAACP v State of Arkansas