UNITED STATES DISTRICT COURT
FOR THE MIDDLE DISTRICT OF FLORIDA
JACKSONVILLE DIVISION

DREW ADAMS, a minor, by and through his next friend and mother, ERICA ADAMS KASPER,

Plaintiff,

v.

THE SCHOOL BOARD OF ST. JOHNS COUNTY, FLORIDA,

Defendant.

Civil Action No. 3:17-cv-00739-TJCJBT

EXPERT DECLARATION of Paul W Hruz, M.D., Ph.D

1. 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 which is attached as Exhibit A to this declaration.

2. I received my doctor of philosophy degree from the Medical College of Wisconsin in 1993. I received my medical degree from the Medical College of Wisconsin in 1994. I am an Associate Professor of Pediatrics in the Division of Pediatric Endocrinology and Diabetes at Washington University School of Medicine. I also have a secondary appointment as Associate Professor of Cellular Biology and Physiology in the Division of Biology and Biological Sciences at Washington University School of Medicine. I served as chief of the Division of Pediatric Endocrinology and Diabetes at Washington University from 2012-2017. I served as the

Director of the Pediatric Endocrinology Fellowship Program at Washington University from 2008-2016.

3. I am board certified in Pediatrics and Pediatric Endocrinology. I have been licensed to practice medicine in Missouri since 2000.

4. My professional memberships include the American Academy of Pediatrics, the Pediatric Endocrine Society, the Endocrine Society, and the American Association for Biochemistry and Molecular Biology.

5. I have extensive experience in treating infants and children with disorders of sexual development and am an active member of the multidisciplinary Disorders of Sexual Development (DSD) program at Washington University. The DSD Team at Washington University is part of the DSD-Translational Research Network, a national multi-institutional research network that investigates the genetic causes and the psychologic consequences of DSD.

6. In the nearly 20 years that I have been in clinical practice I have participated in the care of hundreds of children with disorders of sexual development. In the care of these patients, I have acquired expertise in the understanding and management of associated difficulties in gender identification.

7. In my role as the director of the Division of Pediatric Endocrinology at Washington University, I have extensively studied the existing literature related to the incidence, potential etiology and treatment of gender dysphoria as efforts were made to develop a Transgender clinic at Saint Louis Children's Hospital. I have participated in local and national meetings where the endocrine care of children with gender dysphoria has been discussed and debated. I have met individually with several pediatric endocrinologists, including Dr. Norman Spack, who have developed and led transgender programs in the United States. I have also met with parents of

children with gender dysphoria to understand the unique difficulties experienced by this patient population.

8. Pediatric patients referred to our practice for the evaluation and treatment of gender dysphoria are cared for by an interdisciplinary team of providers that includes a psychologist and pediatric endocrinologist who have been specifically chosen for this role based upon a special interest in this rare patient population. Due to serious concerns regarding the safety, efficacy, and ethics of the current treatment paradigm, I have not directly engaged in hormonal treatment of patients with gender dysphoria.

9. My opinions as detailed in this declaration are based upon my knowledge and direct professional experience in the subject matters discussed. The materials that I have relied upon are the same types of materials that other experts in my field of clinical practice rely upon when forming opinions on the subject. The documents that I have reviewed specifically related to this case are 1.) The first amended complaint for declaratory, injunctive, and other relief for Drew Adams, 2.) The plaintiff's first amended rule 26(a) disclosure and 3.) Drew Adams' medical records. A list of the published literature I have relied on is attached as Exhibit B to this declaration.

10. Over my career, I have provided expert medical record review and testified at deposition in less than a dozen cases. Related to the litigation of issues of sex and gender, I have been designated as an expert witness in *Joaquín Carcaño et al vs. Patrick Mccrory, Jane Doe vs Board of Education of the Highland School District*, and *Ashton Whitaker vs. Kenosha Unified School District*. I have been deposed in the last of these cases. In the past 4 years I have also served as an expert witness in *Dakota Humphrey vs. Stanley Block* and *Liston Ward et al. vs. Janssen Pharmaceuticals*. I have never testified at trial.

11. I am being compensated at an hourly rate for actual time devoted, at the rate of $350 per hour. My compensation does not depend on the outcome of this litigation, the opinions I express, or the testimony I provide.

Case Details

[redacted]

Basic Terminology

13. Biological sex is a term that specifically refers to a member of a species in relation to the member's capacity to either donate (male) or receive (female) genetic material for the purpose of reproduction. This remains the standard definition that has been accepted and used by scientists, medical personnel, and society in general.

14. Gender, a term that had traditionally been reserved for grammatical purposes, is currently used to describe the psychologic and cultural characteristics of a person in relation to biological sex. Gender therefore exists in reference to societal perceptions, not biology.

15. Gender identity refers to a person's individual perception of being male or female.

16. Sexual orientation refers to a person's arousal and desire for sexual intimacy with members of the male or female sex.

Human sexuality in relation to fundamental biology and observed variations

17. Sex is genetically encoded at the moment of conception due to the presence of specific DNA sequences (i.e. genes) that direct the production of signals that influence the formation of bipotential gonad to develop into either a testis or ovary. This genetic information is normally present on X and Y chromosomes. Chromosomal sex refers to the normal complement of X and Y chromosomes (i.e. normal human males have one X and one Y chromosome whereas normal human females have two X chromosomes). Genetic signals are mediated through the activation or deactivation of other genes and through programmed signaling of hormones and cellular transcription factors. The default pattern of development in the absence of external signaling is female. The development of the male appearance (phenotype) depends upon active signaling

processes.

18. For members of the human species, sex is normatively aligned in a binary fashion (i.e., either male or female) in relation to biologic purpose. Medical recognition of an individual as male or female is typically made at birth according to external phenotypic expression of primary sexual traits (i.e., presence of a penis for males and presence of labia and vagina for females).

19. Due to genetic and hormonal variation in the developing fetus, normative development of the external genitalia in any individual differs with respect to size and appearance while maintaining an ability to function with respect to biologic purpose (i.e. reproduction). Internal structures (e.g. gonad, uterus, vas deferens) normatively align with external genitalia.

20. Reliance upon external phenotypic expression of primary sexual traits is a highly accurate means to assign biologic sex. In over 99.9% of cases, this designation will correlate with internal sexual traits and capacity for normal biologic sexual function. Sex is therefore not "assigned at birth" but is rather recognized at birth.

21. Due the complexity of signals that are involved in normal sexual development, it is not surprising that a small number of individuals are born with defects in this process. Defects can occur either through inherited or *de novo* mutations in genes that are involved in sexual determination or through environmental insults during critical states of sexual development. Persons who are born with such abnormalities are considered to have a disorder of sexual development (DSD). Most often, this is first detected as ambiguity in the appearance of the external genitalia.

22. Normal variation in external genital appearance (e.g. phallic size) does not alter the basic biologic nature of sex as a binary trait. "Intersex" conditions represent disorders of normal development, not a third sex.

23. Medical care of persons with DSDs is primarily directed toward identification of the etiology of the defect and treatment of any associated complications. Similar to other diseases, tools such as the Prader scale are used to stage the severity of the deviation from normal. In children with DSDs, characterization based upon phenotype alone does not reliably predict chromosomal sex nor does it necessarily correlate with potential for biological sexual function. Decisions on initial sex assignment in these rare cases require detailed assessment by a team of expert medical providers.

24. Standard medical practice in the treatment of persons with DSDs has evolved with growing understanding of the physical and psychologic needs and outcomes for affected individuals. Previously, it was felt that a definitive sex assignment was necessary shortly after birth with the belief that this would allow patients with DSDs to best conform to the assigned sex. Current practice is to defer sex assignment until the etiology of the disorder is determined and, if possible, a prediction can be made on likely biologic and psychologic outcomes. When this cannot be done with confidence, a presumptive sex assignment is made. Factors used in making such decisions include chromosomal sex, phenotypic appearance of the external genitalia, and parental desires. The availability of new information can in rare circumstances lead to sex reassignment. Decisions on whether to surgically alter the external genitalia to align with sex are generally deferred until the patient is able to provide consent.[1]

Gender Dysphoria in relation to Biological Sex

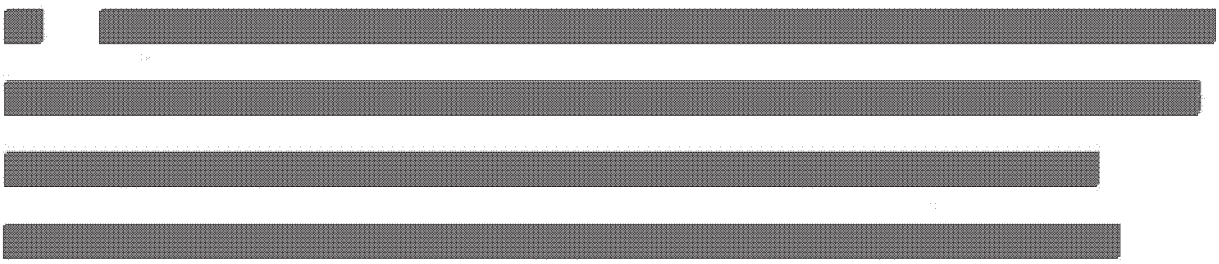
25. Although gender usually aligns with biological sex, some individuals experience discordance in these distinct traits. Specifically, biologic females may identify as males and biologic males may identify as females. As gender by definition is distinct from biological sex,

one's gender identity does not change a person's biological sex.

26. Individuals who experience significant distress due to discordance between gender identity and sex are considered to have "gender dysphoria".[2] Although the prevalence of gender dysphoria has not been established by rigorous scientific analysis, estimates reported in in the DSM-V are between 0.005% to 0.014% for adult males and 0.002% to 0.003% for adult females. Thus, gender dysphoria is a rare condition. It is currently unknown whether these estimates are falsely low due to under-reporting, or if changing societal acceptance of transgenderism and the growing number of medical centers providing medical intervention for gender dysphoria affects the number of persons who identify as transgender. Recent data indicates that the number of people seeking care for gender dysphoria is increasing with some estimates as high as 20-fold.[3,4]

27. There is strong evidence against the theory that gender identity is determined at or before birth and is unchangeable. This comes from identical twin studies where siblings share genetic complements and prenatal environmental exposure but have differing gender identities.[5]

28. Further evidence that gender identity is not fixed comes from established peer reviewed literature demonstrating that the vast majority (80-95%) of children who express gender dysphoria revert to a gender identity concordant with their biological sex by late adolescence.[6,7] It is not known whether individuals with gender dysphoria persistence have differing etiologies or severity of precipitating factors compared to desisting individuals.



30. The etiology of gender dysphoria in individuals with gender dysphoria remains to be identified. Theories include prenatal hormone exposure, genetic variation, and postnatal environmental influences. Based upon the currently available but incomplete dataset, it is likely that gender dysphoria is multifactorial with differing qualitative and quantitative influences in any given individual.

31. The recently coined concept of "neurological sex" as a distinct entity or a basis for classifying individuals as male or female has no scientific justification. Limited emerging data has suggested structural and functional differences between brains from normal and transgender individuals. These data do not establish whether these differences are innate and fixed or acquired and malleable. The remarkable neuronal plasticity of the brain is known and has been studied extensively in gender-independent contexts related to health and disease, learning and behavior.

Gender Ideology

32. The modern attempt to equate gender identity with sex is not based upon sound scientific principles but rather is based upon ideology fueled by advocacy. Although worldviews among scientists and physicians, similar to society at large, differ, science is firmly grounded in physical reality not perception. The inherent link between human sexual biology and teleology is self-evident and fixed.

33. The claims of proponents of transgenderism, which include opinions such as "Gender identity is the primary factor determining a person's sex" and "Gender is the only true determinant of sex" must be viewed in their proper philosophical context. There is no scientific basis for redefining sex on the basis of a person's psychological sense of 'gender'.

34. The prevailing, constant and accurate designation of sex as a biological trait grounded in the inherent purpose of male and female anatomy and as manifested in the appearance of external genitalia at birth remains the proper scientific and medical standard. Redefinition of the classification and meaning of sex based upon pathologic variation is not established medical fact.

Potential Harm Related to Gender Dysphoria Treatments

35. The fundamental purpose of the practice of medicine is to treat disease and alleviate suffering. An essential tenet of medical practice is to avoid doing harm in the process. Due to the frequent lack of clear and definitive evidence on how to best accomplish this goal, treatment approaches can and do frequently differ among highly knowledgeable, competent, and caring physicians.

36. Persons with gender dysphoria as delineated in the DSM-V experience significant psychological distress related to their condition with elevated risk of depression, suicide, and other morbidities. Thus, attempts to provide effective medical care to affected persons are clearly warranted.

37. Efforts to effectively treat persons with gender dysphoria require respect for the inherent dignity of those affected, sensitivity to their suffering, and maintenance of objectivity in assessing etiologies and long-term outcomes. Desistance (i.e. reversion to gender identity concordant with sex) provides the greatest lifelong benefit and is the outcome in the majority of patients and should be maintained as a desired goal. Any forced societal intervention that could interfere with the likelihood of gender dysphoria resolution is unwarranted and potentially harmful.

38. There is an urgent need for high quality controlled clinical research trials to determine

ways to develop supportive dignity affirming social environments that maintain affirmation of biological reality. To date, three approaches have been proposed for managing children with gender dysphoria.[8] The first approach, often referred to as "conversion" or "reparative therapy", is directed toward actively supporting and encouraging children to identify with their biological sex. The second "neutral" approach, motivated by understanding of the natural history of transgender identification in children, is to neither encourage nor discourage transgender identification, recognizing that the majority of affected children if left alone will eventually realign their gender with their sex. The third "affirming" approach is to actively encourage children to embrace transgender identity with social transitioning followed by hormonal therapy.

39. The gender affirming approach, which includes use of a child's preferred pronouns, use of sex-segregated bathrooms, other intimate facilities and sleeping accommodations corresponding to a child's gender identity, has limited scientific support for short-term alleviation of dysphoria and no long-term outcomes data demonstrating superiority over the other approaches. Claims that the other approaches have been scientifically disproven are false. Decades of research, most notably the pioneering work of Dr. Kenneth Zucker, have supported the efficacy of a more conservative approach for the majority of patients experiencing gender dysphoria.[8,9]

40. Feelings of anxiety, depression, isolation, frustration, and embarrassment are not unique to children with gender dysphoria, but rather are common to children who differ physically or psychologically from their peers. Difficulties are accentuated as children progress through the normal stages of neurocognitive and social development. In the clinical practice of pediatric endocrinology, this is most commonly seen in children with diabetes. Attempts to deny or conceal the presence of disease rather than openly acknowledge and address specific needs can

have devastating consequences including death. With proper acknowledgment of the similarity and differences between children with gender dysphoria and other developmental challenges, prior experience can guide the development of effective approaches to both alleviate suffering and minimize harm to school aged children experiencing gender dysphoria.

41. The Endocrine Society published in 2009 clinical guidelines for the treatment of patients with persistent gender dysphoria.[10] The recommendations include temporary suppression of pubertal development of children with GnRH agonists (hormone blockers normally used for children experiencing precocious puberty) followed by hormonal treatments to induce the development of secondary sexual traits consistent with one's gender identity. This guideline was developed using the GRADE (Recommendations, Assessment, Development, and Evaluation) system for rating clinical guidelines. As directly stated in the Endocrine Society publication, "the strength of recommendations and the quality of evidence was low or very low." According to the GRADE system, low recommendations indicate "Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate". Very low recommendations mean that "any estimate of effect is very uncertain". An updated set of guidelines was published in September of 2017.[11] The low quality of evidence presented in this document persist.

42. Clinical Practice Guidelines published by the World Professional Association for Transgender Health (WPATH), which is currently in its 7th iteration, similarly, though less explicitly, acknowledge the limitation of existing scientific data supporting their recommendations given and "the value of harm-reduction approaches".

43. Treatment of gender dysphoric children who experience persistence of symptoms with hormones (pubertal suppression and cross-hormone therapy) carries significant risk. It is

generally accepted, even by advocates of transgender hormone therapy, that hormonal treatment results in sterility which in many cases is irreversible.[12] Emerging data also show that treated patients have lower bone density which may lead to increased fracture risk later in life.[13] Other potential adverse effects include disfiguring acne, high blood pressure, weight gain, abnormal glucose tolerance, breast cancer, liver disease, thrombosis, and cardiovascular disease.[14]

44. Since strategies for the treatment of transgendered children as summarized by the Endocrine Society guidelines are relatively new, long-term outcomes are unknown. Evidence presented as support for short term reductions in psychological distress following social transition in a "gender affirming" environment remains inconclusive. When considered apart from advocacy based agendas, multiple potential confounders are evident. The most notable deficiencies of existing research are the absence of proper control subjects and lack of randomization in study design. Although appropriate caution is warranted in extrapolating the outcomes observed from prior studies with current treatments, adults who have undergone social transition with or without surgical modification of external genitalia continue to have rates of depression, anxiety, substance abuse and suicide far above the background population.[15,16]

45. Evidence cited to support societal measures that promote or encourage gender transition, including the plaintiff's demand for use of multi-user sex-segregated restrooms corresponding with the plaintiff's gender identity, as a medically necessary treatment for gender dysphoria is limited. Recent studies reporting reductions in dysphoria following social transition of adolescent patients are small, poorly controlled and of insufficient duration to draw definitive conclusions regarding long-term efficacy. Long-term follow up of patients with gender dysphoria who have undergone social and hormonal transition with or without surgical intervention has

shown persistent psychological morbidity far above non-transgendered individuals with suicide attempts 7-fold and completed suicides19-fold above the general population.[15,16]

46. Of particular concern is the likelihood that forced societal affirmation including a requirement that the St. John's County School District allow students to use sex-segregated bathrooms corresponding to gender identity rather than access to single unit facilities, will interfere with known rates of gender resolution. Any activity that encourages or perpetuates transgender persistence for those who would otherwise desist can cause significant harm, particularly in light of the current treatment paradigm for persisting individuals. As noted, permanent sterility can be expected with hormonal or surgical disruption of normal gonadal function. This is particularly concerning given that children are likely incapable of making informed consent to castrating treatments.[17]

47. Dignity affirming support for adolescents with gender dysphoria does not necessitate facilitation of a false understanding of human sexuality in schools. Rather, policy requirements that can increase persistence of transgender identification have significant potential for inducing long-term harm to affected children.

48. There remains a significant and unmet need to better understand the biological, psychological, and environmental basis for the manifestation of discordance of gender identity and biological sex in affected individuals.[18] In particular, there is a concerning lack of randomized controlled trials comparing outcomes of youth with gender dysphoria who are provided mandated access to sex-segregated bathroom facilities corresponding with gender identity to youth provided single user facilities. This includes understanding of how forced public encouragement of social gender transition affects the usual progression to resolution of gender dysphoria in affected children. Such studies can be ethically designed and executed with

provision of other dignity affirming measures to both treatment groups. Without this scientific evidence, it is impossible to assert that the approach using sex-segregated bathrooms is an essential component of treatment.

49. Limitations on this report: My opinions and hypotheses in this matter are subject to the limitations of all documentary and related evidence, the impossibility of absolute prediction, as well as the limitations of social and medical science. I have not met with, nor interviewed, plaintiff Drew Adams. As always, I have no expert opinions regarding the veracity of witnesses in this case. I have not yet reviewed all of the evidence in this case and my opinions are subject to change at any time as new information becomes available to me. Only the trier of fact can determine the credibility of witnesses and how scientific research may or may not be related to the specific facts of any particular case. A key role of an expert witness is to help the court, lawyers, parties, and the public understand and apply reliable scientific, technical, and investigative principles, hypotheses, methods, and information. I have transmitted this confidential expert report directly to attorney Michael Spellman, for distribution as consistent with the relevant laws.

Pursuant to 28 U.S.C § 1746, I declare under penalty of perjury under the laws of the United States of America that the foregoing is true and correct.

Date: Paul W Hruz

Signed: November 2, 2017

Paul W. Hruz, M.D., Ph.D.

Curriculum Vitae

Paul W. Hruz, M.D., Ph.D.

Date: 10/18/2017 08:36 AM

Personal Information

Birthplace: WI
Citizenship: USA

Address and Telephone Numbers

University: Washington University in St. Louis
School of Medicine
Department of Pediatrics
Endocrinology and Diabetes
660 S. Euclid Ave.
St. Louis, MO 63110
Campus Box 8208

Phone: 314-454-6051
Fax: 314-286-2892
email: Hruz_P@wustl.edu

Present Positions

Associate Professor of Pediatrics, Endocrinology and Diabetes
Associate Professor of Pediatrics, Cell Biology & Physiology
Researcher, Developmental Biology

Education and Training

1987	BS, Chemistry, Marquette University, Milwaukee, WI
1993	PhD, Biochemistry, Medical College of Wisconsin, Milwaukee, WI
1994	MD, Medicine, Medical College of Wisconsin, Milwaukee, WI
1994 - 1997	Pediatric Residency, University of Washington, Seattle, Washington
1997 - 2000	Pediatric Endocrinology Fellowship, Washington University, Saint Louis, MO

Academic Positions and Employment

1996 - 1997	Locum Tenens Physician, Group Health of Puget Sound Eastside Hospital, Group Health of Puget Sound Eastside Hospital, Seattle , WA
2000 - 2003	Instructor in Pediatrics, Endocrinology and Diabetes, Washington University in St. Louis, St. Louis, MO
2003 - 2011	Assistant Professor of Pediatrics, Endocrinology and Diabetes, Washington University in St. Louis, St. Louis, MO
2004 - 2011	Assistant Professor of Pediatrics, Cell Biology & Physiology, Washington University in St. Louis, St. Louis, MO
2011 - Pres	Associate Professor of Pediatrics, Cell Biology & Physiology, Washington University in St. Louis, St. Louis, MO
2011 - Pres	Associate Professor of Pediatrics, Endocrinology and Diabetes, Washington University in St. Louis, St. Louis, MO
2012 - 2017	Division Chief, Endocrinology and Diabetes, Washington University in St. Louis, St. Louis, MO
2016 - Pres	Researcher, Developmental Biology, Washington University in St. Louis, St. Louis, MO

Exhibit A

Appointments and Committees

NIH Study Sections

2005	NIH- NIDDK Special Emphasis Panel ZDK1 GRB-6
2009	NIH- ACE Competitive Revisions ZRG1 AARR-H (95) S
2009	NIH- AIDS and AIDS Related Research IRG
2011	NIH- Pediatric Endocrinologist K12 ZDK1 GRB-C
2014	NIH- Special Empahsis Panel ZRG1 BBBPY 58
2014	NIH- AIDS and AIDS Related Research IRG
2015	NIH- Cardiovascular and Respiratory Sciences Special Emphasis Panel ZDK1 GRB-J (02)
2015	NIH- NIDDK Special Emphasis Panel ZRG1 CVRS-Q (80)
2016	NIH Special Emphasis Panel ZRG1 AAR-M
2016	American Diabetes Association Research Grant Review Committee

Local Appointments

2017 - Pres	Board of the Catholic Medical Association, St. Louis Guild

University Affiliations

2008 - 2016	Director, Pediatric Endocrinology & Diabetes Fellowship Program
2010 - Pres	Pediatric Computing Facility Advisory Committee
2012 - 2017	Director, Division of Pediatric Endocrinology & Diabetes
2012 - Pres	Disorders of Sexual Development Multidisciplinary Care Program
2013 - Pres	Molecular Cell Biology Graduate Student Admissions Committee
2014 - Pres	Research Consultant, ICTS Research Forum - Child Health
2014 - Pres	Director, Pediatric Diabetes Research Consortium

Hospital Affiliations

2000 - Pres	Attending Physician, St. Louis Children's Hospital

Thesis Committees (*Chair)

		Advisor
2008 - 2011	Kelly Diggs-Andrews	Simon Fisher
2008 - 2010	Irwin Puentes	Simon Fisher
2008 - 2010	Tony Frovola	Kelle Moley
2009 - 2010	Lauren Flessner	Kelle Moley
2010 - 2012	Katie Boehle	Kelle Moley
2010 - 2013	Candace Reno	Simon Fisher
2011 - 2016	Thomas Kraft	Paul Hruz
2013 - 2015	Chi Lun Pui	Audrey Odom
2013 - 2016	Leah Imlay	Audey Odom
2014 - Pres	Anne Robinson	Katie Henzler-Wildman
2015 - Pres	Allyson Mayer	Brian DeBosch

Scholarship Oversight Committees

2013 - 2016	Brittany Knipsein (Advisor: David Rudnick)
2015 - Pres	Pamela Smith (Advisor: Michael Whyte)

Licensure and Certifications

1997 - Pres	Board Certified in General Pediatrics
2000 - Pres	MO State License #2000155004
2001 - Pres	Board Certified in Pediatric Endocrinology & Metabolism

Honors and Awards

1987	National Institute of Chemists Research and Recognition Award
1987	Phi Beta Kappa
1987	Phi Lambda Upsilon (Honorary Chemical Society)
1988	American Heart Association Predoctoral Fellowship Award
1994	Alpha Omega Alpha
1994	Armond J. Quick Award for Excellence in Biochemistry
1994	NIDDK/Diabetes Branch Most Outstanding Resident
1998	Pfizer Postdoctoral Fellowship Award
2002	Scholar, Child Health Research Center of Excellence in Developmental Biology at Washington University
2013	Julio V Santiago, M.D. Scholar in Pediatrics

Editorial Responsibilities

Editorial Ad Hoc Reviews

AIDS
AIDS Research and Human Retroviruses
American Journal of Pathology
American Journal of Physiology
British Journal of Pharmacology
Circulation Research
Clinical Pharmacology & Therapeutics
Comparative Biochemistry and Physiology
Diabetes
Experimental Biology and Medicine
Future Virology
Journal of Antimicrobial Chemotherapy
Journal of Clinical Endocrinology & Metabolism
Journal of Molecular and Cellular Cardiology
Obesity Research
2000 - Pres Journal of Biological Chemistry
2013 - Pres PlosOne
2016 - Pres Scientific Reports

Editorial Boards

2014 - Pres Endocrinology and Metabolism Clinics of North America

Professional Societies and Organizations

1992 - 2004 American Medical Association
1994 - 2005 American Academy of Pediatrics
1995 - 2014 American Association for the Advancement of Science
1998 - Pres American Diabetes Association
1998 - Pres Endocrine Society
1999 - Pres Pediatric Endocrine Society
2004 - Pres American Society for Biochemistry and Molecular Biology
2004 - Pres Society for Pediatric Research
2004 - 2007 American Chemical Society
2005 - Pres Full Fellow of the American Academy of Pediatrics
2013 - Pres International Society for Pediatric and Adolescent Diabetes

Major Invited Professorships and Lectures

2002 St. Louis Children's Hospital, Pediatric Grand Rounds, St. Louis, MO
2004 National Disease Research Interchange, Human Islet Cell Research Conference, Philadelphia, PA
2004 NIDA-NIH Sponsored National Meeting on Hormones, Drug Abuse and Infections, Bethesda, MD
2005 The Collaborative Institute of Virology, Complications Committee Meeting, Boston, MA
2005 University of Indiana, Endocrine Grand Rounds, Indianapolis, IN
2006 Metabolic Syndrome Advisory Board Meeting, Bristol-Meyers Squibb, Pennington, NJ
2007 American Heart Association and American Academy of HIV Medicine State of the Science Conference: Initiative to Decrease Cardiovascular Risk and Increase Quality of Care for Patients Living with HIV/AIDS, Chicago, IL
2007 Medical College of Wisconsin, MSTP Annual Visiting Alumnus Lecture, Milwaukee, WI
2007 St Louis Children's Hospital, Pediatric Grand Rounds, St Louis, MO
2007 University of Arizona, Minority Access to Research Careers Seminar, Tucson AZ
2008 Boston University, Division of Endocrinology, Diabetes and Nutrition, Boston, MA
2009 St Louis Children's Hospital, Pediatric Grand Rounds, St Louis, MO
2010 American Diabetes Association Scientific Sessions, Symposium Lecture Orlando, FL
2010 University of Missouri Kansas City, School of Biological Sciences, Kansas City, MO
2011 Life Cycle Management Advisory Board Meeting, Bristol-Myers Squibb, Chicago, IL.
2013 St Louis Children's Hospital, Pediatric Grand Rounds, St Louis MO
2013 St Louis Children's Hosptial CPU Lecture, St Louis MO
2014 Pediatric Academic Societies Meeting, Vancouver, Canada.
2014 American Diabetes Association 74th Scientific Sessions, San Francisco, CA.
2017 University of Michigan, Division of Pediatric Endocrinology Ann Arbor, MI.
2017 Napa Institute National Conference Napa, CA.
2017 Catholic Medical Association National Conference Denver, CO.

Consulting Relationships and Board Memberships

1996 - 2012 Consultant, Bristol Myers Squibb
1997 - 2012 Consultant, Gilead Sciences

Research Support

Non-Governmental Support

(Hruz)
Gilead Pharma
Novel HIV Protease Inhibitors and GLUT4

MHI-2017-593 (DeBosch) 2/1/2017- 1/31/2020
CDI
Prevention And Treatment Of Hepatic Steatosis Through Selective Targeting Of GLUT8

Completed Support

II (Hruz) 2/1/2012- 1/31/2015
CDI
Solution-State NMR Structure and Dynamics of Facilitative Glucose Transport Proteins

R01 (Hruz) 9/20/2009- 5/31/2014
NIH
Direct Effects of Antiretroviral Therapy on Cardiac Energy Homeostasis
The goal of this project is to characterize the influence of antiretroviral therapies on myocardial energy homeostasis and to elucidate how these changes in substrate delivery adversely affect cardiac function in the stressed heart.

Research Program (Hruz) 6/1/2009- 5/31/2012
MOD
Regulation of GLUT4 Intrinsic Activity
The major goals of this project are to investigate the ability of the GLUT4 tethering protein TUG and an UBL-domain containing N-terminal fragment of this protein to alter the intrinsic activity of the insulin responsive facilitative glucose transporter, to determine whether protein ubiquitination influences this association, and to characterize the role of the GLUT4 binding site on the modulation of glucose transport.

R01 (Hruz) 4/1/2007- 1/31/2012
NIH
Mechanisms for Altered Glucose Homeostasis During HAART
The goal of this project is to identify the cellular targets of HIV protease inhibitors that lead to peripheral insulin resistance, impaired beta-cell function, and alterations in hepatic glucose production and to elucidate the molecular mechanisms of these effects.

R01 Student Supp (Hruz) 6/10/2009- 8/31/2011
NIH
Mechanisms for Altered Glucose Homeostasis During HAART
The goal of this project is to identify the cellular targets of HIV protease inhibitors that lead to peripheral insulin resistance, impaired beta-cell function, and alterations in hepatic glucose production and to elucidate the molecular mechanisms of these effects.

(Hruz) 3/9/2010- 6/8/2011
Bristol-Myers Squibb
Protective Effect of Saxagliptin on a Progressive Deterioration of Cardiovascular Function

II (Hruz) 2/1/2008- 1/31/2011
CDI
Insulin Resistance and Myocardial Glucose Metabolism in Pediatric Heart Failure

Past Trainees

2014 - 2014 David Hannibal, Clinical Research Trainee
2005 - 2005 Dominic Doran, DSc, Postdoctoral Fellow
Study area: HIV Protease Inhibitor Effects on Exercize Tolerance
2002 - 2010 Joseph Koster, PhD, Postdoctoral Fellow
Study area: Researcher
2010 - 2014 Lauren Flessner, PhD, Postdoctoral Fellow
Present position: Instructor, Syracuse University
2008 - 2011 Arpita Vyas, MD, Clinical Fellow
Study area: Research
Present position: Assistant Professor, Michigan State University, Lansing MI
2008 - 2009 Candace Reno, Graduate Student
Study area: Research
Present position: Research Associate, University of Utah
2005 - 2005 Helena Johnson, Graduate Student
2007 - 2008 Kai-Chien Yang, Graduate Student
Study area: Research
Present position: Postdoctoral Research Associate, University of Chicago
2007 - 2007 Paul Buske, Graduate Student
Study area: Research
2006 - 2006 Ramon Jin, Graduate Student
Study area: Research
2009 - 2009 Stephanie Scherer, Graduate Student
Study area: Research
2006 - 2006 Taekyung Kim, Graduate Student
Study area: Research
2008 - 2008 Temitope Aiyejorun, Graduate Student
Study area: Research
2011 - 2016 Thomas Kraft, Graduate Student
Study area: Glucose transporter structure/function
Present position: Postdoctoral Fellow, Roche, Penzberg, Germany
2005 - 2005 Jeremy Etzkorn, Medical Student
Study area: Researcher
2003 - 2004 Johann Hertel, Medical Student
Study area: Research
Present position: Assistant Professor, University of North Carolina, Chapel Hill, NC
2003 - 2003 John Paul Shen, Medical Student
Study area: Research
2007 - 2007 Randy Colvin, Medical Student
Study area: Researcher
2011 - 2011 Amanda Koenig- High School Student, Other
Study area: Research
2009 - 2009 Anne-Sophie Stolle- Undergraduate Student, Other
Study area: Research
2004 - 2005 Carl Cassel- High School Student, Other
Study area: Research
2004 - 2004 Christopher Hawkins- Undergraduate Student, Other
Study area: Researcher
2010 - 2010 Constance Haufe- Undergraduate Student, Other
Study area: Researcher
2010 - 2011 Corinna Wilde- Undergraduate Student, Other
Study area: Researcher
2008 - 2012 Dennis Woo- Undergraduate Student, Other
Study area: Researcher
Present position: MSTP Student, USC, Los Angeles CA
2007 - 2007 Jan Freiss- Undergraduate Student, Other
Study area: Researcher
2004 - 2004 Kaiming Wu- High School Student, Other
Study area: Research
2011 - 2012 Lisa Becker- Undergraduate Student, Other
2009 - 2009 Matthew Hruz- High School Student, Other
Study area: Research
Present position: Computer Programmer, Consumer Affairs, Tulsa OK
2011 - 2011 Melissa Al-Jaoude- High School Students, Other
2002 - 2002 Nishant Raj- Undergraduate Student, Other
Study area: Researcher
2010 - 2010 Samuel Lite- High School Student, Other
Study area: Research

Clinical Responsibilities

	General Pediatrician, General Pediatric Ward Attending: 2-4 weeks per year, St. Louis Children's Hospital
Pres	Pediatric Endocrinologist, Endocrinology Night Telephone Consult Service: Average of 2-6 weeks/per yr, St. Louis Children's Hospital
Pres	Pediatric Endocrinologist, Inpatient Endocrinology Consult Service: 4-6 weeks per year, St. Louis Children's Hospital
Pres	Pediatric Endocrinologist, Outpatient Endocrinology Clinic: Approximately 50 patient visits per month, St. Louis Children's Hospital

Teaching Responsibilities

	Facilitator, Cell Biology Graduate Student Journal Club, 4 hour/year
	Facilitator, Discussion: Pituitary, Growth & Gonadal Cases, 2 hours/year
2000 - Pres	Lecturer, Medical Student Growth Lecture (Women and Children's Health Rotation): Variable
2000 - Pres	Lecturer, Metabolism Clinical Rounds/Research Seminar: Presentations twice yearly
2000 - Pres	Lecturer, Pediatric Endocrinology Journal Club: Presentations yearly
2009 - Pres	Lecturer, Markey Course-Diabetes Module
2009 - Pres	Facilitator, Medical Student Endocrinology and Metabolism Course, Small group
2009 - Pres	Facilitator, Biology 5011- Ethics and Research Science, 6 hours/year
2016 - Pres	Facilitator, Medical Student Endocrinology and Metabolism Course, Small group
2016 - Pres	Lecturer, Cell Signaling Course, Diabetes module, 3 hours/year

Publications

1. Hruz PW, Narasimhan C, Miziorko HM. 3-Hydroxy-3-methylglutaryl coenzyme A lyase: affinity labeling of the Pseudomonas mevalonii enzyme and assignment of cysteine-237 to the active site. *Biochemistry*. 1992;31(29):6842-7. PMID:1637819
2. Hruz PW, Miziorko HM. Avian 3-hydroxy-3-methylglutaryl-CoA lyase: sensitivity of enzyme activity to thiol/disulfide exchange and identification of proximal reactive cysteines. *Protein Sci*. 1992;1(9):1144-53. doi:10.1002/pro.5560010908 PMCID:PMC2142181 PMID:1304393
3. Mitchell GA, Robert MF, Hruz PW, Wang S, Fontaine G, Behnke CE, Mende-Mueller LM, Schappert K, Lee C, Gibson KM, Miziorko HM. 3-Hydroxy-3-methylglutaryl coenzyme A lyase (HL). Cloning of human and chicken liver HL cDNAs and characterization of a mutation causing human HL deficiency. *J Biol Chem*. 1993;268(6):4376-81. PMID:8440722
4. Hruz PW, Anderson VE, Miziorko HM. 3-Hydroxy-3-methylglutaryldithio-CoA: utility of an alternative substrate in elucidation of a role for HMG-CoA lyase's cation activator. *Biochim Biophys Acta*. 1993;1162(1-2):149-54. PMID:8095409
5. Roberts JR, Narasimhan C, Hruz PW, Mitchell GA, Miziorko HM. 3-Hydroxy-3-methylglutaryl-CoA lyase: expression and isolation of the recombinant human enzyme and investigation of a mechanism for regulation of enzyme activity. *J Biol Chem*. 1994;269(27):17841-6. PMID:8027038
6. Hruz PW, Mueckler MM. Cysteine-scanning mutagenesis of transmembrane segment 7 of the GLUT1 glucose transporter. *J Biol Chem*. 1999;274(51):36176-80. PMID:10593902
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10. Hruz PW, Murata H, Mueckler M. Adverse metabolic consequences of HIV protease inhibitor therapy: the search for a central mechanism. *Am J Physiol Endocrinol Metab*. 2001;280(4):E549-53. PMID:11254480
11. Murata H, Hruz PW, Mueckler M. Investigating the cellular targets of HIV protease inhibitors: implications for metabolic disorders and improvements in drug therapy. *Curr Drug Targets Infect Disord*. 2002;2(1):1-8. PMID:12462148
12. Hruz PW, Murata H, Qiu H, Mueckler M. Indinavir induces acute and reversible peripheral insulin resistance in rats. *Diabetes*. 2002;51(4):937-42. PMID:11916910
13. Murata H, Hruz PW, Mueckler M. Indinavir inhibits the glucose transporter isoform Glut4 at physiologic concentrations. *AIDS*. 2002;16(6):859-63. PMID:11919467
14. Koster JC, Remedi MS, Qiu H, Nichols CG, Hruz PW. HIV protease inhibitors acutely impair glucose-stimulated insulin release. *Diabetes*. 2003;52(7):1695-700. PMCID:PMC1403824 PMID:12829635
15. Liao Y, Shikapwashya ON, Shteyer E, Dieckgraefe BK, Hruz PW, Rudnick DA. Delayed hepatocellular mitotic progression and impaired liver regeneration in early growth response-1-deficient mice. *J Biol Chem*. 2004;279(41):43107-16. doi:10.1074/jbc.M407069200 PMID:15265859
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19. Hruz PW. Molecular Mechanisms for Altered Glucose Homeostasis in HIV Infection. *Am J Infect Dis*. 2006;2(3):187-192. PMCID:PMC1716153 PMID:17186064

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39. Kraft TE, Hresko RC, Hruz PW. Expression, purification, and functional characterization of the insulin-responsive facilitative glucose transporter GLUT4. *Protein Sci.* 2015. doi:10.1002/pro.2612 PMID:26402434
40. Kraft TE, Armstrong C, Heitmeier MR, Odom AR, Hruz PW. The Glucose Transporter PfHT1 Is an Antimalarial Target of the HIV Protease Inhibitor Lopinavir. *Antimicrob Agents Chemother.* 2015;59(10):6203-9. doi:10.1128/AAC.00899-15 PMCID:PMC4576095 PMID:26248369
41. Hruz PW. Commentary. *Clin Chem.* 2015;61(12):1444. PMID:26614228
42. DeBosch BJ, Heitmeier MR, Mayer AL, Higgins CB, Crowley JR, Kraft TE, Chi M, Newberry EP, Chen Z, Finck BN, Davidson NO, Yarasheski KE, Hruz PW, Moley KH. Trehalose inhibits solute carrier 2A (SLC2A) proteins to induce autophagy and prevent hepatic steatosis. *Sci Signal.* 2016;9(416):ra21. doi:10.1126/scisignal.aac5472 PMID:26905426
43. Hresko RC, Kraft TE, Quigley A, Carpenter EP, Hruz PW. Mammalian Glucose Transporter Activity is Dependent upon Anionic and Conical Phospholipids. *J Biol Chem.* 2016. doi:10.1074/jbc.M115.730168 PMID:27302066
44. Kraft TE, Heitmeier MR, Putanko M, Edwards RL, Ilagan MX, Payne MA, Autry JM, Thomas DD, Odom AR, Hruz PW. A Novel Fluorescence Resonance Energy Transfer-Based Screen in High-Throughput Format To Identify Inhibitors of Malarial and Human Glucose Transporters. *Antimicrob Agents Chemother.* 2016;60(12):7407-7414. PMCID:PMC5119023 PMID:27736766
45. Mayer AL, Higgins CB, Heitmeier MR, Kraft TE, Qian X, Crowley JR, Hyrc KL, Beatty WL, Yarasheski KE, Hruz PW, DeBosch BJ. SLC2A8 (GLUT8) is a mammalian trehalose transporter required for trehalose-induced autophagy. *Sci Rep.* 2016;6:38586. PMCID:PMC5138640 PMID:27922102
46. Edwards, R, Brothers RC, Wang X, Maron MI, Tsang PS, Kraft TE, Hruz PW, Williamson KC, Dowd CS, Odom John AR. MEPicides: potent antimalarial prodrugs targeting isoprenoid biosynthesis *Sci Rep.* 2017;In press.

47. Shanmugam M, Heilmeier MR, Hruz PW and Schiltz G. Development of selective GLUT4 antagonists for treating multiple myeloma *Eur J Med Chem*. 2017;In press.
48. Zhang Y, Higgins CB, Mayer AL, Mysorekar I, Evans T, Razani B, Graham M, Hruz PW, and DeBosch, BJ. Transcription Factor EB (TFEB)-dependent Induction of Thermogenesis by the Hepatocyte GLUT Inhibitor, Trehalose *EMBO Reports*. 2017;Submitted.

Invited Publications

1. Hruz PW, Mueckler MM. Structural analysis of the GLUT1 facilitative glucose transporter (review). *Mol Membr Biol*. 2001;18(3):183-93. PMID: 11681785
2. Hruz PW, Murata H, Mueckler M. Adverse metabolic consequences of HIV protease inhibitor therapy: the search for a central mechanism. *Am J Physiol Endocrinol Metab*. 2001;280(4):E549-53. PMID: 11254460
3. Murata H, Hruz PW, Mueckler M. Investigating the cellular targets of HIV protease inhibitors: implications for metabolic disorders and improvements in drug therapy. *Curr Drug Targets Infect Disord*. 2002;2(1):1-8. PMID: 12462148
4. Hruz PW. Molecular Mechanisms for Altered Glucose Homeostasis in HIV Infection. *Am J Infect Dis*. 2006;2(3):187-192. PMCID: PMC1716153 PMID: 17186064
5. Grunfeld C, Kotler DP, Arnett DK, Falutz JM, Haffner SM, Hruz P, Masur H, Meigs JB, Mulligan K, Reiss P, Samaras K, Working Group 1. Contribution of metabolic and anthropometric abnormalities to cardiovascular disease risk factors. *Circulation*. 2008;118(2):e20-8. PMCID: PMC3170411 PMID: 18566314
6. Hruz PW. HIV protease inhibitors and insulin resistance: lessons from in-vitro, rodent and healthy human volunteer models. *Curr Opin HIV AIDS*. 2008;3(6):660-5. PMCID: PMC2680222 PMID: 19373039
7. Flint OP, Noor MA, Hruz PW, Hylemon PB, Yarasheski K, Kotler DP, Parker RA, Bellamine A. The role of protease inhibitors in the pathogenesis of HIV-associated lipodystrophy: cellular mechanisms and clinical implications. *Toxicol Pathol*. 2009;37(1):65-77. PMCID: PMC3170469 PMID: 19171928
8. Hruz PW. Molecular mechanisms for insulin resistance in treated HIV-infection. *Best Pract Res Clin Endocrinol Metab*. 2011;25(3):459-68. PMCID: PMC3115529 PMID: 21663839
9. Hruz PW. HIV and endocrine disorders. *Endocrinol Metab Clin North Am*. 2014;43(3): xvii–xviii. PMID: 25169571
10. Hruz PW. Commentary. *Clin Chem*. 2015;61(12):1444. PMID: 26614228
11. Hruz PW, Mayer LS, and McHugh PR. Growing Pains: Problems with Pubertal Suppression in Treating Gender Dysphoria *The New Atlantis*. 2017;52:3-36.

Book Chapters (most recent editions)

1. Henderson KE, Baranski TJ, Bickel PE, Clutter PE, Clutter WE, McGill JB. Endocrine Disorders in HIV/AIDS. In: *The Washington Manual Endocrinology Subspecialty Consult* Philadelphia, PA: Lippincott Williams and Wilkins; 2008:321-328.

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1 Lee, P. A. *et al.* Global Disorders of Sex Development Update since 2006: Perceptions, Approach and Care. *Horm Res Paediatr* **85**, 158-180, doi:10.1159/000442975 (2016).

2 *American Psychiatric Association. Diagnostic and statistical manual of mental disorders*. 5th edn, (2013).

3 Chen, M., Fuqua, J. & Eugster, E. A. Characteristics of Referrals for Gender Dysphoria Over a 13-Year Period. *Journal of Adolescent Health* **58**, 369-371, doi:https://doi.org/10.1016/j.jadohealth.2015.11.010 (2016).

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5 Heylens, G. *et al.* Gender identity disorder in twins: a review of the case report literature. *J Sex Med* **9**, 751-757, doi:10.1111/j.1743-6109.2011.02567.x (2012).

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7 Steensma, T. D., McGuire, J. K., Kreukels, B. P., Beekman, A. J. & Cohen-Kettenis, P. T. Factors associated with desistence and persistence of childhood gender dysphoria: a quantitative follow-up study. *J Am Acad Child Adolesc Psychiatry* **52**, 582-590, doi:10.1016/j.jaac.2013.03.016 (2013).

8 Zucker, K. J. On the "natural history" of gender identity disorder in children. *J Am Acad Child Adolesc Psychiatry* **47**, 1361-1363, doi:10.1097/CHI.0b013e31818960cf (2008).

9 Bradley, S. J. & Zucker, K. J. Gender Identity Disorder: A Review of the Past 10 YearsG. *Journal of the American Academy of Child & Adolescent Psychiatry* **36**, 872-880, doi:10.1097/00004583-199707000-00008.

10 Hembree, W. C. *et al.* Endocrine treatment of transsexual persons: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab* **94**, 3132-3154, doi:10.1210/jc.2009-0345 (2009).

11 Hembree, W. C. *et al.* Endocrine Treatment of Gender-Dysphoric/Gender-Incongruent Persons: An Endocrine Society* Clinical Practice Guideline. *J Clin Endocrinol Metab*, doi:10.1210/jc.2017-01658 (2017).

12 Nahata, L., Tishelman, A. C., Caltabellotta, N. M. & Quinn, G. P. Low Fertility Preservation Utilization Among Transgender Youth. *Journal of Adolescent Health* **61**, 40-44, doi:https://doi.org/10.1016/j.jadohealth.2016.12.012 (2017).

13 Klink, D., Caris, M., Heijboer, A., van Trotsenburg, M. & Rotteveel, J. Bone Mass in Young Adulthood Following Gonadotropin-Releasing Hormone Analog Treatment and Cross-Sex Hormone Treatment in Adolescents With Gender Dysphoria. *The Journal of Clinical Endocrinology & Metabolism* **100**, E270-E275, doi:10.1210/jc.2014-2439 (2015).

14 Seal, L. J. A review of the physical and metabolic effects of cross-sex hormonal therapy in the treatment of gender dysphoria. *Annals of Clinical Biochemistry* **53**, 10-20, doi:10.1177/0004563215587763 (2016).

15 Adams, N., Hitomi, M. & Moody, C. Varied Reports of Adult Transgender Suicidality: Synthesizing and Describing the Peer-Reviewed and Gray Literature. *Transgend Health* **2**, 60-75, doi:10.1089/trgh.2016.0036 (2017).

16 Dhejne, C. *et al.* Long-term follow-up of transsexual persons undergoing sex reassignment surgery: cohort study in Sweden. *PLoS One* **6**, e16885, doi:10.1371/journal.pone.0016885 (2011).

17 Geier, C. F. Adolescent cognitive control and reward processing: Implications for risk taking and substance use. *Hormones and Behavior* **64**, 333-342, doi:https://doi.org/10.1016/j.yhbeh.2013.02.008 (2013).

18 Olson-Kennedy, J. *et al.* Research priorities for gender nonconforming/transgender youth: gender identity development and biopsychosocial outcomes. *Current Opinion in Endocrinology, Diabetes and Obesity* **23**, 172-179, doi:10.1097/med.0000000000000236 (2016).

Exhibit B

1. Adams, N., M. Hitomi, and C. Moody, *Varied Reports of Adult Transgender Suicidality: Synthesizing and Describing the Peer-Reviewed and Gray Literature.* Transgend Health, 2017. 2(1): p. 60-75. PMCID: PMC5436370.
2. Aitken, M., T.D. Steensma, R. Blanchard, D.P. VanderLaan, H. Wood, A. Fuentes, C. Spegg, L. Wasserman, et al., *Evidence for an altered sex ratio in clinic-referred adolescents with gender dysphoria.* J Sex Med, 2015. 12(3): p. 756-63.
3. Aitken, M., D.P. VanderLaan, L. Wasserman, S. Stojanovski, and K.J. Zucker, *Self-Harm and Suicidality in Children Referred for Gender Dysphoria.* J Am Acad Child Adolesc Psychiatry, 2016. 55(6): p. 513-20.
4. Beek, T.F., B.P. Kreukels, P.T. Cohen-Kettenis, and T.D. Steensma, *Partial Treatment Requests and Underlying Motives of Applicants for Gender Affirming Interventions.* J Sex Med, 2015. 12(11): p. 2201-5.
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8. Bradley, S.J., B. Steiner, K. Zucker, R.W. Doering, J. Sullivan, J.K. Finegan, and M. Richardson, *Gender identity problems of children and adolescents: the establishment of a special clinic.* Can Psychiatr Assoc J, 1978. 23(3): p. 175-83.
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11. Buu, A., A. Dabrowska, M. Mygrants, L.I. Puttler, J.M. Jester, and R.A. Zucker, *Gender differences in the developmental risk of onset of alcohol, nicotine, and marijuana use and the effects of nicotine and marijuana use on alcohol outcomes.* J Stud Alcohol Drugs, 2014. 75(5): p. 850-8. PMCID: PMC4161704.
12. Cohen-Kettenis, P.T., A. Owen, V.G. Kaijser, S.J. Bradley, and K.J. Zucker, *Demographic characteristics, social competence, and behavior problems in children with gender identity disorder: a cross-national, cross-clinic comparative analysis.* J Abnorm Child Psychol, 2003. 31(1): p. 41-53.
13. Cohen-Kettenis, P.T., S.E. Schagen, T.D. Steensma, A.L. de Vries, and H.A. Delemarre-van de Waal, *Puberty suppression in a gender-dysphoric adolescent: a 22-year follow-up.* Arch Sex Behav, 2011. 40(4): p. 843-7. PMCID: PMC3114100.
14. Cohen-Kettenis, P.T., T.D. Steensma, and A.L. de Vries, *Treatment of adolescents with gender dysphoria in the Netherlands.* Child Adolesc Psychiatr Clin N Am, 2011. 20(4): p. 689-700.
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