The BASES Expert Statement on Eligibility for Sex Categories in Sport: DSD Athletes

Produced on behalf of the British Association of Sport and Exercise Sciences by Dr Georgina Stebbings, Dr Adam Herbert, Dr Shane Heffernan, Prof Roger Pielke Jr. and Dr Alun Williams.

Introduction

In 2019, the Court of Arbitration for Sport ruled against Caster Semenya, and in favour of World Athletics (formerly IAAF), to uphold their 'Eligibility Regulations for the Female Classification (Athletes with Differences of Sex Development). Consequently, 'relevant athletes' (specifically XY sex chromosome women with naturally occurring serum testosterone ≥5 nmol/L) cannot compete in international races between 400 m and 1 mile, unless they reduce serum testosterone below 5 nmol/L for at least 6 months.

Some consider this decision a victory for female athletes in maintaining a protected category in elite sport, to others it permits unnecessary discrimination. Nonetheless, the ramifications of this decision, and those of possible future challenges, are substantial for all female athletes. Through this expert statement, we provide recommendations for practitioners, researchers and policymakers working in this area.

Background and evidence

Conventionally, biological sex is binary, with differences in sex chromosomes, anatomy, hormone levels and secondary sex characteristics aligning as either male or female. Men typically have greater muscle mass (\sim 37%), strength (\sim 55%), and maximal rate of oxygen consumption (~25%) (Hilton & Lundberg, 2020). Thus, open sport competition between men and women would be unfair and, in some circumstances, unsafe.

Several genetic conditions collectively referred to as differences in sex development (DSDs), however, can lead to atypical development of binary sex phenotypes. Of concern in sport is that unusual genetic variations in some women DSD athletes may confer performance advantages that are ordinarily excluded from the women's category. However, World Athletics' regulations are based on insufficient and flawed empirical evidence.

According to the World Health Organization (WHO), sex refers to multiple biological factors including genetics, hormone levels and anatomy, and while males typically possess XY sex chromosomes and females are typically XX, there are XY females and XX males. Thus, no single factor or formula exists that neatly determines whether an individual is male or female. Yet in establishing their Eligibility Regulations, World Athletics overlooked this reality in favour of using mainly sex chromosomes and serum testosterone levels to differentiate between men and women¹.

Serum testosterone is a strong candidate to differentiate between men and women, with 8-30 nmol/L typical in XY men substantially greater than 0.1-2 nmol/L typical in XX women (Handelsman et al., 2018). Testosterone, and its often more potent metabolite dihydrotestosterone (DHT), increase muscle mass, strength, and other aspects of physical performance. In DSD women, however, due to rare genetic mutations, considerable variability exists in the synthesis of, and response to, circulating testosterone (Table 1).

In DSDs affecting androgen synthesis/action, circulating testosterone is often within the typical male range, yet the ability to process this testosterone is compromised. While women with these DSDs might have natural testosterone levels exceeding the typical female range, and some degree of androgenisation during puberty, the extent of any performance advantage conferred by these biological changes remains unclear. Any advantage may be negligible, but the implicit assumption made in the regulations is that the advantage is similar in magnitude to what typical XY males enjoy over typical XX females. In the absence of evidence to this effect, the existing evidence is insufficient to justify exclusion of these athletes.

Nonetheless, if future research demonstrates conclusively women DSD athletes are advantaged because of their genetics, should this be considered differently to other genetic advantages experienced by most/all elite athletes? The R577X polymorphism on the actinin alpha 3 gene (ACTN3) is a common genetic variant known to convey ~ 1% advantage in elite sprint running (Papadimitriou et al., 2016). A rare mutation to the erythropoietin receptor (EPOR) gene enhances red blood cell synthesis by ~60% (de la Chapelle et al., 1993). Yet for athletes possessing either advantageous variant, there are no eligibility regulations - nor do we believe there should be. Importantly, advantages conferred by variants in ACTN3, EPOR and thousands of other genes, influence athletic performance of both men and women, whereas variants involved in DSDs being regulated by World Athletics affect women only. As the affected DSD women have female gender identity and biological characteristics compatible with the broad WHO definition of female sex, then we do not accept the unscientific recategorisation of these athletes by World Athletics and subsequent use of that recategorisation to justify discrimination against them.

Table 1. Categories of DSDs affected by the World Athletics Eligibility Regulations

DSD	Gene	Consequence of mutation	Likely degree of androgenisation
I7β-hydroxysteroid dehydrogenase type 3 deficiency	HSD17B3	Reduced conversion of androstenedione to testosterone	Partial
5α-reductase type 2 deficiency	SRD5A2	Reduced conversion of testosterone to DHT	Partial
Partial androgen insensitivity syndrome	AR	Reduced androgen receptor binding to testosterone and DHT	Partial
Ovotesticular DSD	SRY, DMRT I or SOX9	Both ovarian and testicular tissue present	Partial

¹Women with XY chromosomes deemed adequately sensitive to testosterone via controversial invasive clinical assessment.









Regardless of whether one accepts discrimination against women DSD athletes is justified, the application of regulations to five middle-distance running events only, and not all athletic events, is paradoxical. It is based primarily on one controversial study, funded by World Athletics (Bermon & Garnier, 2017), which contained extensive data errors leading to calls for retraction (Pielke et al., 2019). The study reported elite women athletes with higher testosterone levels (not a subset of 46 XY women) performed ~2-5% better than those with lower testosterone in five of 22 athletic events. The authors could have concluded there was no overall competitive advantage of higher testosterone within the women's category. Instead, they concluded that in five athletic events only (<25% of events), higher testosterone gives women a competitive advantage. In doing so, the authors incorrectly imply that higher testosterone, and subsequent potential for increased muscle mass and strength, is not advantageous for sprinting, jumping, or throwing events. Similarly, they imply the potential for elevated haemoglobin or reduced fat mass is not advantageous for long-distance events. If this were accurate, we might expect the largest performance differences between XY men and XX women in these same five events, but that is not the case, with men outperforming women by \sim 12% in running events, regardless of distance, and by \sim 20% for most field events (Hilton & Ljundberg, 2020). Furthermore, the five events were running 400-800 m and two field events, whereas the restricted events under World Athletics' regulations are running 400 m to I mile.

Finally, the regulations require affected athletes to lower their natural circulating testosterone to <5 nmol/L for at least 6 months to compete in the restricted events. Testosterone is lowered via hormone administration or surgery; the unsolicited implementation of which for non-pathological conditions violates established medical ethics, may result in harmful side effects, and led the World Medical Association (2019) to advise physicians against administering treatment solely to alter sport performance. Compounding these major ethical concerns, there is poor evidence of 6 months testosterone suppression nullifying any/all competitive performance advantage in women DSD athletes who experienced higher natural testosterone since puberty.

Conclusions and recommendations

- · Exclusion of women DSD athletes from participating in the female category in the five currently restricted running events, or any other event/sport, is not scientifically or ethically
- Before pursuing such restriction, further independent research comparing biochemical, physiological and performance differences between women athletes with and without DSDs is required, and longitudinally in women DSD athletes who voluntarily reduce serum testosterone.
- Medical practitioners should follow World Medical Association guidance and refuse to administer medication or surgery to reduce natural levels of testosterone unless based on medical
- Sport and exercise scientists supporting women athletes should familiarise themselves with the regulations and encourage DSD athletes to train for sports/events not currently restricted.



Dr Georgina Stebbings

Georgina is Senior Lecturer in Sport and Exercise Physiology at Manchester Metropolitan University. She is a BASES accredited sport and exercise scientist.



Dr Adam Herbert

Adam is Lecturer in Sport and Exercise Science at Birmingham City University.



Dr Shane Heffernan

Shane is Senior Lecturer (Research) at A-STEM, Swansea University.



Prof Roger Pielke Jr.

Roger is Professor in Environmental Studies at the University of Colorado Boulder. He was an independent expert witness at the Caster Semenya v IAAF case at CAS in 2019.



Dr Alun Williams

Alun is Reader in Sport and Exercise Genomics at Manchester Metropolitan University. He was an independent expert witness at the Caster Semenya v IAAF case at CAS in 2019.

References:

Bermon, S. & Garnier, P-Y. (2017). Serum androgen levels and their relation to performance in track and field: mass spectrometry results from 2127 observations in male and female elite athletes. British Journal of Sports Medicine, 51, 1309-1314.

de la Chapelle, A., Träskelin, A. L. & Juvonen, E. (1993). Truncated erythropoietin receptor causes dominantly inherited benign human erythrocytosis. Proceedings of the National Academy of Sciences of the United States of America,

Handelsman, D. J., Hirschberg, A. L. & Bermon, S. (2018). Circulating testosterone as the hormonal basis of sex differences in athletic performance. Endocrine Reviews, 39, 803-829.

Hellmann, P. et al. (2012). Male participants with partial androgen insensitivity syndrome: a longitudinal follow-up of growth, reproductive hormones and the development of gynaecomastia. Archives of Disease in Childhood, 97, 403-409.

Hilton, E. N. & Lundberg, T. R. (2020). Transgender women in the female category of sport: perspectives on testosterone suppression and performance advantage. Sports Medicine, doi: 10.1007/s40279-020-01389-3

Papadimitriou, I. D. et al. (2016). ACTN3 R577X and ACE I/D gene variants influence performance in elite sprinters: a multi-cohort study. BMC Genomics, 17,

Pielke, R., Tucker, R., & Boye, E. (2019). Scientific integrity and the IAAF testosterone regulations. The International Sports Law Journal, 19(1), 18-26.

World Medical Association (2019). Physician leaders reaffirm opposition to IAAF rules. Available: https://www.wma.net/news-post/physician-leaders-reaffirmopposition-to-iaaf-rules/

Download a PDF of this article: www.bases.org.uk/BASES-Expert-Statements

Copyright © BASES, 2020

Permission is given for reproduction in substantial part. We ask that the following note be included: "First published in The Sport and Exercise Scientist, Issue 68, Summer 2021 Published by the British Association of Sport and Exercise Sciences - www.bases.org.uk"



