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Perspective

The Impact of Polymorphism on Testosterone Intervention Goals

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DESCRIPTION

There is plenty of evidence from the past ten years to support the increased significance of Androgen Receptor (AR) CAG repeat polymorphism in andrology. The peripheral effects of testosterone can be conditioned by this hereditary factor, which also affects male sexual function and fertility, cardiovascular risk, body composition, bone metabolism, the risk of prostate and testicular cancer, mental health, and the beginning of neurodegenerative illnesses. In this review, we thoroughly evaluate the evidence from the literature and pinpoint how the AR CAG repeat polymorphism affects the effects of systemic testosterone. The primary goal was to provide a text that might clarify the various, frequently contrasting studies claiming that CAG repeat polymorphism has an impact on the targets of testosterone action.

Direct control of gene transcription serves as AR's primary method of action. Heat shock proteins dissociate, move into the nucleus, and dimerize upon the binding of an androgen to its receptor due to a conformational shift. An area of DNA known as a hormone response element is where the AR dimer attaches, up- or down-regulating transcription of a particular gene. A nongenomic mechanism that involves the quick activation of kinase-signaling cascades and the modification of intracellular calcium levels is another way that AR may function. Regarding the direct action, testosterone has an effect *via* AR both directly and indirectly through its metabolite, dihydrotestosterone, which is created when testosterone is transformed by 5-α reductase.

The AR gene has eight exons and is situated at q11–q12 on the X chromosome. The polymorphic CAG repeat sequence found in exon 1 of the AR gene codes for polyglutamine stretches of the AR transactivation domain and typically ranges in number from 10 to 35. Several data point to a negative correlation between CAG number and AR transcriptional activity. Kennedy syndrome individuals really have more than 40 CAG repeats along with diminished virilization, testicular atrophy, decreased sperm production, and infertility. Additionally, other studies have demonstrated a relationship between shorter CAG repeats and prostate illness, notably cancer and benign enlargement, enhanced seminal parameters, and enhanced mineral bone density.

There are racial variations in the CAG repeat length of the AR gene among African, Caucasian, and Asian populations. To comprehend the variations in androgen sensitivity in the general population as well as the variability of the AR gene, it is crucial to evaluate ethnic groupings. Although some African subpopulations appear to have a shorter tract, the allele expansion among Africans was shown to occur between the ages of 18 and 20. Nonetheless, the CAG expansion is lengthier in Caucasian and Asian groups, with the mean number of CAG repeats being, respectively, 21–22 in Caucasians and 23 in Asians. In this review, we'll talk about the evidence from the literature that suggests the AR CAG repeat polymorphism influences how testosterone affects the body's periphery. Our primary goal was to make an updated contribution that could clarify the many, frequently contrasting results on the impact of CAG repeat polymorphism on the targets of testosterone action.

It has not yet been thoroughly investigated how CAG repeat polymorphism affects sexual function. In actuality, not many research have looked into this matter, and the knowledge of this topic is lacking. There is disagreement on the function of CAG polymorphism in transversal studies, which likely caused by the heterogeneity of recruited samples and methodological approaches. All aspects of sexual function measured by IIEF-15 were found to be inversely linked with the amount of CAG repeats in the AR gene. However, this was not seen when total testosterone levels were equal to or lower than 3.40 ng/mL. CAG repeat lengths 25 had a considerably higher chance of developing andropausal symptoms (ADAM questionnaire) than individuals with AR CAG repeat lengths.

On the other hand, there was no significant correlation between the symptoms of erectile dysfunction and the length of the CAG repeat in a population-based study involving 340 controls and 79 males with complaints of erectile dysfunction. It should be noted, nevertheless, that when assessing erectile dysfunction symptoms, these writers only employed one item from the National Institutes of Health Consensus Development Panel on Impotence. Also, another study found that the number of CAG repeats was positively connected with depression in 213 randomly chosen men between the ages of 41 and 70, while those who had CAG repeats higher than or equivalent to 23

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reported decreasing potency less frequently than the others.

Notwithstanding the many contentious findings in each androgen-related activity, a substantial body of evidence suggested that the AR CAG polymorphism had a significant role in conditioning the peripheral impact of testosterone. However, this contribution still needs to be further examined.

Some associations are noteworthy, but they still need to be verified. With respect to the technique (transversal/longitudinal studies), the number of patients evaluated, and the clinical characteristics of the investigated participants, we believe that the disparate results may be justified. Also, it must be noted that not all andrological effects have been thoroughly examined to yet (e.g., sexual function). The paths that scientific research will need to take in order to succeed are uniformity of methodological evaluation and the investigation of seldom considered results to make this critical point clear. Nowadays, routine use of AR CAG polymorphism is not advised. Yet, given the theoretical possibility of identifying individuals who are more or less at risk for specific illnesses and more or less receptive to testosterone treatment, it may soon become of therapeutic consequence. In this latter instance, measuring the length of the CAG repeat could help us specifically personalize testosterone replacement therapy because men with shorter CAG repeats might need lower doses of the hormone while those with longer repetitions might need higher ones.