Testicolopatia fertile

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Membro Consiglio Direttivo Regionale **SIE** (Società Italiana di Endocrinologia)
Academician **EAA** (European Academy of Andrology).

Conflitti di interesse

Ai sensi dell'art. 3.3 sul conflitto di interessi, pag 17 del Regolamento Applicativo Stato-Regioni del 5/11/2009, dichiaro che negli ultimi 2 anni ho avuto rapporti diretti di finanziamento con i seguenti soggetti portatori di interessi commerciali in campo sanitario:

- IBSA
- AstraZeneca

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Ridotta funzione endocrina testicolare

1. Insulino-resistenza e scarso compenso glicemico
2. Aumento rischio CV: maggiore rischio di mortalità
3. Osteoporosi metabolica
4. Ridotta funzione muscolare e avvio processi sarcopenici
5. Declino cognitivo e disturbi tono umore
6. Alterata risposta immunitaria e differente profilo di suscettibilità oncologica

Contesto clinico

1. Disordini della fertilità maschile
2. Disordini della sessualità maschile

Riduzione volume testicolare

J Endocrinol Invest. 2013 May;36(5):287-92. doi: 10.3275/8514. Epub 2012 Jul 9.

Anthropometric, penile and testis measures in post-pubertal Italian males.

Foresta C[1], Garolla A, Frigo AC, Carraro U, Isidori AM, Lenzi A, Ferlin A.

Author information

Abstract

BACKGROUND: Relationships between anthropometric measures, body proportions, weight and penile dimensions in young adult males have not been previously analyzed. Furthermore, although male fertility has declined in last decades, no data on testicular volume (the best surrogate measure for spermatogenic potential) are available for the general population of young men in Italy.

AIM: To analyze anthropometric measures and proportions, testicular volumes, and penile dimensions in a large cohort from the general population of young Italian men aged 18-19 yr. MATERIALS/SUBJECTS: We analyzed 2019 students aged 18-19 years for height, weight, body mass index (BMI), waist circumference, arm span, pubis-to-floor and crown-to-pubis length, and penile dimensions. Testicular volumes were measured by ultrasound in 776 subjects.

RESULTS: Thirty-six percent of the subjects had a pathological arm span-height difference (>3 cm) and 44.7% had a pathological pubis-to-floor/ crown-to-pubis ratio (≤ 0.92). The mean penis length was 8.9 ± 1.4 cm and the penis circumference was 9.5 ± 1.0 cm. BMI was positively associated with arm span-height difference and negatively with penis length; 23.2% of the subjects had low mean testicular volume (<12 ml).

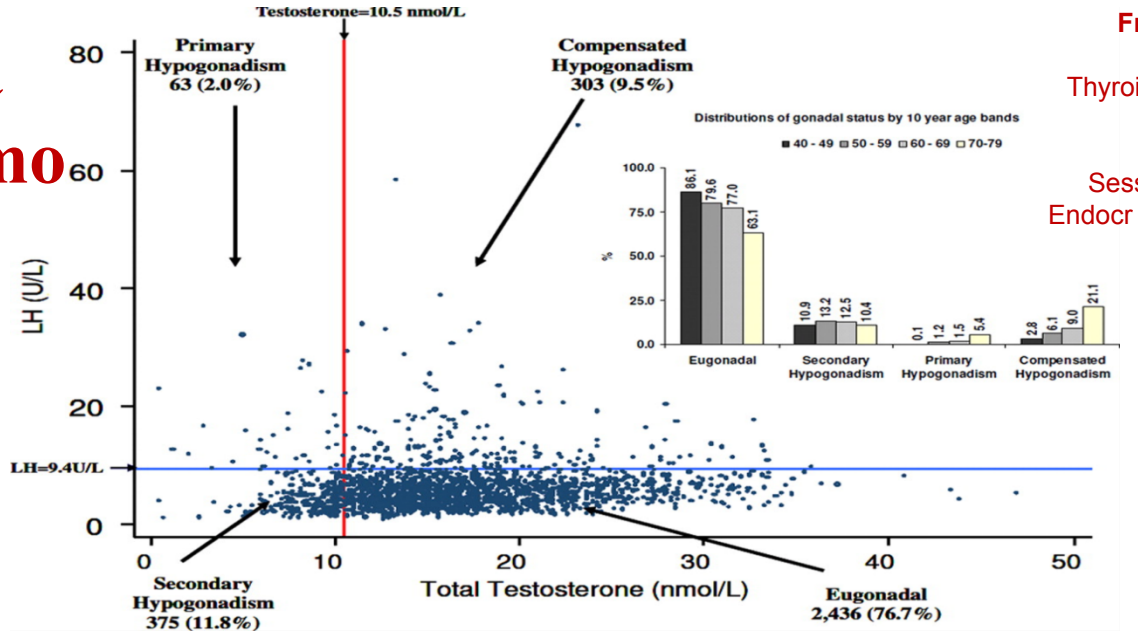
CONCLUSIONS: The findings highlight a strong influence of BMI on skeletal proportions and penis length, identify a large proportion of subjects with testicular hypotrophy at risk for future fertility, and suggest to consider worldwide studies to redefine normal values for arm span-height difference and upper/ lower body segment ratio.

Frequenza ipogonadismo

Characteristics of Secondary, Primary, and Compensated Hypogonadism in Aging Men: Evidence from the European Male Ageing Study

J Clin Endocrinol Metab, April 2010, 95(4):1810–1818

Design: The study was a cross-sectional survey on 3369 community-dwelling men aged 40–79 yr in eight European centers.



Frequenza noduli tiroidei
Sesso maschile = 1%
Thyroid. 2009 Nov;19(11):1167-214

Frequenza DM 2
Sesso maschile > 20 aa = 9.6%
Endocr Rev. 2016 Jun; 37(3): 278–316

From: Characteristics of Secondary, Primary, and Compensated Hypogonadism in Aging Men: Evidence from the European Male Ageing Study
J Clin Endocrinol Metab. 2010;95(4):1810-1818. doi:10.1210/jc.2009-1796

J Clin Endocrinol Metab | Copyright © 2010 by The Endocrine Society

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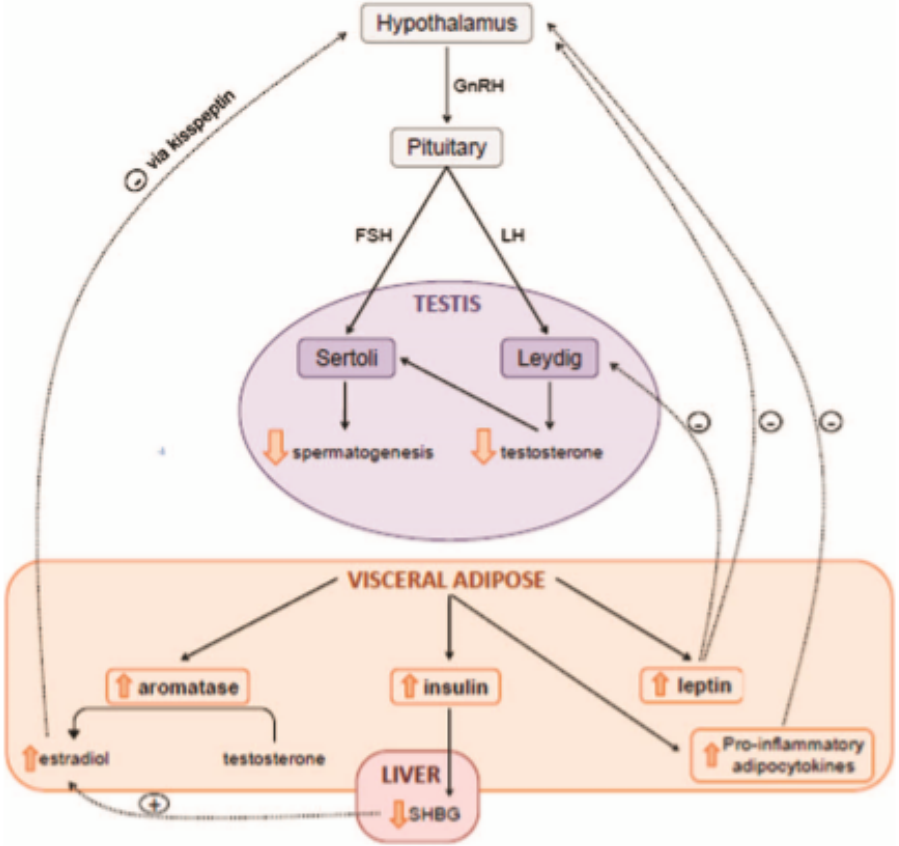
Quante forme di ipogonadismo ?_

Ipogonadismo ipogonadotropo
Ipogonadismo ipergonadotropo
Ipogonadismo da resistenza periferica
Ipogonadismo ad insorgenza tardiva
Ipogonadismo compensato

Ipogonadismo metabolico



Twenty percent up to 64% of obese men have low total T or low free T levels

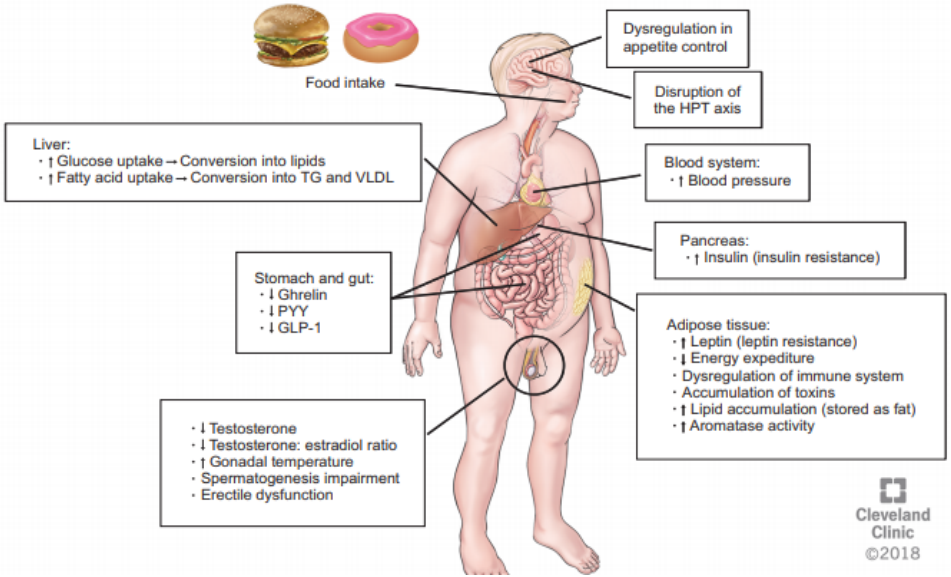
Curr Opin Endocrinol Diabetes Obes, 14 (2007), pp. 226-234

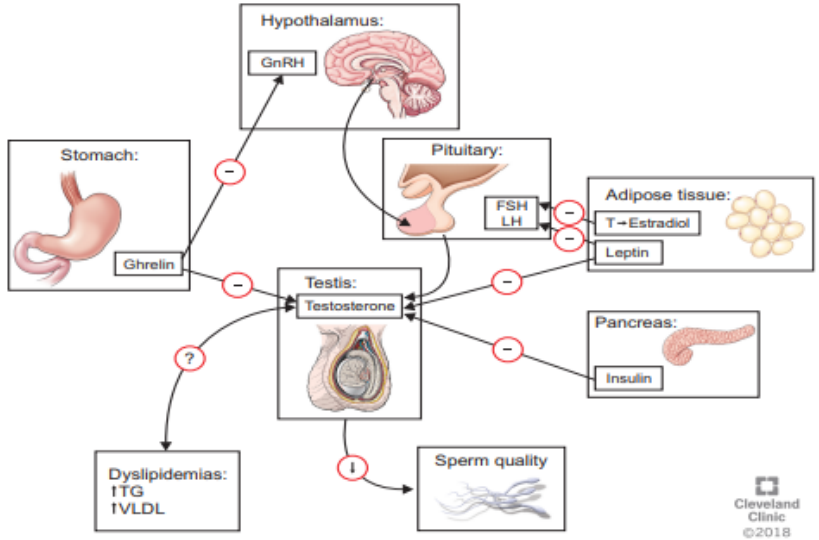
Human Fertility, 2015; 18(3): 184–193
© 2015 The British Fertility Society
ISSN 1464-7273 print/ISSN 1742-8149 online
DOI: 10.3109/14647273.2015.1070438

Review Article

pISSN: 2287-4208 / eISSN: 2287-4690
World J Mens Health Published online Oct 22, 2018
https://doi.org/10.5534/wjmh.180055

The World Journal of
Men's Health





Ma che tipo di ipogonadismo è ?

Ipogonadismo metabolico

(a patogenesi sincrona/mista)

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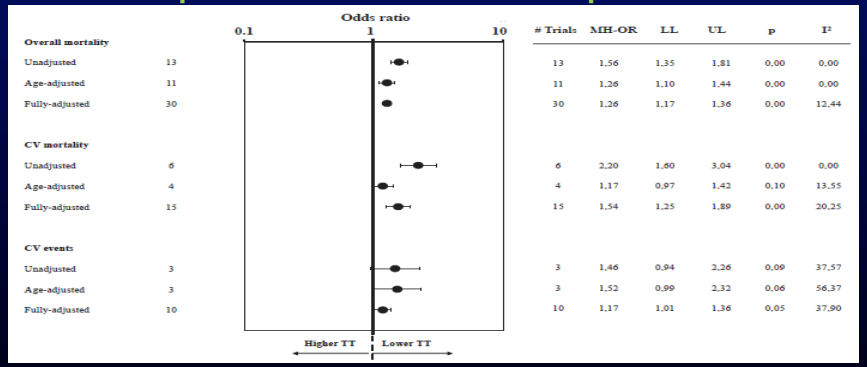
THE JOURNAL OF SEXUAL MEDICINE
An Official Journal of The International Society for Sexual Medicine

September 2018 Volume 15, Issue 9, Pages 1260–1271

Endogenous Testosterone Levels and Cardiovascular Risk: Meta-Analysis of Observational Studies

Giovanni Corona, MD, PhD, Giulia Rastrelli, MD, PhD, Giuseppe Di Pasquale, MD, Alessandra Sforza, MD, Edoardo Mannucci, MD, PhD, Mario Maggi, MD, PhD

- Inclusi **37 studi osservazionali** (1988-2017)
- Casistica di **43041 uomini** (età media pari a 63.5 anni)
- Tempo medio di follow-up: **333 settimane (> 6 anni)**



I livelli di TT al tempo dell'arruolamento predicono un aumento di:

- **mortalità** da causa cardio-vascolare
- **morbilità** cardio-vascolare
- **incidenza** delle malattie cardio-vascolari

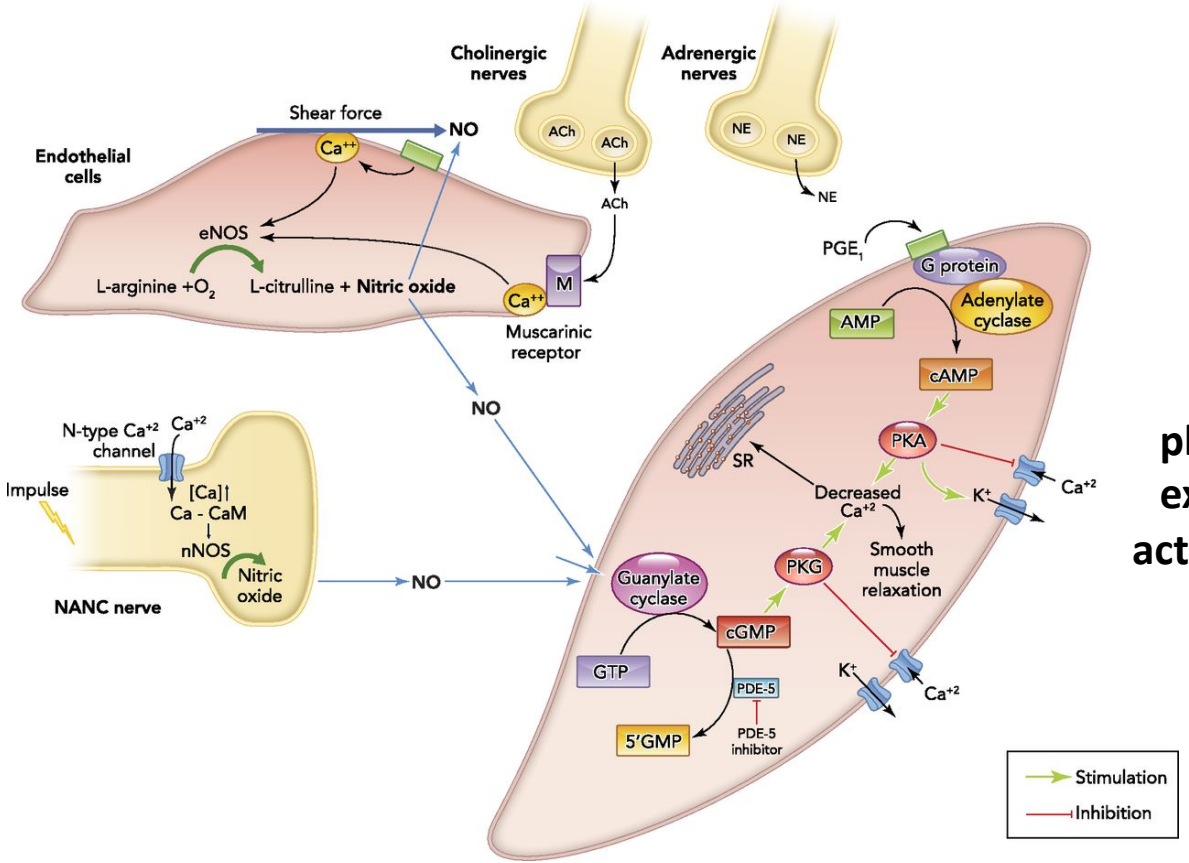
Testosterone: a vascular hormone in health and disease

Journal of Endocrinology
(2013) **217**, R47–R71

Figure 3

Potential mechanisms by which testosterone influences vascular reactivity. Endothelium-independent mechanisms of testosterone are considered to occur primarily via the inhibition of voltage-operated Ca^{2+} channels (VOCCs) and/or activation of K^+ channels (KCs) on smooth muscle cells (SMCs). Testosterone shares the same molecular binding site as nifedipine on the α1-C subunit of L-type Ca^{2+} channels to cause channel blockade at physiological concentrations, thus restricting Ca^{2+} influx and inducing vasodilation (1). Some reports suggest that pharmacological concentrations of testosterone can activate Ca^{2+} channel opening and induce vasoconstriction through enhanced Ca^{2+} influx. Alternatively, testosterone activates voltage-operated K^+ channels and/or large-conductance, Ca^{2+}-activated K^+ channels, increasing intracellular K^+ efflux to induce hyperpolarisation and subsequent vasorelaxation (2). Testosterone may also inhibit intracellular Ca^{2+} influx via store-operated Ca^{2+} channels (SOCCs) by blocking response to prostaglandin F2α (PGF2α) (3). Endothelium-dependent actions of testosterone may be long-term genomic and non-genomic effects. Testosterone increases the expression of endothelial nitric oxide synthase (eNOS) and enhances nitric oxide (NO) production (4). Whether these actions are via direct androgen receptor binding, aromatisation to oestradiol and oestrogen receptor activation, or interaction with alternative intracellular signalling pathways remains unknown. Additionally, testosterone treatment has been shown to increase eNOS activity by enhancing phosphorylation potentially via the non-genomic activation of intracellular signalling pathways and Ca^{2+} influx (5). Increased NO acts on SMCs to induce vasorelaxation by activating cGMP, which in turn activates cGMP-dependent protein kinases (PKG) (6). PKG phosphorylates and activates sarcoplasmic reticulum Ca^{2+}-ATPase (SERCA), which increases the uptake of Ca^{2+} into the superficial sarcoplasmic reticulum (SR) and therefore decreases global cell Ca^{2+}. PKG also increases Ca^{2+} release into the submembrane space where the activation of K^+ channels ensues and PKG may additionally inhibit Ca^{2+} channel activity directly to cause vasorelaxation. T, testosterone.

Journal of Endocrinology (2013) **217**, R47–R71



Lasker G F et al. Physiology 2013;28:262-269

Physiology

©2013 by American Physiological Society

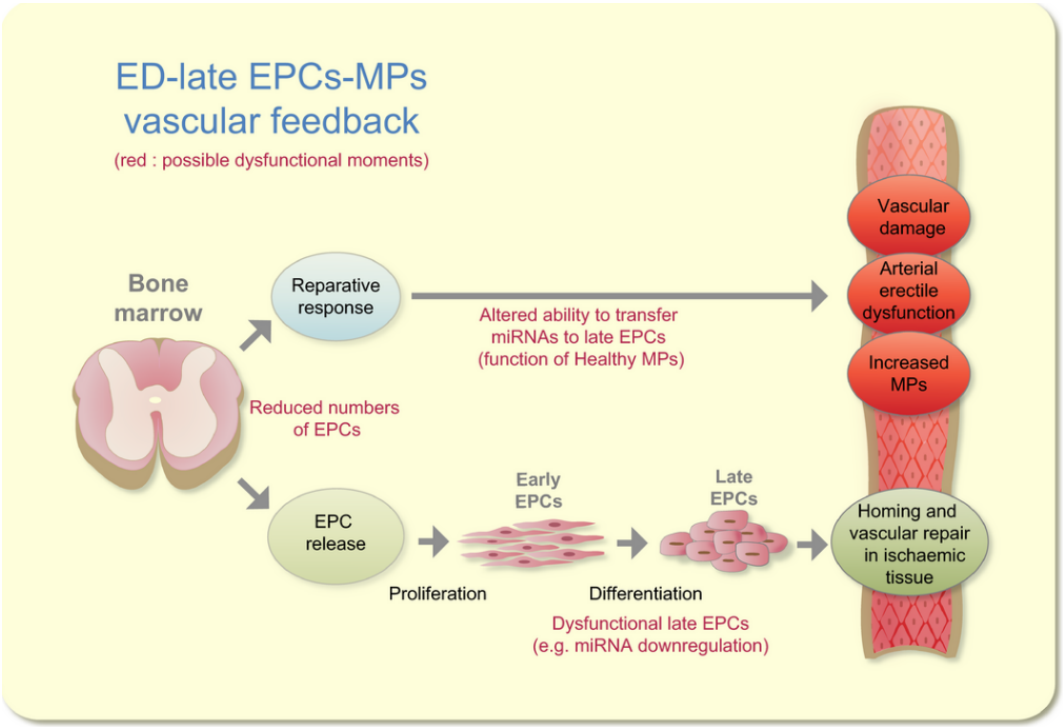
Androgens regulate phosphodiesterase type 5 expression and functional activity in corpora cavernosa

Endocrinology. 2004; 145(5):2253-63.

Androgens stimulate endothelial progenitor cells through an androgen receptor-mediated pathway

Clin Endocrinol (Oxf). 2008;68:284-9

ACTA PHYSIOLOGICA
Acta Physiol 2017

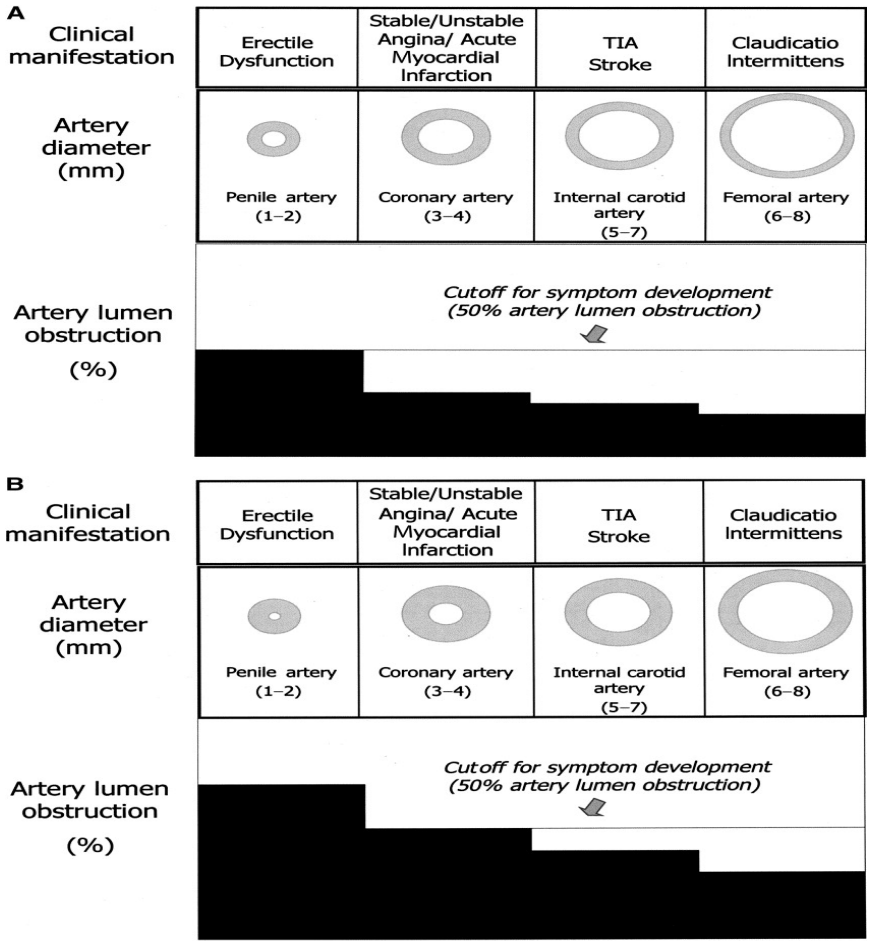


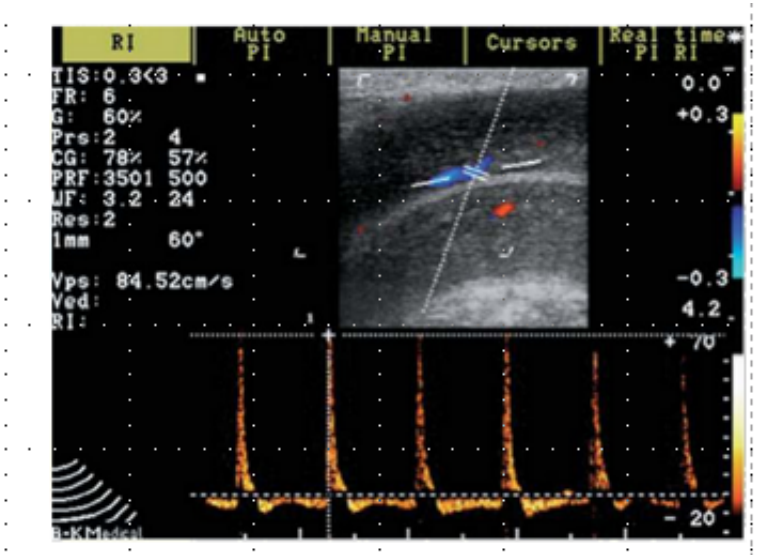
R. A. Condorelli, A. E. Calogero and S. La Vignera
Department of Clinical and Experimental Medicine, Policlinico "G. Rodolico", University of Catania, Catania, Italy
E-mail: sandrolavignera@unict.it

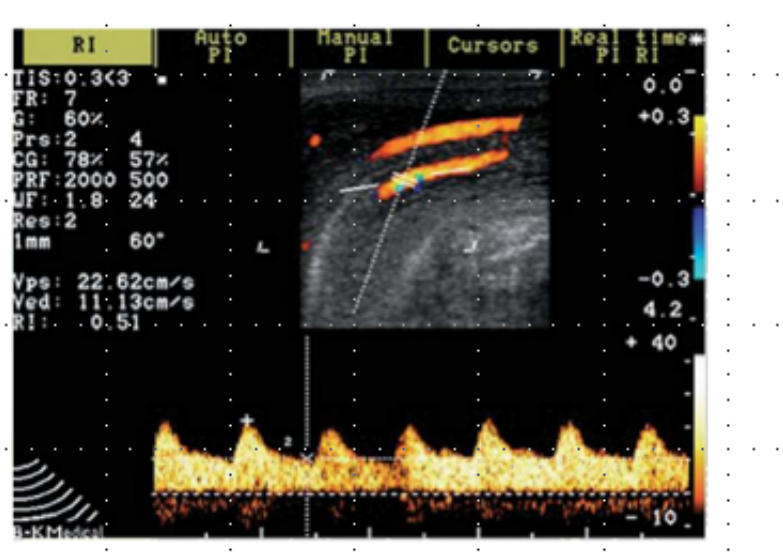
Am J Cardiol. 2005 Dec 26;96(12B):19M-23M. Epub 2005 Nov 4.

The artery size hypothesis: a macrovascular link between erectile dysfunction and coronary artery disease.

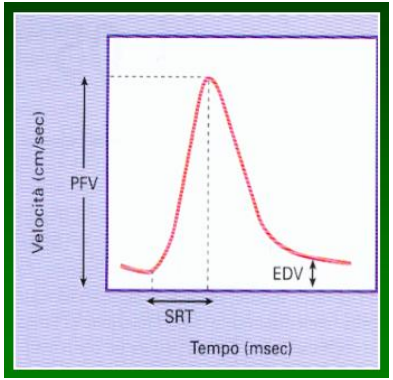
Montorsi P[1], Ravagnani PM, Galli S, Rotatori F, Briganti A, Salonia A, Rigatti P, Montorsi F.







Picco di velocità di flusso	> 30 cm/sec
Tempo di innalzamento sistolico	< 110 m/sec
Velocità diastolica finale	< 5 cm/sec
Indice di resistenza	1.0



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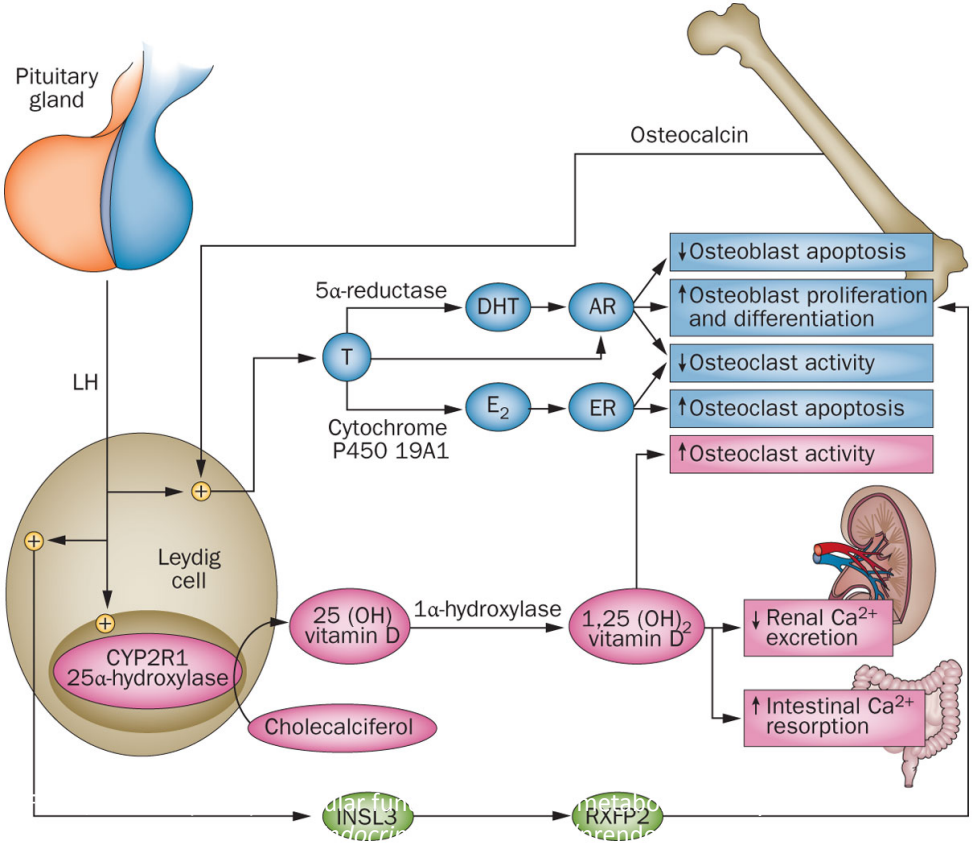

nature REVIEWS ENDOCRINOLOGY

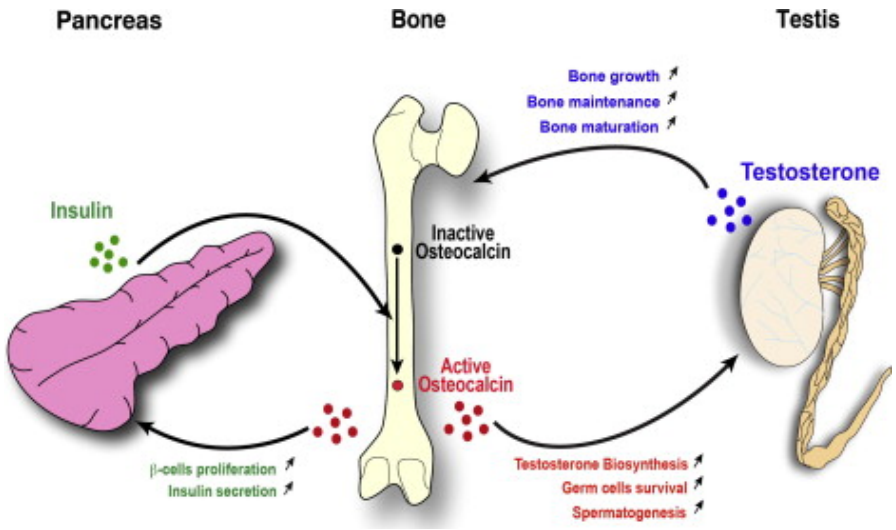

Fig. 3 Osteocalcin-stimulated testosterone biosynthesis is positively regulated by insulin signaling in osteoblasts. Insulin signaling in osteoblasts stimulates the bio-activation of osteocalcin. In a feedback loop control, undercarboxylated active osteoca...

Gerard Karsenty , Franck Oury

Regulation of male fertility by the bone-derived hormone osteocalcin

Molecular and Cellular Endocrinology, Volume 382, Issue 1, 2014, 521 - 526

http://dx.doi.org/10.1016/j.mce.2013.10.008

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Contesto clinico

1. Disordini della fertilità maschile
2. Disordini della sessualità maschile

Testosterone Therapy in Men With Hypogonadism: An Endocrine Society* Clinical Practice Guideline FREE

2018

1.0 Diagnosis of Hypogonadism in Men

Hypogonadism is a clinical syndrome that results from failure of the testis to produce physiological concentrations of testosterone (T) (T deficiency) and/or a normal number of spermatozoa due to pathology at one or more concentrations of the hypothalamic–pituitary–testicular axis (5, 6). Abnormalities at the testicular level cause primary hypogonadism, whereas defects of the hypothalamus or the pituitary cause secondary hypogonadism. Hypogonadism also can result from defects that affect both the testis and the hypothalamus–pituitary unit. This guideline describes the diagnosis, treatment, and monitoring of T deficiency and does not address isolated defects of spermatogenesis.

The Journal of Clinical Endocrinology & Metabolism, Volume 103, Issue 5, 1 May 2018, Pages 1715–1744, https://doi.org/10.1210/jc.2018-00229

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GRAZIE

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