# SAT-041 Testosterone Reduces Atherosclerosis and Plaque Specific Inflammatory Markers in the ApoE-/- Mouse Model [abstract only]

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a Karyotype in fibroblasts culture from oral cavity sample. The results revealed low IGF-1 and mosaic TS in 14%. We preformed 2 provocative tests which revealed low growth hormone peak < 5 ng/ml. A brain and pituitary MRI to exclude pituitary lesions or structural abnormalities revealed gliomas of the optic chiasma and the right optic nerve with characteristic NF1 "spots" (regions of signal abnormality in T2 sequences) involving the basal ganglia, cerebellum and the right temporal lobe. DNA sequencing targeted to a gene panel related to NF1 and NF2 revealed a novel de novo heterozygous NF1 gene mutation in exon 28 [3764A>G];[=] p.[Gln1255Arg].

**Discussion:** NF1- Gliomas are most commonly seen in young children, (mean 4.5 years). Only 1/3 of affected children will require therapeutic intervention. However early diagnosis, of optic gliomas is important. Our patient was completely asymptomatic by the time of diagnosis and no other symptom or sign of NF1 was apparent. Ophthalmologic examination was normal, but visual electrophysiologic testing was abnormal as far the right optic nerve is concerned. The oncology team decided to preform chemotherapy. In TS impaired growth is related to resistance in GH. Some studies suggested that there could be a relationship between GHD and NF1 even in the absence of an organic pituitary damage. In our patient it has been decided not to treat with GH and closely track the patient's growth.

**Conclusion:** Coexistence of NF1 with TS is rare. Awareness is needed as early identification and treatment of CNS gliomas can prevent visual loss and severe co-morbidities.

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Rare Presentation of Neurofibromatosis and Turner Syndrome in a Pediatric Patient. Pediatr Rep. 2017 Jun 26; 9(2): 6810

# Reproductive Endocrinology

## MALE REPRODUCTIVE HEALTH - FROM HORMONES TO GAMETES

### *Testosterone Reduces Atherosclerosis and Plaque Specific Inflammatory Markers in the ApoE-/- Mouse Model*

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**SAT-041**

Low serum testosterone in men is an established cardiovascular risk factor and epidemiological evidence demonstrates an association between low testosterone and with coronary events. Clinical evidence suggests that testosterone therapy (Tth) can improve key cardiovascular risk factors in men and surrogate measures of atherosclerosis, the chronic inflammatory process underlying cardiovascular disease. Atherosclerotic plaque-specific testosterone actions are not fully understood. The present study investigates the influence of testosterone on mediators of vascular inflammation and plaque burden in an *in vivo* model of atherosclerosis. ApoE-/- mice were either sham operated, castrated or castrated and received fortnightly intramuscular injections of physiological doses of testosterone (mixed testosterone esters, Sustanon 100) to create 3 experimental groups; normal testosterone, testosterone deficient and testosterone replaced respectively. All groups were fed a high-fat 'Western' diet for 16 weeks. Lipid deposition in the aortic root was assessed by Oil Red O as an indication of atherosclerotic burden. Plaque composition was assessed immunohistochemically for indicators of stability including collagen content via Masson's trichrome, and α-smooth muscle actin (αSMA) as well as markers of inflammation including vascular cell adhesion molecule-1 (VCAM-1), intracellular adhesion molecule-1 (ICAM-1), endothelial-leukocyte adhesion molecule 1 (E-Selectin), and a pan monocyte/macrophage marker (MOMA2). Testosterone deficient castrated mice had significantly increased lipid accumulation in the aortic root compared to testosterone replete sham-operated littermates (48% intima-media area vs 40%, $p<0.05$). Tth in castrated mice reversed this effect (39%, $p<0.05$). Plaque stability was not altered between groups. MOMA2 staining indicated increased infiltration and localisation of monocytes/macrophages in the plaques of castrated mice compared to sham-operated (positive staining (% of plaque) 77% vs 59%, P=0.062) and Tth treatment reduced this (77% vs 63%, P=0.1). Vascular cell adhesion molecule-1 (VCAM-1) and intracellular adhesion molecule-1 (ICAM-1) expression were reduced in castrated mice receiving Tth compared to castrated mice receiving saline (33% vs 46%, $p<0.05$; and 39% vs 58%, P=0.084 respectively). No significant difference in expression of E-Selectin and αSMA were observed between groups. These findings demonstrate that low testosterone increases aortic root lipid deposition and inflammatory composition in a mouse model of atherosclerosis. Increasing testosterone levels through Tth reduces plaque specific inflammatory markers and atherosclerotic burden. This indicates an anti-inflammatory mechanism by which testosterone can protect against the development and progression of atherosclerosis to reduce cardiovascular risk in men.

# Diabetes Mellitus and Glucose Metabolism

## TYPE 1 DIABETES MELLITUS

### *A Case of Autoimmune Polyglandular Syndrome Type 2 and Guillain-Barré Syndrome*

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**SAT-683**

Background: Autoimmune polyglandular syndrome type 2 (APS2) is defined by the occurrence of two or more autoimmune diseases, with Addison's disease being most prevalent, and autoimmune thyroid disease and type 1 diabetes mellitus also being common. Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyradiculopathy that is also autoimmune in nature, resulting in ascending muscle weakness or paralysis.

Clinical Case: A 49 year old female with past medical history of vitiligo, subclinical hyperthyroidism, and Guillain-Barré