providing it. Of the 31% of residents that correctly identified indications for bariatric surgery, only 9% reported referring patients they considered appropriate for surgery. Notably, a higher CS was significantly correlated with more frequent bariatric surgery referrals (r = 0.29; p = 0.015), lifestyle counselling (r = 0.33; p = 0.004), WMM prescription (r = 0.32; p=0.006), and lifestyle specialist referral (r = 0.25; p = 0.035). Reported barriers to lifestyle counseling were lack of time (93%), poor familiarity with resources (50%), and lack of training in motivational interviewing (36%). Barriers to WMM prescription were unfamiliarity with the medications (84%) and side effect concerns (61%). Finally, 90% desired more training in pharmacotherapy, and 77% wanted more information on referral processes for surgical and medical interventions.

Most residents surveyed do not feel adequately prepared to provide evidence-based management of obesity via lifestyle changes counseling, WMM prescription, or specialty care referral. Comfort and knowledge of system processes/resources and WMMs are critical to resident management of obesity. These are potential targets for educational intervention in residency curricula that may improve care for patients with obesity.

Citations: 1. Ward ZJ, Bleich SN, Cradock AL, et al. Projected U.S. State-Level Prevalence of Adult Obesity and Severe Obesity. *N Engl J Med*. 2019;381(25):2440-2450. doi:10.1056/NEJMsa19093012. Huang J, Yu H, Marin E, Brock S, Carden D, Davis T. Physicians' Weight Loss Counseling in Two Public Hospital Primary Care Clinics. *Acad Med*. 2004. doi:10.1097/00001888-200402000-00012

Neuroendocrinology and Pituitary

ADVANCES IN NEUROENDOCRINOLOGY

Increased Mortality in Acromegaly Is Mainly Due to Vascular and Respiratory Disease and Is Normalized by Control of Growth Hormone Hypersecretion.

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SUN-LB48

Introduction: Epidemiological studies in acromegaly have shown increased mortality rates from vascular, respiratory and malignant diseases. We aimed to use a large national cohort of subjects with acromegaly to determine mortality rates due to the above disease categories and to determine whether suppressing post treatment growth hormone (GH) levels to < 1 ng/ml improved survival compared with GH levels of 1-2.5 ng/ml.**Methods:** We identified 1845 cases from the UK Acromegaly database diagnosed 1970-2016 (31,768 person-yrs.), determined population mortality rates (for the period in question, age and sex of the study population) based on historic data, and death certification data for the study population from NHS digital. We were then able to determine standardized mortality rates (SMR) for the above conditions and determine whether they were associated with with age at diagnosis, duration of disease, post treatment (last recorded random or mean of profile) and mean GH levels (day weighted average of all GH measurements). Data presented as SMR, 95% CI and P value.**Results:** Overall standardised mortality rate (SMR) was increased, Observed 556 vs. Expected 412.74, 1.35 (1.24 -1.46, P <0.001), with increased deaths from cardiovascular 1.38 (1.16-1.63, P<0.0001), cerebrovascular 1.49 (1.10-1.97, P <0.006) and respiratory disease 1.55 (1.22-1.93, P< 0.001), but not malignancy 0.94 (0.78-1.11, P=0.786).We found no increase in mortality from breast 0.62 (0.25-1.27, P=0.936), lung 0.87(0.58-1.26, P=0.789) or colon cancer 1.26(0.63-2.25, P= 0.265).There was no relationship between age at diagnosis and SMR, however, there was a declining relationship between duration of acromegaly and all-cause SMR <5 yrs. 6.02 (4.59-7.75) 5-10 yrs. 2.42 (1.9-3.03) >10yrs.1.13 (1.03-1.25) P <0.001.Lowering post-treatment GH levels lead to a significant reduction in all-cause SMR <2.5 ng/ml 1.15 (1.03-1.28) 2.5- 9.9 ng/ml 1.73 (1.47-2.02) ≥10 ng/ml 2.27 (1.72-2.95) P<0.001 and SMR in all disease categories. A similar relationship between mean GH levels and SMR was found for all disease categories and all-cause SMR < 2.5 ng/ml 1.00 (0.89-1.18) 2.5- 9.9 ng/ml 1.50 (1.33-1.69) ≥ 10 ng/ml 2.28 (1.85-2.79) P<0.001.Lowering post-treatment GH levels to less than 1 ng/ml appeared to have an additional beneficial effect for all-cause SMR, <1ng/ml 1.03 (0.89-1.18) 1-2.5 ng/ml 1.38 (1.16-1.62) P<0.001. A similar relationship was shown in cardiovascular, respiratory and malignant disease, but not cerebrovascular disease.**Conclusion:** Acromegaly is associated with an increased mortality from vascular, respiratory, but not malignant disease. The highest risk is within the first 5 years following diagnosis and the risk is abrogated by lowering GH levels to <1.0 ng/ml.On behalf of UK Acromegaly Register Study Group 2020

Reproductive Endocrinology

MALE REPRODUCTIVE HEALTH - FROM HORMONES TO GAMETES

First Human Trial of an Oral Native Testosterone Shows Physiological Levels of Testosterone and Dht in Both Fasted and Fed State in Hypogonadal Men

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SAT-LB6

Introduction: The prevalence of male hypogonadism is estimated to be 6% in the USA (1). Current therapies have limited acceptability: gels can be messy and risk inadvertent dosing of others; injections are painful; and oral testosterone undecanoate (TU) delivers variable testosterone levels, requires concurrent ingestion of a fatty meal and may produce supraphysiological dihydrotestosterone (DHT) levels. We present the first human trial of an oral native testosterone preparation formulated to deliver

physiological levels of testosterone irrespective of food intake. **Aim:** To compare the pharmacokinetics of DITEST (Diurnal Ltd Cardiff, UK) to an oil based oral TU formulation (Andriol Testocaps MSD, UK) and explore the effect of food on DITEST bioavailability. **Methods:** Single centre, phase 1b study of DITEST in 25 adult males with hypogonadism, one subject withdrawn after single period and only included in safety analysis (Clinicaltrials.gov: NCT02966652). Part 1 compared the pharmacokinetics of 80mg TU with 120mg DITEST after a high fat meal. Part 2 the pharmacokinetics of 200mg of DITEST administered in either fed or fasted states. Results are baseline adjusted. **Results:** DITEST showed a testosterone dose response between 120mg and 200mg with C_{max} 550 (19.1) and 877 (30.4) ng/dl (nmol/l) and $AUC_{0\text{-}10h}$ 59.5 and 88.6 h*nmol/L. DITEST 200mg gave an equivalent C_{max} and $AUC_{0\text{-}10h}$ to TU 80mg: C_{max} 877 (30.4) vs 906 (31.4) ng/dl (nmol/l) and $AUC_{0\text{-}10h}$ 88.6 vs 102 h*nmol/L. Fed and fasted DITEST had similar pharmacokinetics: C_{max} 764 (26.5) vs 877 (30.4) ng/dl (nmol/L), $AUC_{0\text{-}10h}$ 87.0 vs 88.6. DITEST resulted in lower levels of DHT than TU: C_{max} 84 (2.9), 131 (4.5) & 194 (6.7) ng/dl (nmol/l); $AUC_{0\text{-}10h}$ 11.0, 16.7 & 36.3 h*nmol/L for DITEST 120mg, 200mg & 80mg TU, respectively. There was one serious adverse event (urinary retention) in the study during TU dosing. There were no emerging safety concerns, and adverse event frequency and severity was similar between the two treatments. **Discussion:** These results demonstrate that 200mg DITEST provides similar testosterone exposure with more physiological DHT exposure than 80mg TU given with a high fat meal. Administration of DITEST in fed and fasted states provides similar testosterone and DHT exposure. Compared to published literature on a self-emulsifying formulation of TU at 200mg, DITEST at 200mg provides a similar testosterone C_{max} and no requirement for a fatty meal (2). **Conclusion:** DITEST is an oral native testosterone formulation with anticipated advantages over current oral therapy of dosing without food and a lower risk of supraphysiological DHT levels. **References: 1.** Basaria S. Male hypogonadism. Lancet 2014; 383:1250-1263. **2.** Yin AY, et al., J Androl 2012; 33:190-201.

Thyroid

THYROID CANCER CASE REPORTS I

Hyperfunctioning Papillary Thyroid Carcinoma With a BRAF Mutation

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SUN-LB78

Background: Hyperfunctioning papillary thyroid carcinoma (PTC) is a rare tumor and accounts for less than 0.1% of all thyroid tumors. Information about its driver mutations is limited. Our literature search yielded 16 cases wherein a mutational analysis was conducted. Thyrotropin receptor (*TSHR*) mutations were identified in 11 of these cases. One case revealed a combination of *TSHR* and *KRAS* mutations. No mutations were identified in the other four cases. *BRAF*V600E is a prominent oncogene in PTC; however, hyperfunctioning PTC with this mutation has not yet been reported.

Clinical Case: In a 48-year-old man, ultrasonography (US) during an annual medical checkup revealed a nodule at the right lobe of the thyroid gland. He visited the outpatient clinic for further evaluation. Thyroid function tests indicated that he was hyperthyroid with TSH level of 0.01 mIU/L (reference range: 0.05-5.00), free thyroxine level of 1.8 ng/dL (reference range: 0.9-1.7), and free triiodothyronine level of 4.3 pg/mL (reference range: 2.3-4.0). Serum thyroglobulin was 62.1 ng/mL (reference range: <33.7) and TSHR autoantibodies (TRAb) was <0.8 IU/L (reference range: <2.0 IU/L). B-mode US revealed a hypoechoic, heterogeneous nodule with largest diameter of 25 mm, and it had a jagged border and microcalcification. Color Doppler US revealed increased intranodular vascularity. The ^{99m}Tc thyroid scintigram revealed a round, right-sided focus of tracer uptake by the nodule with suppression in the remainder of the gland. These findings were consistent with an autonomously-functioning thyroid nodule. The patient underwent total thyroidectomy because fine-needle aspiration cytology revealed a malignant cytological diagnosis. The histopathological diagnosis of the patient was PTC, tall cell variant, pT2, pEx0, pN1b, and M0. Subsequent mutational analysis of *BRAF* (exon 15), *TSHR* (exons 9 and 10), *GNAS* (exons 7-10), *KRAS*, *NRAS*, *HRAS* (codons 12, 13, and 61), and *TERT* promoter (C250T and C228T) only identified a heterozygous point mutation in *BRAF*V600E in tissue samples.

Conclusion: We report for the first time a case of hyperfunctioning papillary thyroid carcinoma with a *BRAF* mutation.

Neuroendocrinology and Pituitary

PITUITARY TUMORS II

Evaluation of Arterial Stiffness in Acromegaly Patients

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MON-LB52

EVALUATION OF ARTERIAL STIFFNESS IN ACROMEGALY PATIENTS Acromegaly is associated with increased morbidity and mortality primarily attributed