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Bodybuilders and individuals who use anabolic steroids should undergo regular blood tests. Without these tests, there is a risk of developing serious health problems that may not be immediately noticeable but can become critical if not addressed. In this post, we'll walk through common risks, what to test for depending on one's regimen, and reference ranges for test results. Some common risks and issues that could arise include the following:Unnoticed Liver Damage: Without blood work, increases in liver enzymes indicating stress or damage can go undetected. This can lead to liver conditions like hepatitis, fibrosis, or even liver failure, all of which might not show symptoms until advanced stages.Hormonal Imbalances Ignored: Steroids can disrupt the body's natural hormonal balance. Without monitoring, this can lead to conditions like Anabolic Steroid-Induced Hypogonadism (reduced function of the gonads), which affects testosterone production in men, leading to issues like reduced fertility, decreased muscle mass, and mental health challenges.Elevated Cardiovascular Risk: Regular blood tests check cholesterol levels. Without this, imbalances like high LDL (bad cholesterol) and low HDL (good cholesterol) can progress unnoticed, increasing the risk of heart diseases such as atherosclerosis (plaque buildup in arteries), heart attacks, or strokes.Hidden Kidney Damage: Steroids can strain the kidneys, but without blood work, changes in kidney function can go unnoticed. This can lead to chronic kidney disease or acute kidney injury, where the kidneys suddenly stop working properly.Overall Health Risks: Other than these specific risks, a lack of regular blood work means general health concerns, such as changes in blood pressure, blood sugar levels, or immune system function, might not be identified and addressed timely.What to test for while on steroids?The lab tests you need depend on your Performance Enhancing Drug (PED) regimen. This guidance is based on modifications of Bonnecaze et al's recommendations:SARM / Injectable Anabolic Androgenic Steroids (AAS):Complete Blood Count (CBC)estimated Glomerular Filtration Rate (eGFR)Lipid ProfileProstate-Specific Antigen (PSA - only if age-appropriate)Electrocardiogram (ECG)Oral AAS:Injectable AAS screenAlanine Aminotransferase (ALT)Alkaline Phosphatase (ALP)Bilirubin tests.Fat Burning Compounds (like T3):ECG, Thyroid-Stimulating Hormone (TSH)Injectable AAS screen, especially if using DNP.Human Growth Hormone (HGH) and Derivatives:Injectable AAS screenMagnesium (Mg) & Potassium (K) testsFor Human Chorionic Gonadotropin (hCG):Generally, no specific testing is needed unless you suspect product impurity. In such cases, include HGH and Derivative Screen.For Site Oil Enhancement:CBCeGFRLipid ProfileSerum CalciumAdditional tests, like free testosterone and estradiol, are not essential for safety monitoring but many steroid users opt for these to optimize their cycle.Beyond lab tests, a thorough physical examination by a doctor is essential for individuals using steroids. This examination includes checking blood pressure and heart rate, as well as conducting abdominal and cardiovascular evaluations. High blood pressure is a critical indicator, as it may lead to hypertension, increasing the risk of stroke, heart attack, and other cardiovascular diseases. Monitoring heart rate is also vital since steroids can impact heart rhythm. Regular checks help in identifying any arrhythmias or irregular heartbeats, which might indicate underlying cardiac issues. Moreover, steroid use is linked to an elevated risk of heart disease. This includes conditions like left ventricular hypertrophy, an enlargement of the heart's left ventricle, potentially leading to heart failure. Cardiovascular examinations are crucial for detecting early signs of heart strain or damage, ensuring timely intervention and treatment.If your PEDs are sourced illicitly, consider comprehensive screening due to the risk of pharmacologic impurities that can affect your liver, kidneys, and bone marrow.What blood test should you get before a steroid cycle?Before starting a steroid cycle, it's important to conduct several medical tests to assess your current health, as well as to establish baseline measurements for monitoring throughout the cycle. Here are some commonly recommended tests:Blood Tests:Complete Blood Count (CBC): To measure the levels of red cells, white cells, and platelets.Lipid Profile: To check cholesterol levels, including LDL, HDL, and triglycerides.Liver Function Tests: Steroids can impact liver health, so it's crucial to check the levels of liver enzymes.Kidney Function Tests: To ensure the kidneys are functioning properly.Testosterone Levels: Both free and total testosterone levels should be assessed.Estrogen Levels: To monitor the risk of estrogen-related side effects.Follicle Stimulating Hormone (FSH) and Luteinizing Hormone (LH): To understand the baseline state of these hormones which are crucial in the regulation of testosterone.Heart Health Assessments:Electrocardiogram (ECG): To check for heart rhythm abnormalities.Echocardiogram: To assess heart function and structure.Endocrine Evaluation:Thyroid Function Tests: Steroids can affect thyroid hormone levels.Other Hormonal Tests:Cortisol Levels: To understand the adrenal glands' baseline function.SHBG (Sex Hormone Binding Globulin): Which influences the levels of free and bound hormones in the body.These tests can help identify any pre-existing conditions that might be exacerbated by steroid use and provide a basis for comparison during and after the steroid cycle. It's also advisable to have a thorough consultation with a healthcare provider to discuss the potential risks and benefits associated with steroid use.What blood test should you get after a steroid cycle?After completing a steroid cycle, several follow-up tests and measures are crucial to ensure health and monitor any potential side effects or long-term impacts. Here's what is typically recommended:Post-Cycle Blood Tests:Complete Blood Count (CBC): To detect any changes that might have occurred during the cycle.Lipid Profile: Since steroids can negatively impact cholesterol levels.Liver Function Tests: To check for any liver stress or damage post-cycle.Kidney Function Tests: Ensuring that the kidneys are still functioning well.Hormonal Profile:Testosterone Levels: It's important to check if the body has resumed natural testosterone production.Estrogen Levels: To ensure they have returned to normal, reducing the risk of side effects like gynecomastia.FSH and LH: These should ideally return to pre-cycle levels if the body has recovered.Cardiovascular Monitoring:Blood Pressure: Steroid use can raise blood pressure, so it's essential to monitor it after the cycle ends.Electrocardiogram (ECG): To ensure there are no new cardiac issues.Physical Examinations:Physical check-ups: Regular check-ups to monitor for physical changes or developments.Timing of Post-Cycle Blood WorkTiming the blood work after a steroid cycle is crucial to accurately assess the body's response and recovery following the use of anabolic steroids. Here's a guideline on when to conduct various tests after finishing a steroid cycle:Immediate Post-Cycle Tests:Blood work should ideally be done within the first week after the cycle ends to establish immediate post-cycle levels. This helps in understanding the impact of steroids on the body's systems, like liver and kidney functions.Mid-Post-Cycle Tests:Around 3-4 weeks after the cycle, it's advisable to conduct another series of tests, especially if post-cycle therapy (PCT) is being used. This timing helps to evaluate how well the body is recovering and how effective the PCT is in restoring natural hormone levels.End of Post-Cycle Therapy Tests:Once the post-cycle therapy is complete, usually 4-6 weeks after the cycle ends, additional testing should be done. This set of tests should focus on hormonal levels such as testosterone, FSH, and LH to ensure they are returning to normal baseline levels.Follow-Up Tests:Subsequent follow-up tests might be necessary 8-12 weeks post-cycle or longer, depending on recovery progress. These tests monitor the long-term restoration of normal bodily functions and hormone levels.How often should you get blood work?At TeleTest, our focus is on minimizing the potential harm and health risks associated with steroid use. We do not condone the use of PEDs. With this goal in mind, we advise the following schedule for health tests:Quarterly TestsThese tests are recommended every three months to monitor key health indicators.Complete Blood Count (CBC):Liver Enzymes (ALT, ALP)Total TestosteroneCreatinine (eGFR)BilirubinPotassiumSerum CalciumAnnual TestsConducted once a year, these tests provide a broader overview of your health.Electrocardiogram (ECG)Lipid ProfileFasting Blood GlucoseHbA1cNot Required for Harm ReductionWhile these tests can provide additional information, they are not essential for reducing the harm associated with steroid use.EstradiolProlactinSex Hormone Binding Globulin (SHBG)Free TestosteroneProstate Specific Antigen (PSA)Luteinizing Hormone (LH)Follicle Stimulating Hormone (FSH)See here for an estimate of costs of private-pay tests in Ontario, Canada.Targets & Reference Range:Our steroid panel includes a follow up consultation with our physicians to discuss your results but below are the reference ranges of common biomarkers for men:CBCWhite Blood Cells (WBC): 4500 to 11,000/mm3Red Blood Cells (RBC): 4.3 to 5.9 million/mm3Hemoglobin: 13.5 to 17.5 g/dLHematocrit: 41% to 53%Mean corpuscular volume (MCV): 80 to 100 µm3Mean corpuscular hemoglobin (MCH): 25.4-34.6 pg/cellMean corpuscular hemoglobin concentration (MCHC): 31%-36% Hb/cellPlatelets: 150,000-400,000/mm3HormonesTotal Testosterone: >= 500 ng/dLFree Testosterone: >= 2% of Total TestosteroneEstradiol: 20 to 50 pg/mLSex Hormone Binding Globulin (SHBG): 15 to 64 nmol/LLuteinizing Hormone (LH): 1.24 to 7.8 IU/mLFollicle Stimulating Hormone (FSH): 1.6 to 8 mIU/mLLiver Enzymes:Alanine Transaminase (ALT): 0 to 45 IU/LAspartate Transaminase (AST): 0 to 35 IU/LAlkaline Phosphatase (ALP): 30 to 120 IU/LThyroidTSH: = 3.7 pg/mLLipid PanelHDL: >= 40 mg/dLLDL: < 100 mg/dLTriglycerides: < 150mg/dL or 1.7mmol/LBlood GlucoseFasting Blood Glucose: 70 mg/dL (3.9 mmol/L) to 100 mg/dL (5.6 mmol/L)HbA1c: 10gr/day), high consumption of red meat, increased muscle mass (BMI>30), non-steroidal anti-inflammatory drugs (NSAID's) abuse, renal failure, glomerulosclerosis, tubular necrosis,aminoglycosides antibiotics – Uric acid: increased intake of animal proteins, involved in purine's metabolism,Gout – SGOT (AST), SGPT (ALT): Abuse of 17 alkylated AAS per os (pharmaceutical hepatitis), acetaminophen abuse, rhabdomyolysis, over-training,alcohol consumption – γGT, ALP: cholestasis-jaundince, alcoholism, liver cirrhosis – Total/ conjugated bilirubin: Jaundice, Cirrhosis, pharmaceuticalhepatitis,hemolysis – LDL, Total cholesterol: dyslipidemia, atherogenesis, SFA's consumption, absence of EFAs (Ω-3,6,9), – Triglycerides: absence of DHA, EPA (omega 3 PUFA's) – Homocysteine:AAS abuse and high risk of cardiovascular/cerebrovascular events ,low B12,Folate – Ammonia: Cirrhosis, Severe hepatitis, Kidney failure,high protein consumption – Glucose, HbA1c: diabetes mellitus type 2, metabolic syndrome,insulin resistance – INR: AAS abuse – CPK: rhabdomyolysis, overtraining,cocaine use – B12: DECREASE equals to megaloblastic anemia (cyanocobalamine deficiency), as a result of either malnutrition, or alcoholism – TSH: hypothyroidism – T3, T4: hyperthyroidism – CEA, AFP, Ca 19-9: Tumors of lungs, testicles (seminoma), large intestine (bowel), visceral organs (liver, bile, pancreas, stomach) – PSA/free PSA: Benign prostate hypertrophy, prostatitis Laboratory testing is critical to the athlete who abuses AAS and physicians should be accurately informed and provide such monitoring. Each doctor is obligated to inform his patients how to preserve well being. Assessment is crucial firstly to determine the user's current health and risks before any cycle is initiated, then to assess the direct impact of the AAS/PEDsuse and finally to evaluate the distortion or restoration of original state of good health.However,laboratory data is never a substitute for a good physical exam and patient history and clinicians should treat the patient, not only the laboratory results. References: 1. The clinical utility of screening of biochemical parameters in elite athletes: analysis of 100 cases. Fallon KE. Br J Sports Med. 2008 May; 42(5):334-7 2. Outpatient clinic for users of anabolic androgenic steroids: an overview. Smit DL, de Ronde W. Neth J Med. 2018 May; 76(4):167 Heavy testosterone use among bodybuilders: an uncommon cohort of illicit substance users. Westerman ME, Charchenko CM, Ziegelmann MJ, Bailey GC, Nippoldt TB, Trost L. Mayo Clin Proc. 2016;91:175-82 4. Hepatotoxicity associated with illicit use of anabolic androgenic steroids in doping.R. Solimini, M. C. Rotolo,et al. Eur Rev Med Pharmacol Sci 2017; 21 (1 Suppl): 7-16 5.Cardiac and metabolic effects of anabolic-androgenic steroid abuse on lipids, blood pressure, left ventricular dimensions, and rhythm. Achar S, Rostamian A, Narayan SM. Am J Cardiol. 2010 Sep 15; 106 (6): 893-901. Adverse health consequences of performance-enhancing drugs: an Endocrine Society scientific statement. Pope HG Jr., Wood RI, Rogol A, Nyberg F, Bowers L, Bhasin S.Endocr Rev. 2014;35:341-75. 7. Combined effects of androgen anabolic steroids and physical activity on the hypothalamic-pituitary-gonadal axis. Hengevoss J, et al. J Steroid Biochem Mol Biol. 2015 Jun; 150:86-96. 8. Disclaimer: The following article is for educational purposes only and does not promote or endorse the use of illegal steroids. For any questions or concerns, Dr. Touliatos is available for consultation. Testosterone (test) and trenbolone (tren) are both anabolic steroids utilized in bodybuilding today. But which one is the best choice for bulking, and can they be stacked together? We reveal the key differences and the similarities based on existing research and our patient's labs. Test vs. Tren: How Do Their Benefits Compare? Muscle Hypertrophy Test and tren are both very anabolic, although we have found tren to be a slightly more potent muscle builder. Tren's higher androgenic nature also means it will also build more muscle hypertrophy, specifically in the trapezius and deltoid muscles. Testosterone, however, will cause more overall weight gain than trenbolone due to it causing fluid retention, with tren instead flushing out water. After a cycle, when water levels normalize, trenbolone will perhaps cause users to gain an extra 5 pounds in fat-free mass. Tren will also build muscle faster than the most common test esters, such as cypionate and enanthate, as they are longer esters and thus require more time to kick in. Fat Loss It would appear that tren burns considerably more fat than test, as users will look more defined on-cycle. However, this initial appearance is due to tren's diuretic effects and testosterone having the aromatase enzyme present, thus causing a smoother appearance to the muscles with some bloating. Upon cycle cessation, when water levels normalize, fat loss results will be similar. However, tren may still have an edge due to extra stimulation of the androgen receptors, causing a reduction in adipose tissue. For these reasons, tren is more commonly used in cutting cycles than testosterone. Strength Test and tren will significantly increase strength to similar levels. This is a little surprising considering test promotes greater weight gain, but tren's sheer potency means it can rival test in this regard. We have seen users add fifty pounds to their main compound lifts when taking testosterone as a first cycle. Equally, we have seen users add another 20 pounds to their lifts when introducing tren, even after conducting several other steroid cycles. Test vs. Tren Side Effects: Which Compound Is More Toxic? We have found trenbolone's side effects to be significantly harsher than testosterone's. The main concerns we see with patients who have used tren are cholesterol alterations and testosterone suppression. Heart Health Sharp elevations in LDL cholesterol can be experienced on tren, significantly increasing the risk of MI (myocardial infarction). If we can't persuade patients to come off tren, we advise them to: Take 4 g/day of fish oil. Lower their saturated fat intake. Perform regular cardiovascular activity (30 minutes 3–4 times per week). We find this trio to be cardioprotective. Testosterone will cause some shifts in cholesterol (1); however, we have found these to be mild in conservative dosages when compared to tren. Testosterone Suppression Trenbolone is also more suppressive than testosterone based on our SHBG tests, so it will need a comprehensive PCT protocol to recover users' endogenous testosterone levels. Nolvadex and Clomid as a duo, used for 30 days, will accelerate this process and normalize the HPTA (hypothalamic-pituitary-testicular axis). Testosterone is also suppressive; however, just one of these anti-estrogens is typically sufficient in our experience. Gynecomastia Testosterone will cause aromatization (the conversion of testosterone to estrogen), so there is a risk of gynecomastia in sensitive individuals. Tren does not increase aromatase activity, and thus the risks of gyno are considerably less. However, it is not impossible to develop gyno on tren, as it raises progesterone levels (another female sex hormone). We do not recommend using an aromatase inhibitor to combat high levels of estrogen from testosterone, as we have seen this exacerbate blood pressure based on patients' lipid profiles (2). Instead, a SERM (selective estrogen receptor modulator) can be used to block estrogen's effects at a receptor level, thus not affecting cholesterol levels. We typically prescribe Clomid or Nolvadex for users particularly susceptible to gynecomastia or those beginning to experience puffy nipples during a cycle. Nolvadex can also be used to inhibit progesterone's effects and thus be utilized during tren cycles. Hair Loss Testosterone and trenbolone are both androgenic steroids on paper and in practice. Thus, accelerated hair loss may be experienced by genetically susceptible individuals via the elevation of DHT levels. We have seen tren cause more issues in this regard, with it having 5x the androgenic rating of testosterone. Some doctors would prescribe a DHT blocker such as finasteride to prevent hair loss during steroid cycles; however, this often results in reduced muscle and strength gains. Instead, if a user is troubled by hair loss, we encourage them to avoid androgenic steroids. In this case, Dianabol and Deca Durabolin are more suitable alternatives. Acne Individuals with overactive sebaceous glands may be susceptible to acne vulgaris on test and tren, with the latter likely to cause more aggravated episodes. Less androgenic anabolic steroids can help users who are excessively troubled by acne from test or tren. Anxiety Our patients have reported more psychological effects on tren vs. test, which may be attributed to it causing alterations in neurotransmitters due to greater epinephrine production. Consequently, users may be more susceptible to the following on tren: Anxiety Depression Insomnia Can Test and Tren Be Stacked Together? Testosterone is arguably the most optimal anabolic steroid to stack with trenbolone, as it will further enhance muscle and strength gains without additional liver toxicity and without significantly more cardiovascular strain. However, due to tren's toxicity, it is typically only stacked with other steroids by seasoned steroid users who have taken tren before and can tolerate its side effects. And even then, stacking trenbolone with another toxic anabolic steroid, such as Winstrol or Anadrol, can be deleterious to health. Here is a sample cycle of a test and tren stack: Due to testosterone's less toxic side effect profile, it presents more stacking options for users, such as: Anadrol Dianabol Winstrol Anavar Deca Durabolin Summary: Which Is the More Optimal Anabolic Compound? Tren is the more potent anabolic steroid, producing faster improvements in body composition. However, testosterone could be considered the more optimal steroid from a risk-reward perspective. This distinction is also reflected in medicine, with testosterone being an FDA-approved drug for the treatment of hypogonadism (3), whereas trenbolone has never been FDA-approved for human use. (1) (2) (3) Polycyclic organic compound having sterane as a core structure This article is about the family of polycyclic compounds. For the drugs, also used as performance-enhancing substances, see Anabolic steroid. For the scientific journal, see Steroids (journal). For the Death Grips EP, see Steroids (Crouching Tiger Hidden Gabber Megamix). Structure of 24-ethyl-lanostane, a prototypical steroid with 32 carbon atoms. Its core ring system (ABCD), composed of 17 carbon atoms, is shown with IUPAC-approved ring lettering and atom numbering.[1]:1785fA steroid is an organic compound with four fused rings (designated A, B, C, and D) arranged in a specific molecular configuration. Steroids have two principal biological functions: as important components of cell membranes that alter membrane fluidity; and as signaling molecules. Examples include the lipid cholesterol, sex hormones estradiol and testosterone,[2]:10–19 anabolic steroids, and the anti-inflammatory corticosteroid drug dexamethasone.[3] Hundreds of steroids are found in fungi, plants, and animals. All steroids are manufactured in cells from the sterols lanosterol (opisthokonts) or cycloartenol (plants). Lanosterol and cycloartenol are derived from the cyclization of the triterpene squalene.[4] Steroids are named after the steroid cholesterol[5] which was first described in gall stones from Ancient Greek chole- 'bile' and stereos 'solid'.[6][7][8] The steroid nucleus (core structure) is called gonane (cyclopentanoperhydrophenanthrene).[9] It is typically composed of seventeen carbon atoms, bonded in four fused rings: three six-member cyclohexane rings (rings A, B and C in the first illustration) and one five-member cyclopentane ring (the D ring). Steroids vary by the functional groups attached to this four-ring core and by the oxidation state of the rings. Sterols are forms of steroids with a hydroxy group at position three and a skeleton derived from cholestane.[1]:1785f[10] Steroids can also be more radically modified, such as by changes to the ring structure, for example, cutting one of the rings. Cutting Ring B produces secosteroids one of which is vitamin D3. Space-filling representationBall-and-stick representation5α-dihydroprogesterone (5α-DHP), a steroid. The shape of the four rings of most steroids is illustrated (carbon atoms in black, oxygens in red and hydrogens in grey). The nonpolar "slab" of hydrocarbon in the middle (grey, black) and the polar groups at opposing ends (red) are common features of natural steroids. 5α-DHP is an endogenous steroid hormone and a biosynthetic intermediate. See also: Gonane and Sterane Gonane, perhydrocyclopenta[a]phenanthrene, the main structure of steroids, often referred to as the steroid nucleus. Steroid 5α and 5β stereoisomers[1]:1786f Gonane, also known as steran or cyclopentanoperhydrophenanthrene, the nucleus of all steroids and sterols,[11][12] is composed of seventeen carbon atoms in carbon-carbon bonds forming four fused rings in a three-dimensional shape. The three cyclohexane rings (A, B, and C in the first illustration) form the skeleton of a perhydro derivative of phenanthrene. The D ring has a cyclopentane structure. When the two methyl groups and eight carbon side chains (at C-17, as shown for cholesterol) are present, the steroid is said to have a cholestane framework. The two common 5α and 5β stereoisomeric forms of steroids exist because of differences in the side of the largely planar ring system where the hydrogen (H) atom at carbon-5 is attached, which results in a change in steroid A-ring conformation. Isomerisation at the C-21 side chain produces a parallel series of compounds, referred to as isosteroids.[13] Examples of steroid structures are: Testosterone, the principal male sex hormone and an anabolic steroid Cholic acid, a bile acid Dexamethasone, a synthetic corticosteroid drug Lanosterol, the biosynthetic precursor to animal steroids. The number of carbons (30) indicates its triterpenoid classification. Progesterone, a steroid hormone involved in the female menstrual cycle, pregnancy, and embryogenesis Medrogestone, a synthetic drug with effects similar to progesterone β-Sitosterol, a plant or phytosterol, with a fully branched hydrocarbon side chain at C-17 and an hydroxyl group at C-3 In addition to the ring scissions (cleavages), expansions and contractions (cleavage and reclosing to a larger or smaller rings)—all variations in the carbon-carbon bond framework—steroids can also vary: in the bond orders within the rings, in the number of methyl groups attached to the ring (and, when present, on the prominent side chain at C17), in the functional groups attached to the rings and side chain, and in the configuration of groups attached to the rings and chain.[2]:2–9 For instance, sterols such as cholesterol and lanosterol have a hydroxyl group attached at position C-3, while testosterone and progesterone have a carbonyl (oxo substituent) at C-3. Among these compounds, only lanosterol has two methyl groups at C-4. Cholesterol which has a C-5 to C-6 double bond, differs from testosterone and progesterone which have a C-4 to C-5 double bond. Cholesterol, a prototypical animal sterol. This structural lipid and key steroid biosynthetic precursor.[1]:1785f 5α-cholestane, a common steroid core Almost all biologically relevant steroids can be presented as a derivative of a parent cholesterol-like hydrocarbon structure that serves as a skeleton.[14][15] These parent structures have specific names, such as pregnane, androstane, etc. The derivatives carry various functional groups called suffixes or prefixes after the respective numbers, indicating their position in the steroid nucleus.[16] There are widely used trivial steroid names of natural origin with significant biologic activity, such as progesterone, testosterone or cortisol. Some of these names are defined in The Nomenclature of Steroids.[17] These trivial names can also be used as a base to derive new names, however, by adding prefixes only rather than suffixes, e.g., the steroid 17α-hydroxyprogesterone has a hydroxy group (-OH) at position 17 of the steroid nucleus comparing to progesterone. The letters α and β[18] denote absolute stereochemistry at chiral centers—a specific nomenclature distinct from the R/S convention[19] of organic chemistry to denote absolute configuration of functional groups, known as Cahn–Ingold–Prelog priority rules. The R/S convention assigns priorities to substituents on a chiral center based on their atomic number. The highest priority group is assigned to the atom with the highest atomic number, and the lowest priority group is assigned to the atom with the lowest atomic number. The molecule is then oriented so that the lowest priority group points away from the viewer, and the remaining three groups are arranged in order of decreasing priority around the chiral center. If this arrangement is clockwise, it is assigned an R configuration; if it is counterclockwise, it is assigned an S configuration.[20] In contrast, steroid nomenclature uses α and β to denote stereochemistry at chiral centers. The α and β designations are based on the orientation of substituents relative to each other in a specific ring system. In general, α refers to a substituent that is oriented towards the plane of the ring system, while β refers to a substituent that is oriented away from the plane of the ring system. In steroids drawn from the standard perspective used in this paper, α-bonds are depicted on figures as dashed wedges and β-bonds as solid wedges.[14] The name "11-deoxycortisol" is an example of a derived name that uses cortisol as a parent structure without an oxygen atom (hence "deoxy") attached to position 11 (as a part of a hydroxy group).[14][21] The numbering of positions of carbon atoms in the steroid nucleus is set in a template found in the Nomenclature of Steroids[22] that is used regardless of whether an atom is present in the steroid in question.[14] Unsaturated carbons (generally, ones that are part of a double bond) in the steroid nucleus are indicated by changing -ane to -ene.[23] This change was traditionally done in the parent name, adding a prefix to denote the position, with or without Δ (Greek capital delta) which designates unsaturation, for example, 4-pregnene-11β,17α-diol-3,20-dione (also Δ4-pregnene-11β,17α-diol-3,20-dione) or 4-androstene-3,11,17-trione (also Δ4-androstene-3,11,17-trione). However, the Nomenclature of Steroids recommends the locant of a double bond to be always adjacent to the syllable designating the unsaturation, therefore, having it as a suffix rather than a prefix, and without the use of the Δ character, i.e. pregn-4-ene-11β,17α-diol-3,20-dione or androst-4-ene-3,11,17-trione. The double bond is designated by the lower-numbered carbon atom, i.e. "Δ4-" or "4-ene" means the double bond between positions 4 and 5. The saturation of carbons of a parent steroid can be done by adding "dihydro-" prefix,[24] i.e., a saturation of carbons 4 and 5 of testosterone with two hydrogen atoms is 4,5α-dihydrotestosterone or 4,5β-dihydrotestosterone. Generally, when there is no ambiguity, one number of a hydrogen position from a steroid with a saturated bond may be omitted, leaving only the position of the second hydrogen atom, e.g., 5α-dihydrotestosterone or 5β-dihydrotestosterone. The Δ5-steroids are those with a double bond between carbons 5 and 6 and the Δ4 steroids are those with a double bond between carbons 4 and 5.[25][23] The abbreviations like "P4" for progesterone and "A4" for androstenedione for refer to Δ4-steroids, while "P5" for pregnenolone and "A5" for androstenediol refer to Δ5-steroids.[14] The suffix -ol denotes a hydroxy group, while the suffix -one denotes an oxo group. When two or three identical groups are attached to the base structure at different positions, the suffix is indicated as -diol or -triol for hydroxy, and -dione or -trione for oxo groups, respectively. For example, 5α-pregnane-3α,17α-diol-20-one has a hydrogen atom at the 5α position (hence the "5α-" prefix), two hydroxy groups (-OH) at the 3α and 17α positions (hence "3α,17α-diol" suffix) and an oxo group (=O) at the position 20 (hence the "20-one" suffix). However, erroneous use of suffixes can be found, e.g., "5α-pregnan-17α-diol-3,11,20-trione"[26] [sic] — since it has just one hydroxy group (at 17α) rather than two, then the suffix should be -ol, rather than -diol, so that the correct name to be "5α-pregnan-17α-ol-3,11,20-trione". According to the rule set in the Nomenclature of Steroids, the terminal "e" in the parent structure name should be elided before the vowel (the presence or absence of a number does not affect such elision).[14][16] This means, for instance, that if the suffix immediately appended to the parent structure name begins with a vowel, the trailing "e" is removed from that name. An example of such removal is "5α-pregnan-17α-ol-3,20-dione", where the last "e" of "pregnane" is dropped due to the vowel ("o") at the beginning of the suffix -ol. Some authors incorrectly use this rule, eliding the terminal "e" where it should be kept, or vice versa.[27] The term "11-oxygenated" refers to the presence of an oxygen atom as an oxo (=O) or hydroxy (-OH) substituent at carbon 11. "Oxygenated" is consistently used within the chemistry of the steroids[28] since the 1950s.[29] Some studies use the term "11-oxyandrogens"[30][31] as an abbreviation for 11-oxygenated androgens, to emphasize that they all have an oxygen atom attached to carbon at position 11.[32][33] However, in chemical nomenclature, the prefix "oxy" is associated with ether functional groups, i.e., a compound with an oxygen atom connected to two alkyl or aryl groups (R-O-R),[34] therefore, using "oxy" within the name of a steroid class may be misleading. One can find clear examples of "oxygenated" to refer to a broad class of organic molecules containing a variety of oxygen containing functional groups in other domains of organic chemistry,[35] and it is appropriate to use this convention.[14] Even though "keto" is a standard prefix in organic chemistry, the 1989 recommendations of the Joint Commission on Biochemical Nomenclature discourage the application of the prefix "keto" for steroid names, and favor the prefix "oxo" (e.g., 11-oxo steroids rather than 11-keto steroids), because "keto" includes the carbon that is part of the steroid nucleus and the same carbon atom should not be specified twice.[36][14] Steroids are present across all domains of life, including bacteria, archaea, and eukaryotes. In eukaryotes, steroids are particularly abundant in fungi, plants, and animals.[37][38] Eukaryotic cells, encompassing animals, plants, fungi, and protists, are characterized by their complex cellular structures, including a true nucleus and membrane-bound organelles.[39] Sterols, a subgroup of steroids, play crucial roles in maintaining membrane fluidity, supporting cell signaling, and enhancing stress tolerance. These compounds are integral to eukaryotic membranes, where they contribute to membrane integrity and functionality.[40] During eukaryogenesis—the evolutionary process that gave rise to modern eukaryotic cells—steroids likely facilitated the endosymbiotic acquisition of mitochondria.[41] Although sterol biosynthesis is rare in prokaryotes, certain bacteria, including Methylococcus capsulatus, specific methanotrophs, myxobacteria, and the planctomycete Gemmata obscuriglobus, are capable of producing sterols. In G. obscuriglobus, sterols are essential for cell viability, but their roles in other bacteria remain poorly understood.[42] Prokaryotic sterol synthesis involves the tetracyclic steroid framework, as found in myxobacteria,[43] as well as hopanoids, pentacyclic lipids that regulate bacterial membrane functions.[44] These sterol biosynthetic pathways may have originated in bacteria or been transferred from eukaryotes.[45] Sterol synthesis depends on two key enzymes: squalene monooxygenase and oxidosqualene cyclase. Phylogenetic analyses of oxidosqualene cyclase (Osc) suggest that some bacterial Osc genes may have been acquired via horizontal gene transfer from eukaryotes, as certain bacterial Osc proteins closely resemble their eukaryotic homologs.[42] Fungal steroids include the ergosterols, which are involved in maintaining the integrity of the fungal cellular membrane. Various antifungal drugs, such as amphotericin B and azole antifungals, utilize this information to kill pathogenic fungi.[46] Fungi can alter their ergosterol content (e.g. through loss of function mutations in the enzymes ERG3 or ERG6, inducing depletion of ergosterol, or mutations that decrease the ergosterol content) to develop resistance to drugs that target ergosterol.[47] Ergosterol is analogous to the cholesterol found in the cellular membranes of animals (including humans), or the phytosterols found in the cellular membranes of plants.[47] All mushrooms contain large quantities of ergosterol, in the range of tens to hundreds of milligrams per 100 grams of dry weight.[47] Oxygen is necessary for the synthesis of ergosterol in fungi.[47] Ergosterol is responsible for the vitamin D content found in mushrooms; ergosterol is chemically converted into provitamin D2 by exposure to ultraviolet light.[47] Provitamin D2 spontaneously forms vitamin D2.[47] However, not all fungi utilize ergosterol in their cellular membranes; for example, the pathogenic fungal species Pneumocystis jirovecii does not, which has important clinical implications (given the mechanism of action of many antifungal drugs). Using the fungus Saccharomyces cerevisiae as an example, other major steroids include ergosta-5,7,22,24(28)-tetraen-3β-ol, zymosterol, and lanosterol. S. cerevisiae utilizes 5,6-dihydroergosterol in place of ergosterol in its cell membrane.[47] Plant steroids include steroidal alkaloids found in Solanaceae[48] and Melanthiaceae (specially the genus Veratrum),[49] cardiac glycosides,[50] the phytosterols and the brassinosteroids (which include several plant hormones). Animal steroids include compounds of vertebrate and insect origin, the latter including ecdysteroids such as ecdysterone (controlling molting in some species). Vertebrate examples include the steroid hormones and cholesterol; the latter is a structural component of cell membranes that helps determine the fluidity of cell membranes and is a principal constituent of plaque (implicated in atherosclerosis). Steroid hormones include: Sex hormones, which influence sex differences and support reproduction. These include androgens, estrogens, and progestogens. Corticosteroids, including most synthetic steroid drugs, with natural product classes the glucocorticoids (which regulate many aspects of metabolism and immune function) and the mineralocorticoids (which help maintain blood volume and control renal excretion of electrolytes) Anabolic steroids, natural and synthetic, which interact with androgen receptors to increase muscle and bone synthesis. In popular use, the term "steroids" often refers to anabolic steroids. This section needs expansion with: A more detailed explanation of function would also be beneficial. You can help by adding to it. (January 2019) The major classes of steroid hormones, with prominent members and examples of related functions, are:[51][52] Corticosteroids: Glucocorticoids: Cortisol, a glucocorticoid whose functions include immunosuppression Mineralocorticoids: Aldosterone, a mineralocorticoid that helps regulate blood pressure through water and electrolyte balance Sex steroids: Progestogens: Progesterone, which regulates cyclical changes in the endometrium of the uterus and maintains a pregnancy Androgens: Testosterone, which contributes to the development and maintenance of male secondary sex characteristics Estrogens: Estradiol, which contributes to the development and maintenance of female secondary sex characteristics Additional classes of steroids include: Neurosteroids such as DHEATooltip dehydroepiandrosterone and allopregnanolone Bile acids such as taurocholic acid Aminosteroid neuromuscular blocking agents (mainly synthetic) such as pancuronium bromide Steroidal antiandrogens (mainly synthetic) such as cyproterone acetate Steroidogenesis inhibitors (mainly exogenous) such as alfatradiol Membrane sterols such as cholesterol, ergosterol, and various phytosterols Toxins such as steroidal saponins and cardenolides/cardiac glycosides As well as the following class of secosteroids (open-ring steroids): Vitamin D forms such as ergocalciferol, cholecalciferol, and calcitriol Steroids can be classified based on their chemical composition.[53] One example of how MeSH performs this classification is available at the Wikipedia MeSH catalog. Examples of this classification include: Cholecalciferol (vitamin D3), an example of a 9,10-secosteroid Cyclopamine, an example of a complex C-nor-D-homosteroid Class Example Number of carbon atoms Cholestanes Cholesterol 27 Cholanes Cholic acid 24 Pregnanes Progesterone 21 Androstanes Testosterone 19 Estranes Estradiol 18 In biology, it is common to name the above steroid classes by the number of carbon atoms present when referring to hormones: C18-steroids for the estranes (mostly estrogens), C19-steroids for the androstanes (mostly androgens), and C21-steroids for the pregnanes (mostly corticosteroids).[54] The classification "17-ketosteroid" is also important in medicine. The gonane (steroid nucleus) is the parent 17-carbon tetracyclic hydrocarbon molecule with no alkyl sidechains.[55] Secosteroids (Latin seco, "to cut") are a subclass of steroidal compounds resulting, biosynthetically or conceptually, from scission (cleavage) of parent steroid rings (generally one of the four). Major secosteroid subclasses are defined by the steroid carbon atoms where this scission has taken place. For instance, the prototypical secosteroid cholecalciferol, vitamin D3 (shown), is in the 9,10-secosteroid subclass and derives from the cleavage of carbon atoms C-9 and C-10 of the steroid B-ring; 5,6-secosteroids and 13,14-steroids are similar.[56] Norsteroids (nor-, L. norma; "normal" in chemistry, indicating carbon removal)[57] and homosteroids (homo-, Greek homos; "same", indicating carbon addition) are structural subclasses of steroids formed from biosynthetic steps. The former involves enzymic ring expansion-contraction reactions, and the latter is accomplished (biomimetically) or (more frequently) through ring closures of acyclic precursors with more (or fewer) ring atoms than the parent steroid framework.[58] Combinations of these ring alterations are known in nature. For instance, ewes who graze on corn lily ingest cyclopamine (shown) and veratramine, two of a sub-family of steroids where the C- and D-rings are contracted and expanded respectively via a biosynthetic migration of the original C-13 atom. Ingestion of these C-nor-D-homosteroids results in birth defects in lambs: cyclopia from cyclopamine and leg deformity from veratramine.[59] A further C-nor-D-homosteroid (nakiterpiosin) is excreted by Okinawan cyanobacteriosponges. e.g., Terpios hoshinota, leading to coral mortality from black coral disease.[60] Nakiterpiosin-type steroids are active against the signaling pathway involving the smoothened and hedgehog proteins, a pathway which is hyperactive in a number of cancers.[citation needed] Steroids and their metabolites often function as signalling molecules (the most notable examples are steroid hormones), and steroids and phospholipids are components of cell membranes.[61] Steroids such as cholesterol decrease membrane fluidity.[62] Similar to lipids, steroids are highly concentrated energy stores. However, they are not typically sources of energy; in mammals, they are normally metabolized and excreted. Steroids play critical roles in a number of disorders, including malignancies like prostate cancer, where steroid production inside and outside the tumour promotes cancer cell aggressiveness.[63] Simplification of the end of the steroid synthesis pathway, where the intermediates isopentenyl pyrophosphate (PP or IPP) and dimethylallyl pyrophosphate (DMAPP) form geranyl pyrophosphate (GPP), squalene and lanosterol (the first steroid in the pathway) The hundreds of steroids found in animals, fungi, and plants are made from lanosterol (in animals and fungi; see examples above) or cycloartenol (in other eukaryotes). Both lanosterol and cycloartenol derive from cyclization of the triterpenoid squalene.[4] Lanosterol and cycloartenol are sometimes called protosterols because they serve as the starting compounds for all other steroids. Steroid biosynthesis is an anabolic pathway which produces steroids from simple precursors. A unique biosynthetic pathway is followed in animals (compared to many other organisms), making the pathway a common target for antibiotics and other anti-infection drugs. Steroid metabolism in humans is also the target of cholesterol-lowering drugs, such as statins. In humans and other animals the biosynthesis of steroids follows the mevalonate pathway, which uses acetyl-CoA as building blocks for dimethylallyl diphosphate (DMAPP) and isopentenyl diphosphate (IPP).[64][better source needed] In subsequent steps DMAPP and IPP conjugate to form farnesyl diphosphate (FPP), which further conjugates with each other to form the linear triterpenoid squalene. Squalene biosynthesis is catalyzed by squalene synthase, which belongs to the squalene/phytoene synthase family. Subsequent epoxidation and cyclization of squalene generate lanosterol, which is the starting point for additional modifications into other steroids (steroidogenesis).[65] In other eukaryotes, the cyclization product of epoxidized squalene (oxidosqualene) is cycloartenol. Mevalonate pathway Main article: Mevalonate pathway The mevalonate pathway (also called HMG-CoA reductase pathway) begins with acetyl-CoA and ends with dimethylallyl diphosphate (DMAPP) and isopentenyl diphosphate (IPP). DMAPP and IPP donate isoprene units, which are assembled and modified to form terpenes and isoprenoids[66] (a large class of lipids, which include the carotenoids and form the largest class of plant natural products).[67] Here, the activated isoprene units are joined to make squalene and folded into a set of rings to make lanosterol.[68] Lanosterol can then be converted into other steroids, such as cholesterol and ergosterol.[68][69] Two classes of drugs target the mevalonate pathway: statins (like rosuvastatin), which are used to reduce elevated cholesterol levels,[70] and bisphosphonates (like zoledronate), which are used to treat a number of bone-degenerative diseases.[71] Human steroidogenesis, with the major classes of steroid hormones, individual steroids and enzymatic pathways.[72] Changes in molecular structure from a precursor are highlighted in white. See also: Steroidogenic enzyme Steroidogenesis is the biological process by which steroids are generated from cholesterol and changed into other steroids.[73] The pathways of steroidogenesis differ among species. The major classes of steroid hormones, as noted above (with their prominent members and functions), are the progestogens, corticosteroids (corticoids), androgens, and estrogens.[25][74] Human steroidogenesis of these classes occurs in a number of locations: Progestogens are the precursors of all other human steroids, and all human tissues which produce steroids must first convert cholesterol to pregnenolone. This conversion is the rate-limiting step of steroid synthesis, which occurs inside the mitochondrion of the respective tissue. It is catalyzed by the mitochondrial P450scc system.[75][76] Cortisol, corticosterone, aldosterone are produced in the adrenal cortex.[25][74] Estradiol, estrone and progesterone are made primarily in the ovary, estriol in placenta during pregnancy, and testosterone primarily in the testes[25][77][78][79] (some testosterone may also be produced in the adrenal cortex).[25][74] Estradiol is converted from testosterone directly (in males), or via the primary pathway DHEA – androstenedione – estrone and secondarily via testosterone (in females).[25] Stromal cells have been shown to produce steroids in response to signaling produced by androgen-starved prostate cancer cells.[63][non-primary source needed][better source needed] Some neurons and glia in the central nervous system (CNS) express the enzymes required for the local synthesis of pregnenolone, progesterone, DHEA and DHEAS, de novo or from peripheral sources.[25][citation needed] vte Production rates, secretion rates, clearance rates, and blood levels of major sex hormones Sex Sex hormone Reproductivephase Bloodproduction rate Gonadalsecretion rate Metabolicclearance rate Reference range (serum levels) SI units Non-SI units Men Androstenedione – 2.8 mg/day 1.6 mg/day 2200 L/day 2.8–7.3 nmol/L 80–210 ng/dL Testosterone – 6.5 mg/day 6.2 mg/day 950 L/day 6.9–34.7 nmol/L 200–1000 ng/dL Estrone – 150 µg/day 110 µg/day 2050 L/day 37–250 pmol/L 10–70 pg/mL Estradiol – 60 µg/day 50 µg/day 1600 L/day