STANOZOLOL®

Stanozolol USP29, Ph.Eur.5.5, Micronized grade

Molecular Formula: $C_{22}H_{36}N_2O$
Molecular Weight: 344.53 gm/mol
Active life: 8 hours
Detection Time: 3 weeks
Anabolic/Androgenic Ratio (Range): 320:30

DESCRIPTION:
Stanozolol®, brand of Stanozolol tablets, is an anabolic steroid, a synthetic derivative of testosterone. Each tablet contains 5 mg and 10 mg of Stanozolol USP29, Ph.Eur.5.5, micronized grade. It is designated chemically as 17-methyl-2'*H* -5α-androst-2eno[3,2-c]pyrazol-17β-ol.

Each tablet also contains lactose monohydrate, sodium starch glycolate, polyvidone 25,000, microcrystalline cellulose and magnesium stearate as excipients. The 5 mg tablet also contains yellow ferric oxide (E172) and indigo carmine aluminium lake (E132) as colouring agent and the 10 mg tablet also contains yellow ferric oxide (E172)

Stanozolol® is an oral androgen derived from dihydrotestosterone. Stanozolol® acts on androgen receptors to promote anabolism through increased nitrogen retention and protein synthesis in muscle tissue. Stanozolol® is a strong anabolic substance with androgenic action. Stanozolol does not convert to estrogen and therefore does not produce typical estrogen mediated side effects such as water retention. Stanozolol has a large oral bioavailability, due to a C17 a-alkylation which allows the hormone to survive first pass liver metabolism. Stanozolol reduces SHBG increasing free testosterone levels.

CLINICAL PHARMACOLOGY:
Anabolic steroids are synthetic derivatives of testosterone. Certain clinical effects and adverse reactions demonstrate the androgenic properties of this class of drugs. Complete dissociation of anabolic and androgenic effects has not been achieved. The actions of anabolic steroids are therefore similar to those of male sex hormones with the possibility of causing serious disturbances of growth and sexual development if given to young children. They suppress the gonadotropic functions of the pituitary and may exert a direct effect upon the testes.
Stanozolol® has been found to increase low-density lipoproteins and decrease high-density lipoproteins. These changes are not associated with any increase in total cholesterol or triglyceride levels and revert to normal on discontinuation of treatment.
Hereditary angioedema (HAE) is an autosomal dominant disorder caused by a deficient or nonfunctional C1 esterase inhibitor (C1 INH) and clinically characterized by episodes of swelling of the face, extremities, genitalia, bowel wall, and upper respiratory tract.
In small scale clinical studies, Stanozolol® was effective in controlling the frequency and severity of attacks of angiotedema and in increasing serum levels of C1 INH and C4.
Stanozolol® is not effective in stopping HAE attacks while they are under way. The effect of Stanozolol® on increasing serum levels of C1 INH and C4 may be related to an increase in protein anabolism.

INDICATIONS:
Hereditary Angioedema: for prophylactic use to decrease frequency and severity of attacks of angioedema.
Muscle Anabolism: for adjunctive therapy in patients for weight gain following severe muscular atrophy associated with extensive surgery, chronic infections, long term hospitalization, or severe trauma.
Corticosteroid Atrophy: to reduce muscle wasting during prolonged corticosteroid use.

CONTRAINDICATIONS:
The use of Stanozolol® is contraindicated in the following:
1. Female patients due to risk of virilization and fetal harm.
2. Male patients with known or suspected carcinoma of the breast, prostate, or testis.
3. Patients with hypercalcaemia as anabolic steroids may stimulate osteolytic bone resorption.
4. Patients with cardiovascular disorders, renal or hepatic impairment, epilepsy, migraines, or diabetes mellitus.
5. Nephrosis or the nephrotic phase of nephritis.
6. Hypersensitivity to stanozolol,

PRECAUTIONS:
Anabolic steroids may cause suppression of clotting factors II, V, VII and X and an increase in prothrombin time.
Anabolic steroids may increase sensitivity to anticoagulants. Dosage of anticoagulants may have to be decreased in order to maintain the prothrombin time at the desired therapeutic level.
Oral hypoglycemic dosage may need adjustment in diabetic patients who receive anabolic steroids.
Patients using C17 a-alkylated oral steroids should be monitored for hepatotoxicity and jaundicing.
General Women should be observed for signs of virilization (deepening of the voice, hirsutism, acne, and clitoromegaly). To prevent irreversible change, drug therapy must be discontinued, or the dosage significantly reduced when mild virilism is first detected. Such virilization is usual following androgenic anabolic steroid use at high doses. Some virilizing changes in women are irreversible even after prompt discontinuance of therapy and are not prevented by concomitant use of estrogens. Menstrual irregularities may also occur. The insulin or oral hypoglycemic dosage may need adjustment in diabetic patients who receive anabolic steroids.
Information for the patient the physician should instruct patients to report any of the following side effects of androgens:
Adult or Adolescent Males Too frequent or persistent erections of the penis, appearance or aggravation of acne.
Women Hoarseness, acne, changes in menstrual periods, or more hair on the face.
All Patients Any nausea, vomiting, changes in skin color, or ankle swelling.
Laboratory Tests Women with disseminated breast carcinoma should have frequent determination of urine and serum calcium levels during the course of androgenic anabolic steroid therapy (see WARNINGS).
Because of the hepatotoxicity associated with the use of 17-alpha-alkylated androgens, liver function tests should be obtained periodically.
Periodic (every 6 months) x-ray examinations of bone age should be made during treatment of prepubertal patients to determine the rate of bone maturation and the effects of androgenic anabolic steroid therapy on the epiphyseal centers. In common with other anabolic steroids, Stanozolol® has been reported to lower the level of high-density lipoproteins and raise the level of low-density lipoproteins. These changes usually revert to normal on discontinuation of treatment. Increased low-density lipoproteins and decreased high-density lipoproteins are considered cardiovascular risk factors. Serum lipids and high-density lipoprotein cholesterol should be determined periodically.
Carcinogenesis, Mutagenesis, Impairment of Fertility.
Stanozolol has not been tested in laboratory animals for carcinogenic or mutagenic effects. No tumorigenic or cancer inducing properties of Stanozolol were seen in one-year toxicity studies in rats.
Stanozolol administered orally (intragastrically) to pregnant rats at dosages of 2.5 mg/kg/day to 20 mg/kg/day increased the ano-genital distance in rat fetuses, indicative of a masculinizing effect. Stanozolol prevented pregnancy when given orally to rat from the 1st to the 21st day of gestation.
No teratogenic effects or congenital malformation were observed in offspring of rabbits given 0.5 mg/day, 1.0 mg/day, or 5.0 mg/day of Stanozolol from the 8th through the 16th day of pregnancy, nor were there any adverse effects on the course of pregnancy at these dose levels.
Pregnancy Category X See CONTRAINDICATIONS section.
Nursing mothers it is not known whether anabolic steroids are excreted in human milk. Many drugs are excreted in human milk and because of the potential for adverse reactions in nursing infants from Stanozolol®, a decision should be made whether to discontinue nursing or discontinue the drug, taking into account the importance of the drug to the mother.
Pediatric use anabolic agents may accelerate epiphyseal maturation more rapidly than linear growth in children, and the effect may continue for 6 months after the drug has been stopped. Therefore, therapy should be monitored by x-ray studies at 6 month intervals in order to avoid the risk of compromising the adult height. The safety and efficacy of Stanozolol® in children with hereditary angioedema have not been established.

WARNINGS:

LIVER CELL TUMORS ARE REPORTED. MOST OFTEN THESE TUMORS ARE BENIGN AND ANDROGEN DEPENDENT, BUT FATAL MALIGNANT TUMORS HAVE BEEN REPORTED. WITH DRAWAL OF DRUG OFTEN RESULTS IN REGRESSION OR CESSATION OF PROGRESSION OF THE TUMOR. HOWEVER, HEPATIC TUMORS ASSOCIATED WITH ANDROGENS OR ANABOLIC STEROIDS ARE MUCH MORE VASCULAR THAN OTHER HEPATIC TUMORS AND MAY BE SILENT UNTIL LIFE-THREATENING INTRA-ABDOMINAL HEMORRHAGE DEVELOPS.
PELIOSIS HEPATIS, A CONDITION ARE ALSO REPORTED IN WHICH LIVER AND SOMETIMES SPLENIC TISSUE IS REPLACED WITH BLOOD-FILLED CYSTS, HAS BEEN REPORTED IN PATIENTS RECEVING ANDROGENIC ANABOLIC STEROID THERAPY. THESE CYSTS ARE SOMETIMES PRESENT WITH MINIMAL HEPATIC DYSFUNCTION, BUT AT OTHER TIMES THEY HAVE BEEN ASSOCIATED WITH LIVER FAILURE. THEY ARE OFTEN NOT RECOGNIZED UNTIL LIFE-THREATENING LIVER FAILURE OR INTRA-ABDOMINAL HEMORRHAGE DEVELOPS. WITHDRAWAL OF DRUG USUALLY RESULTS IN COMPLETE DISAPPERRANCE OF LESIONS.
BLOOD LIPID CHANGES THAT ARE KNOWN TO BE ASSOCIATED WITH INCREASED RISK OF ATHEROSCLEROSIS ARE SEEN IN PATIENTS TREATED WITH ANDROGENS AND ANABOLIC STEROIDS. THESE CHANGES INCLUDE DECREASED HIGH-DENSITY LIPOPROTEIN AND SOMETIMES INCREASED LOW-DENSITY LIPOPROTEIN. THE CHANGES MAY BE VERY MARKED AND COULD HAVE A SERIOUS IMPACT ON THE RISK OF ATHEROSCLEROSIS AND CORONARY ARTERY DISEASE.

OVERDOSAGE:
Cholestatic hepatitis and jaundice occur with 17-alpha-alkylated androgens at relatively low doses. If cholestatic hepatitis with jaundice appears, the anabolic steroid should be discontinued. If liver function tests become abnormal, the patient should be monitored closely and the etiology determined. Generally, the anabolic steroid should be discontinued although in cases of mild abnormalities, the physician may elect to follow the patient carefully at a reduced drug dosage.
In patients with breast cancer, anabolic steroid therapy may cause hypercalcemia by stimulating osteolysis. In this case, the drug should be discontinued.
Edema with or without congestive heart failure may be a serious complication in patients with preexisting cardiac, renal, or hepatic disease.
Geriatric male patients treated with androgenic anabolic steroids may be at an increased risk for the development of prostatic hypertrophy and prostatic carcinoma.

Anabolic steroids have not been shown to enhance athletic ability.

At the therapeutically dosages, no acute toxicity should be expected.

DRUG INTERACTION:
Anabolic steroids may increase sensitivity to anticoagulants; therefore, dosage of an anticoagulant may have to be decreased in order to maintain the prothrombin time at the desired therapeutic level.

ADVERSE REACTIONS:
Gastrointestinal: Nausea, vomiting, diarrhea.
Hepatic: Cholestatic jaundice with rarely, hepatic necrosis and death. Hepatocellular neoplasms and peliosis hepatis have been reported in association with long term androgenic anabolic steroid use. Reversible changes in liver function tests also occur including increased bromsulphalein (BSP) retention and increases in serum bilirubin, glutamic oxaloacetic transaminase (SGOT), and alkaline phosphatase.
Genitourinary System:
-post pubertal men: Inhibition of testicular functions, testicular atrophy, and oligospermia, impotence, chronic priapism, epididymitis and bladder irritability.
-Women): Clitoral enlargement, menstrual irregularities.
-In both sexes: increased or decreased libido.

Breast: Gynecomastia.
Larynx: Deepening of the vice in women.
Hair: Hirsutism and male pattern baldness in women.
Skin: Acne (especially in women and prepubertal boys).
Skeletal: Premature closure of epiphyses in children.
CNS: Habituation, excitation, insomnia, and depression.
Hematologic: Bleeding in patients on concomitant anticoagulant therapy.
Fluid and Electrolytes: Edema, retention of serum electrolytes (sodium, chloride, potassium, phosphate, calcium).
Metabolic/Endocrine: Decreased glucose tolerance, increased serum levels of low-density lipoproteins and decreased levels of high-density lipoproteins, increased creatine and creatinine excretion, increased serum levels of creatinine phosphokinase (CPK).
Some virilizing changes in women are irreversible even after prompt discontinuance of therapy and are not prevented by concomitant use of estrogens.
CNS: Habituation, excitation, insomnia, and depression.
Hematologic: Bleeding in patients on concomitant anticoagulant therapy.

PATIENT MONITORING:
Lipid profile: Serum Cholesterol, HDL, LDL, TG.
Hemoglobin and Hematocrit,
Liver function test: Total protein, Albumin, Globulin, Total and direct bilirubin, AST, ALT and alkaline phosphatase, tumor marker for liver: AFP and CA19-9
Prostatic specific antigen: PSA, Testosterone: total, free, and bioavailable.
Dihydrotestosterone & Estradiol
Male patients over 40 should undergo a digital rectal examination and evaluate PSA prior to androgen use. Periodic evaluations of the prostate should continue while on androgen therapy, especially in patients with difficulty in urination or with changes in voiding habits.

DOSAGE AND ADMINISTRATION:
The use of anabolic steroids may be associated with serious adverse reactions, many of which are dose related; therefore, patients should be placed on the lowest possible effective dose.
Muscle anabolism: 10-30 mg taken orally each day in divided doses for a duration of 4-6 weeks.
Hereditary angioedema: The dosage requirements for continuous treatment of hereditary angioedema with Stanozolol® should be individualized on the basis of the clinical response of the patient. Commonly 2-6 mg taken orally each day in divided doses initially and reduced by physician thereafter.
For body building: Effective dose of male: 50-100 mg per day.
Effective dose of female: 2.5-10 mg per day

HOW SUPPLIED:
– **Stanozolol®** 5 mg is supplied in bottle of 200 green tablets
– **Stanozolol®** 10 mg is supplied in bottle of 100 yellow tablets

For shelf-life please refer to the imprint on the pack.

Keep out of reach of children.

Should be at controlled room temperatures 15-30°C (59-86°F)

Protect from sun light

This drug has not been shown to be safe and effective for the enhancement of athletic performance!

Manufactured and Distributed by: LA Pharma S.r.l.

Date of approval: 15/2/2015