

RESEARCH MONOGRAPH . COMPOUND PROFILE

Turinabol

A 4-chloro-1,2-dehydro-17alpha-methyltestosterone developed at Jenapharm as a clinical anabolic agent for muscle wasting and bone density disorders, distinguished from its parent compound metandienone by the 4-chloro substitution that eliminates aromatase substrate activity and confers a markedly dissociated anabolic-to-androgenic ratio.

DOCUMENT	KDC-MN-1553
ISSUED	2026-05-15
REVISION	1.0
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COMPOUND CLASS	4-chloro-substituted 17alpha-alkylated anabolic-androgenic steroid derived from metandienone with dissociated anabolic-to-androgenic activity ratio

ABSTRACT

Chlorodehydromethyltestosterone (CDMT), marketed as Oral Turinabol, is a synthetic anabolic-androgenic steroid (AAS) first synthesized by chemist Albert Stachowiak at Jenapharm in the German Democratic Republic and patented in 1961. The compound is the 4-chloro-substituted derivative of metandienone (Dianabol), combining structural elements of clostebol (4-chlorotestosterone) and metandienone (1,2-dehydro-17alpha-methyltestosterone). The 4-chloro substitution at the A-ring prevents aromatization to estrogenic metabolites, while the 1,2-dehydro modification and the 17alpha-methyl group confer oral bioavailability and a dissociated anabolic-to-androgenic ratio of approximately 54:6 relative to methyltestosterone. Introduced for clinical use in 1965 for the treatment of muscle wasting disorders, osteoporosis, and recovery from debilitating illness, CDMT was prescribed at therapeutic doses of 5 to 10 mg per day in adults and 1 to 2.5 mg per day in women and children [1, 2].

The compound occupies a singular position in pharmacological and sporting history as the principal agent of State Plan Topic 14.25 (Staatsplanthema 14.25), the systematic doping program administered by the German Democratic Republic's Sports Medical Service from 1974 through 1989, under which approximately ten thousand athletes received CDMT, frequently without informed consent and often described to recipients as vitamins [3, 4]. The program produced a generation of international athletic dominance and a subsequent generation of adverse health consequences in exposed athletes, including hepatotoxicity, endocrine disruption, virilization in female

athletes, and cardiovascular morbidity. Production was discontinued by Jenapharm in 1994, and the compound holds no current marketing authorization in any jurisdiction.

Pharmacokinetically, CDMT is characterized by complete oral absorption, hepatic first-pass metabolism through multiple cytochrome P450 pathways (principally CYP3A4 for 6beta-hydroxylation and mitochondrial CYP11A1, CYP11B1, and CYP11B2 for 11beta-hydroxylation and additional oxidative transformations), and a plasma elimination half-life of approximately 16 hours [5, 6, 7]. The compound produces extensive phase I and phase II metabolites, with approximately 50 urinary metabolites identified. Of particular significance to anti-doping science, the long-term metabolite 4-chloro-18-nor-17beta-hydroxymethyl,17alpha-methyl-5beta-androst-13-en-3alpha-ol, identified by Schanzer and colleagues and subsequently confirmed in controlled human administration studies, extends the urinary detection window to 40 to 50 days after a single dose and potentially longer with chronic administration, enabling retrospective detection of CDMT use in athletes [8, 9, 10].

The pharmacology of CDMT is that of a moderate-affinity androgen receptor agonist with strong dissociation between anabolic (nitrogen-retentive, myotrophic) and androgenic (virilizing, sebotropic) activities. The compound does not undergo 5alpha-reduction to more potent androgens, does not serve as an aromatase substrate, and binds sex hormone-binding globulin (SHBG) with sufficient affinity to displace endogenous androgens and elevate free testosterone fractions in the presence of concurrent testosterone [11]. The principal adverse effects are dose-dependent hepatotoxicity (consistent with the 17alpha-alkylated steroid class), suppression of the hypothalamic-

pituitary-gonadal axis with reduction of luteinizing hormone, follicle-stimulating hormone, and endogenous testosterone production, unfavorable alterations in serum lipid profiles (suppression of high-density lipoprotein cholesterol and elevation of low-density lipoprotein cholesterol), and, in female subjects, irreversible virilization at supratherapeutic doses [12, 13]. This monograph reviews the chemistry, synthesis, and structural pharmacology of CDMT; the receptor-level mechanism of action; the comprehensive metabolic and pharmacokinetic profile including anti-doping detection science; the preclinical pharmacology; the limited clinical evidence base; sourcing and quality considerations; reconstitution and handling; stack interactions; adverse events and safety signals; and a structured comparative assessment of five alternative oral anabolic-androgenic steroids against CDMT on five competency standards.

1. Compound Identification

1.1 Names, synonyms, and registry identifiers

International Nonproprietary Name: Chlorodehydromethyltestosterone (CDMT). The compound does not hold an INN assignment through the World Health Organization; "chlorodehydromethyltestosterone" is the conventional pharmacological name used in the scientific literature.

Marketed name: Oral Turinabol (Jenapharm, German Democratic Republic). No other branded formulations have been marketed. The compound is frequently abbreviated as OT, Tbol, or DHCMT (dehydrochloromethyltestosterone) in the research and anti-doping literature.

Development origin: Jenapharm, Jena, German Democratic Republic. The compound was conceived by combining the structural features of clostebol (4-chlorotestosterone) and metandienone (1,2-dehydro-17 α -methyltestosterone). The synthesis was attributed to chemist Albert Stachowiak [1].

Chemical Abstracts Service registry number: 2446-23-3.

International Union of Pure and Applied Chemistry name: (8R,9S,10R,13S,14S,17S)-4-chloro-17-hydroxy-10,13,17-trimethyl-1,2,6,7,8,9,11,12,14,15,16,17-dodecahydrocyclopenta[a]phenanthren-3-one. The systematic name reflects the tetracyclic steroid nucleus with the 4-chloro, 1,2-dehydro, and 17 α -methyl modifications.

Other designations: 4-chloro-1-dehydro-17 α -methyltestosterone; 4-chloro-17 α -methyl-1,4-androstadien-17 β -ol-3-one; dehydrochloromethyltestosterone (DHCMT).

1.2 Structural class and chemistry

Chlorodehydromethyltestosterone is a synthetic anabolic-androgenic steroid belonging to the 17 α -alkylated subclass. The compound is derived from the testosterone nucleus through three structural modifications: (a) introduction of a 1,2-double bond (dehydrogenation) in ring A, which reduces androgenic potency and increases oral bioavailability; (b) addition of a 17 α -methyl group, which

prevents hepatic first-pass inactivation of the 17 β -hydroxyl group and confers oral activity; and (c) substitution of a chlorine atom at the 4-position of ring A, which blocks aromatization and further modifies the anabolic-to-androgenic dissociation [1, 2].

Molecular formula: C₂₀H₂₇ClO₂.

Molecular weight: 334.88 g/mol.

Exact mass: 334.1700 g/mol (monoisotopic).

Physical properties: White to off-white crystalline powder. Soluble in ethanol, methanol, dimethyl sulfoxide, and chloroform. Sparingly soluble in water. Melting point approximately 169 to 171 degrees Celsius.

Stereochemistry: The compound retains the natural steroid stereochemistry at carbons 8R, 9S, 10R, 13S, 14S, and 17S. The 17 α -methyl group is in the α configuration (below the plane of the steroid ring system), consistent with all orally active 17-alkylated androgens.

Key structural distinctions from parent compounds: Relative to metandienone (Dianabol), the 4-chloro substitution eliminates aromatase substrate recognition, preventing conversion to estrogenic metabolites and thereby eliminating estrogen-mediated water retention, gynecomastia, and related adverse effects. Relative to clostebol (4-chlorotestosterone), the 1,2-dehydro and 17 α -methyl modifications confer oral bioavailability and alter the anabolic-to-androgenic ratio.

1.3 Synthesis

The published synthesis of CDMT proceeds through modification of readily available steroid intermediates. The general synthetic route involves: (a) preparation of metandienone (1,2-dehydro-17 α -methyltestosterone) from 17 α -methyltestosterone by selective dehydrogenation at the 1,2-position using 2,3-dichloro-5,6-dicyano-1,4-benzoquinone (DDQ) or selenium dioxide as oxidant; (b) electrophilic chlorination at the 4-position of the A-ring using N-chlorosuccinimide, sulfuryl chloride, or molecular chlorine under controlled conditions; and (c) purification by recrystallization from ethanol, ethyl acetate, or acetone [1]. The 4-chlorination step is regioselective for the 4-position owing to the electronic activation of the conjugated dienone system in ring A. Alternatively, the synthesis may proceed from 4-chlorotestosterone (clostebol) by sequential 1,2-dehydrogenation and 17 α -

methylation, though this route is less commonly described in the literature. Industrial synthesis at Jenapharm produced the compound as a bulk pharmaceutical at greater than 98 percent purity by high-performance liquid chromatography. Research-grade material from contemporary chemical suppliers is characterized by nuclear magnetic resonance spectroscopy (^1H and ^{13}C), mass spectrometry, and HPLC purity determination.

2. Abstract

Chlorodehydromethyltestosterone (CDMT; Oral Turinabol) is a synthetic 17alpha-alkylated anabolic-androgenic steroid developed at Jenapharm in the German Democratic Republic and patented in 1961 [1]. The compound was introduced for clinical use in 1965 for muscle wasting disorders, osteoporosis, and convalescent recovery, prescribed at oral doses of 5 to 10 mg per day in men and 1 to 2.5 mg per day in women [2]. The pharmacology is characterized by moderate androgen receptor agonism with a dissociated anabolic-to-androgenic ratio of approximately 54:6, absence of aromatase substrate activity (no estrogenic conversion), minimal 5alpha-reductase substrate activity (reduced dihydrotestosterone formation), and high binding affinity for sex hormone-binding globulin [11].

The compound is historically significant as the central agent of State Plan Topic 14.25, the systematic performance-enhancing drug program of the German Democratic Republic administered from 1974 to 1989, under which approximately ten thousand athletes received CDMT without adequate informed consent [3, 4]. The program produced extensive documentation of both the performance-enhancing effects and the long-term adverse consequences of chronic suprathreshold AAS administration, constituting a unique (though ethically compromised) body of observational pharmacological data.

Production was discontinued in 1994. CDMT holds no current marketing authorization in any jurisdiction and is classified as a prohibited substance under the World Anti-Doping Agency Prohibited List (category S1: anabolic agents) [14]. The compound remains available as a research-grade chemical from multiple commercial suppliers and circulates in the unregulated performance-enhancement market.

The pharmacokinetics are characterized by oral bioavailability (17alpha-alkylation conferring resistance to hepatic first-pass 17-oxidation), a plasma elimination half-life of approximately 16 hours, and extensive hepatic metabolism through CYP3A4 (6beta-hydroxylation), mitochondrial CYP11A1, CYP11B1, and CYP11B2 (11beta-hydroxylation and additional oxidative transformations), and CYP46A1 (16beta-hydroxylation) [5, 6, 7]. Approximately 50 urinary metabolites have been identified. The long-term metabolite profile, characterized principally by Schanzer,

Forsdahl, and colleagues, extends the anti-doping detection window to 40 to 50 days and potentially longer, enabling retrospective detection that has resulted in the retroactive disqualification of multiple Olympic medalists [8, 9, 10].

This monograph provides a comprehensive review of the chemistry, mechanism of action, pharmacokinetics, preclinical and clinical pharmacology, anti-doping detection science, adverse events, and comparative positioning of CDMT within the oral AAS class.

3. Discovery and Development History

3.1 Origin and synthesis

CDMT was synthesized at Jenapharm in Jena, German Democratic Republic, as the first original pharmaceutical product of that company. The concept originated with chemist Albert Stachowiak, who combined the structural features of two existing anabolic-androgenic steroids: clostebol (4-chlorotestosterone, a topical anabolic with reduced androgenic activity due to the 4-chloro substitution) and metandienone (1,2-dehydro-17 α -methyltestosterone, the orally active anabolic marketed as Dianabol by CIBA). The combination was intended to produce an orally active anabolic steroid with reduced androgenic and estrogenic side effects relative to metandienone [1]. The compound was patented in 1961 and assigned the trade name Oral Turinabol.

3.2 Clinical introduction and therapeutic use

CDMT was introduced for clinical use in East Germany in 1965. Therapeutic indications included treatment of muscle wasting in chronic debilitating illness, promotion of bone density in osteoporosis, acceleration of recovery from severe trauma and burns, and management of protein-calorie malnutrition in convalescent patients [2]. Prescribed doses were conservative by later performance-enhancement standards: 5 to 10 mg per day in adult males, 1 to 2.5 mg per day in females and pediatric patients. The compound was considered well tolerated at these doses, with hepatotoxicity, endocrine suppression, and virilization reported as dose-dependent adverse effects manageable at the therapeutic range.

3.3 State Plan Topic 14.25

In 1974, the German Democratic Republic formalized a centralized, clandestine program of systematic performance-enhancing drug administration to elite athletes under the designation Staatsplanthema 14.25 (State Plan Topic 14.25) [3]. The program was administered through the Sports Medical Service (Sportmedizinischer Dienst, SMD) under the direction of Manfred Hoppner and overseen by Manfred Ewald, president of the German Sports Federation (DTSB). CDMT was the principal

anabolic agent employed in the program, selected for its favorable anabolic-to-androgenic dissociation, oral administration route, and relatively short detection window by the analytical methods available in the 1970s and 1980s.

Approximately ten thousand athletes across all Olympic sports received CDMT under the program, typically at doses of 5 to 30 mg per day in males and 5 to 15 mg per day in females, substantially exceeding the therapeutic range [3, 4]. The compound was frequently described to athletes as "vitamins" or "supportive substances" (unterstützende Mittel), and informed consent was not obtained. Female athletes and adolescent athletes were included in the program, resulting in irreversible virilization (voice deepening, clitoral hypertrophy, hirsutism, menstrual irregularity) in a substantial proportion of female recipients [4].

3.4 Discontinuation

Following German reunification in 1990, the State Plan Topic 14.25 program was exposed through access to the Stasi archives and through testimony at the 1998 to 2000 Berlin doping trials, in which Ewald and Hoppner were convicted of causing bodily harm to minors [3]. Jenapharm discontinued production of Oral Turinabol in 1994, coinciding with the broader global withdrawal of multiple oral AAS from clinical markets during the early 1990s. The compound has not been reintroduced to clinical use in any jurisdiction and holds no current marketing authorization.

3.5 Anti-doping legacy

The development of long-term metabolite detection methods, beginning with the work of Schanzer and colleagues in the 1990s and extended by Forsdahl, Thevis, and others through the 2010s, enabled retrospective reanalysis of stored athlete urine samples [8, 9, 10]. This reanalysis resulted in the retroactive disqualification and medal stripping of multiple Olympic athletes, most notably in track and field and weightlifting, when stored samples from the 2008 and 2012 Olympic Games were retested using updated methods capable of detecting the long-term CDMT metabolites. The CDMT detection story is one of the most significant demonstrations of the value of sample storage and retrospective reanalysis in anti-doping science.

4. Mechanism of Action

4.1 Androgen receptor agonism

CDMT exerts its principal pharmacological effects through binding to and activation of the androgen receptor (AR), a member of the nuclear receptor superfamily of ligand-activated transcription factors. Upon binding CDMT, the androgen receptor undergoes conformational change, dissociates from heat-shock protein chaperones, dimerizes, translocates to the nucleus, and binds to androgen response elements (AREs) in the promoter regions of target genes. Transcriptional activation of androgen-responsive genes drives the anabolic effects of the compound: increased protein synthesis, nitrogen retention, and satellite cell activation in skeletal muscle; increased osteoblast activity and bone mineral density; and increased erythropoietin production and red blood cell mass [15, 16].

The binding affinity of CDMT for the androgen receptor is moderate, lower than that of testosterone and substantially lower than that of dihydrotestosterone (DHT). The anabolic-to-androgenic dissociation ratio of approximately 54:6 (relative to methyltestosterone at 100:100) indicates that the compound preferentially activates anabolic pathways (skeletal muscle, bone) relative to androgenic pathways (prostate, sebaceous glands, hair follicles, larynx) [2, 11]. The molecular basis of this tissue selectivity is incompletely characterized but is attributed to the combined effects of the 4-chloro and 1,2-dehydro modifications on receptor conformation, coregulator recruitment, and tissue-specific androgen receptor cofactor interactions.

4.2 Non-aromatizability

The 4-chloro substitution at the A-ring of CDMT prevents recognition by the cytochrome P450 aromatase enzyme (CYP19A1), which catalyzes the conversion of C19 androgens to C18 estrogens [2]. Unlike metandienone (which undergoes partial aromatization) and testosterone (which undergoes substantial aromatization), CDMT does not produce estrogenic metabolites. The clinical consequence is the absence of estrogen-mediated effects including water retention, gynecomastia, and

estrogen-driven adipogenesis. This property distinguishes CDMT from its parent compound metandienone and from testosterone in the context of body composition effects.

4.3 Minimal 5alpha-reduction

CDMT binds weakly to the 5alpha-reductase enzyme (types I and II), which catalyzes the conversion of testosterone to the more potent androgen dihydrotestosterone in androgen-sensitive tissues including the prostate, skin, and hair follicles [11]. The minimal 5alpha-reductase substrate activity of CDMT contributes to the reduced androgenic side-effect profile (lower incidence of acne, male-pattern hair loss, and prostate stimulation) relative to testosterone and to AAS that undergo efficient 5alpha-reduction (such as testosterone itself and boldenone).

4.4 SHBG binding and free androgen modulation

CDMT binds to sex hormone-binding globulin (SHBG) with appreciable affinity [11]. In the presence of concurrent testosterone (either endogenous or exogenous), CDMT displaces testosterone from SHBG binding sites, increasing the free (unbound) fraction of circulating testosterone. This displacement amplifies the effective androgenic and anabolic signal of co-administered testosterone. The SHBG-binding property is pharmacologically relevant in the context of combination (stack) use, where CDMT is frequently co-administered with injectable testosterone preparations.

4.5 Hypothalamic-pituitary-gonadal axis suppression

As with all exogenous androgens, CDMT activates negative feedback on the hypothalamic-pituitary-gonadal (HPG) axis, suppressing gonadotropin-releasing hormone (GnRH) pulsatility and reducing luteinizing hormone (LH) and follicle-stimulating hormone (FSH) secretion from the anterior pituitary [12, 13]. The consequence is reduced endogenous testosterone biosynthesis and spermatogenesis. At doses of 30 to 50 mg per day, endogenous testosterone production may be suppressed by 60 to 70 percent within 6 weeks. Recovery of HPG axis function following cessation is variable and depends on dose, duration of use, and individual factors; anabolic steroid-induced hypogonadism (ASIH) may persist for months following prolonged use.

5. Pharmacokinetics and Routes of Administration

5.1 Absorption

CDMT is administered exclusively by the oral route. The 17alpha-methyl group confers resistance to hepatic first-pass oxidation of the 17beta-hydroxyl group (the principal inactivating transformation for unalkylated androgens), resulting in oral bioavailability sufficient for systemic pharmacological activity. Specific bioavailability values have not been reported in the published literature, but the pharmacological activity at oral doses of 5 to 10 mg per day is consistent with substantial (likely greater than 50 percent) systemic availability [5].

5.2 Distribution

Volume of distribution data for CDMT in humans have not been published in peer-reviewed form. By analogy to structurally related 17alpha-alkylated AAS (metandienone, stanozolol, oxandrolone), the compound is expected to distribute extensively into skeletal muscle, liver, kidney, and adipose tissue. Plasma protein binding has not been quantified but is expected to be moderate, with binding to both albumin and SHBG contributing to the bound fraction.

5.3 Metabolism

The metabolism of CDMT has been characterized in substantial detail, driven primarily by anti-doping detection research rather than by classical clinical pharmacokinetic investigation. The principal metabolic pathways include:

CYP3A4-mediated 6beta-hydroxylation: CYP3A4 catalyzes hydroxylation at the 6beta-position, mirroring the well-characterized 6beta-hydroxylation of testosterone by the same enzyme [6, 7].

Mitochondrial CYP-mediated transformations: The steroid hormone-synthesizing cytochrome P450 enzymes CYP11A1, CYP11B1, and CYP11B2, located in adrenal and gonadal mitochondria, metabolize CDMT through 11beta-hydroxylation and

additional oxidative pathways. CYP11A1 produces at least six metabolites including 2-hydroxy-OT, 16-hydroxy-OT, and 2,16-dihydroxy-OT. These mitochondrial pathways are notable because they are distinct from the conventional microsomal CYP-mediated drug metabolism and involve steroidogenic enzymes not typically associated with xenobiotic metabolism [6].

CYP46A1-mediated 16beta-hydroxylation: The brain cholesterol 24-hydroxylase CYP46A1 metabolizes CDMT to 16beta-hydroxy-CDMT, suggesting central nervous system metabolism of the compound [17].

CYP21A2-mediated metabolism of NorDHCMT: The demethylated metabolite NorDHCMT is further metabolized by CYP21A2 (but not by CYP3A4 or CYP17A1) to 20beta-hydroxy-NorDHCMT, which serves as a long-term urinary metabolite [18].

Phase II conjugation: The oxidized phase I metabolites undergo glucuronidation and, to a lesser extent, sulfation, producing water-soluble conjugates excreted in urine. Approximately 50 distinct urinary metabolites have been identified using gas chromatography-mass spectrometry (GC-MS) and liquid chromatography-tandem mass spectrometry (LC-MS/MS) [8, 9].

5.4 Elimination

The plasma elimination half-life of CDMT is approximately 16 hours, based on pharmacokinetic studies in healthy human subjects [5]. Elimination is predominantly renal, through urinary excretion of glucuronidated and sulfated metabolites. A fraction of the parent compound is excreted unchanged. The long-term metabolite 4-chloro-18-nor-17beta-hydroxymethyl,17alpha-methyl-5beta-androst-13-en-3alpha-ol is excreted for substantially longer periods than the major metabolites, with detection windows of 40 to 50 days demonstrated in controlled administration studies [9, 10]. Some metabolites may remain detectable for several months following chronic high-dose administration.

5.5 Routes of administration

CDMT was manufactured exclusively as an oral tablet (1 mg and 5 mg presentations by Jenapharm). No parenteral formulations have been marketed or described in the published literature. The compound has been detected as a contaminant in topical

preparations of other steroids, and transdermal absorption of CDMT, metandienone, clostebol, and oxandrolone has been demonstrated in controlled studies, with urinary metabolites detectable after skin application [19].

5.6 Anti-doping detection

The detection of CDMT use in athletes has been a major focus of anti-doping analytical chemistry. The evolution of detection methods reflects three phases:

Phase I (1970s to 1990s): Detection relied on identification of the parent compound and major short-lived metabolites by GC-MS. Detection windows were limited to days, enabling athletes to discontinue use before competition and avoid detection. This short detection window was one of the reasons CDMT was selected for State Plan Topic 14.25 [3].

Phase II (1990s to 2010s): Schanzer and colleagues identified long-term metabolites of CDMT, including 4-chloro-18-nor-17 β -hydroxymethyl,17 α -methyl-5 β -androst-13-en-3 α -ol, that persist in urine for weeks to months after administration [8]. These metabolites enabled substantially extended detection windows.

Phase III (2010s to present): Controlled human administration studies by Forsdahl, Thevis, and colleagues confirmed and extended the long-term metabolite detection data, identifying six additional long-term metabolites and establishing detection windows of 40 to 50 days in controlled single-dose studies [9, 10]. LC-MS/MS methods for detection of glucuronidated metabolites without hydrolysis have been developed as alternatives to conventional GC-MS/MS analysis [20]. The World Anti-Doping Agency (WADA) recommends screening for all known CDMT metabolites in both glucuronidated and unconjugated fractions [14].

6. Preclinical Pharmacology

6.1 Myotrophic and androgenic bioassays

The anabolic-to-androgenic dissociation ratio of 54:6 for CDMT was established using the classical Hershberger bioassay in castrated rats, in which the levator ani muscle weight (anabolic endpoint) and the ventral prostate weight (androgenic endpoint) are measured following subcutaneous or oral administration of the test compound relative to methyltestosterone [2, 15]. The ratio indicates that CDMT produces approximately 54 percent of the anabolic activity of methyltestosterone at equimolar doses but only 6 percent of the androgenic activity. This dissociation is among the most favorable in the 17 α -alkylated AAS class, exceeded only by oxandrolone (anabolic:androgenic ratio approximately 322:24 to 630:24 depending on the assay system).

6.2 Nitrogen retention and protein synthesis

CDMT produces dose-dependent nitrogen retention in animal models, consistent with the anabolic mechanism mediated through androgen receptor activation of skeletal muscle protein synthesis [15, 16]. The magnitude of nitrogen retention at therapeutic doses is moderate, reflecting the moderate androgen receptor binding affinity of the compound.

6.3 Bone mineral density

Animal studies in the 1960s and 1970s demonstrated that CDMT increases bone mineral density in ovariectomized and orchidectomized rodent models, supporting the clinical indication for osteoporosis treatment [2]. The mechanism involves androgen receptor-mediated stimulation of osteoblast activity and suppression of osteoclast-mediated bone resorption.

6.4 Hepatotoxicity

The 17alpha-alkylated steroid class produces dose-dependent hepatotoxicity through cholestatic mechanisms. In animal models, CDMT at supratherapeutic doses produces elevation of hepatic transaminases, cholestasis, and, with chronic administration, peliosis hepatis (blood-filled hepatic cysts) [12]. The hepatotoxicity is attributed to the 17alpha-methyl group, which impairs bile salt export pump function and disrupts hepatic bile flow. CDMT is considered less hepatotoxic than oxymetholone and methyltestosterone but retains the class liability inherent to 17alpha-alkylation. In liver and kidney tissues, CDMT may cause fatty degeneration through interference with mitochondrial oxygen transport or mitochondrial cytochrome P450 dysfunction [12].

6.5 Cardiovascular effects

Preclinical and observational data from the East German doping program indicate that chronic supratherapeutic CDMT administration produces unfavorable alterations in serum lipid profiles, including suppression of high-density lipoprotein (HDL) cholesterol and elevation of low-density lipoprotein (LDL) cholesterol [13]. Left ventricular hypertrophy has been documented in former East German athletes who received CDMT under State Plan Topic 14.25, though attribution to CDMT alone is confounded by concurrent use of other performance-enhancing substances and by the effects of intensive athletic training [4].

7. Clinical Evidence Base

7.1 Therapeutic indications (historical)

CDMT was prescribed in East Germany from 1965 to 1994 for the following clinical indications: (a) muscle wasting in chronic debilitating illness, including tuberculosis, cancer cachexia, and post-surgical recovery; (b) osteoporosis, particularly in postmenopausal women; (c) promotion of bone healing in fracture patients; and (d) recovery from severe burns and trauma [2]. Therapeutic doses were 5 to 10 mg per day in adult males and 1 to 2.5 mg per day in females and pediatric patients, administered in cycles of 4 to 6 weeks with monitoring of hepatic function and endocrine parameters.

7.2 Limitations of the clinical evidence base

The clinical evidence base for CDMT is severely limited by several factors. Controlled clinical trials meeting modern standards of randomization, blinding, and statistical analysis were not conducted or, if conducted, were not published in the Western peer-reviewed literature. Much of the clinical documentation from the East German period exists in German-language institutional reports, Stasi archives, and internal Jenapharm documents rather than in indexed medical journals. The clandestine nature of the athletic doping program further compromised the integrity of clinical observations by mixing therapeutic and supratherapeutic dose data, by omitting informed consent, and by introducing selection bias (elite athletes as subjects).

7.3 Performance enhancement data from State Plan Topic 14.25

The documentation recovered from the State Plan Topic 14.25 program provides observational evidence of the performance-enhancing effects of CDMT, though this evidence is scientifically compromised by the absence of controlled conditions, blinding, and randomization [3, 4]. Reported effects in male athletes at doses of 10 to 30 mg per day included increased lean body mass, increased strength, improved recovery from training, and reduced body fat percentage. Reported effects in fe-

male athletes at doses of 5 to 15 mg per day included similar performance enhancements accompanied by progressive virilization, the severity and reversibility of which correlated with dose and duration.

7.4 Modern clinical data

No modern clinical trials of CDMT have been conducted or registered in public trial registries. The compound holds no investigational new drug application and is not under active clinical development for any indication. The pharmacokinetic studies that constitute the modern human evidence base are controlled administration studies conducted for anti-doping purposes rather than for therapeutic investigation [5, 9, 10].

8. Sourcing and Quality Verification

CDMT is not available as a pharmaceutical-grade product from any licensed manufacturer. The Jenapharm production was discontinued in 1994, and no generic pharmaceutical manufacturer has obtained marketing authorization for the compound in any jurisdiction.

Research-grade CDMT is available from multiple chemical and pharmaceutical research suppliers (Sigma-Aldrich, Cayman Chemical, Cerilliant, LGC Standards, TRC Canada, MedChemExpress, and others) at stated purities of 98 percent or greater by HPLC. Supply is in milligram to gram quantities. Pricing varies from approximately \$100 to \$500 per gram depending on supplier and quantity.

The unregulated performance-enhancement market distributes CDMT in oral tablet form from underground laboratories. Quality, purity, and dose accuracy in this market segment are highly variable and frequently unreliable. Independent analytical testing services (Janoshik Analytical, Simec AG, and others) have documented frequent discrepancies between label claims and actual content in underground laboratory products, including incorrect dose, absence of active compound, and substitution with alternative steroids.

Investigators acquiring research-grade CDMT should confirm compound identity and purity on every lot through:

- High-performance liquid chromatography area-percent purity (target greater than 98 percent)
- Mass spectrometry confirmation of molecular weight (335.18 for $[M+H]^+$ of the protonated molecule)
- Nuclear magnetic resonance spectroscopy (1H and ^{13}C) confirmation of structural identity
- Residual solvent analysis
- Absence of related steroid impurities (particularly metandienone and clostebol, which are synthetic precursors)

Certified reference standards for CDMT and its metabolites are available from WADA-accredited reference material suppliers (LGC Standards, Cerilliant) for use in analytical method validation and quantitative analysis.

9. Reconstitution and Handling

CDMT is supplied as a crystalline powder for research applications. The compound is soluble in organic solvents (ethanol, methanol, dimethyl sulfoxide, chloroform, dichloromethane) and sparingly soluble in water.

Stock solution preparation for in vitro work: Dissolve in dimethyl sulfoxide (DMSO) at concentrations of 10 to 100 mM for cell-based assays, or in ethanol at concentrations of 1 to 50 mg/mL for receptor binding assays. Stock solutions should be aliquoted to avoid repeated freeze-thaw cycles and stored at minus 20 degrees Celsius. DMSO stock solutions are stable for at least 6 months at minus 20 degrees Celsius.

Stock solution preparation for in vivo work: Dissolve in a vehicle consisting of 5 to 10 percent DMSO in sesame oil, corn oil, or propylene glycol for oral gavage administration in rodent studies. Alternatively, prepare suspensions in 0.5 percent methylcellulose with 0.1 percent Tween 80 in water for oral dosing.

Storage: Solid material should be stored at room temperature or at 2 to 8 degrees Celsius in a desiccated environment, protected from light. The compound is stable under these conditions for at least 2 years.

Handling precautions: CDMT is a potent androgen. Laboratory personnel should use standard precautions for handling hormonally active compounds: gloves, laboratory coat, and fume hood for weighing and solution preparation. Skin contact should be avoided, as transdermal absorption has been demonstrated [19].

10. Stack Interactions and Combinations

CDMT has been used in combination with other performance-enhancing compounds, and the following interactions and considerations are documented:

Testosterone (injectable esters): The most common combination in the performance-enhancement context. CDMT displaces testosterone from SHBG, increasing the free testosterone fraction and amplifying the androgenic and anabolic effects of co-administered testosterone [11]. The combination produces additive anabolic effects but also additive HPG axis suppression, hepatic strain (from CDMT), and lipid profile deterioration.

Other oral 17alpha-alkylated AAS: Concurrent use with other 17alpha-alkylated steroids (metandienone, stanozolol, oxymetholone, oxandrolone) produces additive hepatotoxicity. The combination is not pharmacologically justified, as the hepatic burden scales with the total 17alpha-alkylated steroid dose without proportional increase in anabolic effect.

Aromatase inhibitors: Because CDMT is non-aromatizable, co-administration with aromatase inhibitors (anastrozole, letrozole, exemestane) targets only the estrogenic conversion of co-administered aromatizable substrates (testosterone, metandienone, boldenone) and does not modify CDMT pharmacology directly.

CYP3A4 inhibitors and inducers: Strong CYP3A4 inhibitors (ketoconazole, itraconazole, ritonavir, clarithromycin) may reduce the 6beta-hydroxylation of CDMT, potentially increasing systemic exposure and hepatic burden. Strong CYP3A4 inducers (rifampin, carbamazepine, phenytoin, St. John's wort) may increase CDMT clearance and reduce systemic exposure. These interactions have not been formally studied in dedicated drug-drug interaction trials but are predicted from the CYP3A4 metabolic dependence [6, 7].

Hepatotoxic agents: Concurrent use with other hepatotoxic drugs (acetaminophen at high doses, methotrexate, valproic acid, isoniazid, some statins) may produce additive liver injury. Monitoring of hepatic transaminases is warranted with any concurrent hepatotoxic agent.

Anticoagulants: As with other AAS, CDMT may potentiate the effects of coumarin anticoagulants (warfarin) through suppression of vitamin K-dependent clotting factor synthesis. Dose adjustment and monitoring of international normalized ratio (INR) are warranted with concurrent use.

Insulin and oral hypoglycemic agents: AAS may improve insulin sensitivity and reduce blood glucose in some contexts. Concurrent use with insulin or sulfonylureas may increase hypoglycemia risk, requiring dose monitoring.

11. Adverse Events and Safety Signal

11.1 Hepatotoxicity

Dose-dependent hepatotoxicity is the principal organ-specific adverse effect of the 17alpha-alkylated steroid class. CDMT produces elevation of hepatic transaminases (alanine aminotransferase, aspartate aminotransferase), alkaline phosphatase, and gamma-glutamyl transferase in a dose-dependent manner [12]. At therapeutic doses (5 to 10 mg per day), transaminase elevations are typically mild and reversible. At supratherapeutic doses (30 to 80 mg per day) or with prolonged administration (greater than 8 weeks), more significant hepatic injury may occur, including cholestatic jaundice and, rarely, peliosis hepatis. Hepatocellular carcinoma has been reported in association with chronic high-dose 17alpha-alkylated AAS use, though a causal relationship specific to CDMT has not been established.

11.2 Endocrine suppression

Suppression of the HPG axis is an expected pharmacological consequence of exogenous androgen administration [12, 13]. At doses of 30 to 50 mg per day, endogenous testosterone production is reduced by approximately 60 to 70 percent within 6 weeks. Gonadotropin (LH and FSH) suppression precedes testosterone decline and is the proximate cause. Spermatogenesis is impaired. Recovery following cessation is variable: most individuals recover HPG axis function within 3 to 6 months, but anabolic steroid-induced hypogonadism (ASIH) may persist longer, particularly following prolonged or high-dose use.

11.3 Cardiovascular effects

CDMT produces dose-dependent unfavorable alterations in serum lipid profiles, including suppression of HDL cholesterol and elevation of LDL cholesterol [13]. The magnitude of the lipid alteration is less pronounced than with some oral AAS (oxymetholone, stanozolol) but is clinically relevant with chronic use. Chronic AAS use is associated with increased risk of atherosclerosis, left ventricular hypertrophy, cardiomyopathy, and thromboembolic events. Blood pressure elevation has been reported but is generally modest at therapeutic doses.

11.4 Virilization in females

Female athletes administered CDMT under State Plan Topic 14.25 at doses of 5 to 15 mg per day experienced progressive virilization including voice deepening, clitoral hypertrophy, hirsutism, acne, menstrual irregularity, and altered body fat distribution [4]. Many of these effects were irreversible, particularly voice changes and clitoral hypertrophy. The virilization burden documented in the East German program remains one of the most extensively reported instances of AAS-induced female virilization in the literature.

11.5 Other adverse effects

Additional reported adverse effects include:

- Acne (mild to moderate, dose-dependent; less pronounced than with aromatizable or 5 α -reducible AAS)
- Male-pattern hair loss (uncommon at therapeutic doses owing to minimal 5 α -reduction)
- Mood and behavioral changes (irritability, aggression, mood lability; dose-dependent)
- Tendon and connective tissue effects (reduced collagen synthesis at supratherapeutic doses)
- Premature epiphyseal closure in adolescents (a class effect of all AAS in growing individuals)

11.6 Safety monitoring

Investigators using CDMT in research or monitoring subjects exposed to the compound should consider:

- Baseline and periodic hepatic function tests (ALT, AST, GGT, alkaline phosphatase, bilirubin)
- Baseline and periodic lipid panel (total cholesterol, LDL, HDL, triglycerides)
- Baseline and periodic hormonal assessment (total and free testosterone, LH, FSH, estradiol)
- Complete blood count (erythrocytosis secondary to androgen-mediated erythropoietin stimulation)

- Blood pressure monitoring
- In females: clinical assessment for virilization signs

12. Comparable Compounds Assessment

This section presents a structured comparative assessment of five oral anabolic-androgenic steroids against CDMT, evaluated on five competency standards: novelty (structural and mechanistic distinctiveness), effect size (anabolic potency relative to androgenic and adverse-effect burden), promising potential (current research or clinical trajectory), side-effect profile, and overall validation (extent and quality of the evidence base). The five comparators were selected as the principal oral AAS with clinical or substantial research documentation, with priority on compounds that share structural or pharmacological features with CDMT.

12.1 Metandienone (Dianabol)

Structural relationship: Parent compound. CDMT is the 4-chloro derivative of metandienone. Both share the 1,2-dehydro and 17alpha-methyl modifications of testosterone [1].

Mechanism: Androgen receptor agonist. Unlike CDMT, metandienone undergoes partial aromatization to 17alpha-methylestradiol, producing estrogenic effects including water retention and gynecomastia. The anabolic-to-androgenic ratio is approximately 90 to 210:40 to 60 (varying by assay), indicating greater anabolic potency than CDMT but also greater androgenic activity.

Novelty: Low. Metandienone was developed before CDMT and is structurally simpler.

Effect size: Greater anabolic potency than CDMT, with correspondingly greater weight gain (a substantial fraction of which is water retention from estrogenic activity). Greater androgenic effect and greater hepatotoxic burden at equianabolic doses.

Promising potential: Low for clinical development. Metandienone holds no current marketing authorization and is not under clinical investigation. It remains the most widely used oral AAS in the unregulated performance-enhancement market.

Side-effect profile: More pronounced than CDMT: estrogenic effects (gynecomastia, water retention), greater hepatotoxicity at typical use doses (25 to 50 mg per day), more significant HPG axis suppression, and greater lipid profile deterioration. The aromatization to methylestradiol is a specific liability that CDMT avoids.

Overall validation: Moderate. Extensive use history (since 1958) but limited controlled clinical data. The pharmacology is well characterized from in vitro and animal studies.

Comparison to CDMT: CDMT was specifically designed to retain the anabolic benefits of metandienone while eliminating the estrogenic liability and reducing the androgenic burden. CDMT achieves this through the 4-chloro substitution. The trade-off is reduced absolute anabolic potency: CDMT produces less total mass gain but leaner (non-water-retentive) gains with a more favorable side-effect profile.

12.2 Oxandrolone (Anavar)

Structural relationship: Both are oral 17alpha-alkylated AAS, but oxandrolone is a dihydrotestosterone (DHT) derivative with a lactone ring (2-oxa modification) rather than a testosterone derivative. The structural classes are distinct.

Mechanism: Androgen receptor agonist with extremely high anabolic-to-androgenic dissociation (approximately 322 to 630:24, the highest in the clinical AAS class). Like CDMT, oxandrolone does not aromatize. Unlike CDMT, oxandrolone stimulates lipolysis through effects on triiodothyronine (T3) and thyroid hormone metabolism [21].

Novelty: Moderate. The 2-oxa (lactone) modification is structurally distinctive and produces metabolic stability that contributes to the favorable therapeutic index.

Effect size: Greater anabolic-to-androgenic dissociation than CDMT. Oxandrolone is the reference compound for dissociated anabolic activity in the clinical AAS literature. Absolute mass gain is less than CDMT at typical doses, but quality of gain (lean tissue without water retention) is comparable or superior.

Promising potential: Moderate to high. Oxandrolone retains FDA approval in the United States for the treatment of involuntary weight loss in catabolic conditions (HIV/AIDS wasting, severe burns, Turner syndrome, constitutional growth delay). It

is the only oral AAS with active marketing authorization in a major regulatory jurisdiction [21].

Side-effect profile: The most favorable in the oral AAS class. Hepatotoxicity is lower than CDMT, metandienone, stanozolol, and oxymetholone at therapeutic doses. Lipid profile effects are present but less severe than stanozolol. Virilization risk in females is low at therapeutic doses (2.5 to 20 mg per day).

Overall validation: High. The most extensively studied oral AAS in controlled clinical trials, with published evidence in burn recovery, HIV wasting, Turner syndrome, hereditary angioedema, and alcoholic hepatitis. The clinical evidence base substantially exceeds that of CDMT.

Comparison to CDMT: Oxandrolone is superior to CDMT on most competency standards: greater anabolic dissociation, lower hepatotoxicity, retained regulatory approval, and a substantially larger clinical evidence base. CDMT has greater absolute anabolic potency at equimolar doses and produces more pronounced lean mass gains in performance-enhancement contexts, but at the cost of greater hepatotoxicity and a more compromised safety profile.

12.3 Stanozolol (Winstrol)

Structural relationship: Both are oral 17alpha-alkylated AAS. Stanozolol is a DHT derivative with a pyrazole ring fused to the A-ring (a 3,2-c pyrazole modification), structurally distinct from the testosterone-derived CDMT.

Mechanism: Androgen receptor agonist. Anabolic-to-androgenic ratio approximately 320:30. Like CDMT, stanozolol does not aromatize. Stanozolol has additional pharmacological activity: it reduces SHBG concentrations (increasing free androgen levels), stimulates fibrinolytic activity, and has reported effects on collagen synthesis [22].

Novelty: Moderate. The pyrazole ring fusion is structurally distinctive.

Effect size: Greater anabolic-to-androgenic dissociation than CDMT. Stanozolol produces lean mass gains without water retention. The compound is particularly associated with strength enhancement and with improvement in speed and power athletic modalities.

Promising potential: Low for new clinical development. Stanozolol retains marketing authorization in some jurisdictions for hereditary angioedema prophylaxis but has been largely supplanted by C1 esterase inhibitor concentrates and kallikrein inhibitors. It is not under active clinical investigation for new indications.

Side-effect profile: More pronounced hepatotoxicity than CDMT at typical performance-enhancement doses (25 to 50 mg per day oral, or 50 mg every other day injectable). More severe HDL cholesterol suppression than most oral AAS. Joint discomfort and tendon desiccation are commonly reported, attributed to anti-estrogenic effects on synovial fluid production.

Overall validation: Moderate. Extensive use history and pharmacological characterization. Limited controlled clinical trial data relative to oxandrolone.

Comparison to CDMT: Stanozolol has greater anabolic dissociation and greater strength-enhancing reputation but produces more pronounced hepatotoxicity and lipid disturbance. CDMT has a more favorable hepatic safety profile at comparable anabolic doses and produces less severe joint and connective tissue effects.

12.4 Clostebol (4-chlorotestosterone)

Structural relationship: Direct structural precursor. Clostebol is 4-chlorotestosterone; CDMT adds the 1,2-dehydro and 17alpha-methyl modifications [1].

Mechanism: Weak androgen receptor agonist. Clostebol has low oral bioavailability (no 17alpha-alkylation) and is used primarily as a topical agent (clostebol acetate in topical dermatological and wound-healing preparations). Anabolic-to-androgenic ratio approximately 46:25.

Novelty: Low. The 4-chloro substitution is shared with CDMT but without the oral-bioavailability and anabolic-potency enhancements.

Effect size: Low systemic anabolic effect owing to poor oral bioavailability. Topical application produces local anabolic effects (wound healing, dermal repair) without significant systemic androgenic or anabolic activity.

Promising potential: Low. Clostebol retains marketing authorization in some European and South American jurisdictions as a topical wound-healing agent (Trofodermin). It is not under clinical development for systemic anabolic

indications.

Side-effect profile: Favorable for topical use. Minimal hepatotoxicity (no 17alpha-alkylation). Minimal systemic endocrine effects at topical doses. The principal safety concern is inadvertent positive anti-doping tests from topical exposure.

Overall validation: Low to moderate. Limited systemic clinical data. Topical use evidence is moderate.

Comparison to CDMT: Clostebol is the structural ancestor of CDMT but is pharmacologically inferior for systemic anabolic applications owing to the absence of oral bioavailability. CDMT represents a successful optimization of the clostebol scaffold for oral anabolic activity.

12.5 Methyltestosterone

Structural relationship: The simplest 17alpha-alkylated androgen (17alpha-methyltestosterone). Both are testosterone-derived 17alpha-alkylated AAS, but methyltestosterone lacks the 1,2-dehydro and 4-chloro modifications of CDMT.

Mechanism: Androgen receptor agonist with minimal anabolic-to-androgenic dissociation (ratio approximately 100:100 by definition, as it is the reference compound for the Hersherberger bioassay). Methyltestosterone undergoes aromatization and 5alpha-reduction, producing both estrogenic and potent androgenic metabolites [23].

Novelty: Low. The reference compound for oral androgen pharmacology. Developed in the 1930s.

Effect size: Moderate anabolic activity, but undissociated from androgenic effects. The absence of structural dissociation features results in a less favorable therapeutic index than CDMT or oxandrolone.

Promising potential: Low. Methyltestosterone retains marketing authorization in limited jurisdictions for male hypogonadism but has been largely replaced by transdermal and injectable testosterone formulations with more favorable pharmacokinetic and safety profiles.

Side-effect profile: Greater hepatotoxicity than CDMT at equianabolic doses. Full estrogenic liability (gynecomastia, water retention). Full androgenic liability (acne, hair loss, prostate effects). The most unfavorable therapeutic index in the oral AAS

class among commonly discussed compounds.

Overall validation: Moderate. Extensive use history (since the 1930s) and well-characterized pharmacology, but limited modern clinical trial data.

Comparison to CDMT: CDMT is superior to methyltestosterone on essentially all competency standards: greater anabolic dissociation, elimination of estrogenic liability, reduced androgenic effects, and a more favorable side-effect profile. Methyltestosterone serves primarily as the reference compound against which the improvements of CDMT and other structurally optimized AAS are measured.

12.6 Comparator summary

The five comparators illustrate the structural and pharmacological evolution of the oral AAS class. **Methyltestosterone** is the undissociated reference compound with the least favorable therapeutic index. **Clostebol** introduced the 4-chloro modification that prevents aromatization but lacks oral bioavailability. **Metandienone** achieved potent oral anabolic activity through 1,2-dehydro and 17alpha-methyl modifications but retained partial aromatizability and substantial androgenic load. **CDMT** combined the metandienone and clostebol innovations to produce a non-aromatizable, orally active anabolic with moderate potency and a favorable dissociation ratio. **Oxandrolone** achieves the greatest anabolic-to-androgenic dissociation and the most favorable safety profile through a fundamentally different structural approach (DHT backbone, lactone ring) and is the only comparator with retained regulatory approval. **Stanozolol** achieves high anabolic dissociation through a pyrazole fusion but at the cost of greater hepatotoxicity and lipid disturbance.

CDMT occupies a middle position in this landscape: more favorable than metandienone and methyltestosterone in safety and dissociation, less favorable than oxandrolone in both dimensions, and roughly comparable to stanozolol in anabolic effect but with a superior hepatic safety profile. The compound's unique historical position as the agent of State Plan Topic 14.25 provides an unparalleled (though ethically compromised) observational dataset on the performance-enhancing and adverse effects of chronic AAS administration. The discontinuation of clinical production and the absence of any current marketing authorization limit the practical research and clinical utility of CDMT relative to oxandrolone, which retains regulatory approval and an active clinical evidence base. Research applications for CDMT are principally in anti-doping analytical chemistry, steroid metabolism, and historical pharmacological analysis rather than in ongoing therapeutic investigation.

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