SOLIAN® 100mg, 200mg and 400mg

Amisulpride Scored Tablets

[Sanofi logo]

COMPOSITION

Solian 100 mg scored tablet
Amisulpride ……..……..... 100 mg,
Solian 200 mg scored tablet
Amisulpride ……..……..... 200 mg,
Solian 400 mg scored tablet
Amisulpride ……..……..... 400 mg

PHARMACEUTICAL FORM

Solian 100 mg scored tablet:
White to off-white scored tablet engraved "AMI 100".
Solian 200 mg scored tablet:
White to off-white scored tablet engraved "AMI 200".
Solian 400 mg scored film-coated tablet:
White scored film-coated tablet engraved "AMI 400".

CLINICAL PARTICULARS

THERAPEUTIC INDICATIONS

Treatment of schizophrenia, particularly acute or chronic schizophrenic disorders, characterised by positive symptoms (e.g. delirium, hallucinations, thought disorders) and/or negative symptoms (e.g. blunted emotions, emotional and social withdrawal), including when the negative symptoms predominate.

DOSAGE AND ADMINISTRATION

Usually, if the daily dose is ≤ 400 mg, it will be administered as a once-daily intake. If the daily dose exceeds 400 mg, it will be administered as two daily intakes.

Predominantly negative episodes
The recommended dosage is 50 to 300 mg/day. Dosage should be adjusted on an individual basis. The optimum dosage is about 100 mg/day.

Mixed episodes with positive and negative symptoms
At the beginning of treatment, the dosage should be that which enables the control of positive symptoms, i.e. 400 to 800 mg/day. The dosage should then be adjusted individually as a function of the patient's response, so as to obtain the minimum effective dose.

Acute psychotic episodes
At the beginning of treatment:
- it is possible to start via the IM route for a few days, at a maximum dose of 400 mg/day, replaced thereafter with oral treatment,
- the recommended dosage via the oral route is 400 to 800 mg; the maximum dosage should never exceed 1 200 mg.

Thereafter:
- the dosage should be maintained or adjusted as a function of the patient's response.

In all cases, the dosage of maintenance therapy should be established individually using the minimum effective dose.

Elderly subjects
Amisulpride should be used with particular caution in this patient population due to the risk of hypotension and sedation (see SPECIAL WARNINGS AND SPECIAL PRECAUTIONS FOR USE).

Renal insufficiency
Because amisulpride is excreted via the kidneys, the dosage should be reduced by half in patients with renal insufficiency whose creatinine clearance (CrCl) is between 30 and 60 ml/min, and to one third if the creatinine clearance is between 10 and 30 ml/min.

In the absence of relevant data on patients with serious renal insufficiency (CrCl < 10 ml/min), amisulpride is contraindicated (see CONTRAINDICATIONS).

Hepatic insufficiency
Amisulpride is poorly metabolised, so it is not necessary to reduce the dose in patients with hepatic insufficiency.

CONTRAINDICATIONS

This medicine MUST NOT BE USED in the following cases:
- Known hypersensitivity to amisulpride or another component of the product.
- Serious hypertensive accidents have been reported in patients with pheochromocytoma using anti-dopaminergic drugs, including some benzamides. It is therefore advisable to abstain from prescribing this product in known or suspected pheochromocytoma carriers.
- Children below the age of 15 years, because no clinical data are available on this age group
- Lactation
- Known or suspected prolactin-dependent tumour, e.g., prolactin-secreting pituitary adenoma and breast cancer.
- Severe renal insufficiency (CrCl < 10 ml/min).In combination with :
  - non-antiparkinsonian dopamine agonists (cabergoline, quinagolide),
  - Sultopride
  - citalopram, escitalopram, domperidone, hydroxyzine, piperaquine

SPECIAL WARNINGS AND SPECIAL PRECAUTIONS FOR USE

Special Warnings

Neuroleptic malignant syndrome
As with other neuroleptics, the onset of malignant syndrome (hyperthermia, muscle rigidity, neurovegetative disorders, impaired consciousness, CPK elevation) is possible and potentially fatal. In the event of hyperthermia and particularly in the context of high daily doses, any drug therapy for psychosis must be discontinued.

Prolongation of the QT interval
Amisulpride prolongs the QT interval in a dose-dependent manner. This effect, which is known to potentiate the risk of onset of serious ventricular arrhythmia, such as torsades de pointes, is increased by the existence of bradycardia, hypokalaemia, or a congenital or acquired long QT interval (combination with a drug increasing the QTc interval).

If the clinical status so permits, it is advisable prior to any administration to ensure the absence of any factors which might favour the onset of this type of arrhythmia:
- Bradycardia slower than 55 bpm,
- Hypokalaemia,
- Congenital prolongation of the QT interval,
- Current treatment with a drug likely to cause marked bradycardia (< 55 bpm), hypokalaemia, a slowing of intra-cardiac conduction or prolongation of the QTc interval.

An ECG should be performed as part of the initial assessment of patients requiring long-term treatment with a neuroleptic.

Stroke
In randomized, placebo-controlled clinical studies in elderly patients with dementia and treated with certain atypical antipsychotic agents, a three-fold risk of stroke was observed versus placebo. The mechanism underlying this increased risk is unknown. Increased risk with other antipsychotic agents or in other patient populations cannot be ruled out. This medicinal product must be used with caution in patients with risk factors for stroke.

Elderly patients with dementia
Elderly patients with dementia-related psychosis treated with antipsychotic drugs are at an increased risk of death. Analyses of seventeen placebo-controlled trials (modal duration of 10 weeks), largely in patients taking atypical antipsychotic drugs, revealed a risk of death in drug-treated patients of between 1.6 to 1.7 times the risk of death in placebo-treated patients. Although the causes of death were varied, most of the deaths appeared to be either cardiovascular (e.g., heart failure, sudden death) or infectious (e.g., pneumonia) in nature. Observational studies suggest that, similar to atypical antipsychotic drugs, treatment with conventional antipsychotic drugs may increase mortality. The extent to which the findings of increased mortality in observational studies may be attributed to the antipsychotic drug as opposed to some characteristic(s) of the patients is not clear.

Venous thromboembolism
Cases of venous thromboembolism (VTE) have been reported with antipsychotic drugs. Since patients treated with antipsychotic drugs often present acquired risk factors for VTE, any potential risk factors for VTE must be identified before and during treatment with **Solian** and preventive measures should be taken if needed (see UNDESIRABLE EFFECTS).

Benign pituitary tumour
Amisulpride may increase prolactin levels. Cases of benign pituitary tumours such as prolactinoma have been observed during amisulpride therapy. (See Section 11) In case of very high levels of prolactin or clinical signs of pituitary tumour (such as visual field defect and headache), pituitary imaging should be performed. If the diagnosis of pituitary tumour is confirmed, the treatment with amisulpride must be stopped.

Hyperglycemia/metabolic syndrome
Increased risk of hyperglycemia or glucose intolerance and onset or exacerbation of diabetes have been reported in patients treated with some antipsychotic drugs, including amisulpride (see UNDESIRABLE EFFECTS).
Clinical and laboratory monitoring should be performed in patients receiving treatment with Solian in compliance with current recommendations. Particular caution should be exercised in patients with diabetes mellitus or with risk factors for diabetes.

Seizure
Amisulpride can lower the seizure threshold. Therefore patients with a history of seizures should be closely monitored during treatment with **Solian**.

Special populations
As amisulpride is eliminated by the renal route, the dose should be decreased or an alternative treatment considered in patients with renal insufficiency (see DOSAGE AND ADMINISTRATION). There are no data concerning patients with serious renal insufficiency (see DOSAGE AND ADMINISTRATION)
Amisulpride, like all antipsychotics, should be used with particular caution in elderly patients due to the potential risk of sedation and hypotension.
Amisulpride, like all antidopaminergic drugs, should be used with caution in patients with Parkinson's disease due to the risk of worsening disease. Amisulpride should be used only if neuroleptic treatment is absolutely necessary.

Withdrawal syndrome
Withdrawal symptoms including nausea, vomiting and insomnia have been described following sudden discontinuation of high doses of antipsychotics. Involuntary movements (e.g. akathisia, dystonia and dyskinesia) have been reported with amisulpride. It is therefore advisable to discontinue amisulpride treatment gradually.
Leukopenia, neutropenia and agranulocytosis have been reported with antipsychotics, including Solian. Unexplained infections or fever may be evidence of blood dyscrasia (see UNDESIRABLE EFFECTS), and requires immediate haematological investigation.

Hyperprolactinemia
Amisulpride may increase prolactin levels (see section 4.8). Patients with a history of hyperprolactinemia or of a potentially prolactin-dependent tumor should be closely monitored during amisulpride treatment

Other
Leukopenia, neutropenia and agranulocytosis have been reported with antipsychotics, including Solian. Unexplained infections or fever may be evidence of leukopenia and require immediate hematological investigation.
Use of this drug is not recommended in combination with alcohol, dopaminergic antiparkinsonian drugs, antiparasitics likely to induce torsades de pointes, methadone, morphine derivatives, levodopa and other neuroleptics and drugs likely to induce torsades de pointes, sodium oxybate and hydroxychloroquine

INTERACTION WITH OTHER MEDICINAL PRODUCTS AND OTHER FORMS OF INTERACTION

+ Sedative drugs
Many drugs or substances can have additive depressant effects on the central nervous system and contribute to a decrease in alertness. This must be taken into account for patients using amisulpride. These drugs/substances include morphine derivatives (analgesics, cough suppressants and substitute treatments), neuroleptics, barbiturates, benzodiazepines, anxiolytics other than benzodiazepines (e.g. meprobamate), hypnotics, sedative antidepressants (amitriptyline, doxepin, mianserin, mirtazapine, trimipramine), sedative H1 antihistamines, centrally acting antihypertensive agents, baclofen and thalidomide.

+ Drugs likely to induce torsades de pointes
This serious cardiac rhythm disorder can be caused by a number of antiarrhythmic and non-antiarrhythmic drugs. Hypokalemia (see Potassium-depleting agents) is a promoting factor, as is bradycardia (see Bradycardia-inducing drugs) or pre-existing congenital or acquired QT interval prolongation. Medicines likely to cause this adverse effect include class Ia and III antiarrhythmics and certain neuroleptics.
Other agents not belonging to these classes are also involved.
For dolasetron, erythromycin, spiramycin, and vincamine, only intravenously administered forms are concerned by this interaction.
Coadministration of two torsadogenic drugs is generally contraindicated.
However, some of these drugs, as their use is unavoidable, are exceptions, and are merely not recommended in combination with other torsadogenic drugs: methadone, hydroxychloroquine, antiparasitic agents (chloroquine, halofantrine, lumefantrine, pentamidine), neuroleptics.
However, citalopram, escitalopram, domperidone, hydroxyzine and piperaquine are not among these exceptions, and are therefore contraindicated when coadministered with all torsadogenic drugs.

Contraindicated combinations
+ **Non-antiparkinsonian dopamine agonists (cabergoline, quinagolide)**
There is mutual antagonism between dopamine agonists and neuroleptics.

+ **Citalopram, escitalopram, domperidone, hydroxyzine, piperaquine**
There is an increased risk of ventricular arrhythmias, especially torsades de pointes

Inadvisable associations

+ Antiparasitics likely to induce torsades de pointes (chloroquine, halofantrine, lumefantrine, pentamidine)
There is an increased risk of ventricular arrhythmias, particularly torsades de pointes.
If possible, one of the two treatments should be discontinued.
If coadministration cannot be avoided, a preliminary QT examination should be carried out and ECG monitoring performed.

+ Antiparkinsonian dopamine agonists (amantadine, apomorphine, bromocriptine, entacapone, lisuride, pergolide, piribedil, pramipexole, rasagiline, ropinirole, rotigotine, selegiline, tolcapone)
There is mutual antagonism between dopamine agonists and neuroleptics.
Dopamine agonists can cause or worsen psychotic disorders. If treatment with neuroleptics is required in patients with Parkinson's disease treated with dopamine agonists, these dopamine agents should be tapered off gradually (sudden discontinuation exposes the patient to a risk of "neuroleptic malignant syndrome").

+ Other drugs likely to induce torsades de pointes: class Ia antiarrhythmics (quinidine, hydroquinidine, disopyramide) and class III antiarrhythmics (amiodarone, dronedarone, sotalol, dofetilide, ibutilide), and other drugs such as arsenic compounds, diphemanil, dolasetron IV, erythromycin IV, levofloxacin, mequitazine, mizolastine, prucalopride, vincamine IV, moxifloxacin, spiramycin IV, toremifene, vandetanib
There is an increased risk of ventricular arrhythmias, particularly torsades de pointes.

+ Other neuroleptics likely to induce torsades de pointes (chlorpromazine, cyamemazine, droperidol, flupenthixol, fluphenazine, haloperidol, levomepromazine, pimozide, pipamperone, pipotiazine, sulpiride, sultopride, tiapride, zuclopenthixol)
There is an increased risk of ventricular arrhythmias, particularly torsades de pointes.

+ Alcohol: Alcohol enhances the sedative effect of neuroleptics. Impaired alertness may make it dangerous to drive or use machinery. Alcoholic drinks and medicines containing alcohol should be avoided during treatment
+ Levodopa
There is mutual antagonism between levodopa and neuroleptics.
In patients with Parkinson's disease, minimum effective doses of each of these drugs should be used.

+ Methadone
There is an increased risk of ventricular arrhythmias, particularly torsades de pointes.

+ Sodium oxybate
The central nervous depressant effect is potentiated. Impaired alertness may make driving vehicles and using machines dangerous.

+ Hydroxychloroquine
There is an increased risk of ventricular arrhythmias, especially torsades de pointes

Associations requiring precautions for use
+ **Anagrelide**
There is an increased risk of ventricular arrhythmias, particularly torsades de pointes.
When coadministering these agents, clinical and ECG monitoring are required

+ **Azithromycin, ciprofloxacin, clarithromycin, levofloxacin, norfloxacin, roxithromycin**
There is an increased risk of ventricular arrhythmias, particularly torsades de pointes.
When coadministering these agents, clinical and ECG monitoring are required.

+ **Beta-blockers in heart failure (bisoprolol, carvedilol, metoprolol, nebivolol)**
There is an increased risk of ventricular arrhythmias, particularly torsades de pointes.

In addition, there is a vasodilator effect and risk of hypotension, particularly postural (additive effect).
Clinical and ECG monitoring are required.

+ **Bradycardia-inducing drugs (in particular class IA antiarrhythmics, beta-blockers, certain class III antiarrhythmics, certain calcium channel blockers, digitalis glycosides, pilocarpine, anticholinesterases)**
There is an increased risk of ventricular arrhythmias, particularly torsades de pointes. Clinical and ECG monitoring are required.

+ **Potassium-depleting agents (potassium-depleting diuretics, alone or in combination, stimulant laxatives, glucocorticoids, tetracosactides and amphotericin B IV)**
There is an increased risk of ventricular arrhythmias, particularly torsades de pointes.
Any existing hypokalemia should be corrected before administration, and clinical, electrolyte and ECG monitoring implemented.

+ **Lithium**
There is a risk of neuropsychiatric signs suggestive of neuroleptic malignant syndrome or lithium poisoning. Regular clinical and laboratory monitoring are required, particularly at the start of coadministration.

+ **Ondansetron**
There is an increased risk of ventricular arrhythmias, particularly torsades de pointes.
When coadministering these agents, clinical and ECG monitoring are required.

Associations to be taken into consideration
+ Antihypertensive drugs (all): Antihypertensive effect and increased risk of orthostatic hypotension (additive effect).
+ **Other sedative drugs**
The central nervous depressant effect is potentiated.
Impaired alertness may make driving vehicles and using machines dangerous.

+ **Orlistat**
There is a risk of treatment failure when the drug is coadministered with orlistat.

PREGNANCY AND LACTATION

Pregnancy
Animal studies do not indicate direct or indirect harmful effects with respect to teratogenicity and embryofetotoxicity. Animal studies are insufficient with respect to neurodevelopmental disorders in pups.

Amisulpride crosses the placenta

Very limited clinical data on exposed pregnancies are available. Therefore, the safety of amisulpride during human pregnancy has not been established. The use of amisulpride is not recommended during pregnancy and in women of child bearing potential not using effective contraception, unless the benefits justify the potential risks.

Neonates exposed to antipsychotic drugs during the third trimester of pregnancy are at risk for extrapyramidal and/or withdrawal symptoms following delivery (see UNDESIRABLE EFFECTS). There have been reports of agitation, hypertonia, hypotonia, tremor, somnolence, respiratory distress and feeding disorder in these neonates. These complications have varied in severity; while in some cases symptoms have been self-limited, in other cases neonates have required intensive care unit support and prolonged hospitalisation.

Consequently, as a precautionary measure, it is preferable to avoid taking this medicinal product during pregnancy.

Fertility
A decrease in fertility linked to the pharmacological effects of the drug (prolactin-mediated effect) was observed in treated animals.

Lactation
Breast-feeding is not recommended during treatment.

EFFECTS ON ABILITY TO DRIVE AND USE MACHINES

Even when used as recommended, amisulpride may cause somnolence and blurred vision so that the ability to drive vehicles or operate machinery can be impaired.

UNDESIRABLE EFFECTS

Undesirable effects have been ranked by incidence using the following convention: very common ≥ 1/10; common ≥ 1/100, < 1/10; uncommon ≥ 1/1 000, < 1/100; rare ≥ 1/10 000, < 1/1 000; very rare < 1/10 000; frequency not known (cannot be estimated from the available data).

Blood and lymphatic system disorders
Uncommon
Leukopenia, neutropenia

Rare
Agranulocytosis (see section 4.4).

Immune system disorders
Uncommon
Allergic reactions.

Endocrine disorders
Common
Increase in plasma prolactin levels which is reversible on treatment discontinuation. This may result in the following clinical signs and symptoms: galactorrhea, amenorrhea, gynecomastia, breast tenderness, erectile dysfunction.
Rare
Benign pituitary tumor, such as prolactinoma

Metabolism and nutrition disorders
Uncommon
Hyperglycemia, hypertriglyceridemia and hypercholesterolemia.

Rare
Hyponatremia, syndrome of inappropriate antidiuretic hormone secretion (SIADH).

Psychiatric disorders
Common
Insomnia, anxiety, agitation, frigidity.

Uncommon
Confusion.

Nervous system disorders
Very common
Etrapyramidal symptoms (tremor, hypertonia, hypersalivation, akathisia, hypokinesia, dyskinesia) may occur. These symptoms are generally moderate at optimal doses and partially reversible with administration of anticholinergic antiparkinsonian medication. Discontinuation of amisulpride treatment is not required
The incidence of extrapyramidal symptoms, which are dose-dependent, is very low in patients being treated for predominantly negative symptoms at doses of 50 to 300 mg/day.

Common
Acute dystonia (spasmodic torticollis, oculogyric crises, trismus, etc.) may appear. This is reversible with administration of an anticholinergic antiparkinsonian agent. Discontinuation of amisulpride treatment is not required.
Drowsiness.

Uncommon
Tardive dyskinesia, characterized by involuntary movements of the tongue and/or face, has been reported, particularly after long-term administration.
Anticholinergic antiparkinsonians have no effect and may cause exacerbation.
Seizures.

Rare
Potentially fatal neuroleptic malignant syndrome

Not known
Restless legs syndrome with or without a context of akathisia.

Eye disorders
Common
Blurred vision

Cardiac disorders

Uncommon
Bradycardia.

Rare
QT interval prolongation, ventricular arrhythmias such as torsades de pointes, ventricular tachycardia, which may result in ventricular fibrillation or cardiac arrest and sudden death

Vascular disorders
Common
Hypotension.

Uncommon
Rise in blood pressure.

Rare
Cases of venous thromboembolism, including cases of pulmonary embolism, sometimes fatal, and deep vein thrombosis, have been reported with antipsychotic drugs

Respiratory, thoracic and mediastinal disorders
Uncommon
Nasal congestion, inhalation pneumonia (mainly in combination with other antipsychotic agents and drugs with a central nervous depressant effect).

Gastrointestinal disorders
Common
Constipation, nausea, vomiting, dry mouth.

Hepatobiliary disorder
Uncommon
hepatocellular injury

Skin and subcutaneous tissue disorders
Rare
Angioedema, urticaria.

Not known
Photosensitivity reaction

Musculoskeletal and systemic disorders
Uncommon
Osteopenia, osteoporosis.

Renal and urinary disorders
Uncommon
Urinary retention.

Pregnancy, puerperium and perinatal conditions
Frequency not known
Neonatal withdrawal syndrome (see section 4.6).

Investigations
Common
Weight gain.

Uncommon
Elevated liver enzymes, mainly transaminases.

OVERDOSE
At present, data concerning acute overdose with **Solian** are limited. The signs and symptoms reported result generally from an increase in pharmacological effects of the medicinal product, and the clinical picture includes: drowsiness, sedation, coma, hypotension and extrapyramidal symptoms.

Cases with a fatal outcome have been reported mainly in combination with other antipsychotic drugs.

There is no known specific antidote to amisulpride. In the event of acute overdose, the combination with other products must be investigated and appropriate measures taken:
- Close monitoring of vital signs.
- Cardiac monitoring (risk of lengthening of QT interval) until the patient recovers.
- In the event of severe extrapyramidal symptoms, anticholinergic treatment must be administered.
- Amisulpride is poorly dialysable, so it is not effectively eliminated by haemodialysis.

PHARMACOLOGICAL PROPERTIES

Pharmacodynamic properties
Antipsychotic
ATC code: N05AL05

Amisulpride is an antipsychotic drug belonging to the class of substituted benzamides.

Its pharmacodynamic profile is characterised by selective and predominant affinity for the D2 and D3 dopaminergic receptors of the limbic system. Amisulpride has no affinity for serotoninergic receptors or other neuroreceptors such as histaminic, cholinergic and adrenergic receptors.

At high doses, in animal studies, amisulpride preferentially blocks the dopaminergic neurones of the mesolimbic system compared with those of the striatal system. This specific affinity could explain the predominant antipsychotic effects of amisulpride compared with its extrapyramidal effects.

At low doses, amisulpride preferentially blocks the presynaptic D2/D3 dopaminergic receptors, which could explain its action on negative symptoms.

In a controlled, double-blind study versus haloperidol in 191 patients presenting with acute schizophrenia, amisulpride significantly improved secondary negative symptoms in comparison with haloperidol.

Pharmacokinetic properties
In man, amisulpride exhibits two absorption peaks: the first is reached rapidly one hour after dosing and the second occurs three to four hours after administration.

The corresponding plasma levels are 39 ± 3 and 54 ± 4 ng/ml, respectively, following the administration of a 50 mg dose.

The distribution volume is 5.8 l/kg. Plasma protein binding is low (16%), and does not suggest any drug interactions at this level. Absolute bioavailability is 48%.

Amisulpride is weakly metabolised: two inactive metabolites have been identified and represent 4% of the total quantity eliminated. Following repeated doses, amisulpride does not accumulate and the pharmacokinetic parameters remain unchanged.

The elimination half-life is approximately 12 hours after oral administration.

Amisulpride is eliminated in the urine in unchanged form. Half (50%) of the IV dose is eliminated in the urine, usually over the first 24 hours (90% of urinary excretion).

Renal clearance is about 330 ml/min. A high-carbohydrate meal significantly lowers the AUC, T_{max} and C_{max} values for amisulpride, while a high-fat meal does not affect these parameters. The effect of these results during treatment with amisulpride is not known.

Hepatic insufficiency
Amisulpride is poorly metabolised, so it is not necessary to reduce dosage in patients with hepatic insufficiency.

Renal insufficiency
The elimination half-life is not modified in patients with renal insufficiency whilst total clearance is reduced by a factor of 2.5 to 3. The AUC of amisulpride is doubled in patients with mild renal insufficiency and is nearly 10 times greater in patients with moderate renal insufficiency. However, experience remains limited and there are few data available for doses exceeding 50 mg. Amisulpride is poorly dialysable.

Elderly subjects
The pharmacokinetic data available for elderly subjects aged above 65 years show an increase of between 10 to 30% for C_{max}, $T_{1/2}$ and AUC after a single 50-mg dose.
No data are available for repeated doses

PRECLINICAL SAFETY DATA

The toxicological profile of amisulpride is dominated by the pharmacological effects of the compound. Toxicity studies after repeated administrations showed no target organ impairment. The compound has no teratogenic or genotoxic effects.
Carcinogenesis studies have demonstrated hormone-dependent tumours in rodents. These are not of any clinical relevance in man.

Decreased fertility related to the pharmacological properties of the product (prolactinmediated effects) was observed in animals.

PHARMACEUTICAL PARTICULARS

Shelf life
3 years

Nature and contents of container
15, 30 or 150 scored tablets in blisters (PVC/aluminium).
- Not all pack sizes are available in the local market

Manufactured by:
Delpharm Dijon
6 Boulevard de l'Europe 21800 Quetigny
FRANCE

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References: CCDS v13