

## Caprolactam; CASRN 105-60-2

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the [IRIS assessment development process](#). Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the [guidance documents located on the IRIS website](#).

### STATUS OF DATA FOR Caprolactam

**File First On-Line 09/07/1988**

| Category (section)                      | Assessment Available?  | Last Revised |
|---|------------------------|--------------|
| <b>Oral RfD (I.A.)</b>                  | yes                    | 09/07/1988   |
| <b>Inhalation RfC (I.B.)</b>            | qualitative discussion | 09/01/1994   |
| <b>Carcinogenicity Assessment (II.)</b> | not evaluated          |              |

### I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

#### I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Caprolactam

CASRN — 105-60-2

Last Revised — 09/07/1988

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of

information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

### I.A.1. Oral RfD Summary

| Critical Effect                                     | Experimental Doses*                        | UF  | MF | RfD            |
|---|--|-----|----|----------------|
| <b>Reduced offspring body weight</b>                | NOAEL: 1000 ppm of diet<br>(50 mg/kg/day)  | 100 | 1  | 5E-1 mg/kg/day |
| <b>Rat Oral Three Generation Reproduction Study</b> | LOAEL: 5000 ppm of diet<br>(250 mg/kg/day) |     |    |                |
| <b>Serotta et al, 1984</b>                          |  |     |    |                |

\*Conversion Factor: Assumed food consumption equivalent to 5% of body weight/day.

### I.A.2. Principal and Supporting Studies (Oral RfD)

Serota, C.G., A.M. Hoberman and S.C. Gad. 1984. A three-generation reproduction study with caprolactam in rats. In: Proc. Symp. Ind. Approach Chem. Risk Assess.: Caprolactam Relat. Compd. Case Study. Ind. Health Found., Pittsburgh, PA. p. 191-204.

In this study, groups of 10 male and 20 female F344 rats were fed diets containing caprolactam at 0, 1000, 5000 or 10,000 ppm for three-generations. Mean body weights and food consumption were reduced in both parental generations at 5000 and 10,000 ppm. Body weights of offspring were also reduced at these dietary concentrations. A slight increase in the severity of spontaneous nephropathy was observed on histopathologic examination of males in the high-dose group of the first parental generation. No adverse effects were noted at 1000 ppm (50 mg/kg/day), which was chosen as the NOAEL to serve as the basis for the RfD. Application of an uncertainty factor of 100 resulted in an RfD of 0.5 mg/kg/day or 35 mg/day for a 70 kg human.

### I.A.3. Uncertainty and Modifying Factors (Oral RfD)

UF — The UF of 100 includes 10 for uncertainty in the extrapolation of dose levels for animals to humans, and 10 for uncertainty in the threshold for sensitive humans.

MF — None

#### **I.A.4. Additional Studies/Comments (Oral RfD)**

Rat and mouse chronic feeding bioassay: rat doses 0, 3750 and 7500 ppm; mouse doses 0, 7500 and 15000 ppm; LOAEL for rat is 125 mg/kg/day and LOAEL for mouse is 650 mg/kg/day for body weight depression (NTP, 1982).

Rat developmental study: oral gavage doses 0, 100, 500, 1000 mg/kg/day on gestation days 6-20; 1000 mg/kg/day is LOAEL for fetal resorption and 500 mg/kg/day NOAEL (Gad et al., 1984).

Rabbit developmental study: oral gavage doses 0, 50, 150, 250 mg/kg/day on gestation days 6-28; 150 mg/kg/day is LOAEL for maternal and fetal body weight depression (Gad et al., 1984).

90-day rat feeding study, three strains of male rats: doses 0, 0.01, 0.05, 0.1 and 0.5%; 0.5% (250 mg/kg/day) is LOAEL for slightly increased BUN and NOAEL at 0.1% (50 mg/kg/day) (Powers et al., 1984).

#### **I.A.5. Confidence in the Oral RfD**

Study — High

Database — High

RfD — High

Confidence in the study is high because the threshold for the most sensitive reproductive effect, reduced body weight of offspring, was clearly identified. Confidence in the database is high because subchronic and chronic dietary studies identified no effect levels for kidney effects in rats, another critical effect in the most sensitive species. The carcinogenicity, developmental and reproductive toxicity have been adequately studied. Therefore, confidence in the RfD is high.

#### **I.A.6. EPA Documentation and Review of the Oral RfD**

Source Document — U.S. EPA, 1987

Limited peer review and extensive Agency-wide review, 1987.

Other EPA Documentation — None

Agency Work Group Review — 09/17/1987, 03/24/1988

Verification Date — 03/24/1988

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfD for Caprolactam conducted in November 2001 identified one or more significant new studies. IRIS users may request the references for those studies from the IRIS Hotline at [hotline.iris@epa.gov](mailto:hotline.iris@epa.gov) or (202)566-1676.

#### **I.A.7. EPA Contacts (Oral RfD)**

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or [hotline.iris@epa.gov](mailto:hotline.iris@epa.gov) (internet address).

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#### **I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)**

Substance Name — Caprolactam  
CASRN — 105-60-2

The health effects data for caprolactam were reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for the derivation of an inhalation RfC. The verification status for this chemical is currently NOT VERIFIABLE. For additional information on the health effects of this chemical, interested parties are referred to the U.S. EPA documentation listed below.

NOT VERIFIABLE status indicates that the U.S. EPA RfD/RfC Work Group deemed the database at the time of review to be insufficient to derive an inhalation RfC according to the Interim Methods for Development of Inhalation Reference Concentrations (U.S. EPA, 1990). This status does not preclude the use of information in cited references for assessment by others.

Derivation of an inhalation RfC for caprolactam is not recommended at this time. Because no adequate long-term studies examining the effects of inhalation exposure to caprolactam exist, the requirements for a minimal database have not been met (U.S. EPA, 1990). Caprolactam is a respiratory tract irritant (ACGIH, 1991; U.S. EPA, 1988), and no data exist to definitively rule out portal-of-entry effects associated with long-term chronic inhalation exposure. No inhalation pharmacokinetic data exist for this compound.

Caprolactam is a highly water soluble, hygroscopic powder with a vapor pressure sufficient to allow it to exist in the air as a vapor at up to 13 mg/cu.m at 25 C and 29 mg/cu.m at 35 C. Unless

otherwise indicated, the discussions in this document regarding caprolactam toxicity are for caprolactam vapor, not the dust.

Ferguson and Wheeler (1973) present retrospective information on occupational exposures to caprolactam. Little actual data on workers is presented in this study, although some corroborative documentation on this study (Ferguson, 1972) shows that medical records of 155 workers (several of whom apparently had worked in the plants for more than 17 years) in two plants were examined for possible indications of reported health effects due to caprolactam over a period of 17 years prior to the study. The search yielded only three cases of skin irritation, all relating to direct exposure to caprolactam. TWA exposures of workers, based on area samples, in various plant locations ranged up to 4.8 ppm (23 mg/cu.m). As an experiment, effects of caprolactam were investigated in five volunteer workers who were stationed at various distances from a caprolactam source, and their subjective complaints were noted after momentary exposure. These workers had not been exposed continuously in their work duties. Irritation associated with caprolactam exposure was characterized as transient, ceasing promptly after termination of exposure. The authors claim a concentration response for these effects because transient irritant effects of the eyes, nose, and throat were noted in workers at 100 ppm with distress decreasing as concentration decreased such that no eye irritation was noted below 25 ppm. The majority (4/5) experienced upper airway irritation at 10 ppm (46 mg/cu.m) caprolactam, the lowest concentration tested. Transient nose and throat irritation occurred in some subjects at all levels above 10 ppm, and no distress was noted at concentrations ranging up to about 7 ppm. The authors claim that discomfort from irritation generally was absent in workers located where these lower exposures (<7 ppm) were measured. Reported complaints varied greatly with respect to perceived magnitude of discomfort, however. Somewhat higher discomfort levels were reported from subjects in the polymer plant versus those in the monomer plant. The authors suggest that the higher humidity in the monomer plant may have protected those subjects from the irritant effects of caprolactam by ensuring tissue hydration and biological clearance.

Both components of the study by Ferguson and Wheeler (1973) have significant deficiencies. Neither the number of workers nor the average duration or distribution of exposure are given in the occupational portion of the study. No historical air levels are given, and all exposures are determined from area rather than personal samplers. No attempt to reconstruct individual exposure histories was made. The relationship of some of the supporting data on worker medical and employment records (Ferguson, 1972) to the formal study is not linked clearly. No firm basis exists for consideration of this study as chronic in duration because no definite worker population is present and no average exposure duration is reported. Although judgment about the onset and intensity of irritation is known to have substantial subjective components (OSHA, 1989), only five individuals were used in the irritation portion of the study and no concentration was examined at which irritation was absent in all. The animal-based evaluation for sensory irritation (Alarie, 1973) provides a more objective measure, although this methodology recently

has been criticized (Bos et al., 1992). These deficiencies preclude the use of this study in the derivation of an RfC.

Billmaier et al. (1992) examined medical records of 39 workers from two plants for health effects related to caprolactam exposure. Each of the exposed workers was matched for age, sex, race, and smoking history with at least one control who had not been exposed to caprolactam. Worker selection was also based on a minimum of 10 years work exposure (mean 18.7 +/- 7.0, range 8.2-31.7 years) and the existence of pulmonary function test data for this period. Pulmonary function test data consisted of expiratory spirographs from which forced vital capacity, forced expiratory volume in one second, and other values related to these measures were obtained. Other health record information, available for all shifts from 1980-1991, was examined for worker complaints associated with exposure to caprolactam. Historical industrial hygiene monitoring of one plant indicated caprolactam air concentrations to be 3.7 mg/cu.m and for the areas of highest exposure in the other plant, 4.5 and 9.9 mg/cu.m. No personal air samples were taken. In comparing exposed workers and their controls, no statistically significant alterations were noted in any pulmonary function test. Possible detection of a smoking effect indicates that the study may have been sensitive enough to detect pulmonary obstruction. Of 878 worker visits to the medical clinic from 1980-1991, two could be related to direct dermal contact with solid caprolactam, and there was one episode of eye irritation and one episode of inhalation of material possibly containing caprolactam. Although the results from this study suggest that prolonged inhalation exposure to caprolactam vapors at concentrations as high as 9.9 mg/cu.m are without adverse consequence for the lower respiratory tract, the small sample size precludes further conclusions. Furthermore, it appears that the spirometry performed and evaluated in this study was not in accordance with current guidelines (ATS, 1987) and quality assurance procedures (Gardner et al., 1986). Upper respiratory tract symptomatology was not investigated in this report.

Spirometry was performed on 173 caprolactam plant workers who had an average exposure of 12 years (Patel, 1990). After adjusting for age, height, and smoking habits, no differences were noted between the exposed cohort and 60 nonexposed workers. No exposure information was given in this study.

Guirguis (1990) reports on a case control study of six workers who developed respiratory problems within a year after being exposed to a mixture of emissions of which one was caprolactam at 0.46 mg/cu.m. The respiratory problems (including symptoms of bronchial hyperreactivity, asthmatic responses, and deficits in pulmonary functions tests) were preceded by eye, nasal, and upper respiratory tract irritation.

Information on human exposure to caprolactam dust is limited. Kelman (1986) reported that workers exposed to caprolactam dust/vapor at 68-84 mg/cu.m for an average of 4.8 years showed

evidence of dermal damage but not systemic toxicity. Hohensee (1951) states that worker complaints at the end of an 8-hour work shift where caprolactam vapor/dust was claimed to be present at 61 mg/cu.m included irritability, nervousness, nosebleeds, irritation/inflammation of the upper respiratory passages, dry nose, abdominal gas, and heartburn. Ferguson and Wheeler (1973) reported that exposure to caprolactam dust produces skin irritation, although no concentrations are given. OSHA and ACGIH both promulgate a TLV-TWA for caprolactam dust of 1 mg/cu.m for avoidance of dermal irritation in workers, this value being 20-fold less than for the vapor.

Male albino rats (15/group) were exposed either to air or to 0.06, 0.6, or 6.0 mg/cu.m caprolactam for 82 days (Krichevskaya, 1968). Information regarding length of daily exposure is not given. Alterations in whole blood cholinesterase, "chronaxial ratio," and several biochemical measures were claimed to occur in rats exposed to the highest concentration. Effects on cerebral electrical activity in three volunteers also were claimed at concentrations as low as 0.11 mg/cu.m. Little data is available in this report, and the significance of the results is unclear.

Albino mice (sex not specified) were exposed either to air or to 10 mg/cu.m caprolactam for 4 hours/day for 4 months. Body weights were monitored, various behavioral tests and some pathology were performed. Little data is presented in this study. No effects attributable to caprolactam were reported (Lomonova, 1966).

Alarie and Stock (1990) exposed guinea pigs (groups of four) for 0.5 hours on 5 consecutive days to air or to 3, 10, or 30 mg/cu.m aerosols generated from a 15% aqueous solution of caprolactam. Animals were monitored with whole-body plethysmography for indications of irritation, coughing, pulmonary hypersensitivity, and airway hyperreactivity. No significant respiratory responses were noted during the 5-day repeated exposure period, including sensory or pulmonary irritation, even at the highest concentration. No tissues were examined in this study.

Three successive generations of Fischer 344 rats (20 females and 10 males/generation) were mated after a 10-week dietary exposure to 0, 1000, 5000, or 10,000 ppm (500 mg/kg/day) caprolactam (Serota et al., 1984, 1988). The number of live and dead pups was noted for each litter, with individual body weights and any abnormalities noted on days 1, 7, and 21 of lactation. In pups chosen as parents for the following generations, all reproduction indices were noted. Lower mean body weights (accompanied by concomitant decreases in food consumption) were observed in the P2 and P3 generations of both sexes treated at the highest dose level. All pregnancy and fertility indices were unaffected by caprolactam treatment. There was a consistently lower mean body weight in both female and male pups in all filial generations at both the 10,000 ppm and 5000 ppm (500 and 250 mg/kg/day, respectively), but not at the 1000-ppm dose level (50 mg/kg/day). The results in this study were used in the derivation of the current RfD for caprolactam, 0.5 mg/kg/day.

Pregnant Fischer 344 rats (20/dose group) were intubated with caprolactam at 0, 100, 500, or 1000 mg/kg/day on gestation days 6-15. Dams in the highest dose group experienced mortality (>50%). The mean body weight changes (and food consumption) in the two highest dose groups were less ( $p = 0.05$ ) than controls and the lowest dose group on days 6-11. The mean incidence of resorption in the highest dose group was nearly 10-fold higher in the controls and all other dose groups. No dose-related malformations or anomalies were noted among the offspring of any exposure group, although skeletal variants (including incomplete ossification of the skull or vertebral column, and the presence of extra ribs) were markedly increased among offspring from animals exposed to the highest dose (Gad et al., 1987). This study identifies a NOAEL of 100 mg/kg/day for developmental effects in rats and an FEL of 1000 mg/kg/day.

Pregnant New Zealand white rabbits (25/group) were intubated with water or 50, 150, or 250 mg/kg caprolactam/day on gestation days 6-28. Mortality was observed (4/25) in the highest dose group only, and maternal body weight gain was significantly depressed ( $p < 0.05$ ) on gestation days 6-9. Lower mean fetal weights ( $p < 0.05$ ) were noted among fetuses in the two highest dose groups. An increased incidence of unilateral or bilateral thirteenth ribs was noted among fetuses whose mothers had been exposed to the highest concentration of caprolactam (Gad et al., 1987). A NOAEL of 50 mg/kg is identified by this study, with 250 mg/kg being an FEL.

A number of limited studies exist on reproductive effects in both humans and animals from inhalation of caprolactam vapors/dust. The human studies, many of which have been reviewed by Gross (1984), are confounded due mostly to coexposures. The studies do, however, report effects that are internally consistent with one another and with the animal studies. The numerous deficiencies in these studies in reporting, data presentation, and methods preclude their use in a concentration-response assessment. They do, however, indicate an area of uncertainty on reproductive endpoints (ovarian-menstrual functions and male gonadal parameters) that were not evaluated specifically in the long-term oral studies of NTP (1982) or the three-generation reproductive study of Serota et al. (1988). These studies are described briefly below.

The human occupational studies in which female workers were exposed to caprolactam vapors/dusts consistently report alterations in ovarian-menstrual functions and condition. Nadezhdina and Talakina (1971) also reported in Livke et al., 1971) report unspecified disturbances in ovarian menstrual function occurring in 37.1% of 170 pregnant workers exposed to caprolactam (no levels given) versus 12.8% in a control population of 101 pregnant women. Petrov (1975) reported that inflammatory diseases of the uterus and "uterine appendages" were more prevalent in a female worker population ( $n = 492$ ) exposed to <10 mg/cu.m caprolactam and biphenyl than in a control population (8.9% versus 1.08%). Martynova et al. (1972) reported a 48.2% incidence of menstrual function disorders, the most frequent being hypomenstrual syndrome, in a group ( $n = 300$ ) of female caprolactam workers; the authors give no exposure

levels but do state that this rate was 2.5 times that of the controls. In a cohort of 304 female workers exposed to <10 mg/cu.m caprolactam (no duration given), irregular menstruation was significantly greater in paired controls (34.3% vs. 25%;  $p < 0.005$ ) (Liu et al., 1988). In a cross-sectional study of 200 female workers exposed to <10 mg/cu.m caprolactam, Angelov (1988) noted that the incidence of uterine myoma (a tumor containing muscle tissue) in a cohort of 616 female workers exposed to a number of compounds including caprolactam, was 2-3 times higher than in a control population of 182 women. Martynova et al. (1972) also claim that the number of pregnancy/birth complications occurred at a higher rate in a group of women ( $n = 137$ ) exposed to caprolactam than in a group of 150 control women, including hemorrhage at 33.8% in exposed vs. 18.1% in controls.

The animal studies available on inhalation exposure to caprolactam also have numerous deficiencies. The studies are not, however, confounded by coexposure. Khadzhieva (1969a) exposed inseminated female rats either to air ( $n = 22$ ) or to 139.2 ( $n = 40$ ) or 473 ( $n = 46$ ) mg/cu.m caprolactam vapor/dust. The daily duration of exposure was 4 hours, but the number of days is not clearly stated; some animals were exposed during the preimplantation phase, some during the period of organogenesis, and still others during fetal development. The results show concentration responses in the percent impregnated, in alteration of pregnancy duration, in mean birth weights, and in the percent of live-born young (based on corpora lutea). These parameters were clearly different from controls at both exposure levels. In a report apparently conducted on the same animals prior to their insemination, this author reports significant shortening of the rutting stage and prolongation of the dormancy phase of the estrous cycle (assumed to be estrus and diestrus, respectively), the latter effect occurring at both levels of exposure (Khadzhieva, 1969b). Gabpielyan et al. (1975) exposed three groups (number unspecified) of male rats either to air or to 10.6 or 124.6 mg/cu.m caprolactam dust/vapor for 4 hours/day for 2.5 months, and various measures of the gonads were taken at the end of this period. Statistically significant alterations relative to controls ( $p < 0.05$ ) were noted in the spermatogenesis index (unspecified), the total quantity of normal spermatogonia, and the number of tubules at twelfth-stage meiosis in those animals exposed to the higher concentration. No significant alterations relative to controls were noted in those animals exposed to the lower concentration. In an effort to explain his observation of increased uterine hemorrhage during and after birth in female workers exposed to caprolactam, Martynova et al. (1972) showed a reduction in spontaneous uterine contractions in pregnant rabbits after injection with an unspecified volume of a 10% solution of caprolactam.

The excretion and tissue distribution of caprolactam has been examined in male Fischer 344 rats after a single oral dose (Unger et al., 1981). By 24 hours post dosing, over 75% of the radiolabel had been excreted in the urine, predominantly as two unidentified metabolites. Small amounts of radiolabel were also present in feces and expired air. Concentration of radiolabel in tissues was substantially the same as blood except for portal-of-entry (stomach) and excretory (bladder and kidney) tissues.

Waddell et al. (1984) examined the excretion and tissue distribution of radiolabeled caprolactam in male and pregnant female Swiss-Webster mice by whole-body autoradiography. Caprolactam was administered by oral intubation to five pregnant and one nonpregnant female mice and intravenously to two male mice. The radioactivity was distributed throughout the animals (including fetuses) and, by 24 hours, had been nearly eliminated through renal secretion. Small amounts of radioactivity were retained in the cephalic region (including nasal epithelium, optic lens, and olfactory lobe). These data are not suitable for purposes of oral-to-inhalation extrapolation because they are not the appropriate route and provide no information on identity of circulating metabolites. The latter issue may be especially relevant because portal-of-entry tissues capable of metabolism (nasal epithelium) show retention of radiolabeled caprolactam.

#### **I.B.6. EPA Documentation and Review of the Inhalation RfC**

Agency Work Group Review — 08/03/1994

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the RfC for Caprolactam conducted in November 2001 identified one or more significant new studies. IRIS users may request the references for those studies from the IRIS Hotline at [hotline.iris@epa.gov](mailto:hotline.iris@epa.gov) or (202)566-1676.

#### **I.B.7. EPA Contacts (Inhalation RfC)**

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or [hotline.iris@epa.gov](mailto:hotline.iris@epa.gov) (internet address).

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## **II. Carcinogenicity Assessment for Lifetime Exposure**

Substance Name — Caprolactam  
CASRN — 105-60-2

This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.

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**III. [reserved]**

**IV. [reserved]**

**V. [reserved]**

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## **VI. Bibliography**

Substance Name — Caprolactam

CASRN — 105-60-2

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### VI.C. Carcinogenicity Assessment References

None

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### VII. Revision History

Substance Name — Caprolactam  
CASRN — 105-60-2

| Date       | Section | Description              |
|------------|---------|--------------------------|
| 09/07/1988 | I.A.    | Oral RfD summary on-line |

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| Date       | Section           | Description  |
|------------|-------------------|--|
| 09/01/1994 | I.B.              | Inhalation RfC under review and discussion on-line                 |
| 12/03/2002 | I.A.6.,<br>I.B.6. | Screening-Level Literature Review Findings message has been added. |

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### VIII. Synonyms

Substance Name — Caprolactam

CASRN — 105-60-2

Last Revised — 09/07/1988

- 105-60-2
- A1030
- A1030N0
- akulon
- akulon M 2W
- alkamid
- amilan CM 1001
- amilan CM 1001C
- amilan CM 1001G
- amilan CM 1011
- 6-aminocaproic acid lactam
- aminocaproic lactam
- 6-aminohexanoic acid cyclic lactam
- ATM 2(nylon)
- 1-aza-2-cycloheptanone
- 2-azacycloheptanone
- 2H-azepin-2-one,hexahydro-
- 2H-azepin-7-one, hexahydro-
- bonamid
- capran 77C
- capran 80
- Caprolactam
- 6-caprolactam
- epsilon-caprolactam
- caprolactam monomer

- caprolattame
- caprolon B
- caprolon V
- capron
- capron 8250
- capron 8252
- capron 8253
- capron 8256
- capron 8257
- capron B
- capron GR 8256
- capron GR 8258
- capron PK4
- chemlon
- cyclohexanone iso-oxime
- danamid
- dull 704
- durethan BK
- epsylon kaprolaktam
- ertalon 6SA
- extrom 6N
- grilon
- hexahydro-2-azepinone
- hexahydro-2H-azepin-2-one
- hexamethylenimine, 2-oxo-
- 6-hexanelactam
- hexanoic acid, 6-amino-, cyclic lactam
- hexanoic acid, 6-amino-, lactam
- hexanolactam
- hexanone isoxime
- hexanonisoxim
- 1,6-hexolactam
- itamid
- e-kaprolaktam
- kaprolit
- kaprolon
- kapromine
- kapron
- 2-ketohexamethyleneimine
- 2-ketohexamethylenimine
- KS 30P
- metamid
- NCI-C50646
- nylon A1035SF
- nylon CM 1031

- nylon X 1051
- omega-caprolactam
- orgamide
- orgamid RMNOCD
- 2-oxohexamethyleneimine
- P 6
- PA 6
- 2-perhydroazepinone
- PK 4
- PKA
- polyamide PK 4
- relon P
- renyl MV
- sipas 60
- steelon
- stilon
- stylon
- tarnamid T
- TNK 2G5
- torayca N 6
- UBE 1022B
- vidlon
- widlon
- zytel 211