www.pdr.net

PHYSICIANS' DESK REFERENCE®

PDR®

53

EDITION

1999

PHYSICIANS' DESK REFERENCE®

CONTENTS

To Prepare Admixture:

1. Squeeze the bottom of the diluent container gently to inflate the portion of the container surrounding the end of the drug vial.

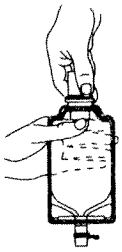


Figure 4

2. With the other hand, push the drug vial down into the container telescoping the walls of the container. Grasp the inner cap of the vial through the walls of the container. (See Figure 5.)

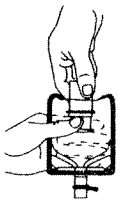


Figure 5

3. Pull the inner cap from the drug vial. (See Figure 6.) Verify that the rubber stopper has been pulled out and invert the system several times, allowing the drug and diluent to mix.

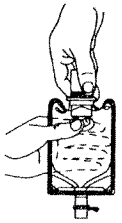


Figure 6

4. Mix contents thoroughly and use within the specified time.

Preparation For Administration: (Use Aseptic Technique)

1. Confirm the activation and admixture of vial contents.
2. Check for leaks by squeezing container firmly. If leaks are found, discard unit as sterility may be impaired.
3. Close flow control clamp of administration set.
4. Remove cover from outlet port at bottom of container.
5. Insert piercing pin of administration set into port with a twisting motion until the pin is firmly seated. **NOTE:** See full directions on administration set carton.
6. Lift the free end of the hanger loop on the bottom of the vial, breaking the two tie strings. Bend the loop outward to lock it in the upright position, then suspend container from hanger.
7. Squeeze and release drip chamber to establish proper fluid level in chamber.
8. Open flow control clamp and clear air from set. Close clamp.
9. Attach set to venipuncture device. If device is not indwelling, prime and make venipuncture.
10. Regulate rate of administration with flow control clamp.

WARNING: Do not use flexible container in series connections.

COMPATIBILITY AND STABILITY

Compatibility of MERREM I.V. with other drugs has not been established. MERREM I.V. should not be mixed with or physically added to solutions containing other drugs.

Freshly prepared solutions of MERREM I.V. should be used whenever possible. However, constituted solutions of MERREM I.V. maintain satisfactory potency at controlled room temperature 15–25°C (59–77°F) or under refrigeration at 4°C (39°F) as described below. Solutions of intravenous MERREM I.V. should not be frozen.

Intravenous Bolus Administration

MERREM I.V.) may be stored for up to 2 hours at controlled room temperature 15–25°C (59–77°F) or for up to 12 hours at 4°C (39°F).

Intravenous Infusion Administration

Stability in Infusion Vials: MERREM I.V. infusion vials constituted with Sodium Chloride Injection 0.9% (MERREM I.V. concentrations ranging from 2.5 to 50 mg/mL) are stable for up to 2 hours at controlled room temperature 15–25°C (55–77°F) or for up to 18 hours at 4°C (39°F). Infusion vials of MERREM I.V. constituted with Dextrose Injection 5% (MERREM I.V. concentrations ranging from 2.5 to 50 mg/mL) are stable for up to 1 hour at controlled room temperature 15–25°C (59–77°F) or for up to 8 hours at 4°C (39°F).

Stability in Plastic I.V. Bags: Solutions prepared for infusion (MERREM I.V. concentrations ranging from 1 to 20 mg/mL) may be stored in plastic intravenous bags with diluents as shown below:

	Number of Hours Stable at Controlled Room Temperature 15–25°C (59–77°F)	Number of Hours Stable at 4°C (39°F)
Sodium Chloride Injection 0.9%	4	24
Dextrose Injection 5.0%	1	4
Dextrose Injection 10.0%	1	2
Dextrose and Sodium Chloride Injection 5.0%/0.9%	1	2
Dextrose and Sodium Chloride Injection 5.0%/0.2%	1	4
Potassium Chloride in Dextrose Injection 0.15%/5.0%	1	6
Sodium Bicarbonate in Dextrose Injection 0.02%/5.0%	1	6
Dextrose Injection 5.0% in Normosol®-M	1	8
Dextrose Injection 5.0% in Ringers Lactate Injection	1	4
Dextrose and Sodium Chloride Injection 2.5%/0.45%	3	12
Mannitol Injection 2.5%	2	16
Ringers Injection	4	24
Ringers Lactate Injection	4	12
Sodium Lactate Injection 1/6 N	2	24
Sodium Bicarbonate Injection 5.0%	1	4

Stability in Baxter Minibag Plus: Solutions of MERREM I.V. (MERREM I.V. concentrations ranging from 2.5 to 20 mg/mL) in Baxter Minibag Plus bags with Sodium Chloride Injection 0.9% may be stored for up to 4 hours at controlled room temperatures 15–25°C (59–77°F) or for up to 24 hours at 4°C (39°F). Solutions of MERREM I.V. (MERREM I.V. concentrations ranging from 2.5 to 20 mg/mL) in Baxter Minibag Plus bags with Dextrose Injection 5.0% may be stored up to 1 hour at controlled room temperatures 15–25°C (59–77°F) or for up to 6 hours at 4°C (39°F).

Stability in Plastic Syringes, Tubing and Intravenous Infusion Sets: Solutions of MERREM I.V. (MERREM I.V. concentrations ranging from 1 to 20 mg/mL) in Water for Injection or Sodium Chloride Injection 0.9% (for up to 4 hours) or in Dextrose Injection 5.0% (for up to 2 hours) at controlled room temperatures 15–25°C (59–77°F) are stable in plastic syringes, plastic tubing, drip chambers, and volume control devices of common intravenous infusion sets.

ADD-Vantage Vials: ADD-Vantage vials diluted in Sodium Chloride Injection 0.45% (MERREM I.V. concentrations ranging from 5 to 20 mg/mL) may be stored for up to 6 hours at controlled room temperature 15–25°C (59–77°F) or for 24 hours at 4°C (39°F). ADD-Vantage vials diluted in Sodium Chloride Injection 0.9% (MERREM I.V. concentrations ranging from 1–20 mg/mL) may be stored for up to 4 hours at controlled room temperature 15–25°C (59–77°F) or for 24 hours at 4°C (39°F). ADD-Vantage vials diluted with Dextrose Injection 5.0% (MERREM I.V. concentrations ranging from 1–20 mg/mL) may be stored for up to 1 hour at controlled room temperature 15–25°C (59–77°F) or for 8 hours at 4°C (39°F).

NOTE: Parenteral drug products should be inspected visually for particulate matter and discoloration prior to administration, whenever solution and container permit.

HOW SUPPLIED

MERREM I.V. is supplied in 20 mL and 30 mL injection vials containing sufficient meropenem to deliver 500 mg or 1 g for intravenous administration, respectively. MERREM I.V. is supplied in 100 mL infusion vials containing sufficient meropenem to deliver 500 mg or 1 g for intravenous administration. MERREM I.V. is also supplied as ADD-Vantage Vials containing sufficient meropenem to deliver 500 mg or 1 g for intravenous administration.

500 mg/20 mL Injection Vial (NDC 0310-0325-20)
500 mg/100 mL Infusion Vial (NDC 0310-0325-11)
1 g/30 mL Injection Vial (NDC 0310-0321-30)
1 g/100 mL Infusion Vial (NDC 0310-0321-11)
500 mg/15 mL ADD-Vantage (NDC 0310-0325-15)
1 g/15 mL ADD-Vantage (NDC 0310-0321-15)

REFERENCES

1. National Committee for Clinical Laboratory Standards. Methods for Dilution Antimicrobial Susceptibility Tests for Bacteria that Grow Aerobically — Third Edition. Approved Standard NCCLS Document M7-A3, Vol. 13, No. 25, NCCLS, Villanova, PA, December, 1993.
2. National Committee for Clinical Laboratory Standards. Performance Standards for Antimicrobial Disk Susceptibility Tests — Fifth Edition. Approved Standard NCCLS Document M2-A5, Vol. 13, No. 24, NCCLS, Villanova, PA. December 1993.
3. National Committee for Clinical Laboratory Standards. Methods for Antimicrobial Susceptibility Testing of Anaerobic Bacteria – Third Edition. Approved Standard NCCLS Document M11-A3, Vol. 13, No. 26, NCCLS, Villanova, PA. December 1993.
4. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron. 1976; 16.31-41.

†ADD-Vantage is a registered trademark of Abbott Laboratories Inc.

MERREM® (meropenem for injection) is manufactured by:
Sumitomo Pharmaceuticals Co. Ltd
Oita Works
Tsurusaki 2200
Oita-shi
Oita
Japan

Manufactured for:
Zeneca Pharmaceuticals
A business unit of Zeneca Inc.
Wilmington, DE 19850-5437
Rev F 07/96 SIC 64041-00

Shown in Product Identification Guide, page 346

NOLVADEX® ℞

[nol 'va-dex]
tamoxifen citrate

DESCRIPTION

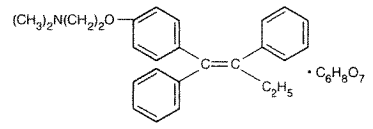
NOLVADEX® (tamoxifen citrate) Tablets, a nonsteroidal antiestrogen, are for oral administration. NOLVADEX Tablets are available as:

10 mg Tablets. Each tablet contains 15.2 mg of tamoxifen citrate which is equivalent to 10 mg of tamoxifen.

20 mg Tablets. Each tablet contains 30.4 mg of tamoxifen citrate which is equivalent to 20 mg of tamoxifen.

Inactive Ingredients: carboxymethylcellulose calcium, magnesium stearate, mannitol and starch.

Chemically, NOLVADEX is the trans-isomer of a triphenylethylene derivative. The chemical name is (Z)2-[4-(1,2-diphenyl-1-butenyl) phenoxy]-N, N-dimethylethanamine 2-hydroxy-1,2,3-propanetricarboxylate (1:1). The structural and empirical formulas are:



$(C_{32}H_{37}NO_8)$

Tamoxifen citrate has a molecular weight of 563.62, the pKa' is 8.85, the equilibrium solubility in water at 37°C is 0.5 mg/mL and in 0.02 N HCl at 37°C, it is 0.2 mg/mL.

CLINICAL PHARMACOLOGY

NOLVADEX is a nonsteroidal agent which has demonstrated potent antiestrogenic properties in animal test systems. The antiestrogenic effects may be related to its ability to compete with estrogen for binding sites in target tissues such as breast. Tamoxifen inhibits the induction of rat mammary carcinoma induced by dimethylbenzanthracene (DMBA) and causes the regression of already established DMBA-induced tumors. In this rat model, tamoxifen appears to exert its antitumor effects by binding the estrogen receptors.

In cytosols derived from human breast adenocarcinomas, tamoxifen competes with estradiol for estrogen receptor protein.

Nolvadex—Cont.

Tamoxifen is extensively metabolized after oral administration. Studies in women receiving 20 mg of ^{14}C tamoxifen have shown that approximately 65% of the administered dose was excreted from the body over a period of 2 weeks with fecal excretion as the primary route of elimination. The drug was excreted mainly as polar conjugates, with unchanged drug and unconjugated metabolites accounting for less than 30% of the total fecal radioactivity.

N-desmethyl tamoxifen was the major metabolite found in patients' plasma. The biological activity of N-desmethyl tamoxifen appears to be similar to tamoxifen. 4-Hydroxytamoxifen and a side chain primary alcohol derivative of tamoxifen have been identified as minor metabolities in plasma.

Following a single oral dose of 20 mg tamoxifen, an average peak plasma concentration of 40 ng/mL (range 35 to 45 ng/mL) occurred approximately 5 hours after dosing. The decline in plasma concentrations of tamoxifen is biphasic with a terminal elimination half-life of about 5 to 7 days. The average peak plasma concentration for N-desmethyl tamoxifen is 15 ng/mL (range 10 to 20 ng/mL). Chronic administration of 10 mg tamoxifen given twice daily for three months to patients results in average steady-state plasma concentrations of 120 ng/mL (range 67–183 ng/mL) for tamoxifen and 336 ng/mL (range 148–654 ng/mL) for N-desmethyl tamoxifen. The average steady-state plasma concentrations of tamoxifen and N-desmethyl tamoxifen after administration of 20 mg tamoxifen once daily for three months are 122 ng/mL (range 71–183 ng/mL) and 353 ng/mL (range 152–706 ng/mL), respectively. After initiation of therapy, steady state concentrations for tamoxifen are achieved in about 4 weeks and steady state concentrations for N-desmethyl tamoxifen are achieved in about 8 weeks, suggesting a half-life of approximately 14 days for this metabolite.

In a 3-month crossover steady-state bioavailability study with NOLVADEX 10 mg twice a day versus NOLVADEX 20 mg given once daily, the results deomonstrated that NOLVADEX 20 mg taken once daily has comparable bioavailability to NOLVADEX 10 mg taken twice a day.

Clinical Studies: The Early Breast Cancer Trialists' Collaborative Group (EBCTCG) conducted worldwide overviews of systemic adjuvant therapy for early breast cancer in 1985 and again in 1990. In 1992, 10-year outcome data were reported for 29,892 women in 40 randomized trials of adjuvant tamoxifen using doses of 20–40 mg/day for 1–5+ years (median 2 years). Fifty-one percent were entered into trials comparing tamoxifen to no adjuvant therapy and 49% were entered into trials of tamoxifen in combination with chemotherapy vs. the same chemotherapy alone. Twenty-nine percent were <50 years of age and 71% were ≥50 years. Fifty-seven percent were node-positive and 43% were node-negative. Fifty percent of the tumors were estrogen receptor (ER) positive (≥10 fmol/mg), 18% were ER poor (<10 fmol/mg), and 32% were ER unknown.

The overall recurrence-free survival at 10 years of follow-up was 51.2% for tamoxifen versus 44.7% for control (logrank 2p <0.00001). Overall survival at 10 years was 58.8% for tamoxifen versus 52.6% for control (logrank 2p <0.00001). Both the absolute risk of relapse and the absolute benefit of treatment with tamoxifen were greater in women with positive nodes than in women with negative nodes. In women with positive nodes, 10-year recurrence-free survival was 41.9% for tamoxifen versus 33.1% for control (logrank 1p <0.00001). Ten-year survival was 50.4% for tamoxifen versus 42.2% for control (logrank 1p <0.00001). In women with negative nodes, recurrence-free survival was 68.1% for tamoxifen versus 63.1% for control (logrank 1p <0.00001). Survival at 10 years was 74.5% for tamoxifen versus 71.0% for control (logrank 1p = 0.0002).

The reduction in the annual odds of recurrence with tamoxifen was 12% in women <50 years of age versus 29% in women ≥50 years. Similarly, the reduction in the annual odds of death was 6% versus 20%. The reduction in the annual odds of recurrence with tamoxifen was significantly greater in ER positive (32%) than in ER poor (13%) tumors (1p <0.00001). The reduction in recurrence and mortality was greater in those studies that used tamoxifen for longer (≥2 years) rather than shorter (<2 years) periods. There was no indication that doses greater than 20 mg per day were more effective.

Two studies (Hubay and NSABP B-09) demonstrated an improved disease-free survival following radical or modified radical mastectomy in postmenopausal women or women 50 years of age or older with surgically curable breast cancer with positive axillary nodes when NOLVADEX was added to adjuvant cytotoxic chemotherapy. In the Hubay study, NOLVADEX was added to "low-dose" CMF (cyclophosphamide, methotrexate and fluorouracil). In the NSABP B-09 study, NOLVADEX was added to melphalan [L-phenylalanine mustard (P)] and fluorouracil (F).

In the Hubay study, patients with a positive (more than 3 NSABP B-09 study in women age 50–59 years, only women with both estrogen and progesterone receptor levels 10 fmol or greater clearly benefited, while there was a nonstatistically significant trend toward adverse effect in women with both estrogen and progesterone receptor levels less than 10 fmol. In women age 60–70 years, there was a trend toward a beneficial effect of NOLVADEX without any clear relationship to estrogen or progesterone receptor status.

Three prospective studies (ECOG-1178, Toronto, NATO) using NOLVADEX adjuvantly as a single agent demonstrated an improved disease-free survival following total mastectomy and axillary dissection for postmenopausal women with positive axillary nodes compared to placebo/no treatment controls. The NATO study also demonstrated an overall survival benefit.

NSABP B-14, a prospective, double-blind, randomized study, evaluated NOLVADEX versus placebo in the treatment of women with axillary node-negative, estrogen-receptor positive (≥ 10 fmol/mg cytosol protein) breast cancer (as adjuvant therapy, following total mastectomy and axillary dissection, or segmental resection, axillary dissection, and breast radiation). After five years of treatment, a significant improvement in disease-free survival was demonstrated in women receiving NOLVADEX. This benefit was apparent both in women under age 50 and in women at or beyond age 50. In this trial women who received tamoxifen for five years and were disease-free at the end of this 5-year period were offered an additional five years of NOLVADEX, or placebo in a double-blind randomized scheme. With four years of follow-up after this rerandomization, 92% of the women that received five years of NOLVADEX followed by placebo are alive and disease-free, compared to 86% of the women scheduled to receive 10 years of NOLVADEX. This difference was not statistically significant. One additional randomized study (NATO) demonstrated improved disease-free survival for NOLVADEX compared to no adjuvant therapy following total mastectomy and axillary dissection in postmenopausal women with axillary node-negative breast cancer. In this study, the benefits of NOLVADEX appeared to be independent of estrogen receptor status.

Three prospective, randomized studies (Ingle, Pritchard, Buchanan) compared NOLVADEX to ovarian ablation (oophorectomy or ovarian irradiation) in premenopausal women with advanced breast cancer. Although the objective response rate, time to treatment failure, and survival were similar with both treatments, the limited patient accrual prevented a demonstration of equivalence. In an overview analysis of survival data from the three studies, the hazard ratio for death (NOLVADEX/ovarian ablation) was 1.00 with two-sided 95% confidence intervals of 0.73 to 1.37. Elevated serum and plasma estrogens have been observed in premenopausal women receiving NOLVADEX. However, the data from the randomized studies do not suggest an adverse effect. A limited number of premenopausal patients with disease progression during NOLVADEX therapy responded to subsequent ovarian ablation.

In a large randomized trial in Sweden of adjuvant NOLVADEX 40 mg/day for 2–5 years, the incidence of second primary breast tumors was reduced in the tamoxifen arm (p<0.05). In the NSABP B-14 trial in which patients were randomized to NOLVADEX 20 mg/day for 5 years versus placebo, the incidence of second primary breast cancers is also reduced.

Published results from 122 patients (119 evaluable) and case reports in 16 patients (13 evaluable) treated with NOLVADEX have shown that NOLVADEX is effective for the palliative treatment of male breast cancer. Sixty-six of these 132 evaluable patients responded to NOLVADEX which constitutes a 50% objective response rate.

INDICATIONS AND USAGE

Adjuvant Therapy: NOLVADEX is indicated for the treatment of axillary node-negative breast cancer in women following total mastectomy or segmental mastectomy, axillary dissection, and breast irradiation. Data are insufficient to predict which women are most likely to benefit and to determine if NOLVADEX provides any benefit in women with tumors less than 1 cm.

NOLVADEX is indicated for the treatment of node-positive breast cancer in postmenopausal women following total mastectomy, or segmental mastectomy, axillary dissection, and breast irradiation. In some NOLVADEX adjuvant studies, most of the benefit to date has been in the subgroup with 4 or more positive axillary nodes.

The estrogen and progesterone receptor values may help to predict whether adjuvant NOLVADEX therapy is likely to be beneficial.

Therapy for Advanced Disease: NOLVADEX is effective in the treatment of metastatic breast cancer in women and men. In premenopausal women with metastatic breast cancer, NOLVADEX is an alternative to oophorectomy or ovarian irradiation. Available evidence indicates that patients whose tumors are estrogen receptor positive are more likely to benefit from NOLVADEX therapy.

CONTRAINDICATIONS

NOLVADEX is contraindicated in patients with known hy-

WARNINGS

Visual disturbance including corneal changes, cataracts and retinopathy have been reported in patients receiving NOLVADEX.

As with other additive hormonal therapy (estrogens and androgens), hypercalcemia has been reported in some breast cancer patients with bone metastases within a few weeks of starting treatment with NOLVADEX. If hypercalcemia does occur, appropriate measures should be taken and, if severe, NOLVADEX should be discontinued.

An increased incidence of endometrial cancer has been reported in association with NOLVADEX treatment. The underlying mechanism is unknown, but may be related to the estrogen-like effect of NOLVADEX. Any patients receiving or having previously received NOLVADEX, who report abnormal vaginal bleeding should be promptly evaluated. Patients receiving or having previously received NOLVADEX should have routine gynecological care and they should promptly inform their physician if they experience any abnormal gynecological symptoms, eg, menstrual irregularities, abnormal vaginal bleeding, changes in vaginal discharge, or pelvic pain or pressure.

An increased incidence of endometrial changes including hyperplasia and polyps have been reported in association with NOLVADEX treatment. The incidence and pattern of this increase suggest that the underlying mechanism is related to the estrogenic properties of NOLVADEX.

In a large randomized trial in Sweden of adjuvant NOLVADEX 40 mg/day for 2–5 years, an increased incidence of uterine cancer was noted. Twenty-three of 1,372 patients randomized to receive NOLVADEX versus 4 of 1,357 patients randomized to the observation group developed cancer of the uterus [RR = 5.6 (1.9–16.2), p<.001]. One of the patients with cancer of the uterus who was randomized to receive NOLVADEX never took the drug. After approximately 6.8 years of follow-up in the NSABP B-14 trial, 15 of 1,419 women randomized to receive NOLVADEX 20 mg/day for 5 years developed uterine cancer and 2 of the 1,424 women randomized to receive placebo, who subsequently were treated with NOLVADEX, also developed uterine cancer. Most of the uterine cancers were diagnosed at an early stage, but deaths from uterine cancer have been reported.

NOLVADEX has been associated with changes in liver enzyme levels, and on rare occasions, a spectrum of more severe liver abnormalities including fatty liver, cholestasis, hepatitis and hepatic necrosis. A few of these serious cases included fatalities. In most reported cases the relationship to NOLVADEX is uncertain. However, some positive rechallenges and dechallenges have been reported.

In the Swedish trial using adjuvant NOLVADEX 40 mg/day for 2–5 years, 3 cases of liver cancer have been reported in the NOLVADEX-treated group versus 1 case in the observation group. In other clinical trials evaluating NOLVADEX, no other cases of liver cancer have been reported to date.

Data from the NSABP B-14 study show no increase in other (non-uterine) cancers among patients receiving NOLVADEX. However, a number of second primary tumors, occurring at sites other than the endometrium, have been reported following the treatment of breast cancer with NOLVADEX in clinical trials. Whether an increased risk for other (non-uterine) cancers is associated with NOLVADEX is still uncertain and continues to be evaluated.

Pregnancy Category D: NOLVADEX may cause fetal harm when administered to a pregnant woman. Women should be advised not to become pregnant while taking NOLVADEX and should use barrier or nonhormonal contraceptive measures if sexually active. Effects on reproductive functions are expected from the antiestrogenic properties of the drug. In reproductive studies in rats at dose levels equal to or below the human dose, nonteratogenic developmental skeletal changes were seen and were found reversible. In addition, in fertility studies in rats and in teratology studies in rabbits using doses at or below those used in humans, a lower incidence of embryo implantation and a higher incidence of fetal death or retarded in utero growth were observed, with slower learning behavior in some rat pups when compared to historical controls. Several pregnant marmosets were dosed during organogenesis or in the last half of pregnancy. No deformations were seen and, although the dose was high enough to terminate pregnancy in some animals, those that did maintain pregnancy showed no evidence of teratogenic malformations.

In rodent models of fetal reproductive tract development, tamoxifen (at doses 0.3 to 2.4-fold the human maximum recommended dose on a mg/m^2 basis) caused changes in both sexes that are similar to those caused by estradiol, ethynylestradiol and diethylstilbestrol. Although the clinical relevance of these changes is unknown, some of these changes, especially vaginal adenosis, are similar to those seen in young women who were exposed to diethylstilbestrol *in utero* and who have a 1 in 1000 risk of developing clear-cell adenocarcinoma of the vagina or cervix. To date, *in utero* exposure to tamoxifen has not been shown to cause vaginal

DOCKET ALARM

Explore Litigation Insights

Docket Alarm provides insights to develop a more informed litigation strategy and the peace of mind of knowing you're on top of things.

Real-Time Litigation Alerts

Keep your litigation team up-to-date with **real-time alerts** and advanced team management tools built for the enterprise, all while greatly reducing PACER spend.

Our comprehensive service means we can handle Federal, State, and Administrative courts across the country.

Advanced Docket Research

With over 230 million records, Docket Alarm's cloud-native docket research platform finds what other services can't. Coverage includes Federal, State, plus PTAB, TTAB, ITC and NLRB decisions, all in one place.

Identify arguments that have been successful in the past with full text, pinpoint searching. Link to case law cited within any court document via Fastcase.

Analytics At Your Fingertips

Learn what happened the last time a particular judge, opposing counsel or company faced cases similar to yours.

Advanced out-of-the-box PTAB and TTAB analytics are always at your fingertips.

API

Docket Alarm offers a powerful API (application programming interface) to developers that want to integrate case filings into their apps.

LAW FIRMS
Build custom dashboards for your attorneys and clients with live data direct from the court.

Automate many repetitive legal tasks like conflict checks, document management, and marketing.

FINANCIAL INSTITUTIONS
Litigation and bankruptcy checks for companies and debtors.

E-DISCOVERY AND LEGAL VENDORS
Sync your system to PACER to automate legal marketing.