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Atossa Therapeutics, Inc.

(ATOS-NASDAQ)

ATOS: Multiple Catalysts Converge in 2H26

Based on our probability adjusted DCF model that takes into account potential future revenues from endoxifen, ATOS is valued at $22.00/share. This model is highly dependent upon continued clinical success of endoxifen and will be adjusted accordingly based upon future clinical results.

Current Price (08/14/26)	$2.56
Valuation	**$22.00**

OUTLOOK

On Aug. 7, 2026, Atossa Therapeutics, Inc. (ATOS) announced financial results for the second quarter of 2026 and provided a business update. The company is approaching a potentially transformative period as two Phase 2 studies of (Z)-endoxifen are expected to produce data by year-end. In addition, we anticipate durability data to be reported from the Phase 2 KARISMA trial in the near-term along with development updates for potential applications in Duchenne muscular dystrophy (DMD) and McCune-Albright syndrome (MAS). Lastly, cash burn is expected to materially decline in the second half of 2026, which should extend the company's operating runway into the first half of 2027.

SUMMARY DATA

52-Week High	**$17.69**
52-Week Low	**$1.75**
One-Year Return (%)	**-78.11**
Beta	**1.29**
Average Daily Volume (sh)	**53,804**
Shares Outstanding (mil)	**10**
Market Capitalization ($mil)	**$26**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**13**
Insider Ownership (%)	**10**
Annual Cash Dividend	**$0.00**
Dividend Yield (%)	**0.00**
5-Yr. Historical Growth Rates	
Sales (%)	**N/A**
Earnings Per Share (%)	**N/A**
Dividend (%)	**N/A**
P/E using TTM EPS	**N/A**
P/E using 2026 Estimate	**-0.4**
P/E using 2027 Estimate	**-0.5**

Risk Level	**High**
Type of Stock	**Small-Value**
Industry	**Med/Biomed-Gene**

ZACKS ESTIMATES

Revenue
(in millions of $)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2025	0 A	0 A	0 A	0 A	0 A
2026	0 A	0 A	0 E	0 E	0 E
2027					0 E
2028					0 E

Earnings per Share

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2025	-$0.78 A	-$0.98 A	-$1.01 A	-$1.27 A	-$4.04 A
2026	-$1.11 A	-$0.95 A	-$0.74 E	-$0.73 E	-$3.49 E
2027					-$2.73 E
2028					-$2.38 E

WHAT'S NEW

Business Update

2H26 Shaping Up to be Catalyst-Rich

Atossa Therapeutics, Inc. (ATOS) is approaching a potentially important inflection point as its two most advanced clinical programs are now fully enrolled and expected to generate data by year-end. The company's lead asset, (Z)-endoxifen, is being evaluated across multiple potential applications in breast cancer, including neoadjuvant treatment of perimenopausal women with ER+/HER2- disease in the Phase 2 EVANGELINE study and in combination with established therapies in the I-SPY 2 trial. Those studies are expected to have data before the end of 2026. In addition, the company expects to report durability data from the Phase 2 KARISMA study of (Z)-endoxifen in breast cancer prevention and provide development updates for potential applications in Duchenne muscular dystrophy (DMD) and McCune-Albright syndrome (MAS) in the second half of 2026.

EVANGELINE Trial

EVANGELINE represents Atossa's most direct test of whether the pharmacologic properties of (Z)-endoxifen can translate into meaningful clinical activity in premenopausal women with ER+/HER2- breast cancer. The Phase 2 study is evaluating 40 mg of oral (Z)-endoxifen daily in combination with goserelin, an ovarian function suppressant, as neoadjuvant therapy prior to surgery. We anticipate data from EVANGELINE before the end of 2026.

Premenopausal women with ER+/HER2- breast cancer represent a particularly interesting population for (Z)-endoxifen. Unlike postmenopausal women, premenopausal patients continue to produce substantial amounts of estrogen from the ovaries, meaning that aromatase inhibition alone is generally insufficient to achieve complete estrogen suppression. Consequently, premenopausal patients receiving endocrine therapy often require ovarian function suppression (OFS) in addition to either aromatase inhibitors or tamoxifen.

Although OFS-based regimens can be effective, the additional hormonal suppression can substantially increase treatment burden. Vasomotor symptoms, sexual dysfunction, bone loss, and other endocrine-related adverse effects can negatively affect quality of life and treatment adherence. Atossa's thesis is that directly administering (Z)-endoxifen could provide potent ER antagonism without relying on conversion of tamoxifen to its active metabolite, potentially creating an alternative endocrine approach for this population.

The trial consists of two cohorts. Cohort A is the primary signal-seeking population that uses a Simon two-stage design based on the proportion of patients whose Ki-67 falls to 10% or less after four weeks of treatment. The first stage enrolls 20 patients, with another 25 patients added if the initial results are sufficiently promising. The trial is designed to determine whether at least 65% of evaluable patients can achieve the predefined Ki-67 response threshold. Cohort B is enrolling patients whose baseline Ki-67 is already 10% or lower and will evaluate objective tumor response after 24 weeks using RECIST 1.1. Secondary endpoints include safety and tolerability, residual cancer burden and PEPI score, while additional analyses will examine tumor and plasma biomarkers. Rather than being a randomized registration-enabling efficacy study, it is intended to establish a sufficiently strong biological and clinical signal to justify further development.

The rationale for this study is predicated on previous results with (Z)-endoxifen. In a prior Phase 2 window-of-opportunity study, treatment reduced average Ki-67 from 25.6% at baseline to 6.0% at surgery, representing a 65.1% reduction. More directly relevant to EVANGELINE, the pharmacodynamic

run-in for the current study produced a Week-4 Ki-67 ≤10% response rate of 86% across evaluated (Z)-endoxifen dose levels with and without OFS. These results supported the selection of the 40 mg dose in combination with OFS for the Phase 2 study.

When the results are announced, we will be watching for the proportion of Cohort A patients with baseline Ki-67 >10% who achieve Ki-67 ≤10% after four weeks. A result comfortably above the 65% threshold would provide the clearest validation of the company's development hypothesis. Given the 86% response rate observed in the earlier run-in, a result in that general range would likely be viewed very favorably. The 24-week data could ultimately be even more important. While Ki-67 provides a rapid measure of pharmacodynamic activity, objective tumor response provides a more direct indication that treatment is affecting tumor burden. Thus, we will be very interested in the objective responses in Cohort B, with a favorable combination of strong Ki-67 suppression and meaningful tumor responses substantially strengthening the case for advancing (Z)-endoxifen into a larger controlled study.

I-SPY 2 Trial

While EVANGELINE is designed to establish the biological activity of (Z)-endoxifen in premenopausal ER+/HER2- breast cancer, Atossa's participation in the I-SPY 2 trial addresses a potentially broader question: can (Z)-endoxifen enhance the activity of established targeted therapies when used as part of a combination regimen? We believe this question could ultimately be more important to the commercial opportunity than the activity of (Z)-endoxifen as a standalone endocrine therapy, because combination treatment could allow Atossa to position the drug as a backbone for multiple therapeutic regimens rather than competing directly with the existing endocrine therapy market.

I-SPY 2 is an adaptive platform trial evaluating multiple investigational therapies in patients with high-risk, locally advanced breast cancer in the neoadjuvant setting. Rather than requiring each sponsor to independently conduct a large randomized trial, the platform allows experimental regimens to be evaluated against a common control framework, with treatments advancing or graduating based on predefined probability thresholds.

Atossa is evaluating (Z)-endoxifen in combination with abemaciclib, a CDK4/6 inhibitor marketed by Eli Lilly as Verzenio®, and with elagolix, a gonadotropin-releasing hormone antagonist marketed by Abbvie as Orilissa®. (Z)-endoxifen is intended to suppress estrogen receptor signaling while the combination partner inhibits complementary pathways that contribute to tumor cell proliferation or estrogen production. The abemaciclib combination is particularly intriguing from an investment perspective. CDK4/6 inhibitors are already an established component of treatment for HR+/HER2- breast cancer, creating a commercially validated therapeutic class. If adding (Z)-endoxifen to a CDK4/6 inhibitor produces a meaningful improvement in response, Atossa would have evidence that its drug can potentially complement rather than simply replace existing endocrine therapies. The elagolix combination provides a different test of the company's strategy. Elagolix suppresses ovarian hormone production, while (Z)-endoxifen directly antagonizes estrogen receptor signaling. This combination therefore offers the opportunity to examine whether more complete suppression of the estrogen axis can translate into greater antitumor activity in premenopausal patients.

KARISMA Trial

KARISMA represents the most clinically advanced opportunity outside Atossa's therapeutic breast cancer programs. Rather than treating established tumors, the program is evaluating whether low-dose (Z)-endoxifen can reduce mammographic breast density (MBD), a well-established biomarker associated with future breast cancer risk.

The trial enrolled 240 healthy premenopausal women with elevated MBD and randomized them to placebo, 1 mg/day (Z)-endoxifen, or 2 mg/day (Z)-endoxifen for six months. Both active treatment groups demonstrated statistically significant reductions in MBD compared with placebo, with the 1 mg dose producing a mean 19.3% reduction from baseline (P<0.01) and the 2 mg dose producing a mean 26.5%

reduction (*P*<0.01). Importantly, the 1 mg dose had a tolerability profile broadly comparable to placebo, which could be particularly important for a drug intended for use in otherwise healthy women (Hall *et al.*, 2026).

The apparent efficacy of the 1 mg dose is particularly notable because systemic exposure was relatively low, with mean endoxifen concentrations of approximately 5 ng/mL. In addition, treatment-related discontinuations occurred in five patients in the 1 mg group versus 11 in the 2 mg group and four with placebo, while vasomotor symptoms were significantly more pronounced at 2 mg. These findings suggest that 1 mg may provide an attractive balance between biological activity and tolerability for a prevention population consisting of otherwise healthy women.

The next important question is durability. Management expects to report additional KARISMA data in the near term. The durability analysis could help determine whether the reduction in MBD persists after treatment and therefore whether women might require continuous long-term treatment to maintain the effect. This distinction has meaningful implications for the commercial opportunity. A preventive endocrine therapy that requires continuous administration for many years would face a substantial tolerability and adherence hurdle, particularly in healthy women. Conversely, if (Z)-endoxifen produces a durable reduction in breast density following a relatively limited treatment course, the potential treatment paradigm could be considerably more attractive. While positive results would not by themselves establish that reducing MBD prevents breast cancer, and additional clinical studies would be required to demonstrate a reduction in breast cancer incidence, durable MBD reduction combined with the favorable tolerability profile observed at the 1 mg dose could strengthen the rationale for advancing (Z)-endoxifen as a potential breast cancer prevention therapy.

McCune-Albright Syndrome

Atossa's development strategy has also expanded into McCune-Albright syndrome (MAS), a rare disorder caused by activating mutations in the GNAS gene that result in constitutive signaling of the stimulatory G protein α-subunit (Gsα). In girls with MAS, autonomous ovarian estrogen production can cause peripheral precocious puberty, including recurrent vaginal bleeding, breast development, and accelerated bone maturation.

The company's interest in MAS is based on the possibility that (Z)-endoxifen could address more than one component of the disease biology. Atossa's recent preclinical work suggests that (Z)-endoxifen can suppress estrogen receptor-mediated transcription while also inhibiting PKCβ/AKT-associated proliferative signaling. The company believes this dual mechanism could potentially address both the consequences of autonomous estrogen production and downstream cellular proliferation.

Atossa has received Rare Pediatric Disease designation from the FDA for (Z)-endoxifen in MAS, which could potentially lead to the issuance of a Priority Review Voucher (PRV) if the drug is ultimately approved for treating MAS. PRV's are fully transferrable and can be used by a drug company to reduce the review time for an NDA or BLA from the standard 10 months to six months. Recently, two PRV's have each sold for more than $200 million.

Duchenne Muscular Dystrophy

Duchenne muscular dystrophy (DMD) represents an even more unconventional application of (Z)-endoxifen, however the company's rationale extends beyond the recently published hypothesis that (Z)-endoxifen may increase expression of utrophin (Remmel *et al.*, 2026). Atossa believes the compound could potentially address multiple downstream consequences of dystrophin deficiency through a combination of estrogen receptor modulation, PKC inhibition, and effects on several signaling pathways involved in inflammation, fibrosis, calcium homeostasis, mitochondrial function, and muscle regeneration.

The most recent work has focused on utrophin, a structural and functional homolog of dystrophin that can partially compensate for the absence of dystrophin by helping maintain muscle-cell membrane stability.

The May 2026 publication used transcriptomic and mechanistic analyses to propose that (Z)-endoxifen may increase utrophin expression and modulate pathways associated with myogenesis, oxidative phosphorylation, ERβ signaling, inflammation, and epithelial-to-mesenchymal transition. This hypothesis is particularly attractive because pharmacologic upregulation of utrophin would theoretically be independent of the specific dystrophin mutation carried by an individual patient.

However, the utrophin hypothesis represents only on component of Atossa's broader DMD rationale. The company's earlier work proposed (Z)-endoxifen could influence DMD through both estrogen-dependent and estrogen-independent mechanisms (Remmel *et al.*, 2025). In muscle, the effects of estrogen receptor modulation may differ from anti-estrogenic effects that underpin (Z)-endoxifen's activity in breast cancer. Estrogen receptors, particularly ERβ, are expressed in skeletal muscle and have been implicated in muscle regeneration, cellular survival, and adaptation to injury. Atossa hypothesizes that (Z)-endoxifen could promote protective estrogen signaling in muscle while simultaneously exerting effects through non-estrogen receptor pathways.

A second component of the hypothesis involves protein kinase C (PKC) signaling. Endoxifen is an allosteric inhibitor of PKC, and Atossa highlighted PKCβ1 as one potential target in DMD. PKC signaling intersects with downstream AKT/mTOR and NF-κB pathways, which are involved in muscle growth, inflammation, and cellular stress. The company believes that inhibiting dysregulated PKC signaling could therefore complement the effects of utrophin upregulation by reducing some of the inflammatory and degenerative processes that contribute to progressive muscle loss.

This multidimensional hypothesis is potentially important because DMD is not simply a disease of absent dystrophin. Loss of dystrophin initiates a cascade of membrane instability, muscle injury, inflammation, impaired regeneration, and fibrosis that progressively destroys functional muscle tissue. A therapy capable of simultaneously increasing compensatory structural proteins and attenuating downstream inflammatory and metabolic abnormalities could, in theory, provide broader disease modification than an approach directed at a single pathway.

Atossa has received both Rare Pediatric Disease designation and Orphan Drug designation from the FDA for (Z)-endoxifen in the treatment of DMD. We anticipate an update from the company on both the MAS and DMD programs in the third quarter of 2026.

Financial Update

On August 7, 2026, Atossa announced financial results for the second quarter of 2026. As expected, the company did not report any revenues for the quarter ending June 30, 2026. R&D expenses in the second quarter of 2026 totaled $4.9 million compared to $5.5 million for the second quarter of 2025. The decrease was primarily due to decreased costs for preclinical studies. G&A expenses for the second quarter of 2026 were $3.8 million compared to $3.5 million for the second quarter of 2025. The increase was primarily due to increased legal fees.

Atossa exited the second quarter of 2026 with approximately $26.1 million in cash and cash equivalents, partially due to the closing of a registered direct offering that resulted in aggregate gross proceeds to the company of $4.5 million with the potential for an additional approximately $12 million if the Series warrants are fully exercised on a cash basis. We estimate the company has sufficient capital to fund operations into the first half of 2026. As of July 31, 2026, Atossa had approximately 10.0 million shares outstanding and, when factoring in stock options and warrants, a fully diluted share count of 14.4 million.

Conclusion

Atossa is entering what we believe could be a very consequential period. With both EVANGELINE and I-SPY now fully enrolled and data from each expected before year-end, investors should soon have substantially greater visibility into whether the differentiated pharmacology of (Z)-endoxifen can translate into meaningful clinical benefit. In addition, near-term KARISMA durability data and the planned updates

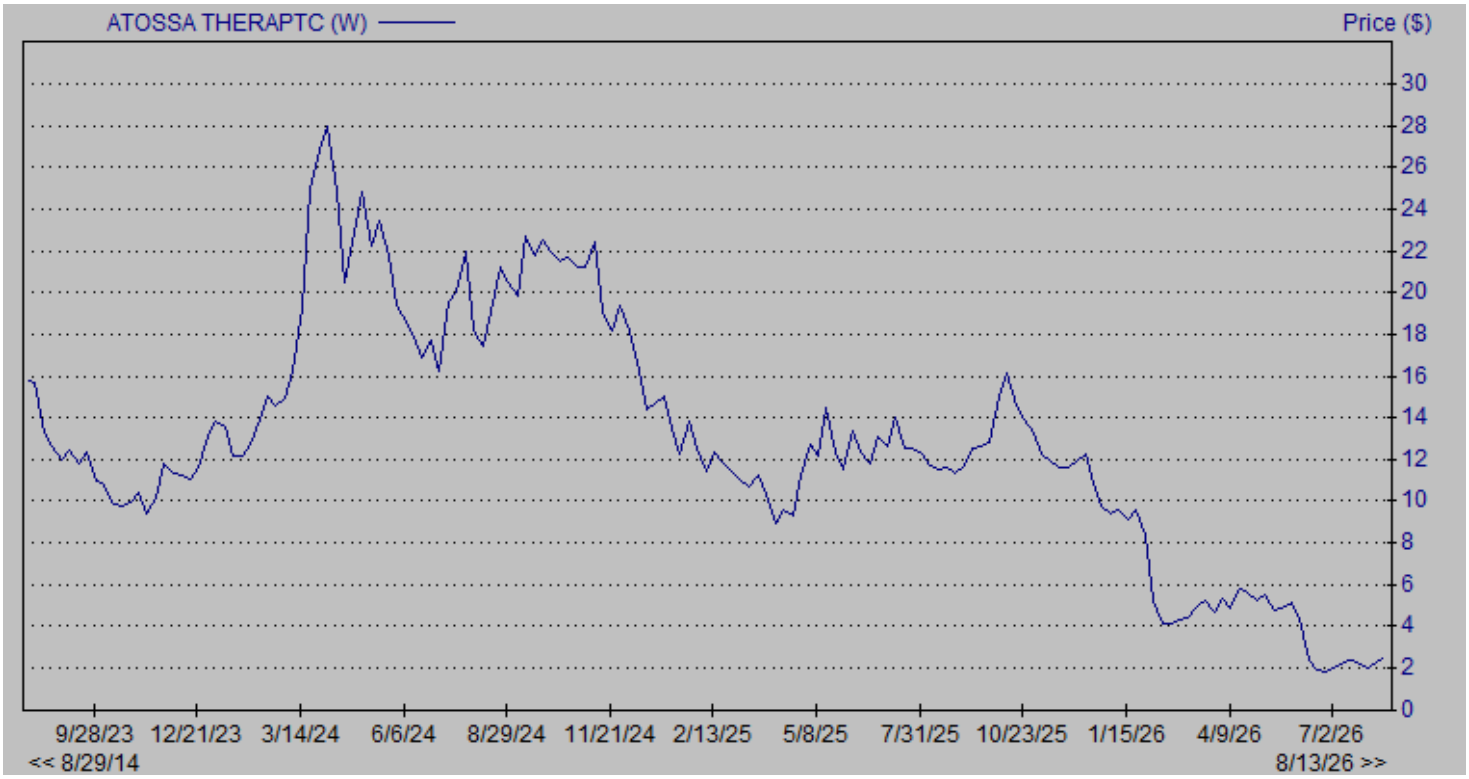
for the MAS and DMD programs provide additional opportunities to expand the company's addressable market. While meaningful clinical risk remains, we believe positive data from either EVANGELINE or I-SPY could materially improve the company's strategic and financial position. Our current valuation is $22 per share.

PROJECTED FINANCIALS

Atossa Therapeutics, Inc.	2025 A	Q1 A	Q2 A	Q3 E	Q4 E	2026 E	2027 E	2028 E
(Z)-Endoxifen	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
License and other revenues	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Total Revenues	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**	**$0.0**
Cost of revenues	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Research & development	$21.2	$4.8	$4.9	$4.0	$4.0	$17.7	$18.0	$20.0
General & administrative	$16.0	$5.1	$3.8	$3.8	$3.8	$16.5	$17.5	$18.0
Operating Income	($37.1)	($9.9)	($8.7)	($7.8)	($7.8)	($34.2)	($35.5)	($38.0)
Non-Operating Expenses (Net)	$2.4	$0.3	$0.2	$0.5	$0.5	$1.5	$0.0	$0.0
Pre-Tax Income	($34.8)	($9.6)	($8.5)	($7.3)	($7.3)	($32.7)	($35.5)	($38.0)
Income Taxes	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Net Income	**($34.8)**	**($9.6)**	**($8.5)**	**($7.3)**	**($7.3)**	**($32.7)**	**($35.5)**	**($38.0)**
Net Margin	-	-	-	-	-	-	-	-
Reported EPS	**($4.04)**	**($1.11)**	**($0.95)**	**($0.74)**	**($0.73)**	**($3.49)**	**($2.73)**	**($2.38)**
YOY Growth	-	-	-	-	-	-	-	-
Basic Shares Outstanding	8.6	8.6	8.9	9.9	10.0	9.4	13.0	16.0

Source: Zacks Investment Research, Inc. *David Bautz, PhD*

HISTORICAL STOCK PRICE



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