

Atossa Therapeutics, Inc.

Reports Q2. Breast cancer and rare diseases key data and milestones over the next year should be strong catalysts for stock. Raising P/T to \$25.

Reports Q2: Atossa recently (on August 7) reported its Q2 2026 (ending June) results. Net loss was \$8.5 million or EPS of \$(0.95), compared with our and consensus estimates of \$(1.02) – (1.10). There was no Q2 guidance. Atossa is a clinical stage drug development company so it generates no revenue.

Operating expenses: Operating expenses were \$8.7 million, down from \$9.9 million in Q1.

No guidance: Management did not provide forward guidance, but we believe ~\$7 million to be a reasonable near term quarterly cash burn rate.

Adjusting estimates: We are adjusting our 2026 EPS estimate to \$(3.56) from \$(4.40).

Endoxifen in development: Atossa has one main therapeutic drug candidate, Endoxifen for breast cancer and rare disease.

Breast cancer: Atossa's breast cancer drug under development is its proprietary form of Endoxifen which is being developed primarily in two settings: 1) to reduce tumor cell activity in breast cancer patients in the "window of opportunity" between diagnosis of breast cancer and surgery; and 2) for women with high mammographic breast density ("MBD") to reduce the density and/or to act as an adjunct to mammography. Endoxifen is being developed for use across the breast cancer spectrum, including prevention, neoadjuvant, adjuvant, and metastatic settings.

Key data upcoming: Key data from its Karisma-(Z)-endoxifen study (~Q3 2026), I-SPY (2H 2026), and EVANGELINE clinical trials (enrollment was complete in June 2026) are expected over the next year.

Rare disease focus: The company is progressing clinical development of Endoxifen for potentially broader utility as a therapeutic platform in serious and rare diseases with significant unmet medical need.

Duchenne Muscular Dystrophy: DMD is a progressive neuromuscular disease that primarily affects boys, leading to loss of muscle function, loss of ambulation, and life-threatening heart and respiratory complications. Atossa believes that (Z)-endoxifen's direct estrogen-receptor modulation, protein kinase C inhibition, and effects on key signaling pathways could be relevant in addressing various pathologies associated with DMD, including inflammation, fibrosis, and cardiomyopathy.

McCune-Albright Syndrome: MAS is a rare, non-inherited genetic disorder caused by a postzygotic activating mutation in GNAS, resulting in abnormal hormone signaling in affected tissues and involving bone, skin, and the endocrine system, with symptoms typically appearing in early childhood. Atossa is evaluating whether (Z)-endoxifen could reduce estrogen-mediated manifestations.

Other trials and indications: The company is making solid progress on its ongoing clinical trials. The company is also working with the FDA on regulatory strategies for other breast cancer indications and rare diseases with significant unmet needs.

Clinical data can be catalysts: Atossa anticipates finishing or making significant milestones in its various clinical trials over the next year. We believe achieving key clinical milestones and data will likely be strong catalysts for the stock.

To issue PRV rights: Atossa plans to Issue CVR (contingent value right) to shareholders tied to a potential receipt of a Rare Pediatric Disease Priority Review Voucher (PRV). These vouchers have sold for \$100 – 220 million in the past 2 years.

Positive high risks versus high rewards: Atossa's drugs still have long development roads left and the high risks of clinical trials failures, but we believe the ~billion dollars market potential presents high rewards for the risks.

Current valuation attractive: We are maintaining our BUY rating, but raising our 12-month price target to \$25 from \$24 based on a NPV analysis. This represents significant upside from the current share price. We believe this valuation appropriately balances out the company's high risks with the company's high growth prospects and large upside opportunities.

Company Description

Based in Seattle, WA, Atossa Therapeutics is a clinical-stage biopharmaceutical company developing innovative medicines in oncology and other areas of significant unmet need with its lead drug candidate (Z)-endoxifen.

United States
Healthcare

September 30, 2026

Edward Woo, CFA
(561) 327-9435
ewoo@ascendant.com

COMPANY UPDATE

Rating: BUY

Ticker: ATOS

Price: \$1.95

Target: \$25
(from \$24)

Stock Data

Exchange:	NasdaqCM
52-week Range:	\$1.70 – 19.35
Shares Outstanding (million):	12
Market cap (\$million):	\$24
EV (\$million):	\$(2)
Debt (\$million):	\$0
Cash (\$million):	\$26
Avg. Daily Trading Vol. (\$million):	\$0.3
Float (million shares):	10
Short Interest (million shares):	~0
Dividend, annual (yield):	\$0 (NA%)

Revenues (US\$ million)

	2026E (Cur.)	2026E (Old)	2027E (Cur.)	2027E (Old)
Q1 Mar	0A		0E	
Q2 Jun	0A	0E	0E	
Q3 Sep	0E		0E	
Q4 Dec	0E		0E	
Total	0E		0E	
EV/Revs	N/A		N/A	

Earnings per Share (pro forma)

	2026E (Cur.)	2026E (Old)	2027E (Cur.)	2027E (Old)
Q1 Mar	(1.11)A		(0.83)E	(1.10)E
Q2 Jun	(0.95)A	(1.10)E	(0.83)E	(1.10)E
Q3 Sep	(0.78)E	(1.10)E	(0.83)E	(1.09)E
Q4 Dec	(0.78)E	(1.10)E	(0.83)E	(1.09)E
Total	(3.56)E	(4.40)E	(3.30)E	(4.38)E
P/E	N/A		N/A	

*Reflects a 1:15 reverse stock split in February 2026.

Important Disclosures

Ascendant Capital Markets LLC seeks to do business with companies covered by its research team. Consequently, investors should be aware that the firm may have a conflict of interest that could affect the objectivity of this report. Investors should consider this report as only a single factor in making an investment decision.

For analyst certification and other important disclosures, refer to the Disclosure Section, located at the end of this report, beginning on page 20.

Exhibit 1: Atossa Therapeutics Investment Highlights

Investment Thesis: One Molecule, Multiple Value Streams

\$26.1M

cash and equivalents at June 30, 2026

No debt

no debt outstanding at June 30, 2026

~800

patients dosed with (Z)-endoxifen to date

3

FDA RPD for both MAS and DMD; FDA ODD for DMD

\$100 – \$220M

disclosed PRV sale range, last 18–24 months*

1

A Differentiated Molecule

- Oral SERM/SERD delivered as the active metabolite, bypassing the need for liver metabolism to achieve more consistent drug levels and improved tolerability
- Dual mechanism of action: blocks estrogen receptor (ER) signaling, promotes degradation of ER
- Growing global patent portfolio

2

Oncology – the Underwritten Core

- ER+/HER2 – breast cancer across neoadjuvant, adjuvant and metastatic settings
- EVANGELINE Phase 2 enrollment completed as of June 30, 2026
- Partnered I-SPY2 combinations with abemaciclib and elagolix keep oncology capital-sparing

3

Rare Disease – Regulatory-Validated Expansion

- Efficient development model: smaller patient pools allow streamlined, lower-cost studies with accelerated timelines to pivotal data readouts
- RPD designation for both MAS and DMD; ODD for DMD
- PRV eligibility upon approval of a qualifying marketing application
- IND clearance in MAS and DMD anticipated in 2H26

4

Disciplined Capital Deployment

- \$26.1M cash and cash equivalents and no debt at June 30, 2026; June registered direct offering raised \$4.0M (net)
- Up to \$12M additional from Series A and short-term Series B warrants, assuming full cash exercise
- Total operating expenses of \$8.7M in Q2 2026; spend directed behind defined regulatory pathways

Oncology underwrites the core while rare disease adds regulatory-validated optionality both from a single molecule, a single manufacturing process and one capital base.

Source: Company reports

Exhibit 2: Upcoming Milestones (as of October 2025)

Anticipated Upcoming Milestones

**Mid
2025**

- ✓ mBC Global Steering Committee
- ✓ mBC FDA Meeting
- ✓ mBC CRO Selection
- ✓ mBC PI Selection

**Fourth Quarter
2025***

- ✓ Early Disease FDA Meeting
- ✓ mBC 1st patient on path
- ✓ mBC IND Submission

**1st HALF
2026***

- ✓ Continued regulatory readouts for Early Disease
- ✓ I-Spy Combination Readout (neo)

**2nd HALF
2026***

- ✓ Evangeline Trial Update
- ✓ mBC Clinical Trial Update
- ✓ Continued regulatory readouts on Early Disease



More than a year of cash on hand at current burn rate and zero debt

Source: Company reports

Exhibit 3: Drug Pipeline (as of September 2026)

(Z)-Endoxifen – One Molecule, Two Markets

	PRECLINICAL	PHASE I	PHASE II	PHASE III
Rare Diseases				
McCune-Albright Syndrome (MAS) ER-targeting of precocious puberty in young girls	✓ 	IND clearance anticipated in 2H26, Ph 2 initiation in 2027		
Duchenne Muscular Dystrophy (DMD) Not specific to exon defect	✓	IND clearance anticipated in 2H26, Ph 2 initiation in 2027		
Adults with Dystrophinopathies Women carriers of DMD gene; men with Becker's disease				
Oncology - Neoadjuvant				
EVANGELINE ((Z)-Endoxifen + Goserelin) Premenopausal women with ER+/HER2- breast cancer		Final Ph2 data anticipated in 1H27		
I-SPY2 EOP ((Z)-Endoxifen Monotherapy) High-risk breast cancer (stage II-III)		Manuscript published in 2Q26		
I-SPY2 EOP ((Z)-Endoxifen + Abemaciclib) High-risk breast cancer (stage II-III)		Enrollment completed, top line data anticipated in 4Q26		
I-SPY2 EOP ((Z)-Endoxifen + Elagolix) High-risk breast cancer (stage II-III)		Enrollment completion anticipated in 4Q26		

✓ - Obtained Rare Pediatric Disease for DMD and MAS, Orphan Drug Designation for DMD
 - Orphan Drug Designation requested Q1 2026

(Z)-Endoxifen – Non-Oncology Indications

Indication	PRECLINICAL	PHASE I	PHASE II	PHASE III
Translational				
Duchenne Muscular Dystrophy ✓ Not specific to exon defect	Pre-IND Meeting, Q1 '26	Targeted FDA Meetings in '26		
Women carriers of DMD gene  Carriers with cardiomyopathy due to carrier gene expression	Pre-IND Meeting, H1 '26			
McCune-Albright Syndrome (MAS)  ER-targeting of precocious puberty in young girls	Pre-IND Meeting, H1 '26			
ESR1 Mutations In breast cancer	Pre-IND Meeting, Q3 '26			
Gynecological cancer Efficacy in ER+ ovarian, endometrial, etc. cancers	Pre-IND Meeting, Q3 '26			

✓ - Obtained Rare Pediatric Disease and Orphan Drug Designations
 - Will be seeking Pediatric Rare Disease and/or Orphan Drug Designations in 2026

Source: Company reports

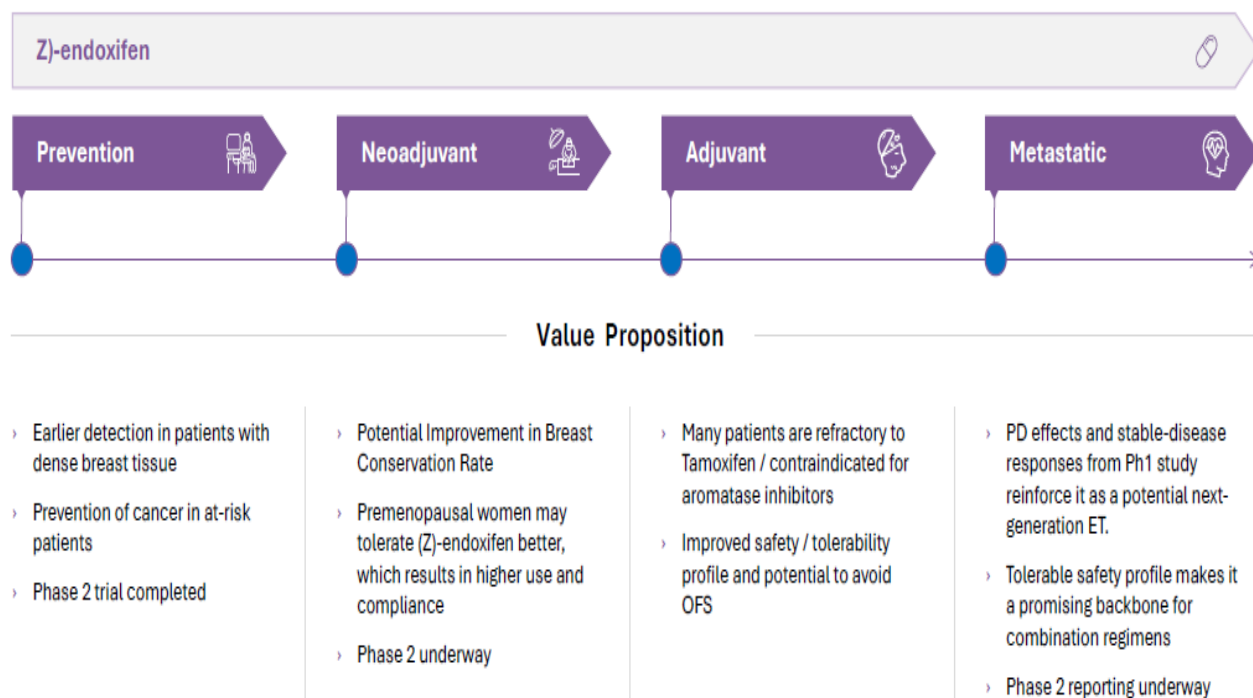
Exhibit 4: Atossa Market Opportunities - Oncology

Large Market Opportunities



PROGRAM		OPPORTUNITY
Mammographic Breast Density	➔	27 Million Women in U.S. with dense breasts ¹
Ductal Carcinoma In Situ (DCIS)	➔	60,000 DCIS diagnoses annually in U.S. ²
Neoadjuvant	➔	250,000 ER+ Breast Cancers/Yr. in U.S. ²

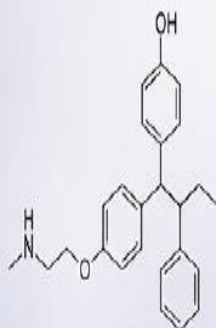
(Z)-endoxifen can be positioned
in all stages of ER+/HER2- breast cancer treatment paradigm



Source: Company reports.

(Z)-Endoxifen Is More Potent and Direct-Acting than Parent Drug Tamoxifen

(Z)-endoxifen is **30x to 100x** more potent as an estrogen receptor-targeted therapy compared with parent drug tamoxifen



2 Promotes degradation of the estrogen receptor

(Z)-Endoxifen delivers the active molecule directly—bypassing the need for liver metabolism to achieve more consistent drug levels and improved tolerability.

Blocked

Receptor (ER)

degradation

Exploring the therapeutic potential of our platform in rare diseases with significant unmet need.

Patients at the Center

Greater Potency
Engineered for increased
therapeutic activity

A line graph with a vertical y-axis and a horizontal x-axis. The line starts at a low point on the y-axis, rises to a peak, dips slightly, and then rises again to a higher peak than the first one.

More Consistent Drug Levels
Designed for predictable
absorption and exposure



Improved Tolerability
Optimized profile to support
long-term treatment

Source: Company reports.

Exhibit 6: Atossa – Rare Disease

(Z)-Endoxifen Targets Distinct Disease Biology Through Multiple Pathways

Estrogen-Driven Diseases

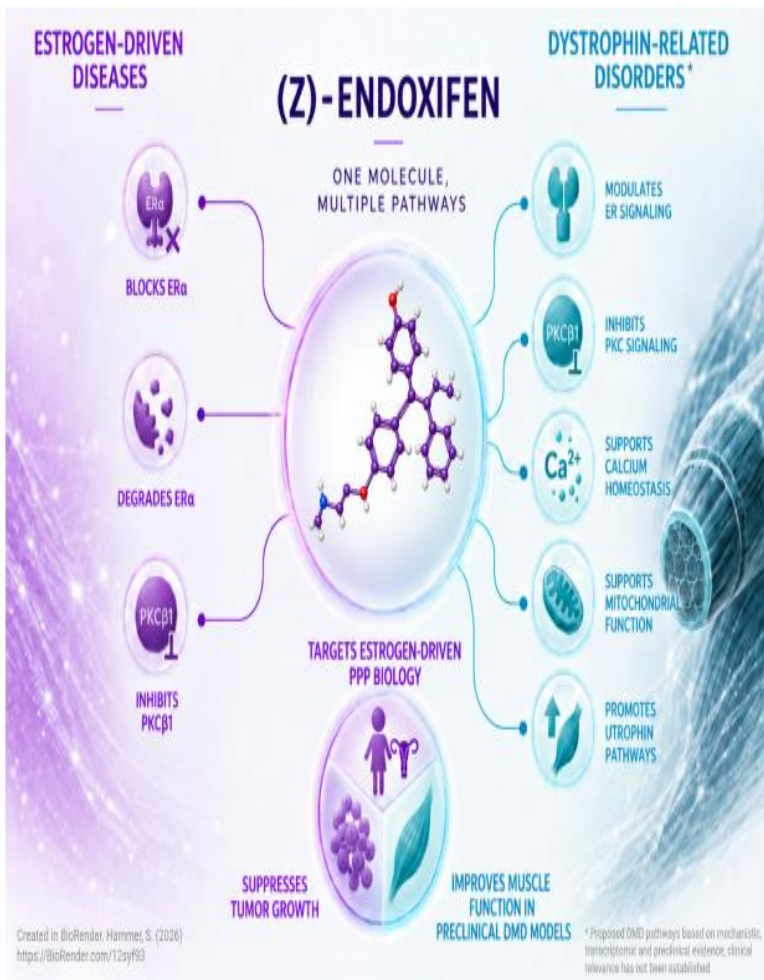
MAS and ER+ Breast Cancer

- Antagonizes ER α signaling and promotes ER α degradation, suppressing estrogen-driven proliferation
- Inhibits PKC β 1 and downstream AKT-associated proliferative signaling
- Targets estrogen-driven biology relevant to tumor growth and MAS-associated peripheral precocious puberty

Dystrophin-Related Disorders

DMD and Related Dystrophinopathies

- Mutation-agnostic therapeutic rationale, independent of specific DMD mutation class
- Modulates ER and inhibits PKC signaling to support calcium homeostasis and mitochondrial function
- May promote utrophin-associated compensatory pathways
- Improves muscle function in preclinical dystrophin-deficient models



Source: Company reports.

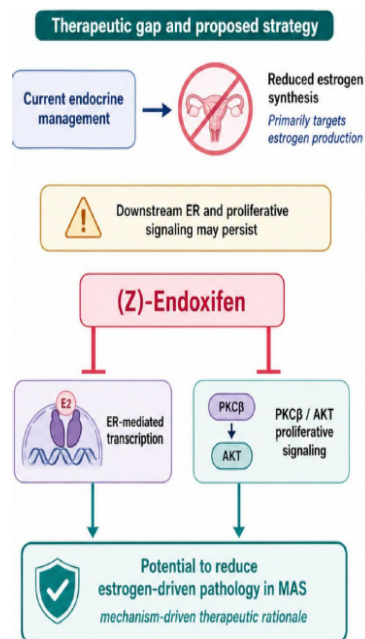
Exhibit 7: Atossa Market Opportunity – MAS and DMD Rare Diseases

Off-Label Agents Slow the Disease but None Deliver Complete Estrogen Receptor Blockade

The unmet need: no FDA- or EMA-approved therapy exists for MAS-PPP, and no agent provides consistent, complete estrogen receptor blockade at the growth plate. Every option available is off-label use of a generic molecule.

	Demonstrated Effect	Why It Falls Short
Aromatase Inhibitors (letrozole, anastrozole)	↓ bone age progression ($P \leq 0.004$) ↓ growth velocity ($P \leq 0.01$) - Fewer bleeding episodes	- Does not prevent ovarian cyst formation, estrogen suppression incomplete and variable - Bone age often continues to advance on therapy - Systemic estrogen suppression raises bone-accrual concern in a critical developmental window
SERMs (tamoxifen)	- Bleeding reduced ~65% - Bone age advancement slowed: $\Delta BA/\Delta CA$ improved from 1.21 to 0.72	- Partial agonist — uterine stimulation, increased uterine volume, endometrial proliferation - Ovarian volumes stay enlarged, reflecting incomplete ER blockade in target tissues - CYP2D6 prodrug dependence; ~7–10% poor metabolizers get inconsistent exposure
GnRH Agonists	Suppresses the central axis if secondary central precocious puberty emerges	Adjunct only — does not address the peripheral estrogen source driving the disease

SERM: selective estrogen receptor modulator; GnRH: gonadotropin-releasing hormone



(Z)-Endoxifen: One molecule, Six Downstream Pathways

Published preclinical studies and early clinical reports in DMD models suggest benefit across membrane integrity, calcium handling, muscle preservation and cardiac function

(Z)-Endoxifen may act downstream of the dystrophin mutation on six disease pathways, supported by SERM-class, clinical cardiac, and human iPSC-cardiomyocyte data

1 Utrophin pathway support

Tamoxifen upregulates utrophin, the dystrophin-like protein that compensates for absent dystrophin · Membrane localization ↑ · Membrane stability ↑

2 Membrane & calcium stabilization

Arrhythmic Ca^{2+} events 64% → 38% of cells (DMD1 iPSC-CMs) · Resting Ca^{2+} ratio ↓ · Faster time-to-peak at 1 Hz pacing

3 Reduced muscle damage

Cell survival at day 42: DMD1 17% → 29%, DMD2 16% → 34% · CK and histologic injury markers ↓ · Resistance to injury ↑

4 Inflammation & fibrosis

Anti-inflammatory and anti-fibrotic across preclinical DMD models · Oxidative stress ↓ · Collagen deposition ↓

5 PKCβ1 & cellular signaling

PKCβ1 activity ↓ · AKT signaling ↓ · Concordant with PKCβ1-silencing RNA-seq and phosphoproteomic data

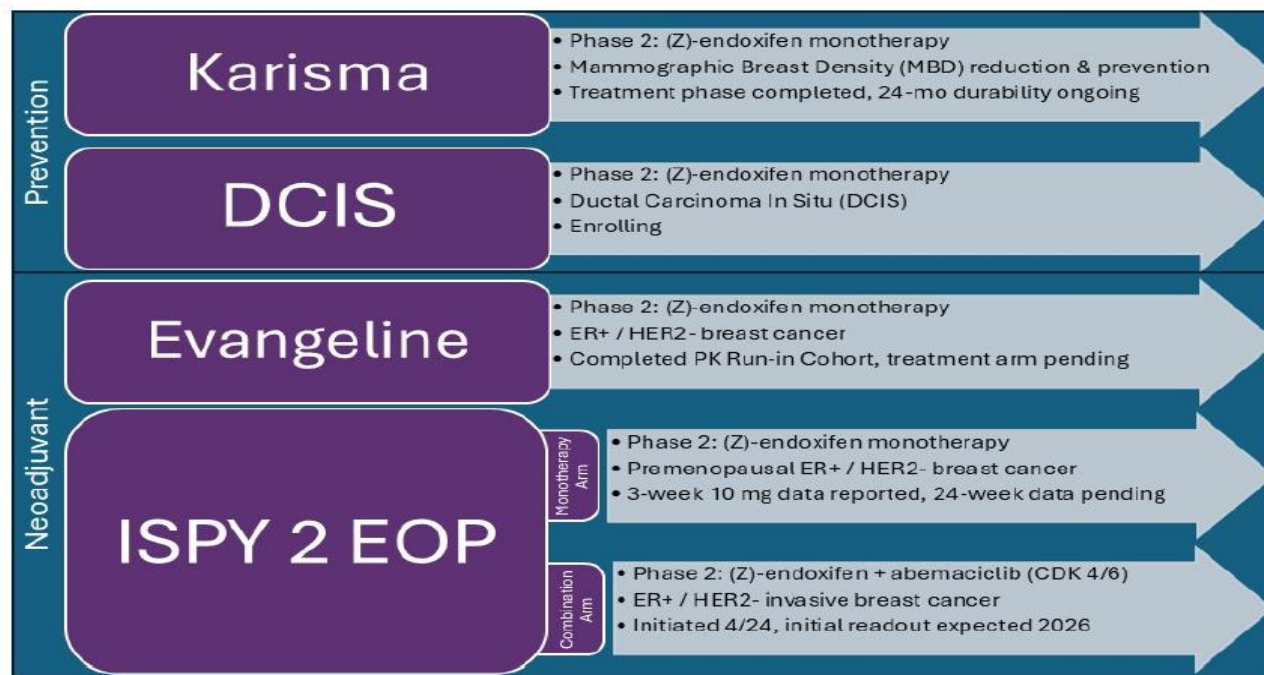
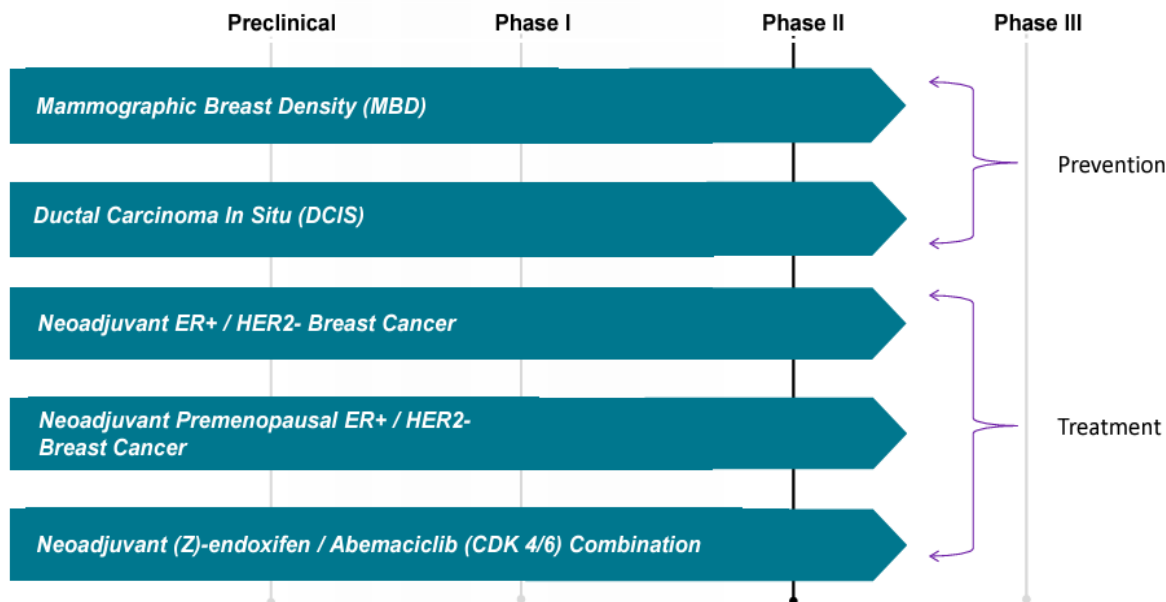
6 Cardiac function & muscle quality

Tamoxifen LVEDd 44 → 41 mm and FS 34 → 35%, vs placebo 39 → 43 mm and 35 → 33% · Beating velocity restored toward healthy · Lean mass ↑

Source: Company reports.

Exhibit 8: Atossa Drug Development Pipeline (as of March 2025)

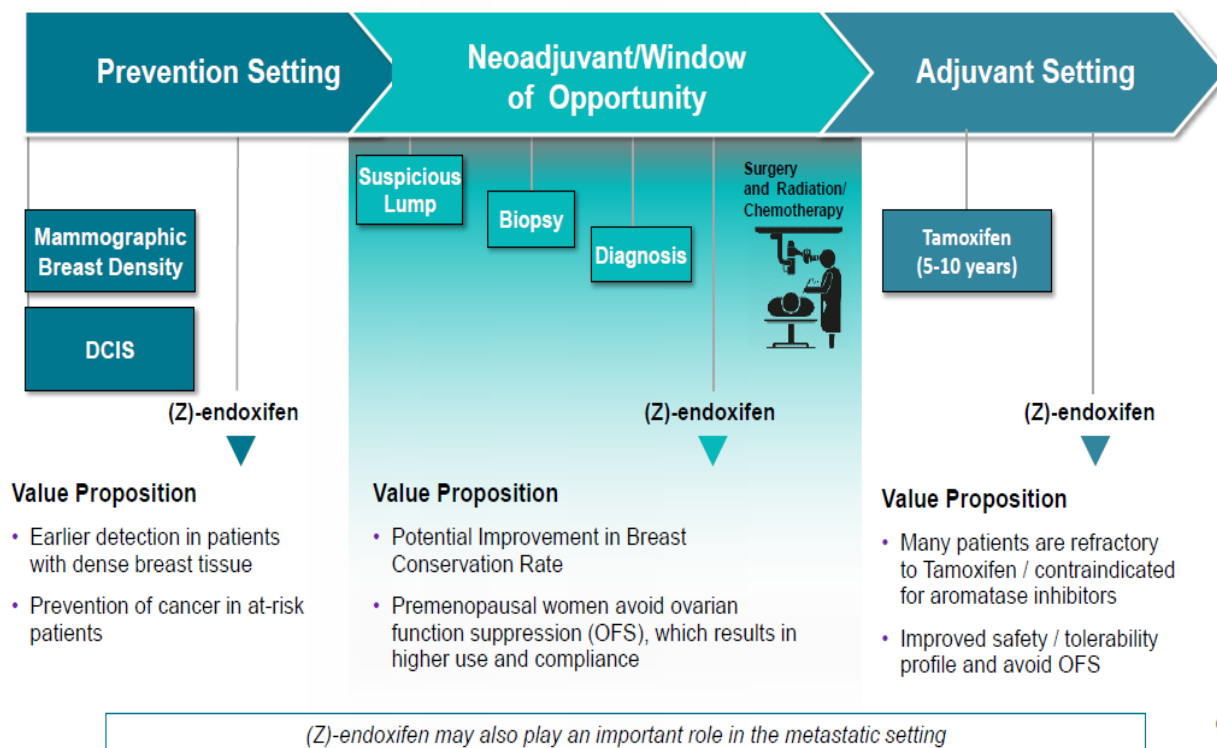
Development Pipeline



Source: Company reports

Exhibit 9: ENDOXIFEN

Clinical Positioning In Breast Cancer



6

The Breast Cancer Problem



Source: Company reports

Exhibit 10: (Z)-endoxifen's Points of Differentiation

(Z)-Endoxifen: Potential Best-in-Class SERM/SERD

Superior ER Antagonist



30x to 100x more potent vs other SERMs
Apoptosis in tumors
Estrogen receptor degradation (SERD)

PKCβ1 Inhibition



Binds to and inhibits protein kinase C beta one (PKCβ1, a known oncogenic protein)
Downregulates AKT pathway and induces apoptosis in breast cancer cells

ESR1 Mutant Inhibition



Inhibits clinically relevant *ESR1* mutants, an acquired resistance mechanism to aromatase inhibitors

Improved safety & tolerability



Potential to reduce current negative "on target off tissue" effects
May increase patient adherence

Superior Combination Partner



Potential to be preferred endocrine combination partner



(Z)-Endoxifen is a novel, next-generation anti-estrogen with best-in-class potential across breast cancer and other rare diseases with significant unmet need

Source: Company reports

Exhibit 11: (Z)-endoxifen

(Z)-Endoxifen Represents an Ideal Combination Partner in Breast Cancer



(Z)-Endoxifen with CDK4/6 inhibitors

- CDK4/6 inhibitors are FDA-approved combination partners for tamoxifen, aromatase inhibitors and fulvestrant
- The I-SPY2 trials will help confirm the hypothesis that (Z)-endoxifen could be a preferred endocrine therapy in combination with abemaciclib (CDK4/6 inhibitor)
- Neoadjuvant/adjuvant therapy for high-risk, node positive ER+/HER2- breast cancer, post surgery
- Other potential combinations have been preclinically tested and suggested, including PI3Ki, MDM2i and others



(Z)-Endoxifen plus Ovarian Function Suppression

- GnRH receptor antagonist for management of pain associated with endometriosis; FDA approved elagolix
- Off label usage in premenopausal women with HR+/HER2- breast cancer where ovarian function suppression (OFS) is required, but comes with severe side effects
- The I-SPY2 trials will help test the hypothesis that (Z)-endoxifen could be a preferred endocrine therapy in combination with elagolix for achieving and maintaining OFS for ~6 months

Source: Company reports

(Z)-endoxifen has the potential to be the preferred endocrine therapy for combination partners

The diagram illustrates the regulation of E2F and the cell cycle by the Rb protein. ERI (Estrogen Receptor Inhibitor) inhibits ESR1 (Estrogen Receptor 1), which in turn inhibits CCND1 (Cyclin D1). CCND1 forms a complex with CDK4/6 (Cyclin-Dependent Kinase 4/6), which is inhibited by p18 (p18^{INK4A}) and CDKI (Cyclin-Dependent Kinase Inhibitor). The Rb protein (Retinoblastoma protein), which is inhibited by E2F (E2F transcription factor), forms a complex with CDK4/6. The Rb protein is phosphorylated by CDK4/6, leading to the release of E2F. E2F then promotes the expression of E2F target genes, including those involved in DNA methylation and PD-L1 expression. The cell cycle is regulated by the E2F pathway, with G1, S, and M phases shown.

Improved adherence

Page 11

Exhibit 13: (Z)-Endoxifen Treatment Opportunities

Emerging (Z)-Endoxifen Treatment Opportunities



New Opportunities

(Z)-Endoxifen's mechanism of action goes beyond BC

Duchenne Muscular Dystrophy (DMD)

- Upregulation of utrophin
- Utrophin is not mutated in DMD (like dystrophin)
- Mutation agnostic approach with naturally expressed, functionally competent protein
- Not limited to specific exon mutations or nonsense errors

McCune-Albright Syndrome (MAS)

- Rare, non-inherited genetic disorder caused by a postzygotic GNAS mutation, onset at ages ≤ 2
- Affects bones, skin, and the endocrine system, including early onset puberty (precocious puberty)
- (Z)-endoxifen could prove to be an effective hormone blocker significantly reducing the effects of precocious puberty

Symptomatic Women Carriers of DMD

- Female DMD carriers have mosaic dystrophin loss, driving subclinical muscle and cardiac fibrosis.
- Endoxifen modulates estrogen signaling to reduce fibrosis and inflammation, independent of dystrophin restoration.
- Lowering the dystrophin functional threshold may preserve cardiac and muscle function in carriers.

Gynecological Cancers

- Modulates ER α transcriptional activity at low concentrations, inhibiting proliferation in ER+ endometrial cancer models.
- Inhibits PKC β , suppresses AKT signaling and induces apoptosis, with enhanced anti-tumor activity when combined with CDK4/6 inhibition (abemaciclib)

(Z)-Endoxifen for Duchenne Muscular Dystrophy (DMD)

- Potent SERM with muscle-protective, anti-inflammatory, and anti-fibrotic effects
- MDX Mouse Model of DMD
 - Improves muscle strength, motor performance, and phasic force in dystrophic models
 - Reduces plasma CK as well as muscle necrosis toward wild-type profiles
 - Provides cardiac functional benefits
- Builds on and shown to exceed clinical findings observed with tamoxifen
- Strong mechanistic and translational rationale for clinical development



Source: CureDuchenne

Source: Company reports

Exhibit 14: Q1 2026 Results and Recent Highlights (as of August 7, 2026)

Atossa Therapeutics Reports Second Quarter 2026 Financial Results and Provides a Corporate Update

SEATTLE, Aug. 7, 2026 /PRNewswire/ -- Atossa Therapeutics, Inc. (Nasdaq: ATOS) (Atossa or the Company), a clinical-stage biopharmaceutical company developing novel therapies in oncology and other areas of high unmet clinical need, today announced its financial results and provided an update on recent corporate developments for the second quarter ended June 30, 2026.

"During the quarter, we executed well across the business," stated Dr. Steven Quay, M.D., Ph.D., Atossa Therapeutics' President and Chief Executive Officer. "We continued to advance the scientific rationale for (Z)-endoxifen's potential in rare pediatric diseases, such as Duchenne Muscular Dystrophy and McCune-Albright Syndrome, while also strengthening its clinical and scientific foundation in breast cancer, all through new data presented at important industry conferences, including ASCO and AACR, as well as publications in well-regarded peer-reviewed journals, *Degenerative Neurological and Neuromuscular Disease* and *npj Breast Cancer*."

Dr. Quay continued, "These accomplishments, together with additional capital from our registered direct offering, underscore the breadth of potential we see for (Z)-endoxifen, and investors' support in our ability to continue advancing our programs."

Second Quarter 2026 & Recent Highlights

Rare Diseases

- **Atossa Participated at the 2026 American Association for Cancer Research (AACR) Special Conference in Cancer Research: Cancer Evolution**
 - In a poster presentation titled, "Dual estrogen receptor and PKC- β signaling modulation by (Z)-Endoxifen: A mechanism-driven therapeutic strategy for estrogen-driven pathology in McCune-Albright Syndrome," the Company discussed a dual mechanism of action for (Z)-endoxifen in estrogen-driven pathology relevant to McCune-Albright Syndrome-associated Peripheral Precocious Puberty (MAS-PPP).
 - The dual mechanism includes the blockade of estrogen receptor (ER)-mediated transcription downstream of autonomous estrogen production and suppression of PKC- β /AKT-associated proliferative and cell-cycle signaling.
 - This multi-pathway profile may address a key therapeutic gap in MAS-PPP, where estrogen suppression alone may not fully mitigate downstream proliferative signaling.
 - The Company previously received Rare Pediatric Disease (RPD) designation for (Z)-endoxifen from the U.S. Food and Drug Administration (FDA) for (Z)-endoxifen for the treatment of McCune-Albright Syndrome (MAS).
- **The Company Announced Acceptance of Manuscript Highlighting the Utrophin-Modulation Potential of (Z)-Endoxifen in Duchenne Muscular Dystrophy (DMD)**
 - A paper details how (Z)-endoxifen may support the expression of utrophin, a paralog of dystrophin. (Z)-Endoxifen therefore represents a potential dystrophin mutation-agnostic treatment for DMD. The paper citation is: Remmel HL, Hammer SS, Blackburn SM, Quay SC. (Z)-Endoxifen as a Potential Modulator of Utrophin Pathways in Duchenne Muscular Dystrophy: A Mechanistic and Transcriptomic Perspective. *Degener Neurol Neuromuscular Dis.* 2026;16:574524 <https://doi.org/10.2147/DNND.S574524>
 - The results support further investigation of (Z)-endoxifen in dystrophin-deficient models, as well as biomarker development. These data also build upon the Company's previously published manuscript, "[A Hypothesized Therapeutic Role of \(Z\)-Endoxifen in Duchenne Muscular Dystrophy](#)," also published in *Degenerative Neurological and Neuromuscular Disease*.
 - The Company previously received Orphan Drug Designation (ODD) and RPD designation for (Z)-endoxifen from the FDA for the treatment of DMD.

Source: Company reports

Exhibit 15: Q1 2026 Results and Recent Highlights (as of August 7, 2026)

Oncology

- **The Company Published Manuscript Highlighting the Anti-cancer Activity of (Z)-Endoxifen-related Compounds**
 - In the peer-reviewed journal, *npj Breast Cancer*, the Company published an article titled, "[Novel \(Z\)-endoxifen-related new chemical entities exhibit potent anti-cancer activity in ERα+ breast cancer.](#)"
 - The investigators evaluated five previously uncharacterized compounds generated during the synthesis of (Z)-endoxifen, alongside (Z)-endoxifen in a broad panel of laboratory assays, as well as in combination with the CDK4/6 inhibitor abemaciclib.
 - The publication reported anti-estrogenic and anti-cancer activity across multiple ER-positive (ER+) breast cancer models, including models harboring clinically relevant activating mutations in ESR1.
 - In certain experimental settings and models, selected compounds combined with abemaciclib demonstrated additive to synergistic activity that was comparable to or greater than the activity observed with abemaciclib plus (Z)-endoxifen.
 - The authors concluded that select compounds warrant further in vivo safety evaluation, as well as efficacy studies, including as potential second- or third-line approaches for recurrent disease.
- **Atossa Participated in the 2026 American Society of Clinical Oncology (ASCO) Annual Meeting**
 - In a poster presentation titled, "A Phase 2 Clinical Trial in Progress of (Z)-Endoxifen Plus Goserelin as Neoadjuvant Therapy in Premenopausal Women With ER+/HER2- Breast Cancer (EVANGELINE)," the Company described EVANGELINE (NCT05607004), an ongoing, multicenter, open-label Phase 2 study evaluating daily 40 mg (Z)-endoxifen plus goserelin administered every 28 days as neoadjuvant therapy in premenopausal women with ER+/human epidermal growth factor receptor 2 negative (HER2-), cT2-3, cN0-1 breast cancer. Enrollment in this study was completed as of June 30, 2026.
 - In an online publication titled, "Effect of (Z)-endoxifen Demonstrates Robust Estrogen Receptor Signaling Inhibition Across Clinically Relevant ESR1 Mutations," the Company highlighted new preclinical data demonstrating that (Z)-endoxifen delivers robust ER inhibition across clinically relevant estrogen receptor alpha gene (ESR1) mutations. ESR1 mutations are a major mechanism of acquired endocrine resistance in ER-positive breast cancer and remain associated with limited treatment options despite the emergence of next-generation endocrine therapies. These data support the ongoing clinical development of (Z)-endoxifen, as well as its potential as a promising treatment option for breast cancer patients with limited therapeutic alternatives.

Corporate

- **Atossa Strengthened its Balance Sheet with a Registered Direct Offering, Potentially Providing Up to \$16.5 Million in Gross Proceeds**
 - The Company entered into a securities purchase agreement with institutional investors, which provided for the issuance and sale by the Company, in a registered direct offering of (i) 1,363,637 shares of the Company's common stock and (ii) Series A warrants to purchase up to 1,363,637 shares of common stock and short-term Series B warrants to purchase up to 1,363,637 shares of common stock, raising \$4.5 million in upfront gross proceeds, with the potential to receive up to an additional \$12 million, assuming the full cash exercise of the warrants. Net proceeds are designated to support the clinical development of (Z)-endoxifen and for general corporate working capital.

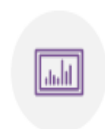
Source: Company reports

Exhibit 16: Investment Highlights Summary

(Z)-Endoxifen: Closing Summary



- **Oncology:** Underwritten core
- **Rare Disease:** Clinical-stage expansion, low-cost studies with accelerated timelines to pivotal data readouts



- **Platform**
 - Oral SERM/SERD; ~800 patients exposed, no maximum tolerated dose reached
 - Given as the active metabolite, consistent exposure, no dependence on conversion in liver
- **Rare Disease**
 - IND clearance in MAS and DMD anticipated in 2H26, with study initiations in 1H27
 - RPD designation for DMD and MAS; ODD for DMD; PRV eligible upon approval
- **Oncology**
 - Combination strategy is intentional and capital-sparing
 - EVANGELINE Phase 2 enrollment complete; I-SPY2 combinations partnered
- **Financials**
 - \$26.1M cash and equivalents and no debt outstanding at June 30, 2026
 - Funded into 2027; up to \$12M from Series A and Series B warrants if fully exercised
 - Opportunistically raising capital to initiate DMD and MAS studies

Source: Company reports

Exhibit 17: Atossa Therapeutics, Inc. Stock Price (5-years)



*Reflects a 1:15 reverse stock split in February 2026.

Source: <https://www.nasdaq.com/>, Chart IQ, EDGAR ONLINE.

Exhibit 18: Consensus Expectations (as of August 7, 2026)

Revenue (mils)			EPS		
	2026E	2027E		2026E	2027E
Q1 Mar	\$0A		Q1 Mar	\$(1.11)A	
Q2 Jun	\$0E		Q2 Jun	\$(1.02)E	
Q3 Sep	\$0E		Q3 Sep	\$(1.08)E	
Q4 Dec			Q4 Dec		
Total	\$0E	\$0E	Total	\$(4.30)E	\$(3.00)E

*Quarterly estimates may not add to annual estimates due to variations in contributing estimates and rounding

*Reflects a 1:15 reverse stock split in February 2026.

Source: Company report, LSEG, and Ascendant Capital Markets estimates

FINANCIAL MODEL

Atossa Therapeutics, Inc.

Income Statement (\$ mils)	Mar-24	Jun-24	Sep-24	Dec-24	2024	Mar-25	Jun-25	Sep-25	Dec-25	2025	Mar-26	Jun-26	Sep-26	Dec-26	2026	Mar-27	Jun-27	Sep-27	Dec-27	2027
Fiscal Year End: December 31	Q1A	Q2A	Q3A	Q4A	FY-A	Q1A	Q2A	Q3A	Q4A	FY-A	Q1A	Q2A	Q3E	Q4E	FY-E	Q1E	Q2E	Q3E	Q4E	FY-E
Total Revenue	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	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	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gross Profit	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Research and development	3.7	3.6	3.4	3.4	14.1	4.2	5.5	5.4	6.2	21.2	4.8	4.9	5.0	5.0	19.7	5.5	5.5	5.5	5.5	22.0
General and administrative	3.2	3.6	3.0	3.7	13.5	3.3	3.5	3.9	5.3	16.0	5.1	3.8	4.0	4.0	16.9	4.0	4.0	4.0	4.0	16.0
Restructuring and other					0.0					0.0					0.0					0.0
Total operating expenses	7.0	7.1	6.4	7.2	27.6	7.4	9.0	9.3	11.4	37.1	9.9	8.7	9.0	9.0	36.6	9.5	9.5	9.5	9.5	38.0
Operating income (loss)	(7.0)	(7.1)	(6.4)	(7.2)	(27.6)	(7.4)	(9.0)	(9.3)	(11.4)	(37.1)	(9.9)	(8.7)	(9.0)	(9.0)	(36.6)	(9.5)	(9.5)	(9.5)	(9.5)	(38.0)
Interest income (expense)	1.1	1.1	1.0	0.9	4.1	0.7	0.6	0.6	0.4	2.4	0.3	0.2	0.0	0.0	0.5	0.0	0.0	0.0	0.0	0.0
Other income (expense)			(1.8)	(0.1)	(1.9)	(0.0)	(0.0)	(0.0)	0.1	(0.0)	(0.0)	(0.0)	0.0	0.0	(0.1)	0.0	0.0	0.0	0.0	0.0
Income before income taxes	(5.9)	(6.0)	(7.2)	(6.3)	(25.5)	(6.7)	(8.4)	(8.7)	(10.9)	(34.8)	(9.6)	(8.5)	(9.0)	(9.0)	(36.1)	(9.5)	(9.5)	(9.5)	(9.5)	(38.0)
Income taxes					0.0					0.0					0.0					0.0
Net income (loss)	(5.9)	(6.0)	(7.2)	(6.3)	(25.5)	(6.7)	(8.4)	(8.7)	(10.9)	(34.8)	(9.6)	(8.5)	(9.0)	(9.0)	(36.1)	(9.5)	(9.5)	(9.5)	(9.5)	(38.0)
Nonrecurring/noncash adjustments					0.0					0.0					0.0					0.0
Net income (pro forma)	(5.9)	(6.0)	(7.2)	(6.3)	(25.5)	(6.7)	(8.4)	(8.7)	(10.9)	(34.8)	(9.6)	(8.5)	(9.0)	(9.0)	(36.1)	(9.5)	(9.5)	(9.5)	(9.5)	(38.0)
EBITDA																				
Shares, Basic	8.4	8.4	8.4	8.4	8.4	8.6	8.6	8.6	8.6	8.6	8.6	8.9	11.5	11.5	10.1	11.5	11.5	11.5	11.5	11.5
Shares, Diluted	8.4	8.4	8.4	8.4	8.4	8.6	8.6	8.6	8.6	8.6	8.6	8.9	11.5	11.5	10.1	11.5	11.5	11.5	11.5	11.5
EPS Basic (pro forma)	(\$0.70)	(\$0.72)	(\$0.86)	(\$0.75)	(\$3.04)	(\$0.78)	(\$0.98)	(\$1.01)	(\$1.27)	(\$4.04)	(\$1.11)	(\$0.95)	(\$0.78)	(\$0.78)	(\$3.56)	(\$0.83)	(\$0.83)	(\$0.83)	(\$0.83)	(\$3.30)
EPS Diluted (pro forma)	(\$0.70)	(\$0.72)	(\$0.86)	(\$0.75)	(\$3.04)	(\$0.78)	(\$0.98)	(\$1.01)	(\$1.27)	(\$4.04)	(\$1.11)	(\$0.95)	(\$0.78)	(\$0.78)	(\$3.56)	(\$0.83)	(\$0.83)	(\$0.83)	(\$0.83)	(\$3.30)
Margins																				
Gross margin																				
Research and development																				
General and administrative																				
Operating margin	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
Tax rate, GAAP	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%	0%
Net margin	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM	NM
Y/Y % change																				
Total Revenue																				
Gross margin																				
Research and development	7%	-4%	-24%	-40%	-19%	11%	55%	57%	81%	50%	15%	-11%	-7%	-19%	-7%	15%	12%	10%	10%	12%
General and administrative	-10%	-13%	-1%	11%	-4%	1%	0%	31%	41%	18%	56%	7%	3%	-24%	6%	-21%	5%	0%	0%	-5%
Operating income (loss)	-2%	-9%	-15%	-21%	-12%	6%	27%	45%	60%	34%	33%	-4%	-3%	-21%	-2%	-4%	9%	6%	6%	4%
Net income (loss)	-6%	-38%	16%	-18%	-15%	14%	39%	20%	72%	36%	43%	1%	4%	-18%	4%	-1%	12%	6%	6%	5%
EPS Diluted (pro forma)	-5%	-38%	16%	-19%	-15%	11%	36%	17%	69%	33%	43%	-3%	-22%	-38%	-12%	-26%	-13%	6%	6%	-7%

Source: Company reports and Ascendant Capital Markets estimates.

Reflects a 1:15 reverse stock split in February 2026

Atossa Therapeutics, Inc.

Balance Sheet (\$ mils)	Mar-24	Jun-24	Sep-24	Dec-24	Mar-25	Jun-25	Sep-25	Dec-25	Mar-26	Jun-26	Sep-26	Dec-26	Mar-27	Jun-27	Sep-27	Dec-27
Fiscal Year End: December 31	Q1A	Q2A	Q3A	Q4A	Q1A	Q2A	Q3A	Q4A	Q1A	Q2A	Q3E	Q4E	Q1E	Q2E	Q3E	Q4E
Assets																
Cash and cash equivalents	84.0	79.5	74.8	71.1	65.1	57.9	51.8	41.3	31.7	26.1	18.0	9.8	1.2	(7.5)	(16.2)	(24.9)
Short term investments											0.0	0.0	0.0	0.0	0.0	0.0
Restricted cash	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
Research and development tax rebate receivable											0.0	0.0	0.0	0.0	0.0	0.0
Deferred income taxes											0.0	0.0	0.0	0.0	0.0	0.0
Prepaid expenses and other	3.0	2.1	2.2	3.3	3.5	4.6	3.7	4.2	4.8	4.8	4.8	4.8	4.8	4.8	4.8	4.8
Total current assets	87.1	81.7	77.1	74.5	68.7	62.5	55.7	45.6	36.7	31.0	22.9	14.8	6.1	(2.6)	(11.3)	(20.0)
Property and equipment, net											(0.0)	(0.0)	(0.0)	(0.0)	(0.0)	(0.0)
Intangibles, net											0.0	0.0	0.0	0.0	0.0	0.0
Deferred income tax											0.0	0.0	0.0	0.0	0.0	0.0
Other	4.0	4.1	2.4	2.0	2.0	2.0	2.3	2.0	1.3	1.3	1.3	1.3	1.3	1.3	1.3	1.3
Total assets	91.1	85.9	79.5	76.4	70.7	64.5	58.0	47.6	37.9	32.3	24.2	16.0	7.3	(1.3)	(10.0)	(18.7)
Liabilities and stockholders' equity																
Accounts payable	1.2	1.1	1.6	0.7	1.2	2.0	3.5	4.3	2.6	2.0	2.0	2.0	2.0	2.0	2.0	2.0
Accrued expenses	1.6	1.9	1.7	2.8	1.8	2.2	2.0	2.9	2.8	1.4	1.4	1.4	1.4	1.4	1.4	1.4
Deferred income tax											0.0	0.0	0.0	0.0	0.0	0.0
Warrant liabilities											0.0	0.0	0.0	0.0	0.0	0.0
Other	2.5	2.7	2.5	1.5	2.5	2.6	2.7	1.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1	2.1
Short term debt											0.0	0.0	0.0	0.0	0.0	0.0
Total current liabilities	5.3	5.7	5.8	5.0	5.4	6.8	8.2	8.3	7.5	5.5	5.5	5.5	5.5	5.5	5.5	5.5
Deferred income taxes											0.0	0.0	0.0	0.0	0.0	0.0
Warrant liabilities											0.0	0.0	0.0	0.0	0.0	0.0
Other long term liabilities											0.0	0.0	0.0	0.0	0.0	0.0
Long term debt											0.0	0.0	0.0	0.0	0.0	0.0
Total other liabilities	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Preferred stock											0.0	0.0	0.0	0.0	0.0	0.0
Common stock	22.829	22.9	22.9	23.5	23.5	23.5	23.5	1.6	1.6	1.8	2.6	3.4	4.2	5.1	5.9	6.7
Additional paid-in capital	255.096	255.5	256.2	259.8	260.3	261.1	261.9	284.4	285.1	289.7	289.7	289.7	289.7	289.7	289.7	289.7
Retained earnings	(192.166)	(198.2)	(205.4)	(211.8)	(218.5)	(226.9)	(235.6)	(246.6)	(256.2)	(264.6)	(273.6)	(282.6)	(292.1)	(301.6)	(311.1)	(320.6)
Accumulated other comprehensive income											0.1	0.1	0.1	0.1	0.1	0.1
Total stockholders' equity	85.759	80.2	73.7	71.5	65.3	57.7	49.8	39.4	30.5	26.8	18.8	10.6	1.9	(6.8)	(15.5)	(24.2)
Total stockholders' equity and liabilities	91.087	85.9	79.5	76.4	70.7	64.5	58.0	47.6	37.9	32.3	24.2	16.0	7.3	(1.3)	(10.0)	(18.7)

Balance Sheet Drivers

	Mar-24	Jun-24	Sep-24	Dec-24	Mar-25	Jun-25	Sep-25	Dec-25	Mar-26	Jun-26	Sep-26	Dec-26	Mar-27	Jun-27	Sep-27	Dec-27
	Q1A	Q2A	Q3A	Q4A	Q1A	Q2A	Q3A	Q4A	Q1A	Q2A	Q3E	Q4E	Q1E	Q2E	Q3E	Q4E
Book & Cash Value (per share)																
Book Value per Share (diluted)	\$10.26	\$9.56	\$8.79	\$8.46	\$7.59	\$6.70	\$5.78	\$4.57	\$3.54	\$3.01	\$1.63	\$0.92	\$0.16	-\$0.59	-\$1.35	-\$2.10
Cash per Share (diluted)	\$10.05	\$9.49	\$8.92	\$8.42	\$7.56	\$6.72	\$6.02	\$4.80	\$3.68	\$2.93	\$1.57	\$0.86	\$0.10	-\$0.65	-\$1.41	-\$2.16
Net cash per Share (diluted)	\$10.05	\$9.49	\$8.92	\$8.42	\$7.56	\$6.72	\$6.02	\$4.80	\$3.68	\$2.93	\$1.57	\$0.86	\$0.10	-\$0.65	-\$1.41	-\$2.16

Source: Company reports and Ascendant Capital Markets estimates

Atossa Therapeutics, Inc.

Cash Flow Statement (\$ mils)	Mar-24	Jun-24	Sep-24	Dec-24	2024	Mar-25	Jun-25	Sep-25	Dec-25	2025	Mar-26	Jun-26	Sep-26	Dec-26	2026	Mar-27	Jun-27	Sep-27	Dec-27	2027
Fiscal Year End: December 31	Q1A	Q2A	Q3A	Q4A	FY-A	Q1A	Q2A	Q3A	Q4A	FY-A	Q1A	Q2A	Q3E	Q4E	FY-E	Q1E	Q2E	Q3E	Q4E	FY-E
Cash flow from operating activities																				
Net income	(5.9)	(6.0)	(7.2)	(6.3)	(25.5)	(6.7)	(8.4)	(8.7)	(10.9)	(34.8)	(9.6)	(8.5)	(9.0)	(9.0)	(36.1)	(9.5)	(9.5)	(9.5)	(9.5)	(38.0)
Depreciation	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Amortization					0.0					0.0					0.0					0.0
Debt related amortization expense					0.0					0.0					0.0					0.0
Stock comp	0.4	0.4	0.7	0.8	2.3	0.6	0.8	0.8	0.5	2.6	0.7	0.8	0.8	0.8	3.2	0.8	0.8	0.8	0.8	3.3
Deferred rent					0.0					0.0					0.0					0.0
A/R reserves					0.0					0.0					0.0					0.0
Deferred income taxes					0.0					0.0				0.0	0.0	0.0	0.0	0.0	0.0	0.0
Change in fair value of warrant liability					0.0					0.0					0.0					0.0
Writedowns and impairments			1.7		1.7					0.0					0.0					0.0
Other gains/losses	0.0	0.0	(0.0)	0.0	0.0					0.0					0.0					0.0
Other					0.0					0.0					0.0					0.0
Changes in operating assets and liabilities:																				
Prepaid expenses & other curre	0.7	0.9	(0.2)	(1.0)	0.4	0.0	(1.3)	0.2	0.1	(1.0)	0.1	(0.7)	0.0	0.0	(0.6)	0.0	0.0	0.0	0.0	0.0
Research and development tax rebate receivable					0.0					0.0					0.0					0.0
Income tax					0.0					0.0	0.7				0.7					0.0
Other assets		(0.1)	0.1	0.4	0.3	(0.3)	0.3	0.2	(0.2)	0.0	(0.7)	0.7	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Accounts payable	0.4	(0.2)	0.5	(0.9)	(0.1)	0.5	0.8	1.5	0.8	3.6	(1.7)	(0.6)	0.0	0.0	(2.3)	0.0	0.0	0.0	0.0	0.0
Accrued expenses	(0.4)	1.3	(0.2)	(0.8)	(0.1)	0.9	0.4	(0.2)	(0.7)	0.4	1.5	(1.4)	0.0	0.0	0.1	0.0	0.0	0.0	0.0	0.0
Other liabilities	0.0	(0.7)	(0.2)	0.8	(0.1)	(0.9)	0.1	0.1	(0.0)	(0.7)	(0.6)	0.0	0.0	0.0	(0.6)	0.0	0.0	0.0	0.0	0.0
Net cash (used in) provided by	(4.7)	(4.5)	(4.8)	(7.1)	(21.0)	(6.0)	(7.3)	(6.0)	(10.5)	(29.8)	(9.581)	(9.644)	(8.2)	(8.2)	(35.6)	(8.7)	(8.7)	(8.7)	(8.7)	(34.7)
Cash flow from investing activities																				
Purchases of property and equip	(0.0)	(0.0)	(0.0)		(0.0)	(0.0)	0.0	(0.0)	(0.0)	(0.0)				0.0	0.0	0.0	0.0	0.0	0.0	0.0
Purchases of short-term investments					0.0					0.0					0.0					0.0
Acquisitions					0.0					0.0					0.0					0.0
Other					0.0					0.0					0.0					0.0
Net cash used in investing acth	(0.0)	(0.0)	(0.0)	0.0	(0.0)	(0.0)	0.0	(0.0)	(0.0)	(0.0)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Cash flow from financing activities																				
Issuance of debt					0.0					0.0				0.0	0.0	0.0	0.0	0.0	0.0	0.0
Repayment of debt					0.0					0.0					0.0					0.0
Issuance of stock					0.0					0.0			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Proceeds from stock option exe	0.2	0.1		3.4	3.7					0.0		4.0			4.0					0.0
Other					0.0					0.0					0.0					0.0
Dividends and distributions					0.0					0.0					0.0					0.0
Cash provided by (used in) fina	0.2	0.1	0.0	3.4	3.7	0.0	0.0	0.0	0.0	0.0	0.0	4.0	0.0	0.0	4.0	0.0	0.0	0.0	0.0	0.0
Effect of exchange rate on cash					0.0					0.0					0.0					0.0
Net increase (decrease) in cash	(4.5)	(4.4)	(4.8)	(3.7)	(17.4)	(6.0)	(7.3)	(6.0)	(10.5)	(29.8)	(9.6)	(5.6)	(8.2)	(8.2)	(31.6)	(8.7)	(8.7)	(8.7)	(8.7)	(34.7)
Beginning cash and equivalents	88.6	84.1	79.6	74.9	88.6	71.2	65.2	58.0	52.0	71.2	41.4	31.8	26.2	18.0	41.4	9.8	1.2	(7.5)	(16.2)	9.8
Ending cash and equivalents	84.1	79.6	74.9	71.2	71.2	65.2	58.0	52.0	41.4	41.4	31.8	26.2	18.0	9.8	9.8	1.2	(7.5)	(16.2)	(24.9)	(24.9)

Source: Company reports and Ascendant Capital Markets estimates

ANALYST CERTIFICATION

Each analyst hereby certifies that the views expressed in this report reflect the analyst's personal views about the subject securities or issuers. Each analyst also certifies that no part of the analyst's compensation was, is, or will be, directly or indirectly, related to the specific recommendations or views expressed in this report. The analyst who prepared this report is compensated based upon the overall profitability of Ascendant Capital Markets, LLC, which may, from time to time, include the provision of investment banking, financial advisory and consulting services. Compensation for research is based on effectiveness in generating new ideas for clients, performance of recommendations, accuracy of earnings estimates, and service to clients.

Atossa Therapeutics, Inc.



*Reflects a 1:15 reverse stock split in February 2026.

Source: <https://www.nasdaq.com/>, Chart IQ, EDGAR ONLINE.

	Report Date		Price
Report	Date	Rating	Target
11	3/27/2023	Buy	82.50
12	5/23/2023	Buy	78.75
13	9/6/2023	Buy	82.50
14	12/22/2023	Buy	86.25
15	5/1/2024	Buy	90.00
16	6/5/2024	Buy	93.75
17	9/10/2024	Buy	97.50
18	12/7/2024	Buy	105.00
19	4/20/2025	Buy	108.75
20	6/5/2025	Buy	112.50
21	9/21/2025	Buy	116.25
22	12/7/2025	Buy	120.00
23	4/9/2026	Buy	24.00

- Ascendant Capital Markets, LLC has not received compensation for advisory or investment banking services from the company in the past 12 months.

IMPORTANT DISCLOSURES

This report has been distributed by Ascendant Capital Markets, LLC and is for the sole use of our clients. This report is based on current public information that we consider reliable, but we do not represent it is accurate or complete, and it should not be relied on as such. This report contains information from various sources, including United States government publications, The Wall Street Journal and other periodicals, Yahoo! Finance and other sources, and is for informational purposes only and is not a recommendation to trade in the securities of the companies mentioned within the report. We seek to update our research and recommendations as appropriate, but the large majority of reports are published at irregular intervals as we consider appropriate and, in some cases, as constrained by industry regulations.

We may have a business relationship with companies covered in this report. Ascendant Capital Markets, LLC may make a market in the securities of the subject company. We and our affiliates, officers, directors, and employees will from time to time have long or short positions in, act as principal in, and buy or sell, the securities or derivatives (including options and warrants) thereof of covered companies referred to in this report. This report is not an offer to sell or the solicitation of an offer to buy any security in any jurisdiction where such an offer or solicitation would be illegal. It does not constitute a personal recommendation or take into account the particular investment objectives, financial situations, or needs of individual clients. Clients should consider whether any information in this report is suitable for their particular circumstances and, if appropriate, seek professional advice, including tax advice. The price and value of the investments referred to in this report may fluctuate.

Following are some general risks that can adversely impact future operational and financial performance and share price valuation: (1) industry fundamentals with respect to legislation, mandates, incentives, customer demand, or product pricing; (2) issues relating to competing companies or products; (3) unforeseen developments with respect to management, financial condition or accounting policies or practices; or (4) external factors that affect the interest rates, currency, the economy or major segments of the economy. Past performance is not a guide to future performance, future returns are not guaranteed, and loss of original capital may occur. Certain transactions, including those involving futures, options, and other derivatives, give rise to substantial risk and are not suitable for all investors. Our report is disseminated primarily electronically, and, in some cases, in printed form. The information contained in this report is not incorporated into the contents of our website and should be read independently thereof. Copyright Ascendant Capital Markets, LLC. No part of this material may be copied, photocopied or duplicated by any means or redistributed without the prior written consent of Ascendant Capital Markets, LLC.

Risks & Considerations

Risks to attainment of our share price target include balance sheet/liquidity risks, failure of product candidates to demonstrate safety and efficacy in clinical trials, failure to gain regulatory approvals, ability to commercialize product, failure to obtain suitable reimbursement, competition, changing macroeconomic factors, investor sentiment for investing in biotech stocks, and changes in consumer or government priorities for healthcare.

Ascendant Capital Markets, LLC Rating System

BUY: We expect the stock to provide a total return of 15% or more within a 12-month period.

HOLD: We expect the stock to provide a total return of negative 15% to positive 15% within a 12-month period.

SELL: We expect the stock to have a negative total return of more than 15% within a 12-month period.

Total return is defined as price appreciation plus dividend yield.

Ascendant Capital Markets, LLC Distribution of Investment Ratings (as of July 18, 2026)

Rating	Count	Percent	Investment Banking Services Past 12 months	
			Count	Percent
Buy	49	98%	19	39%
Hold	0	0%	0	0%
Sell	1	2%	0	0%
Total	50	100%	19	38%

Other Important Disclosures

Our analysts use various valuation methodologies including discounted cash flow, price/earnings (P/E), enterprise value/EBITDAS, and P/E to growth rate, among others. Risks to our price targets include failure to achieve financial results, product risk, regulatory risk, general market conditions, and the risk of a change in economic conditions.

Dissemination of Research

Ascendant Capital Markets, LLC research is distributed electronically via the Thomson Reuters platforms, Bloomberg, Capital IQ and FactSet. Please contact your investment advisor or institutional salesperson for more information.

General Disclaimer

The information and opinions in this report were prepared by Ascendant Capital Markets, LLC. This information is not intended to be used as the primary basis of investment decisions and because of individual client objectives it should not be construed as advice designed to meet the particular investment needs of any investor. This material is for information purposes only and is not an offer or solicitation with respect to the purchase or sale of any security. The reader should assume that Ascendant Capital Markets, LLC may have a conflict of interest and should not rely solely on this report in evaluating whether or not to buy or sell securities of issuers discussed herein. The opinions, estimates, and projections contained in this report are those of Ascendant Capital Markets, LLC as of the date of this report and are subject to change without notice. Ascendant Capital Markets, LLC endeavors to ensure that the contents have been compiled or derived from sources that we believe are reliable and contain information and opinions that are accurate and complete. However, Ascendant Capital Markets, LLC makes no representation or warranty, express or implied, in respect thereof, takes no responsibility for any errors and omissions contained herein, and accepts no liability whatsoever for any loss arising from any use of, or reliance on, this report or its contents. Information may be available to Ascendant Capital Markets, LLC, or its affiliates that is not reflected in this report. This report is not to be construed as an offer or solicitation to buy or sell any security.

Additional Disclosures

Ascendant Capital Markets, LLC is a broker-dealer registered with the United States Securities and Exchange Commission (SEC) and a member of the FINRA and SIPC. Ascendant Capital Markets, LLC is not a Registered Investment Advisor nor is it an investment advisor registered with the Securities and Exchange Commission or with the securities regulators of any state, and at the present time is not eligible to file for federal registration.