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David Bautz, PhD
312-265-9471
dbautz@zacks.com

scr.zacks.com
101 N. Wacker Drive, Chicago, IL 60606

Atossa Therapeutics, Inc.

(ATOS-NASDAQ)

ATOS: Initiating Coverage of Atossa Therapeutics; Endoxifen's Expanding Opportunity from Oncology to Orphan Disease

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 (05/13/26)	$5.50
Valuation	**$22.00**

OUTLOOK

We are initiating coverage of Atossa Therapeutics, Inc. (ATOS) with a valuation of $22.00. Atossa is a clinical-stage biopharmaceutical company focused on rare diseases and oncology with a significant unmet need. The lead development compound is (Z)-endoxifen, which is being advanced across multiple indications, including McCune-Albright Syndrome (MAS), breast cancer, and other endocrine-driven conditions. (Z)-endoxifen is an active metabolite of tamoxifen, an FDA-approved drug used to treat and prevent breast cancer. Many patients lack the ability to adequately metabolize tamoxifen to (Z)-endoxifen, thus direct administration of the drug can achieve consistent therapeutic levels across patients. The company is currently evaluating (Z)-endoxifen in multiple Phase 2 trials, with a strategic focus on rare disease indications and combination therapy approaches in breast cancer.

SUMMARY DATA

52-Week High	**$17.69**
52-Week Low	**$3.94**
One-Year Return (%)	**-56.17**
Beta	**1.25**
Average Daily Volume (sh)	**59,834**
Shares Outstanding (mil)	**9**
Market Capitalization ($mil)	**$47**
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	**-1.2**
P/E using 2027 Estimate	**-2.2**

Risk Level	**Above Avg.**
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 E	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	-$0.98 E	-$1.02 E	-$1.08 E	-$4.19 E
2027					-$3.59 E
2028					-$3.00 E

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WHAT'S NEW

Initiating Coverage

Source: Atossa Therapeutics, Inc.

We are initiating coverage of Atossa Therapeutics, Inc. (ATOS) with a valuation of $22.00. Atossa is a clinical-stage biopharmaceutical company that is repositioning (Z)-endoxifen, an active metabolite of tamoxifen, as a multi-indication endocrine therapy with applications in both rare diseases and oncology. Tamoxifen is a pro-drug and must be metabolized to its active components, however many patients lack the liver enzymes to adequately metabolize the drug and thus do not reach therapeutic levels of (Z)-endoxifen. The direct administration of (Z)-Endoxifen addresses many of the shortcomings of tamoxifen due to the fact that it is not a pro-drug and does not require liver metabolism, thus adequate therapeutic levels of the compound are more easily attained with fewer side effects compared to tamoxifen.

Strategic Pivot to Rare Diseases – Atossa has recently shifted its development focus toward rare disease indications, with McCune-Albright Syndrome (MAS) emerging as a potential lead program. MAS is a rare pediatric endocrine disorder characterized by precocious puberty and other hormone-driven abnormalities, with no approved disease-modifying therapies currently available. We believe MAS represents the most efficient and potentially expedited path to market for (Z)-endoxifen given its well-defined biology and high unmet medical need. In addition, the company recently received Rare Pediatric Disease designation (which makes it eligible for a Priority Review Voucher upon approval) and will be pursuing Orphan Drug Designation.

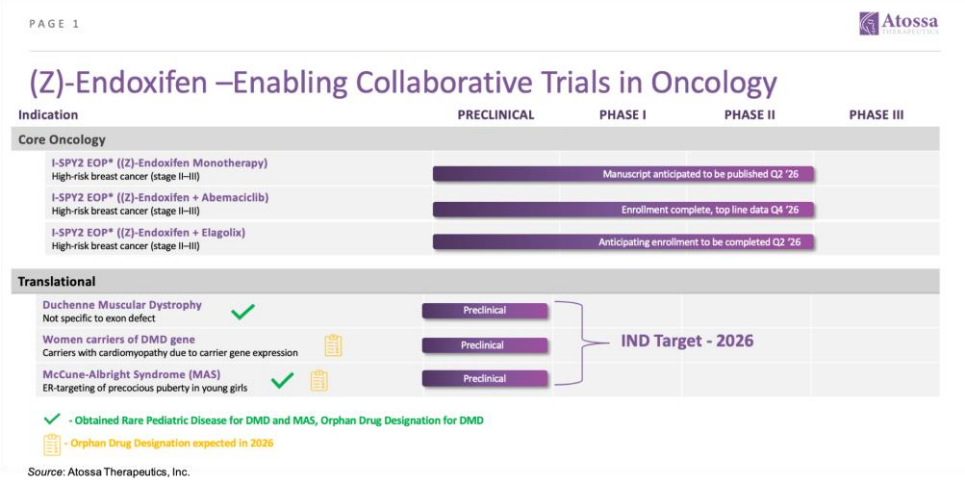
Expansion into Additional Rare Disease Indications – Beyond MAS, Atossa is expanding into other rare disease opportunities, including Duchenne muscular dystrophy (DMD) and symptomatic female carriers of DMD (D-CAP). D-CAP in particular lacks approved therapies and shares key pathological features with DMD, including inflammation, fibrosis, and muscle degeneration. The dual mechanism of (Z)-endoxifen, combining estrogen receptor modulation with protein kinase C (PKC) inhibition, provides a strong rationale for therapeutic activity in these conditions.

Refocused Oncology Strategy Centered on Partnerships – While breast cancer remains an important component of the pipeline, Atossa is shifting away from a single-agent commercialization strategy in metastatic disease toward a partnership-driven approach focused on combination therapy. The company's participation in the I-SPY 2 trial platform highlights this strategy, with (Z)-endoxifen being evaluated both as a monotherapy and in combination with agents such as abemaciclib (Verzenio®) and elagolix (Orlissa®). These studies are designed to optimize endocrine therapy, particularly in premenopausal women, where current treatment approaches often require ovarian suppression and are associated with significant side effects.

Regulatory and Development Progress – Following a Type C meeting with the U.S. Food and Drug Administration (FDA) in November 2025, Atossa received feedback on potential expedited regulatory pathways and development strategies for (Z)-endoxifen. The company is expected to pursue additional regulatory interactions in 2026, including pre-IND discussions related to its rare disease programs. We anticipate that the shift toward indications such as MAS could enable more streamlined clinical development relative to larger oncology indications.

INVESTMENT THESIS

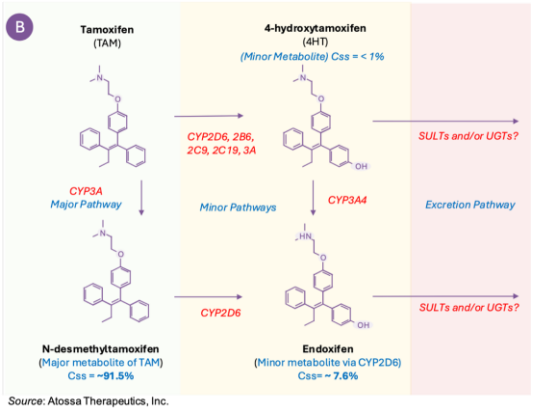
Atossa Therapeutics, Inc. (ATOS) is a clinical-stage biopharmaceutical company developing oral (Z)-endoxifen as a multi-indication endocrine therapy across rare diseases and oncology. (Z)-endoxifen is an active metabolite of tamoxifen, an FDA-approved therapy for the prevention and treatment of estrogen receptor (ER)-positive breast cancer. Tamoxifen itself has weak affinity for the ER and requires metabolic activation via hepatic cytochrome P450 enzymes to generate active metabolites, including (Z)-endoxifen, which exhibit significantly higher receptor affinity. Direct administration of (Z)-endoxifen eliminates the need for metabolic conversion, enabling more consistent therapeutic exposure across patient populations. (Z)-endoxifen has been evaluated in multiple Phase 1 and Phase 2 clinical trials and is currently being studied in several ongoing Phase 2 programs. Following recent regulatory interactions, Atossa has expanded its development strategy to prioritize rare disease indications, including McCune-Albright Syndrome (MAS), while continuing to advance breast health and cancer programs through combination and partnering approaches.



Source: Atossa Therapeutics, Inc.

(Z)-Endoxifen

(Z)-endoxifen is the primary active metabolite of tamoxifen and functions as a potent modulator of estrogen receptor (ER) signaling. Unlike tamoxifen, which is a pro-drug requiring hepatic metabolism via cytochrome P450 enzymes (predominantly CYP2D6) to generate active metabolites (see figure below), (Z)-endoxifen is administered directly, thereby eliminating variability associated with metabolic activation and enabling more consistent systemic exposure across patient populations (Higgins *et al.*, 2010; Ahmad *et al.*, 2010).).



Source: Atossa Therapeutics, Inc.

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CYP2D6 is responsible for metabolizing approximately 25% of all pharmaceutical agents, and more than 100 different genetic variants of *CYP2D6* have been identified that can be pooled into four different phenotypes: extensive (normal activity), intermediate (reduced activity), poor (no activity), and ultrarapid (high activity) metabolism (Ingelman-Sundberg *et al*., 2007). This is particularly important given that polymorphisms in CYP2D6, the primary enzyme responsible for converting tamoxifen to endoxifen, can significantly impact drug efficacy, with reduced metabolizers exhibiting lower circulating endoxifen levels and poorer clinical outcomes (Schroth *et al*., 2009; Madlensky *et al*., 2011).

(Z)-endoxifen exhibits significantly higher affinity (>10x) for the ER compared to tamoxifen and has demonstrated potent antiestrogenic activity across multiple preclinical and clinical settings (Johnson *et al*., 2004; Wu *et al*., 2009). In addition to its direct effects on ER signaling, (Z)-endoxifen has been shown to exert ER-independent activity through inhibition of protein kinase C (PKC), a family of serine/threonine kinases involved in regulating cell proliferation, apoptosis, inflammation, and cellular differentiation (Jayaraman *et al*., 2023). This dual mechanism that combines endocrine modulation with kinase pathway inhibition differentiates (Z)-endoxifen from traditional selective estrogen receptor modulators (SERMs) and may broaden its therapeutic applicability beyond hormone-driven cancers.

Preclinical studies have demonstrated that (Z)-endoxifen inhibits cellular proliferation and modulates signaling pathways such as PI3K/AKT, which are implicated in a wide range of disease processes including oncology, fibrosis, and inflammatory disorders (Jayaraman *et al*., 2020). In addition, transcriptomic and proteomic analyses have shown that (Z)-endoxifen alters phosphorylation signaling networks without significantly affecting total protein expression, supporting a targeted mechanism of action at the signaling pathway level (Jayaraman *et al*., 2023). These findings suggest that (Z)-endoxifen may be capable of modulating multiple disease-relevant pathways simultaneously.

Beyond its effects on cellular signaling, (Z)-endoxifen has also demonstrated activity in physiological systems relevant to both endocrine and musculoskeletal health. Preclinical models have shown positive effects on bone density and cortical bone mass, consistent with the known effects of tamoxifen, indicating potential utility in conditions where bone health is compromised (Gingery *et al*., 2014; Gingery *et al*., 2017). In addition, its influence on estrogen-regulated pathways suggests potential applicability in diseases characterized by hormonal dysregulation, including rare endocrine disorders.

Clinically, (Z)-endoxifen has been evaluated in multiple Phase 1 and Phase 2 trials and has been administered to hundreds of patients, demonstrating a well-characterized safety and pharmacokinetic profile. Across studies, the compound has been generally well tolerated, with no maximum tolerated dose identified in dose-escalation trials and a safety profile consistent with endocrine modulation (Goetz *et al*., 2017; Takebe *et al*., 2021). Importantly, therapeutic drug levels are achieved rapidly and predictably following oral administration, supporting its use in indications requiring consistent systemic exposure.

In summary, the pharmacological profile of (Z)-endoxifen supports its development across multiple disease settings, particularly those driven by aberrant estrogen signaling. We believe this is most clearly exemplified in McCune-Albright Syndrome (MAS), a rare pediatric endocrine disorder characterized by estrogen-driven precocious puberty and a lack of effective treatment options. As such, Atossa is prioritizing MAS as a lead indication, where the mechanism of (Z)-endoxifen is directly aligned with disease biology and may enable a more streamlined clinical and regulatory path to market.

McCune-Albright Syndrome

McCune-Albright Syndrome (MAS) is a rare, non-inherited mosaic genetic disorder characterized by a triad of fibrous dysplasia (FD) of bone, café-au-lait skin pigmentation, and endocrine hyperfunction, most notably gonadotropin-independent precocious puberty (Boyce *et al*., 2020). MAS is caused by postzygotic activating mutations in the *GNAS* gene, which encodes the stimulatory G protein α-subunit (Gsα), leading to constitutive activation of cyclic AMP (cAMP) signaling pathways in affected tissues (Weinstein *et al*., 1991). Because the mutation occurs after fertilization, the disease exhibits a mosaic distribution, resulting in highly variable clinical presentation depending on the tissues affected.

There is little reliable epidemiological data on the prevalence of MAS, however the literature suggests a prevalence estimated to range from approximately 1 in 100,000 to 1 in 1,000,000 individuals, corresponding to up to approximately 80,000 patients worldwide and roughly 3,500 patients in the United States (Dumitrescu *et al*., 2008). Clinical manifestations typically arise in early childhood, with endocrine abnormalities representing a major source of morbidity. Among these, precocious puberty, particularly in girls, is one of the most common and clinically significant features, often presenting as early as two years of age (Collins *et al*., 2012). This early estrogen exposure can result in accelerated skeletal maturation, reduced adult height, psychological distress, and long-term reproductive complications.

Management of MAS remains largely symptomatic and multidisciplinary, with no approved disease-modifying therapies currently available. Treatments for endocrine manifestations, including precocious puberty in girls, often involve off-label use of aromatase inhibitors, selective estrogen receptor modulators (SERMs), or gonadotropin-releasing hormone (GnRH) analogs, each of which has variable efficacy and potential safety limitations (Spencer *et al*., 2019). As such, there remains a significant unmet medical need for safe and effective therapies that can modulate estrogen signaling in this patient population.

(Z)-endoxifen may be particularly well-suited for the treatment of MAS-associated precocious puberty due to its direct and potent antiestrogenic activity. As the active metabolite of tamoxifen, (Z)-endoxifen exhibits high affinity binding to the estrogen receptor (ER) and does not require metabolic activation, allowing for more consistent pharmacokinetics and therapeutic exposure across patients (Johnson *et al*., 2004; Madlensky *et al*., 2011). In MAS, where estrogen-driven pathology is a central feature, (Z)-endoxifen could function as a targeted endocrine modulator to suppress premature estrogen signaling and delay pubertal progression and premature bone maturation.

Prior Experience with Tamoxifen in MAS

Importantly, tamoxifen and aromatase inhibitors (AIs) have previously been evaluated in the treatment of precocious puberty in MAS, demonstrating the clinical relevance of estrogen modulation in this disease. A selection of these studies is discussed below.

Eugster *et al*., 2003: This was a one-year multicenter study of tamoxifen treatment to evaluate its effect on precocious puberty in girls with MAS. The study enrolled 28 girls ≤ 10 years with classic or atypical MAS and 25 competed the 12 months treatment. Participants were treated with 20 mg tamoxifen daily. Outcomes were assessed through diaries to record bleeding along with physical examinations, pelvic ultrasound, hormone levels, and safety assessments. Compared with before the study, vaginal bleeding episodes decreased (3.42 ± 3.36/year vs 1.17 ± 1.41/year), growth velocity slowed (SDS 1.22 ± 2.65 vs -0.59 ± 3.06, P=0.005), and rate of bone maturation decreased (1.21 ± 0.78 vs 0.72 ± 0.36, P=0.02). Tamoxifen was well tolerated by all the participants and there were no significant adverse events, no effect on liver enzymes, and no apparent side effects.

Passone *et al*., 2015: This was a retrospective analysis of eight female patients with MAS that initially presented with café-au-lait spots and gonadotropin-independent precocious puberty. Patients were treated with tamoxifen (10-20 mg/day) for 3-8 years. Treatment resulted in cessation of vaginal bleeding in all patients and stabilization of bone age (BA) maturation based on the ratio of BA/chronological age (1.82 ± 0.56 vs 1.14 ± 0.11, P<0.01) along with a significant difference between the final height prediction at the beginning of treatment and at the end of treatment (145.1 ± 8.6 cm vs 153.5 ± 9.6 cm, P<0.01). Tamoxifen was well tolerated by all participants and there were no reports of adverse events.

Estrada *et al*., 2016: This was a retrospective cohort analysis of 28 girls suffering from MAS that were treated with letrozole for an average of 4.1 ± 2.6 years with a mean follow up of 6.0 ± 3.3 years. The results showed that letrozole was effective at decreasing the rate of skeletal maturation and predicted adult height Z-scores increased from -2.9 ± 3.2 to -0.8 ± 1.5 (P=0.004). In addition, four subjects who completed treatment reached adult height Z-scores ranging from -1.5 to 1.7, which was increased

compared to untreated historical controls (*P*=0.02). There were no adverse events reported over the treatment period

While the results described above for tamoxifen are encouraging, variability in its metabolism may limit its utility. Letrozole has also demonstrated clinical efficacy, including decrease skeletal maturation and improvements in predicted adult height. However, the mechanistic difference between (Z)-endoxifen (direct targeting of estrogen receptor) and letrozole (suppression of systemic estrogen production) may favor endoxifen.

- In MAS, estrogen production is intermittent and driven by autonomously functioning ovarian cysts. (Z)-endoxifen may provide a more physiologically balanced approach to estrogen signaling compared to complete estrogen suppression, potentially preserving beneficial effects in tissues such as bone.

- (Z)-endoxifen also functions as a PKC signaling inhibitor, which introduces a secondary mechanism that may address broader disease biology beyond estrogen alone. Letrozole does not offer a secondary mechanism of action aside from systemic estrogen suppression.

We believe the aforementioned features support the potential for (Z)-endoxifen to offer a differentiated therapeutic approach in MAS, either as an alternative or complement to existing off-label treatments such as AIs and could become the first approved therapy specifically for MAS. Atossa recently announced the FDA granted Rare Pediatric Disease designation to (Z)-endoxifen for the treatment of MAS, which makes the company eligible to receive a Priority Review Voucher (PRV) upon approval. PRVs are fully transferrable and have recently sold for $150 million-$200 million, which could be a valuable source of non-dilutive capital. Atossa will also be pursuing Orphan Drug Designation, which offers development incentives including market exclusivity and the potential for accelerated regulatory pathways.

Given the relatively small patient population and significant unmet need, we believe clinical development in MAS could be more streamlined compared to larger oncology indications. From a commercial standpoint, orphan drug pricing dynamics support a compelling opportunity despite the limited patient population. Assuming an annual treatment cost in the range of $100,000 to $150,000, consistent with other rare endocrine and pediatric indications, and peak penetration of approximately 30–40% of the addressable population, we estimate that MAS could represent a meaningful revenue opportunity within the broader rare disease and endocrinology markets.

(Z)-Endoxifen for the Treatment of DMD

In November 2025, Atossa announced the potential for (Z)-endoxifen in Duchenne muscular dystrophy (DMD), a severe, progressive, and fatal neuromuscular disease, along with Duchenne carrier-associated pathologies (D-CAPs) that affect a subset of female carriers. A new publication titled 'A Hypothesized Therapeutic Role of (Z)-Endoxifen in Duchenne Muscular Dystrophy (DMD)' discusses the current DMD treatment landscape and how (Z)-endoxifen could target a number of disease drivers, including inflammation, fibrosis, calcium dysregulation, mitochondrial dysfunction, and lipid abnormalities (Remmel *et al*., 2025).

Tamoxifen has previously been tested in DMD patients. In a Phase 1 trial of tamoxifen up to 20 mg/day, patients exhibited increased muscle retention and respiratory function when compared to age- and performance-matched historical controls (Tsabari *et al*., 2021). These encouraging results were followed up in a Phase 3 trial that evaluated tamoxifen treatment over 48 weeks. While slower disease progression was noted in the tamoxifen group, the trial did not reach the primary endpoint of change in patient's ambulation or any of the secondary outcomes (Henzi *et al*., 2023). It should be noted that the trial was terminated early due to the COVID-19 pandemic before the planned number of patients could be recruited. In addition, the trial did not evaluate the effects of tamoxifen on respiratory function or cardiovascular function, both of which are known to be impacted by tamoxifen treatment.

(Z)-Endoxifen is likely a better candidate to test as a DMD therapy than tamoxifen due to its variability in bioavailability following tamoxifen dosing. The use of (Z)-endoxifen eliminates this issue and provides for a more reliable circulating endoxifen level. There are a number of different pathways that are postulated to be affected by (Z)-endoxifen in the context of DMD, including:

- Estrogen Receptors – There are two main types of ERs that mediate cellular estrogen activity: nuclear ERs (ERα and ERβ) and membrane ERs (GPER/GPR30) (Fuentes *et al*., 2019). Both ERα and ERβ are expressed in cardiac and skeletal muscle of men and women, with ERα predominant in adipose tissue and ERβ predominant in skeletal muscle. In men, ERα and ERβ mRNA expression increases in skeletal muscle with endurance training, suggesting a possible role for them in adaptation to exercise (Wiik *et al*., 2005).

- Calcium Signaling – DMD causes disruptions in muscle calcium homeostasis along with increased intracellular calcium levels. This alteration leads to cellular damage, necrosis, and contraction-induced muscle damage. Over the long term, this damage leads to fibrosis and increased adipose tissue, thus therapies that can regulate calcium levels in muscle cells could mitigate some of the symptoms of DMD. The effect of (Z)-endoxifen on intracellular calcium level is unknown, however a similar selective estrogen receptor modulator (SERM) showed a dose-dependent protective effect against calcium influx in U2OS cells (Getter *et al*., 2019).

- Protein Kinase C (PKC) – The PKC family of protein is implicated not just in DMD but in other muscular diseases as well. PKC-Θ is of particular interest in DMD as it is involved in skeletal muscle regulation and chronic inflammatory responses. In a mouse model of DMD, genetic ablation of *Prkcq* (the PKC-Θ gene) led to reduced inflammation and improved muscle healing and regeneration along with a reduction in NF-κB activation (Madaro *et al*., 2012). PKC-β, a known target of (Z)-endoxifen, is also involved in NF-kB activation, however how this relates to DMD pathogenesis is currently unknown. The role of PKC-β in DMD progression is actively being studied, however several studies have shown that activation of PKC-β leads to muscle damage in DMD by promoting inflammatory processes and modifying calcium signaling in muscle fibers.

- Bone Mineralization – Both tamoxifen and (Z)-endoxifen are known to have positive effects on skeletal bone, including higher bone mineral density and bone mineral content along with increased expression of osteoblast, osteoclast, and osteocyte marker genes (Gingery *et al*., 2014). Promoting bone density and preventing bone loss are both important factors that could benefit DMD patients.

In summary, there is a large body of both theoretical and clinical evidence to support testing (Z)-endoxifen in DMD patients. The goal of endoxifen therapy would not be curative, but instead to be used as a safe, palliative treatment to aid in mitigating DMD symptoms, which could provide patients time in which curative therapies are developed. The company is planning a pre-IND meeting with the FDA in the first half of 2026 to discuss the development of (Z)-endoxifen for DMD.

Female Carriers of DMD

In addition to DMD, Atossa is expanding its development focus to include female carriers of DMD who exhibit clinical manifestations of the disease, often referred to as Duchenne carrier-associated pathologies (D-CAPs). While DMD is an X-linked recessive disorder that primarily affects males, a subset of female carriers develop symptoms due to skewed X-chromosome inactivation, leading to reduced dystrophin expression in skeletal and cardiac muscle (Hoogerwaard *et al*., 1999; Florian *et al*., 2016).

It is estimated that approximately 10–20% of female DMD carriers exhibit clinically significant symptoms, including progressive muscle weakness, fatigue, and cardiomyopathy, with cardiac involvement representing a major source of morbidity and mortality in this population. Despite this, there are currently

no approved therapies specifically indicated for symptomatic female carriers, and treatment is generally limited to supportive care and management of cardiac complications.

The pathophysiology of D-CAP shares many features with classical DMD, including chronic inflammation, fibrosis, calcium dysregulation, and progressive muscle degeneration. These overlapping mechanisms provide a strong rationale for evaluating therapies that target multiple downstream pathways involved in muscle damage and repair.

(Z)-endoxifen may offer a novel therapeutic approach for D-CAP due to its combined estrogen receptor modulation and protein kinase C (PKC) inhibition. Estrogen signaling plays an important role in muscle physiology, with both ERα and ERβ expressed in skeletal and cardiac muscle tissue (Wiik *et al.*, 2005). Modulation of estrogen pathways has been shown to influence muscle regeneration, inflammation, and metabolic function, suggesting a potential role for SERMs in neuromuscular diseases.

In addition, (Z)-endoxifen has been shown to inhibit PKCβ signaling, which is implicated in inflammatory and fibrotic pathways relevant to muscular dystrophies (Jayaraman *et al.*, 2023). PKC-mediated signaling contributes to NF-κB activation and chronic inflammation, both of which are central drivers of muscle degeneration in DMD and related conditions (Madaro *et al.*, 2012). By targeting both ER-dependent and ER-independent pathways, (Z)-endoxifen may provide a multifaceted approach to slowing disease progression and improving muscle function.

Clinical data supporting the use of tamoxifen in DMD further strengthens the rationale for endoxifen in this setting. Previous studies have demonstrated that tamoxifen treatment can improve muscle strength and respiratory function in DMD patients, although variability in metabolism and study limitations have constrained its broader application (Tsabari *et al.*, 2021; Henzi *et al.*, 2023). Direct administration of (Z)-endoxifen may overcome these limitations by ensuring consistent therapeutic exposure independent of CYP2D6 metabolism.

From a regulatory and commercial perspective, D-CAP represents an attractive extension of Atossa's rare disease strategy. Similar to MAS, this indication may qualify for Orphan Drug Designation and other regulatory incentives. While the patient population is relatively small, orphan pricing dynamics and the absence of approved therapies support a meaningful commercial opportunity.

In summary, female carriers of DMD represent an underserved patient population with significant unmet medical need. The mechanistic profile of (Z)-endoxifen, combined with existing clinical and preclinical evidence, supports further investigation of the drug in this indication as part of Atossa's broader expansion into rare diseases.

Background on DMD

DMD is an X-linked recessive disorder and the most common form of muscular dystrophy, a group of diseases characterized by progressive weakness and loss of muscle mass. DMD occurs in approximately 1 in 5000 boys and the average age of diagnosis is five years of age (Moat *et al.*, 2013).

Patients typically present with gross motor delay, gait abnormalities, and frequent falls. Most boys with DMD will gain strength until approximately six years of age, however after this progressive deterioration in strength will occur and without treatment most will need a wheelchair around age 10 (Falzarano *et al.*, 2015). Cardiomyopathy presents in all patients by the age of 18, however most are asymptomatic due to limited physical activity (Nigro *et al.*, 1990). Respiratory complications are found in all patients and respiratory failure is the leading cause of death for DMD patients (Phillips *et al.*, 2001).

DMD is caused by a mutation in the *DMD* gene, which encodes the dystrophin protein (Hoffman *et al.*, 1987). The *DMD* gene contains more than 2.5 million base pairs of the X chromosome (approximately 1.5% of the X chromosome). The coding sequence is 86 exons, which leads to a 14,000 base pair messenger RNA that is predominantly expressed in skeletal and cardiac muscle, with lesser amounts

expressed in the brain (Muntoni *et al*., 2003). Three different promoters give rise to different full-length isoforms, with additional isoforms derived from alternative splicing events (Sadoulet-Puccio *et al*., 1996).

The dystrophin protein is 427 kDa and comprises four domains. It is associated with the plasma membrane of cardiac and skeletal muscle (sarcolemma) where it interacts with various integral membrane proteins that together form the dystrophin-glycoprotein complex (DGC). The main role of the DGC is to stabilize the muscle fibers during contractions as well as propagate cell survival and cellular defense signaling pathways (Rando 2001).

Mutations in the *DMD* gene include intragenic deletions (60-65%), duplications (5-15%), and various combinations of point mutations, intronic deletions, and exonic insertions. While deletions can happen anywhere in the gene, two "hotspots" exist within exons 2-19 toward the 5' end of the gene and exons 45-55 in the middle of the gene (Muntoni *et al*., 2003). There is no direct correlation between the size of the deletion in the *DMD* gene and disease severity, with the phenotype for DMD patients being dependent upon whether the mutation disrupts the reading frame.

Currently, treatment for DMD consists of managing symptoms, treating complications, and improving a patient's quality of life as much as possible. Standard of care for DMD patients begins with corticosteroids. Multiple short-term and long-term studies have shown that their use leads to muscle strength improvement across a number of parameters (Matthews *et al*., 2016). In addition, corticosteroid therapy has been shown to increase ambulation by approximately three years (Biggar *et al*., 2006). Emflaza® (deflazacort) was approved by the FDA for the treatment of DMD in 2017 and is currently approved to treat DMD patients 2 years of age and older, regardless of mutation status. In 2024, Emflaza generated revenues of $207 million for PTC Therapeutics (EvaluatePharam).

While offering a number of positive benefits to patients, corticosteroid therapy results in a number of potentially serious side effects that must be effectively managed. These side effects include excessive weight gain, gastric complications, cataracts, hypertension, behavioral changes, bone fracture, and growth suppression (Bushby *et al*., 2010).

Newer therapies developed for DMD include gene addition, exon skipping, stop codon readthrough, and genome editing techniques, which are all designed to restore some level of dystrophin expression in genetically defined patient subets. In contrast (Z)-endoxifen is not mutation-specific, and therefore could potentially be used across a broader DMD population regardless of the underlying genetic defect. In addition, due to its anti-inflammatory and signaling pathway effects, (Z)-endoxifen could potentially be used in combination with genetic therapies to address ongoing muscle inflammation and degeneration.

Breast Cancer

While Atossa is increasingly prioritizing rare disease indications, breast cancer remains an important component of the company's development strategy, particularly through combination therapy and partnering opportunities. Rather than pursuing endoxifen as a standalone therapy in highly competitive metastatic settings, the company is now focusing on its integration into multi-drug regimens, most notably through participation in the I-SPY 2 clinical trial platform.

The I-SPY 2 trial is an adaptive, multicenter Phase 2 platform study designed to rapidly evaluate novel agents and combinations in the neoadjuvant treatment of women with locally advanced ER+/HER2- breast cancer (NCT01042379). The trial investigating (Z)-endoxifen is a study arm of the ongoing I-SPY 2 Endocrine Optimization Pilot, a collaborative effort among academic investigators across the U.S. The study evaluated 20 women who received 10 mg (Z)-endoxifen once daily for six cycles (28-day cycle) or up to 24 weeks prior to surgery. In May 2025, Atossa reported updated results from the study that showed a) 95% of participants completed at least 75% of planned dosing; b) median Ki-67 fell from 10.5% at baseline to 5% by Week 3; c) 68.5% (13/19) of patients achieved Ki-67 <10% at Week 3 with suppression maintained at surgery; d) median functional tumor volume measurement decreased 77.7%

from baseline to surgery; and e) the longest tumor diameter in the participants from baseline to preoperative MRI was reduced by 36.8%.

In April 2024, Atossa announced the initiation of a new study arm in I-SPY 2 to evaluate (Z)-endoxifen in combination with Verzenio® (abemaciclib), a CDK4/6 inhibitor. Enrollment of a planned 88 participants in this study is ongoing in which participants will receive 40 mg (Z)-endoxifen once daily in combination with 150 mg abemaciclib twice daily.

A key focus of the combination strategy is addressing the challenges associated with endocrine therapy in premenopausal women. Standard approaches often require ovarian function suppression, which induces a menopausal state and is associated with significant side effects, including hot flashes, bone loss, and reduced quality of life (Francis *et al*., 2015). By combining (Z)-endoxifen with agents such as abemaciclib or elagolix, Atossa aims to develop treatment regimens that maintain efficacy while improving tolerability and patient quality of life.

This combination-based approach enhances the strategic value of (Z)-endoxifen by positioning it as a foundational endocrine therapy that can be paired with other targeted agents. This creates multiple potential partnering opportunities with larger pharmaceutical companies developing complementary therapies, allowing Atossa to participate in the breast cancer market through collaborations rather than direct commercialization.

Intellectual Property

Atossa owns and maintains 13 issued patents (five U.S. patents and eight international patents) and are pursuing 119 pending patent applications (28 U.S. patent applications and 91 international patent applications) directed to (Z)-endoxifen therapies, immunotherapies (e.g., CAR-T), and other therapies. Specifically for (Z)-endoxifen, the company has four U.S. patents and eight international patents along with 15 U.S. pending applications and 70 international pending applications with estimated expiry dates ranging from 2038-2046.

Two of Atossa's patents (U.S. Patent 11,261,151 and U.S. Patent 12,071,391) are currently the subject of post-grant challenges, however Atossa recently announced a settlement agreement that is intended to terminate the post-grant challenges, and even if the proceedings are not terminated the settlement calls for neither party to participate further in the proceeding or any related appeal, thus it appears that the issue is resolved. In addition, Atossa holds four additional U.S. Patents (U.S. Patent Nos. 11,680,036; 12,201,591; 12,275,684; and 12,281,056) with claims to endoxifen that cover proprietary manufacturing methods, stable crystalline forms, and multiple sustained-release and enteric oral formulations.

Financials and Capital Structure

In May 2026, Atossa announced financial results for the first quarter of 2026. As expected, the company did not report any revenues in the first quarter of 2026. R&D expenses for the three months ending March 31, 2026 were approximately $4.8 million compared to $4.2 million in the first quarter of 2025. The increase was primarily due to increased clinical and pre-clinical trial expenses along with increased non-cash stock-based compensation. G&A expenses in the first quarter of 2026 were approximately $5.1 million compared to $3.3 million in the first quarter of 2025. The increase was primarily due to increased professional fees partially offset by a decrease in compensation costs.

As of March 31, 2026, Atossa had approximately $31.7 million in cash and cash equivalents. We estimate this is sufficient to cover operating expenses for greater than one year. In addition, the company has no debt. As of May 1, 2026, Atossa had approximately 8.6 million shares outstanding and, when factoring in stock options, a fully diluted share count of approximately 10.2 million.

Risks to Consider

In addition to the risks listed below, investors are encouraged to read the company's latest 10-K filing that discusses additional risk factors.

Development Risk: Atossa has announced positive results for a number of clinical trials testing (Z)-endoxifen, however just because the compound was successful in earlier clinical trials does not mean it is guaranteed to be successful in future trials. Clinical research is inherently risky, and there are a number of unforeseen circumstances that may arise during the multiple phases of clinical development, including delays in patient recruitment, failure of the contract research organization (CRO) conducting the trial to effectively oversee its conduct, and severe or unexpected side effects experienced by patients in the trials. These risks may be amplified in rare disease indications such as MAS, where small patient populations and limited historical precedent may complicate trial design and interpretation.

Regulatory Risk: Atossa is pursuing development in rare disease indications that may qualify for Orphan Drug and Rare Pediatric Disease designations. However, there is no guarantee that such designations will be granted or that they will result in an expedited regulatory pathway. Regulatory agencies may require additional clinical data, including larger or longer-duration studies, which could delay development timelines. In pediatric populations, regulatory requirements may be particularly stringent given safety considerations, and failure to meet these requirements could limit or delay approval.

Commercial Risk: While rare disease indications may benefit from favorable pricing dynamics, commercial success is not guaranteed. Even if approved, (Z)-endoxifen may face competition from existing off-label therapies, such as aromatase inhibitors in MAS, which have demonstrated clinical efficacy and are widely available as low-cost generics. In oncology, particularly breast cancer, the market remains highly competitive, and Atossa's strategy is increasingly dependent on positioning (Z)-endoxifen with combination regimens. There is no assurance that the drug will achieve sufficient differentiation, physician adoption, or payer reimbursement to drive meaningful commercial uptake.

Partnering Risk: Atossa's oncology strategy is increasingly reliant on partnerships with larger pharmaceutical companies, particularly for combination therapy development in breast cancer. While ongoing clinical trials, including those conducted as part of the I-SPY platform, may generate supportive data, there is no guarantee that these efforts will result in collaboration agreements. Failure to secure partnerships could limit the company's ability to fully capitalize on the breast cancer market and may require additional capital investment to advance these programs independently.

Intellectual Property Risk: Atossa owns and maintains 13 issued patents and is pursuing 119 pending patent applications, however there is no guarantee that the pending applications will result in issued patents or that issued patents will withstand legal challenges. For example, in August 2023, Intas Pharmaceuticals LTD. filed a Petition for Post Grant Review (PGR) with the Patent Trial and Appeal Board (PTAB) of the U.S. Patent and Trademark Office seeking to invalidate all claims related to U.S. Patent 11,572,334. While Atossa vigorously contested the PGR petition, in January 2025 the PTAB issued a final written decision that found all claims under the Patent were unpatentable. Recently, two additional Atossa patents (U.S. Patent Nos. 11,261,151 and 12,071,391) were the subject of PGRs, however Atossa recently announced a settlement agreement with Intas that is intended to terminate the PTAB proceedings, and even if the proceeding is not terminated the settlement calls for neither party to participate further in the proceeding or any related appeal, thus it appears that the issue is resolved.

Financial Risk: As of December 31, 2025, Atossa had approximately $40 million in cash and cash equivalents, which we estimate is sufficient to fund operations for at least one year. However, clinical development is very expensive and the company will need to raise additional capital in order to finish the development of (Z)-endoxifen. There is no guarantee that the company will be able to raise additional capital, or that the capital will be available on terms that are agreeable to the company. In addition, the sale of shares of common stock to raise additional capital may result in significant dilution to current shareholders.

MANAGEMENT PROFILES

Steven C. Quay, MD, PhD – Chairman of the Board and Chief Executive Officer

Dr. Quay has served as Chief Executive Officer, President, and Chairman of the Board of Directors of Atossa since the company was incorporated in April 2009. Dr. Quay is certified in Anatomic Pathology with the American Board of Pathology, completed both an internship and residency in anatomic pathology at Massachusetts General Hospital, a Harvard Medical School teaching hospital, and is a former faculty member of the Department of Pathology, Stanford University School of Medicine.

Dr. Quay is a named inventor on 96 U.S. patents, 130 pending U.S. patent applications, and is named inventor on patents covering five pharmaceutical products that have been approved by the U.S. Food and Drug Administration. Dr. Quay received an M.D. in 1977 and a Ph.D. in 1975 from the University of Michigan. He received his B.A. degree in biology, chemistry and mathematics from Western Michigan University in 1971. He was selected to serve on the Company's Board of Directors because of his role as a founder of the Company, as well as his qualifications as a physician and the principal researcher overseeing the clinical and regulatory development of the Company's pharmaceutical programs.

Mark Daniel – Chief Financial Officer

Mr. Daniel is an accomplished finance executive with over 25 years of experience guiding global life science organizations through growth, transformation, and strategic transactions. Before joining Atossa, Mr. Daniel served as Senior Vice President of Finance at NanoString Technologies, where he played a key leadership role in the company's growth, strategic financings, and ultimate acquisition by Bruker Corporation. Earlier in his career, he held senior finance and accounting leadership roles at publicly held and private equity-backed companies, including HaloSource, Cardiac Science, and Numera (Newyu), supporting capital markets transactions and divestitures, and leading all aspects of corporate accounting and finance, including SEC reporting and operational efficiency programs across global organizations. Mr. Daniel began his career with KPMG's assurance practice and holds a Bachelor of Science in Business Administration from Washington State University. He is a Certified Public Accountant (Washington State, inactive).

Janet R. Rea, MSPH – Senior Vice President, Research & Development

Ms. Rea brings over 35 years of industry leadership experience in clinical development through commercialization in biologics and small molecules, with a focus on oncology, infectious diseases, and orphan and rare-diseases. A Washington native, she obtained her B.S. degree in Microbiology from the University of Washington and was conferred a Master of Science of Public Health from the same institution. Prior to rejoining Atossa, she was an independent consultant to biopharmaceutical companies. She has held senior leadership positions with AVM Biopharma, Poniard Pharmaceuticals, AVI BioPharma (Sarepta), Protein Sciences (Glaxo), and Atossa Therapeutics, playing key roles in the approval of Immunex's initial product, LEUKINE® and Sanofi's rDNA influenza vaccine, FLU-BLOK®. She has lectured at both Shoreline Community College and the University of Washington for the Biomedical Regulatory Affairs Certificate and Master's Program, where she also served as a part-time Assistant Clinical Professor.

Delly Behen, PHR, SHRM-CP – Senior Vice President, Business Operations

Ms. Behen has served as Atossa's Senior Vice President, Business Operations since July 2014. She brings over 20 years of human resources, administrative, and operational experience to the company. Her experience includes leading people, culture and administration at various biotech companies throughout the Puget Sound. Most recently, she served as Impel NeuroPharma's HR Consultant, where she helped grow the company and implement HR policies and procedures. She also held positions with increasing responsibilities at CTI Biopharma. Delly received her B.A. degree from the University of Washington and her HR certification from Seattle Pacific University.

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VALUATION

We are initiating coverage of Atossa Therapteutics, Inc. (ATOS) with a valuation of $22.00. Atossa is a clinical-stage biopharmaceutical company developing oral (Z)-endoxifen as a multi-indication endocrine therapy across rare diseases and oncology. (Z)-endoxifen is an active metabolite of tamoxifen, an FDA-approved therapy for the prevention and treatment of estrogen receptor (ER)-positive breast cancer. Tamoxifen itself has weak affinity for the ER and requires metabolic activation via hepatic cytochrome P450 enzymes to generate active metabolites, including (Z)-endoxifen, which exhibit significantly higher receptor affinity. Direct administration of (Z)-endoxifen eliminates the need for metabolic conversion, enabling more consistent therapeutic exposure across patient populations.

(Z)-Endoxifen

(Z)-endoxifen is the primary active metabolite of tamoxifen and functions as a potent modulator of ER signaling. Unlike tamoxifen, which is a pro-drug requiring hepatic metabolism via cytochrome P450 enzymes (predominantly CYP2D6) to generate active metabolites, (Z)-endoxifen is administered directly, thereby eliminating variability associated with metabolic activation and enabling more consistent systemic exposure across patient populations.

Compared to tamoxifen, (Z)-endoxifen exhibits significantly higher affinity (>10x) for the ER and has demonstrated potent antiestrogenic activity across multiple preclinical and clinical settings. In addition to its direct effects on ER signaling, (Z)-endoxifen has been shown to exert ER-independent activity through inhibition of protein kinase C (PKC), a family of serine/threonine kinases involved in regulating cell proliferation, apoptosis, inflammation, and cellular differentiation. This dual mechanism that combines endocrine modulation with kinase pathway inhibition differentiates (Z)-endoxifen from traditional selective estrogen receptor modulators (SERMs) and may broaden its therapeutic applicability beyond hormone-driven cancers.

(Z)-endoxifen has been evaluated in multiple Phase 1 and Phase 2 trials and has been administered to hundreds of patients, demonstrating a well-characterized safety and pharmacokinetic profile. Across studies, the compound has been generally well tolerated, with no maximum tolerated dose identified in dose-escalation trials and a safety profile consistent with endocrine modulation. Importantly, therapeutic drug levels are achieved rapidly and predictably following oral administration, supporting its use in indications requiring consistent systemic exposure. We believe the pharmacological profile of (Z)-endoxifen supports its development across multiple disease settings, particularly those driven by aberrant estrogen signaling.

Atossa has recently shifted its development focus toward rare disease indications, with McCune-Albright Syndrome (MAS) emerging as a potential lead program. MAS is a rare pediatric endocrine disorder characterized by precocious puberty and other hormone-driven abnormalities, with no approved disease-modifying therapies currently available. We believe MAS represents the most efficient and potentially expedited path to market for (Z)-endoxifen given its well-defined biology and high unmet medical need. In addition, the company recently received Rare Pediatric Disease designation (which makes it eligible for a Priority Review Voucher upon approval) and will be pursuing Orphan Drug Designation.

Beyond MAS, Atossa is expanding into other rare disease opportunities, including Duchenne muscular dystrophy (DMD) and symptomatic female carriers of DMD (D-CAP). The pathophysiology of D-CAP shares many features with classical DMD, including chronic inflammation, fibrosis, calcium dysregulation, and progressive muscle degeneration. These overlapping mechanisms provide a strong rationale for evaluating therapies that target multiple downstream pathways involved in muscle damage and repair.

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Valuation

We value Atossa using a probability-adjusted discounted cash flow model that takes into account potential future revenues for (Z)-endoxifen in MAS, DMD, and D-CAP along with the potential sale of PRVs obtained from approvals in MAS and DMD, for which Rare Pediatric Disease Designation has been conferred by the FDA for both indications.

For MAS, we estimate there are approximately 3,500 individuals in the U.S. and approximately 8,500 in the E.U. and Japan. Given it is an ultra-orphan indication, we model for a yearly cost for (Z)-endoxifen of $120,000. Using a peak market share of 20% results in peak sales of approximately $60 million in the U.S. and $75 million outside the U.S. We model for approval in the U.S. in 2029 and outside the U.S. in 2030 and for peak sales to occur seven years after launch. We assume that Atossa will market the drug in the U.S. and enter into a commercialization partnership for sales outside the U.S. and receive a 12% royalty on net sales. Using a 15% discount rate and a 50% probability of approval leads to an NPV for MAS of $25 million.

For DMD, we estimate there are approximately 14,000 boys affected by the disease in the U.S. and approximately 15,000 in the E.U. and Japan. Using a peak market share of 40% in all jurisdictions results in peak sales estimates of $400 million in the U.S. and $250 million outside the U.S. We model for approval in the U.S. in 2030 and outside the U.S. in 2031 and for peak sales to occur seven years after launch. We model for Atossa to market the drug in the U.S. and to enter into a commercialization partnership for sales outside the U.S. and receive a 12% royalty on net sales. Using a 15% discount rate and a 25% probability of approval leads to an NPV for DMD of $95 million.

For D-CAP, we estimate there are approximately 12,000 symptomatic female carriers of DMD in the U.S. and approximately 18,000 in the E.U. and Japan. Using a peak market share of 15% in all jurisdictions results in peak sales estimates of $250 million in the U.S. and $175 million outside the U.S. We model for approval in the U.S. in 2030 and outside the U.S. in 2031 and for peak sales to occur seven years after launch. We model for Atossa to market the drug in the U.S. and to enter into a commercialization partnership for sales outside the U.S. and receive a 12% royalty on net sales. Using a 15% discount rate and a 25% probability of approval leads to an NPV for D-CAP of $65 million.

For the PRVs, we model for them to be sold shortly after they are issued to the company in 2029 for MAS and 2030 for DMD. Based on recent sales of PRVs, we model for them to be sold for $150 million each. Using a 15% discount rate and a 50% probability in MAS and a 33% probability in DMD leads to an NPV of $49 million and $28 million, respectively.

Combining the NPVs for each of the indications, PRVs, the current cash on hand, and the potential cash from stock options leads to a net present value for Atossa of approximately $330 million. Dividing by the fully diluted share count of 10.2 million shares, plus an additional 5.0 million shares for future dilution, leads to a valuation of $22.00.

PROJECTED FINANCIALS

Atossa Therapeutics, Inc.	2025 A	Q1 A	Q2 E	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	$5.0	$5.3	$5.7	$20.8	$22.0	$24.0
General & administrative	$16.0	$5.1	$3.9	$4.0	$4.1	$17.1	$17.5	$18.0
Operating Income	($37.1)	($9.9)	($8.9)	($9.3)	($9.8)	($37.9)	($39.5)	($42.0)
Non-Operating Expenses (Net)	$2.4	$0.3	$0.5	$0.5	$0.5	$1.8	$0.0	$0.0
Pre-Tax Income	($34.8)	($9.6)	($8.4)	($8.8)	($9.3)	($36.1)	($39.5)	($42.0)
Income Taxes	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Net Income	**($34.8)**	**($9.6)**	**($8.4)**	**($8.8)**	**($9.3)**	**($36.1)**	**($39.5)**	**($42.0)**
Net Margin	-	-	-	-	-	-	-	-
Reported EPS	**($4.04)**	**($1.11)**	**($0.98)**	**($1.02)**	**($1.08)**	**($4.19)**	**($3.59)**	**($3.00)**
YOY Growth	-	-	-	-	-	-	-	-
Basic Shares Outstanding	8.6	8.6	8.6	8.6	8.6	8.6	11.0	14.0

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

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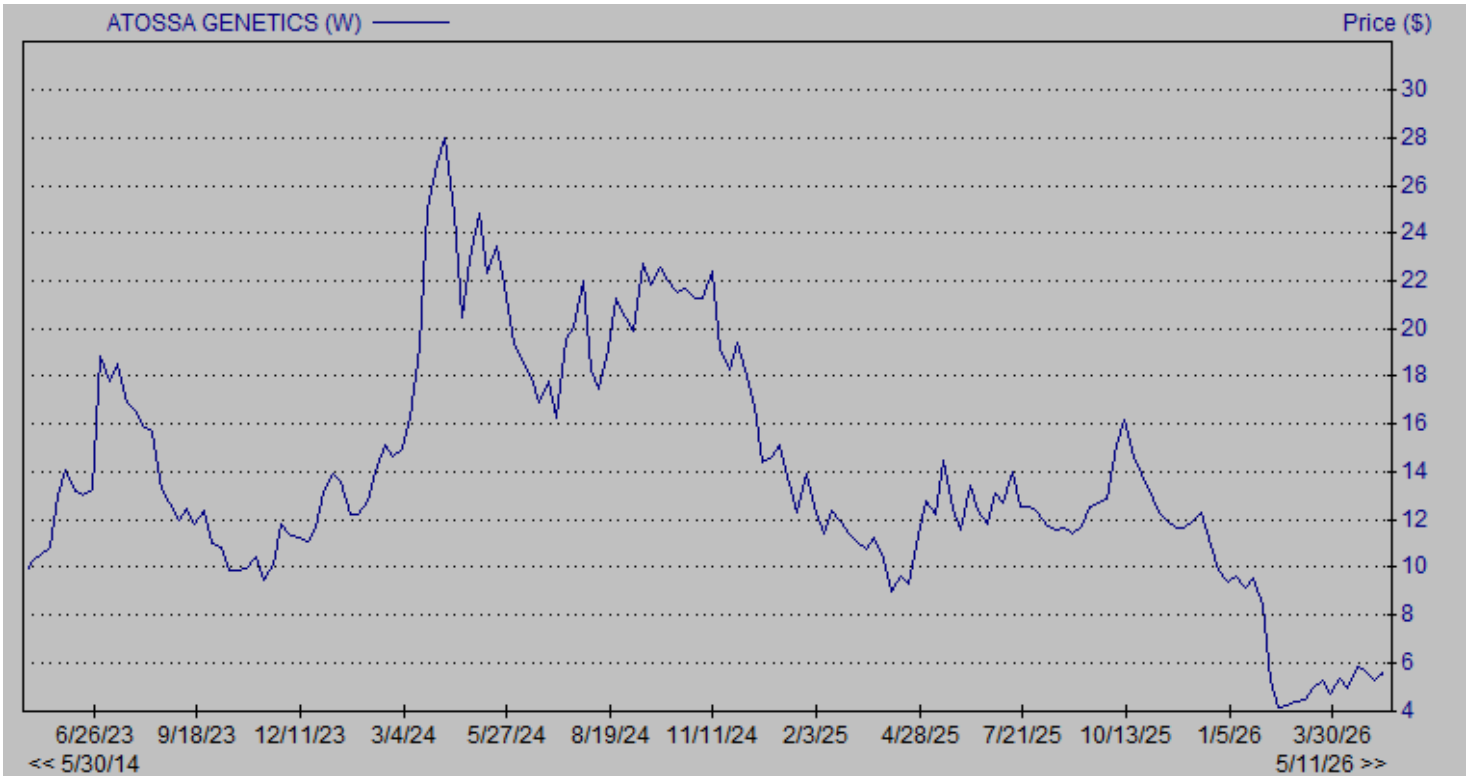
HISTORICAL STOCK PRICE



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