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Estrella Immunopharma, Inc. (ESLA-NASDAQ)

ESLA: Can Next-Generation CAR-T Therapy Improve Disease Control in Extranodal Lymphoma?

Based on our probability adjusted DCF model that takes into account potential future revenues for EB103, ESLA is valued at $12.00/share. This model is dependent upon continued clinical success of EB103 and will be adjusted accordingly based upon future clinical results.

Current Price (07/14/26)	$0.88
Valuation	**$12.00**

OUTLOOK

On May 18, 2026, Estrella Immunopharma, Inc. (ESLA) filed form 10-Q with financial results for the first quarter of 2026. We recently initiated coverage of Estrella, highlighting the company's ARTEMIS® platform as a differentiated next-generation T cell technology designed to address several of the biological limitations associated with conventional CAR-T therapies. One of the most significant remaining challenges in cellular therapy is achieving durable disease control in patients with extranodal lymphoma, where emerging evidence suggests that tissue-specific relapse remains a key obstacle despite high initial response rates. In this report, we review the evolving clinical and mechanistic evidence surrounding extranodal disease and discuss why the biological characteristics of the ARTEMIS platform may provide a compelling rationale for evaluating EB103 in this high unmet need setting.

SUMMARY DATA

52-Week High	**$3.05**
52-Week Low	**$0.82**
One-Year Return (%)	**-1.06**
Beta	**0.85**
Average Daily Volume (sh)	**37,973**
Shares Outstanding (mil)	**43**
Market Capitalization ($mil)	**$38**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**0**
Insider Ownership (%)	**6**
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	**-2.2**
P/E using 2027 Estimate	**-2.7**

Risk Level	**Above Avg.**
Type of Stock	**Small-Blend**
Industry	**N/A**

ZACKS ESTIMATES

Revenue
(in millions of $)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2025	0.0 A	0.0 A	0.0 A	0.0 A	0.0 A
2026	0.0 A	0.0 E	0.0 E	0.0 E	0.0 E
2027					0.0 E
2028					0.0 E

Earnings Per Share

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2025	-$0.06 A	-$0.15 A	-$0.13 A	-$0.02 A	-$0.35 A
2026	-$0.05 A	-$0.09 E	-$0.08 E	-$0.07 E	-$0.29 E
2027					-$0.32 E
2028					-$0.31 E

WHAT'S NEW

Business Update

ARTEMIS Platform Potential in Treating Extramedullary Disease

Extramedullary disease (EMD) remains one of the most significant unresolved challenges in cellular therapy. EMD, or extranodal disease, in aggressive B-cell non-Hodgkin lymphoma (NHL) broadly refers to lymphoma involvement outside traditional lymphatic structures, including sites such as the central nervous system (CNS), liver, lung, pleura, gastrointestinal (GI) tract, skin, or soft tissue structures (Krol *et al*., 2003). Extranodal dissemination is particularly common in relapsed/refractory diffuse large B-cell lymphoma (DLBCL), where it is seen in up to 50% of patients at diagnosis (St-Pierre *et al*., 2025), and is generally associated with aggressive biology and inferior clinical outcomes.

While currently approved CAR-T products have transformed outcomes in hematologic malignancies, multiple studies have demonstrated inferior durability, shorter progression-free survival, and higher relapse rates in patients with extranodal disease involvement. Emerging evidence suggests these inferior outcomes may be driven by a combination of impaired T cell trafficking, immunosuppressive tissue microenvironments, and accelerated T cell exhaustion within EMD lesions.

Estrella Immunopharma's (ESLA) ARTEMIS® platform may represent a differentiated next-generation cellular therapy approach. The company's platform architecture is designed to promote more physiologic T cell activation and potentially reduce exhaustion-associated dysfunction that may limit conventional CAR-T performance in difficult tissue environments. We believe that previous experience from solid tumor trials, including hepatocellular carcinoma studies, may support the hypothesis that ARTEMIS-based therapies could demonstrate improved activity against EMD.

Although clinical validation in EMD remains early, the biological rationale underlying the platform's potential differentiation appears increasingly aligned with the mechanisms now believed to drive CAR-T failure in EMD.

CD19 CAR-T Therapy Relapse Challenges

The introduction of CD19-directed chimeric antigen receptor T cell (CAR-T) therapy has fundamentally altered the treatment paradigm for patients with relapsed or refractory large B-cell lymphoma (LBCL). Prior to the availability of CAR-T therapy, patients who relapsed following second-line treatment had a median overall survival measured in months, particularly if they were ineligible for autologous stem cell transplantation. Today, commercially available CD19-directed CAR-T products, including axicabtagene ciloleucel (Yescarta®), tisagenlecleucel (Kymriah®), and lisocabtagene maraleucel (Breyanzi®), have demonstrated the ability to induce deep and durable remissions in heavily pretreated patients, with approximately 35-45% of treated patients remaining progression-free several years after treatment (Neelapu *et al*., 2023; Abramson *et al*., 2024; Schuster *et al*., 2019).

Despite remarkable advances, relapse following CAR-T therapy remains the principle obstacle to further improving patient outcomes. Approximately one-half of patients treated with currently approved CD19 CAR-T products ultimately experience disease progression, with most relapses occurring during the first year after infusion (Toro-Mijares *et al*., 2023). Considerable research has therefore shifted from maximizing initial response rates toward understanding the biological mechanisms responsible for treatment failure and identifying strategies capable of improving long-term disease control.

Historically, investigations of CAR-T resistance have focused primarily on tumor-intrinsic mechanisms such as CD19 antigen loss, inadequate CAR-T expansion, or limited cellular persistence. However, recent clinical evidence suggests that anatomic distribution of lymphoma itself may significantly influence

treatment durability. Specifically, recent multicenter analyses have demonstrated that patients with concomitant nodal and extranodal LBCL experience inferior progression-free survival and overall survival following commercial CD19-directed CAR-T therapy compared with patients with nodal disease alone. Moreover, the risk of treatment failure appears to depend not only on the presence of extranodal disease but also on the specific organs involved, with increasing numbers of extranodal sites and involvement of the liver, adrenal glands, and pancreas associated with worse outcomes. Importantly, extranodal disease becomes more prevalent at relapse, suggesting that certain tissue compartments may function as relative sanctuary sites where durable immune surveillance following CAR-T therapy is more difficult to achieve (Iacoboni *et al.*, 2025).

Supporting this concept, a second multicenter imaging study demonstrated that local response rates varied substantially by anatomic site, with lung/pleural/pericardial lesions and gastrointestinal/peritoneal lesions exhibiting the lowest complete response rates and the highest risk of relapse within the same tissue compartment. These findings suggest that CAR-T efficacy is heterogeneous across extranodal sites and that tissue-specific biology may influence long-term disease control (Luna *et al.*, 2025).

These observations have shifted attention toward understanding the unique biology of extranodal disease and raise an important question for next-generation cellular therapies: rather than simply improving initial tumor cytotoxicity, can CAR-T platforms be engineered to achieve more durable immune surveillance within difficult tissue microenvironments? As discussed below, this question may represent one of the most compelling opportunities for differentiation among next-generation CAR-T platforms, including Estrella's ARTEMIS technology.

Understanding Why Certain Tissue Compartments Become CAR-T Sanctuary Sites

The emerging pattern of site-specific relapse following CD19-directed CAR-T therapy suggests that durable disease control is influenced by more than tumor burden or antigen expression alone. Rather, accumulating evidence indicates that the local tissue microenvironment plays a critical role in determining whether infused CAR-T cells can successfully traffic to, infiltrate, expand within, and persist inside individual extranodal lesions. While these processes remain incompletely understood, several complementary mechanisms have emerged that may explain why certain tissue compartments function as relative sanctuary sites following CAR-T therapy.

A) Tissue Trafficking and Tumor Infiltration

The first requirement for effective CAR-T therapy is successful migration of engineered T cells from the circulation into sites of active disease. Unlike lymphoma confined to lymph nodes or the bloodstream, extranodal lesions frequently reside within complex tissue environments that present both physical and biological barriers to immune-cell infiltration.

Abnormal tumor-associated vasculature, dense stromal architecture, altered chemokine gradients, and increased interstitial pressure can all limit the ability of circulating T cells to penetrate tumor tissue. Similar barriers have long been recognized as major obstacles to CAR-T therapy in solid tumors, where inadequate trafficking is considered one of the principal determinants of treatment failure (Newick *et al.*, 2017). Although these mechanisms have not been fully characterized in extranodal lymphoma, they provide a plausible biological explanation for the heterogeneous response rates observed across different organ systems following CD19-directed CAR-T therapy.

B) Immunosuppressive Tissue Microenvironments

Successful CAR-T therapy requires more than simply reaching the tumor. Once inside an extranodal lesion, engineered T cells must remain functionally active despite exposure to an immunosuppressive microenvironment. Many extranodal tissues contain abundant regulatory immune cells, including tumor-associated macrophages and regulatory T cells, together with inhibitory cytokines such as transforming growth factor-β (TGF-β) and interleukin-10 (IL-10). Chronic antigen exposure within these environments

may further promote expression of inhibitory immune checkpoint molecules including PD-1, LAG-3, and TIM-3, collectively reducing T cell effector function (Joyce *et al*., 2015). These local suppressive mechanisms may limit the duration of CAR-T activity even after an initial antitumor response has been achieved.

C) T Cell Exhaustion and Loss of Functional Persistence

Among the mechanisms proposed to underlie CAR-T failure, T cell exhaustion has emerged as one of the most compelling. CAR-T cells undergo rapid activation and expansion following antigen recognition. However, prolonged stimulation can drive a progressive loss of proliferative capacity, cytokine production, and cytotoxic activity. This process is commonly referred to as T cell exhaustion (Blank *et al*., 2019).

Direct evidence supporting this mechanism has recently been reported in patients with extramedullary multiple myeloma treated with BCMA-directed CAR-T therapy (Qi *et al*., 2024). Qi *et al*. demonstrated that extramedullary lesions contained markedly higher levels of exhausted CD8+ T cells than corresponding medullary disease sites and that these exhausted immune phenotypes were associated with delayed responses, inferior progression-free survival, and earlier relapse. Although this study evaluated BCMA-targeted therapy rather than CD19-directed lymphoma treatment, it provides important mechanistic evidence that hostile tissue microenvironments may actively promote CAR-T dysfunction and loss of durable immune surveillance.

Taken together, these findings suggest that successful treatment of extranodal lymphoma depends on a sequence of biological events extending well beyond initial tumor recognition. CAR-T cells must successfully traffic into difficult tissue compartments, infiltrate established tumor deposits, resist local immunosuppressive signals, maintain functional persistence despite chronic antigen exposure, and continue to provide long-term immune surveillance after the initial tumor burden has been reduced.

Failure at any point along this continuum may allow residual lymphoma cells to survive and ultimately drive site-specific relapse. This framework is consistent with the emerging clinical observation that relapse following CAR-T therapy frequently occurs within previously involved extranodal tissues rather than representing random systemic disease recurrence.

Thus, the next generation of CAR-T therapies may ultimately be differentiated not by their ability to induce higher initial response rates, but by their capacity to maintain functional activity within the hostile tissue microenvironments that appear to limit durable disease control.

ARTEMIS was Designed to Improve T Cell Fitness and Functional Persistence

The recent recognition that relapse following CD19-directed CAR-T therapy may occur preferentially within specific extranodal tissue compartments raises an important question for the next generation of cellular therapies: what biological characteristics are required to achieve durable immune surveillance within hostile tissue microenvironments? While definitive answers remain elusive, the emerging literature increasingly points toward T cell fitness, resistance to exhaustion, and long-term functional persistence as key determinants of durable disease control. These concepts closely align with the biological rationale underlying Estrella's ARTEMIS platform.

Unlike conventional CAR-T constructs, which rely on synthetic intracellular signaling domains such as CD28 or 4-1BB linked to CD3ζ, ARTEMIS utilizes the company's proprietary Antibody-TCR (AbTCR) architecture, in which an antibody-derived antigen recognition domain is coupled to the endogenous γδ T cell receptor (TCR) signaling complex. Rather than activating only selected intracellular signaling pathways, the AbTCR receptor engages the native TCR/CD3 complex, allowing T cell activation to occur through the physiologic signaling machinery that has evolved to regulate normal T cell function (Xu *et al*., 2018).

This distinction may have important biological implications. As discussed in the previous section, durable CAR-T activity within extranodal tissues likely depends not only on initial tumor recognition but also on the ability of engineered T cells to remain functional despite prolonged antigen exposure and an immunosuppressive tissue microenvironment. Chronic stimulation can promote progressive T cell differentiation and exhaustion, ultimately limiting persistence and long-term immune surveillance.

Preclinical studies comparing the AbTCR platform with conventional second-generation CD19-directed CAR-T cells demonstrated that the two approaches produced comparable antitumor cytotoxicity against CD19-positive leukemia and lymphoma models both *in vitro* and *in vivo*. Importantly, however, the AbTCR platform achieved this antitumor activity while exhibiting several biological characteristics that may favor improved long-term T cell fitness.

First, AbTCR T cells produced substantially lower levels of inflammatory cytokines, including interferon-γ (IFN-γ), tumor necrosis factor-α (TNF-α), interleukin-2 (IL-2), and interleukin-10 (IL-10), than conventional CAR-T cells following antigen stimulation (Xu *et al.*, 2018). Although reduced cytokine production alone does not establish superior clinical efficacy, it suggests that effective tumor killing may be achieved through a more physiologic pattern of T cell activation rather than through excessive immune stimulation.

Second, AbTCR T cells demonstrated a less exhausted immune phenotype. Prior to antigen exposure, expression of the inhibitory receptors PD-1, LAG-3, and TIM-3 was consistently lower than observed in conventional CAR-T cells, and reduced PD-1 expression persisted following tumor clearance in mouse models (Xu *et al.*, 2018). Because inhibitory receptors are widely recognized markers of T cell exhaustion, these findings suggest that endogenous TCR signaling may better preserve functional T cell activity during prolonged immune responses.

Lastly, the AbTCR platform generated a greater proportion of naïve T cells and stem cell memory T cells than conventional CAR-T products (Xu *et al.*, 2018). Less differentiated T cell populations possess greater proliferative capacity, self-renewal potential, and long-term persistence following adoptive transfer and have repeatedly been associated with improved durability of cellular immunotherapies. Preservation of these populations therefore represents another mechanistic feature that could prove advantageous in clinical settings requiring prolonged immune surveillance.

All together, these observations provide a biologically plausible framework linking the ARTEMIS platform to the mechanisms increasingly implicated in site-specific relapse following contemporary CD19-directed CAR-T therapy. The emerging clinical literature suggests that durable disease control within extranodal lymphoma depends on sustained T cell functionality inside hostile tissue microenvironments rather than simply achieving an initial cytotoxic response. The preclinical characteristics demonstrated by the AbTCR platform, including preservation of less differentiated T cell populations, reduced expression of exhaustion markers, and maintenance of potent antitumor activity with lower inflammatory cytokine production, are consistent with the biological attributes that may be required to overcome these limitations.

Importantly, this hypothesis remains to be validated clinically. The company has generated very early data for patients with extranodal disease, showing that of the five complete responses in dose level 2 reported in February 2026 from the Phase 1 STARLIGHT-1 trial, two of those were EMD patients. However, additional data will need to be generated to demonstrate whether ARTEMIS-derived therapies produce superior outcomes in patients with extranodal lymphoma or reduce site-specific relapse following treatment. Nevertheless, the mechanistic profile of the platform appears well aligned with the biological challenges identified in recent clinical studies, providing a compelling scientific rationale for evaluating EB103 in patient populations where durable tissue-specific immune surveillance remains an important unmet need.

Early Clinical Evidence Supporting ARTEMIS Platform in Challenging Tissue Microenvironments

The mechanistic advantages demonstrated by the ARTEMIS platform in preclinical CD19 models leads to an important hypothesis: that improved T cell fitness will translate into meaningful activity within the hostile tissue microenvironments that have historically limited adoptive cellular therapy. While this hypothesis is currently being tested for EB103 in lymphoma, early clinical experience with another ARTEMIS-derived cellular therapy provides encouraging proof-of-concept that the platform can function within a difficult solid tumor setting.

Investigators from Eureka Therapeutics evaluated an AbTCR-based T cell therapy targeting an alpha-fetoprotein (AFP)-derived peptide presented by HLA-A*02 in patients with advanced hepatocellular carcinoma (HCC), one of the most immunosuppressive solid tumors encountered in clinical oncology. To enhance tumor specificity and reduce the risk of off-target activation, the AFP-specific AbTCR construct was paired with a glypican-3 (GPC3)-directed CD28 co-stimulatory receptor, creating a dual-recognition system that required engagement of both tumor-associated antigens for optimal activation (Liu *et al.*, 2022).

Preclinical studies demonstrated potent and highly specific antitumor activity against AFP-positive HCC models both *in vitro* and *in vivo*. Importantly, the engineered T cells showed improved infiltration into established solid tumors while maintaining strict antigen specificity, with minimal activity observed against AFP-negative targets. Structural analyses further demonstrated that the TCR-mimic antibody recognized the AFP/HLA-A*02 peptide complex with high specificity, providing confidence that the platform could selectively target intracellular tumor antigens while minimizing off-target recognition.

Perhaps most importantly, the investigators translated these findings into a first-in-human safety study involving six patients with advanced metastatic HCC. Although the study was primarily designed to evaluate safety and feasibility, the results provided encouraging early evidence of clinical activity. Treatment was well tolerated, with no cases of cytokine release syndrome or other serious treatment-related toxicities reported. Evidence of antitumor activity was observed in multiple patients, including one individual with metastatic HCC involving the lung who achieved a complete radiographic remission after approximately nine months of treatment and subsequently underwent potentially curative liver transplantation.

For investors, the importance of these findings extends beyond the clinical outcome observed in a small number of HCC patients. Rather, the study demonstrates that the ARTEMIS/AbTCR platform can mediate durable antitumor responses within a highly immunosuppressive solid tumor microenvironment while maintaining a favorable safety profile. Although HCC and extranodal lymphoma are biologically distinct diseases, both present significant challenges for adoptive cellular therapy, including impaired immune-cell trafficking, chronic antigen exposure, and local immunosuppressive signaling. As discussed previously, these same biological features are increasingly believed to contribute to tissue-specific relapse following conventional CD19-targeted CAR-T therapy.

Accordingly, the HCC experience should not be viewed as direct evidence that EB103 will demonstrate superior efficacy in extranodal lymphoma. Rather, it provides an important proof-of-concept that the ARTEMIS platform retains functional activity in a tissue microenvironment where conventional cellular therapies have historically faced substantial biological barriers. This observation strengthens the scientific rationale for evaluating EB103 in patients with extranodal lymphoma, where improving long-term immune surveillance within difficult tissue compartments may represent a meaningful opportunity for clinical differentiation.

Taken together, the available evidence supports a coherent biological hypothesis. Recent clinical studies have identified tissue-specific relapse as an emerging limitation of first-generation CD19-directed CAR-T therapy. Mechanistic studies suggest that impaired persistence and progressive T cell exhaustion contribute to this phenomenon, while preclinical studies demonstrate that the ARTEMIS platform preserves key features associated with long-term T cell fitness. Finally, early clinical experience in HCC

indicates that these biological characteristics may translate into functional activity within a hostile tissue microenvironment. Whether these mechanistic advantages ultimately improve the durability of response in patients with extranodal lymphoma remains an important question that ongoing clinical development of EB103 will begin to address.

Financial Update

On May 18, 2026, Estrella filed form 10-Q with financial results for the first quarter of 2026. As expected, the company did not report any revenues for the first quarter of 2026. R&D expenses for the first quarters of both 2026 and 2025 were $1.4 million. G&A expenses for the first quarter of 2026 were $0.9 million compared to $0.7 million for the first quarter of 2025. The increase was primarily due to higher legal and professional fees.

As of March 31, 2026, Estrella had approximately $1.9 million in cash and cash equivalents. As of May 11, 2026, Estrella had approximately 43.0 million shares outstanding and, when factoring in stock options and warrants, a fully diluted share count of 54.7 million.

Conclusion

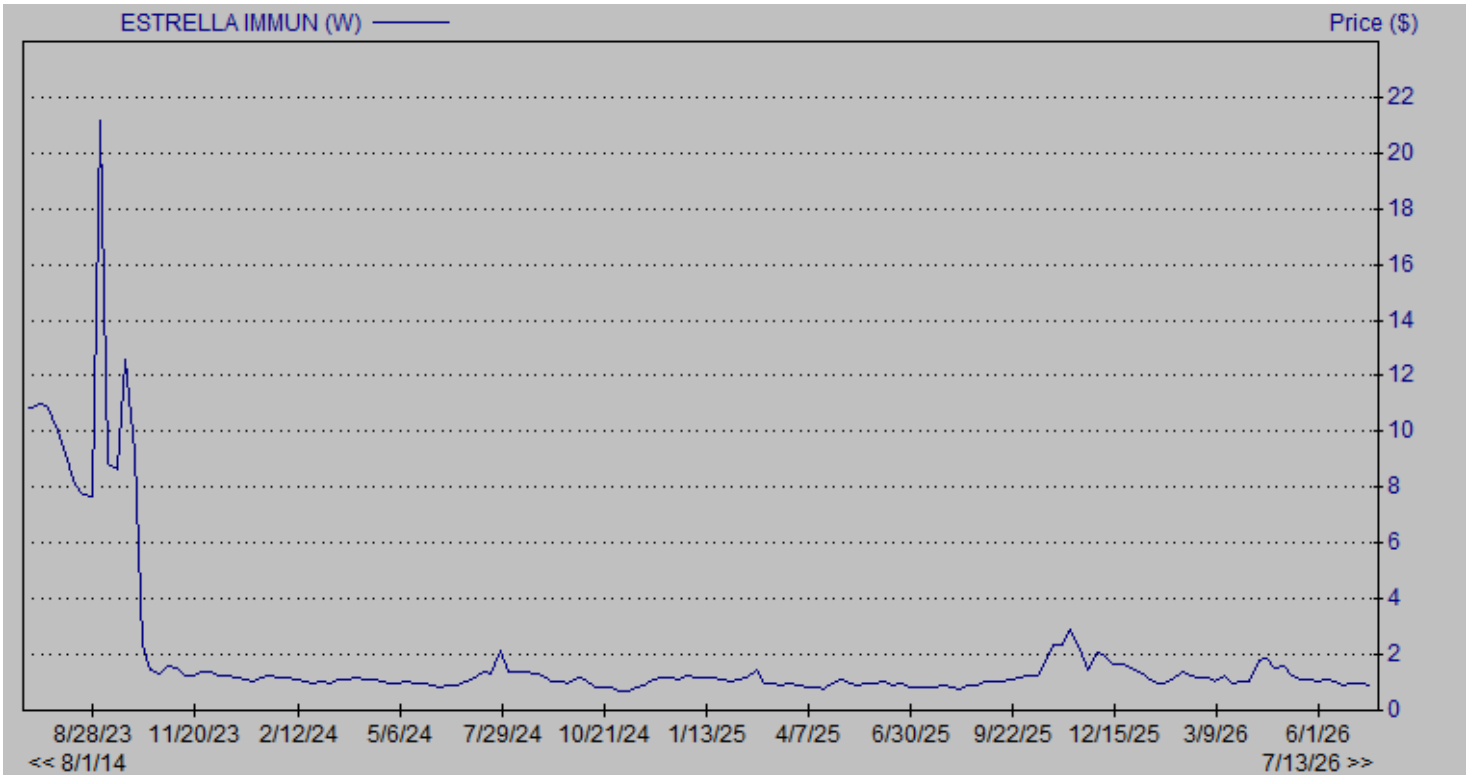
EB103 does not necessarily need to demonstrate higher initial response rates than currently approved CD19 CAR-T therapies to be clinically differentiated. If the ARTEMIS platform improves the durability of response by maintaining functional T cell activity within tissue compartments that currently serve as sites of relapse, the resulting extension in progression-free survival could represent a meaningful advance for patients with high-risk extranodal lymphoma. We anticipate additional updates from the company's ongoing Phase 1 STARLIGHT-1 trial of EB103 in the second half of 2026. With no changes to our model our valuation remains at $12 per share.

PROJECTED FINANCIALS

Estrella Immunopharma, Inc.	2025 A	Q1 A	Q2 E	Q3 E	Q4 E	2026 E	2027 E	2028 E
EB103	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
EB104	$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	$10.2	$1.4	$3.0	$3.0	$3.0	$10.4	$15.0	$17.0
General & administrative	$2.8	$0.9	$0.7	$0.8	$0.8	$3.2	$4.0	$4.0
Depreciation Expense	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Operating Income	($13.1)	($2.3)	($3.7)	($3.8)	($3.8)	($13.6)	($19.0)	($21.0)
Non-Operating Expenses (Net)	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0
Pre-Tax Income	($13.1)	($2.3)	($3.7)	($3.8)	($3.8)	($13.6)	($19.0)	($21.0)
Income Taxes	($0.0)	$0.0	$0.0	$0.0	$0.0	$0.0	$0.0	$1.0
Net Income	**($13.1)**	**($2.3)**	**($3.7)**	**($3.8)**	**($3.8)**	**($13.6)**	**($19.0)**	**($22.0)**
Net Margin	-	-	-	-	-	-	-	-
Reported EPS	**($0.35)**	**($0.05)**	**($0.09)**	**($0.08)**	**($0.07)**	**($0.29)**	**($0.32)**	**($0.31)**
YOY Growth	-	-	-	-	-	-	-	-
Basic Shares Outstanding	36.8	42.1	43.0	50.0	52.0	46.8	60.0	70.0

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

HISTORICAL STOCK PRICE



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