2026年9月29日

H.C. Wainwright & Co.による当社レポートの発表に関するお知らせ

現地時間の9月28日、当社が公表した2型糖尿病に起因する高中性脂肪血症及び脂肪肝疾患を対象とするフェーズ2治験のトップラインデータに関するプレスリリースを受け、米国ニューヨークに本拠を置く投資銀行 H.C. Wainwright & Co.のアナリストである Lander Egaña Gorroño 氏による、当社レポートが発表されましたので、参考情報としてお知らせいたします。

なお、当該レポートは、恐れ入りますが、権利の都合上、英文のままでのご案内となりますので、ご了承ください。

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※当該レポートは、本書の下部にございますので、スクロールしてご確認ください。

MediciNova, Inc.（メディシノバ・インク）
東京事務所 IR担当
E-mail infojapan@medicinova.com
URL https://medicinova.jp/

H.C.WAINWRIGHT&CO.

Company Update
Healthcare
September 28, 2026

MediciNova, Inc. (MNOV)
Rating: Buy

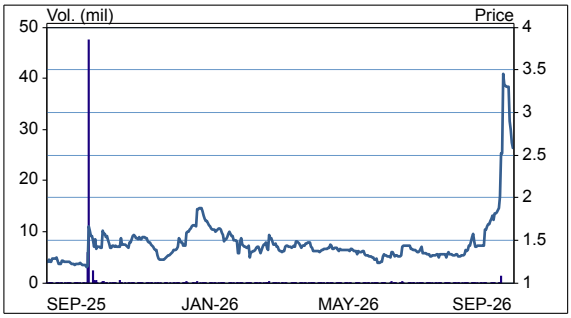
Lander Egaña Gorroño, Ph.D.
212-916-3977
legana@hcwresearch.com

Mixed Phase 2 Efficacy Signals Support Potential Evaluation of Tipelukast's Broader Metabolic Effects

Stock Data	9/25/2026
Price	$2.58
Exchange	NASDAQ
Price Target	$10.00
52-Week High	$3.90
52-Week Low	$1.17
Enterprise Value (M)	$102
Market Cap (M)	$127
Shares Outstanding (M)	49.2
3 Month Avg Volume	274,449
Short Interest (M)	0.31
Balance Sheet Metrics	
Cash (M)	$25.4
Total Debt (M)	$0.0
Total Cash/Share	$0.52

EPS ($) Diluted			
Full Year - Dec	2025A	2026E	2027E
1Q	(0.06)	(0.05)A	--
2Q	(0.07)	(0.05)A	--
3Q	(0.06)	(0.06)	--
4Q	(0.06)	(0.11)	--
FY	(0.24)	(0.28)	(0.45)

Revenue ($M)			
Full Year - Dec	2025A	2026E	2027E
1Q	0.0	0.2A	--
2Q	0.1	0.5A	--
3Q	0.1	0.2	--
4Q	0.2	0.1	--
FY	0.4	0.9	1.0



Our takeaways from the PoC Phase 2 NATG-202 topline readout:

- Tipelukast misses 24-week co-primary endpoints, but exhibits an early reduction in serum TG and a numerical reduction in liver fat
- Statistically significant improvements in HDL-C and HDL-P, alongside a trend toward lower body weight, point to potentially broader metabolic effects that warrant further evaluation
- Changes in TG, HDL-C, and HDL-P are consistent with findings from preclinical MoA studies
- The company plans to use the findings to inform the potential next stage of tipelukast development, including the appropriate patient population and study design (pending guidance)
- Main focus is ibudilast's Phase 2b/3 COMBAT-ALS data by YE26

Mixed week 24 results; missing on TG and liver fat, but offering broader metabolic insight. This morning, MediciNova announced topline results from the Phase 2 NATG-202 study evaluating tipelukast (MN-001) for the treatment of hypertriglyceridemia (HTG) and nonalcoholic fatty liver disease (NAFLD) associated with type 2 diabetes (T2D). As a reminder, NATG-202 was a multi-center (U.S.), randomized, double-blind, placebo-controlled study evaluating the safety and efficacy of tipelukast in 40 T2D patients with HTG and NAFLD (NCT05464784). Participants (FibroScan score ≥ 248 dB/m, HbA1c >6.5 and ≤10% at screening, oral anti-diabetic therapy for ≥3 months, fasting TG levels >150 mg/dL at screening) were randomized to receive either 500 mg/day of tipelukast or placebo for 24 weeks. The co-primary endpoints were change from baseline in liver fat content (FibroScan; controlled attenuation parameter (CAP) score) and change from baseline in fasting serum TG levels at week 24. Secondary endpoints included safety and tolerability, and changes in lipid profile. Results showed:

- At week 4, mean serum TG decreased from baseline by 54.7 mg/dL (26.05%) in the tipelukast arm vs. 23.8 mg/dL (11.54%) in the placebo group, representing a 30.96 mg/dL greater reduction with tipelukast vs. placebo (p=0.015).
- At week 24, mean serum TG decreased from baseline by 45.8 mg/dL (21.78%) in the tipelukast arm vs. 14.4 mg/dL (6.97%) in the placebo group, representing a 31.4 mg/dL greater reduction with tipelukast vs. placebo (p=0.113).
- At week 24, CAP score decreased from baseline by 14.1 dB/m (4.24%) in the tipelukast group vs. 4.3 dB/m (1.27%) with placebo, representing a 9.7 dB/m greater reduction with tipelukast vs. placebo (p=0.2438).

- At week 24, mean HDL-C increased by 3.3 mg/dL (8.39%) with tipelukast vs. a 2.4 mg/dL (6.23%) decline with placebo, corresponding to a 5.7 mg/dL treatment difference (p=0.0048).
- At week 24, HDL particle (HDL-P) concentration increased by 3.62 µmol/L (11.80%) with tipelukast vs. a 0.9 µmol/L (3.00%) decline with placebo, representing a 4.52 µmol/L treatment difference (p=0.018).
- Body weight tended to favor tipelukast, with a decrease of 4.91 lb (2.28%) vs. 0.55 lb (0.25%) with placebo at the end of the study (p=0.082).
- Tipelukast was generally safe and well tolerated, with mild to moderate TRAEs and no drug-related SAEs.

Overall, we view NATG-202 as providing a mixed efficacy signal for tipelukast in this small, PoC, exploratory study. While the numerical magnitude of the TG treatment difference was broadly maintained through week 24, the study did not demonstrate a statistically significant between-group difference at the prespecified co-primary endpoints of change in TG and liver fat content. The week 4 TG result provides evidence of an early lipid-lowering effect, but the lack of statistical significance at week 24 may limit the strength of the evidence for a sustained treatment effect. Similarly, while CAP score decreased numerically more with tipelukast vs. placebo, the difference did not reach statistical significance. The more notable findings came from the broader lipid profile. The statistically significant increases in HDL-C and HDL-P, together with the numerical reduction in body weight, suggest broader metabolic effects that may warrant further investigation in a larger study with an appropriately selected patient population, endpoints, time points, and sample size. Mechanistically, we believe these findings are directionally consistent with tipelukast's proposed multimodal MoA (see below). The company expects to conduct further detailed analyses of the study data and use these findings to inform the design and target population of a potential follow-on study.

Tipelukast aims to reshape the HTG-NAFLD-T2D axis. Tipelukast was in-licensed from Kyorin in 2002 as an orally bioavailable small molecule with multiple MoA targeting inflammation (5-lipoxygenase (5-LO)/ leukotriene (LTs), PDE, and CD36 pathways), fibrosis, and lipid metabolism. Preclinical studies demonstrated anti-fibrotic effects in livers from NAFLD animal models, and inhibition of TG biosynthesis and lipid-modifying activity in hepatotoxic and monocytic human cell lines, respectively. Of note, HTG, NAFLD, and T2D are pathophysiologically interconnected through systemic insulin resistance, dysregulated lipid metabolism, and chronic inflammation, forming a self-reinforcing metabolic axis. An open-label Phase 2 trial in patients with NAFLD and HTG (n=14) showed that tipelukast treatment led to early serum TG reductions (28.8%; p=0.00006) at week eight. Interestingly, subgroup analyses demonstrated that compared to participants without T2D (n=9), T2D patients (n=10) showed greater TG reductions at week eight (50.8% vs. 17.8% reduction in non-T2D), and a greater high-density lipoprotein cholesterol (HDL-C) increases (15.8% vs. 1.0% in non-T2D; p<0.0002), strengthening the case for prioritizing T2D patients in further clinical evaluation. There were no clinically significant safety and tolerability issues related to tipelukast. Details on tipelukast's MoA, preclinical data demonstrating significant anti-fibrotic effects, and clinically meaningful TG reductions in NAFLD patients with HTG can be found in: *A Novo Approach to Multi-Target Medicine; Initiating at Buy and $10 PT*.

Ibudilast completes double-blind COMBAT-ALS portion; topline data in December. Recently, MediciNova announced the completion of the last patient last visit (LPLV) in the double-blind portion of the Phase 2b/3 COMBAT-ALS trial evaluating ibudilast in ALS. A total of 234 participants were randomized across clinical sites in the U.S. and Canada and have now completed the 12-month double-blind treatment period, with patients remaining in the study continuing active treatment in the six-month open-label extension (OLE). Recall, the trial's primary endpoint is the Combined Assessment of Function and Survival (CAFS), with secondary endpoints including change in the ALS Functional Rating Scale–Revised (ALSFRS-R), muscle strength (hand-held dynamometry), and QoL, which could provide additional support for the treatment effect. With enrollment and dosing complete, the company's next step is database lock and analysis, with topline results from the double-blind portion expected by YE26, most likely in December. LPLV for the full study, including the OLE, is anticipated in March 2027. As a reminder, the company recently announced that target enrollment (n=200) was completed in ibudilast's SEANOBI EAP (see next). With the two programs expected to provide complementary datasets, we see ibudilast well positioned for a pivotal period and look forward to upcoming clinical and regulatory updates.

SEANOBI EAP fully enrolled and on track to complement COMBAT-ALS. Recently, MediciNova announced that the target enrollment of 200 patients has been achieved in the SEANOBI Expanded Access Program (EAP) evaluating ibudilast in ALS patients. With funding from the National Institute of Neurological Disorders and Stroke (NINDS/NIH), the multicenter, open-label EAP offers access to ibudilast treatment to ALS patients who are not eligible to participate in the COMBAT-ALS randomized clinical trial, while also generating important biomarker (NfL) and clinical outcome (ALSFRS-R) data from a real-world ALS population (NCT06743776). Active participants in SEANOBI remain on track with treatment and protocol-specified follow-ups. Final patient follow-ups are projected to conclude in early 2027, triggering the data analysis phase. We believe this enrollment milestone underscores strong interest from the ALS community in ibudilast and its potential as a novel therapeutic option for ALS. Together with Orphan Drug Designations from the FDA and EMA, and Fast Track Designation from the FDA, ibudilast continues to advance toward potential approval for the treatment of ALS.

Early functional and survival signals meet ibudilast's late-stage validation; Phase 2b/3 COMBAT-ALS data by YE26. MediciNova in-licensed ibudilast from Kyorin Pharmaceutical (TSE:4569; OTC:KYRNF; not rated) in 2004, following approval in Japan and South Korea for the treatment of asthma and post-stroke complications (i.e. dizziness) in the late 1980s. Ibudilast is a multi-target, oral small molecule inhibitor of phosphodiesterase (PDE), macrophage migration inhibitory factor (MIF), and toll-like receptor (TLR)-mediated pathways, that presents a multi-modal anti-inflammatory and neuroprotective MoA. The responder analysis from the Phase 2 ALS-1201 clinical trial evaluating ibudilast, in combination with riluzole SoC, in 49 ALS patients showed higher rates of stable or improved ALS Functional Rating Scale-Revised (ALSFRS-R), manual muscle testing (MMT), and five-item ALS Assessment Questionnaire (ALSAQ-5) scores at month six compared to the placebo + riluzole group. In addition, subjects who completed six or 12 months of treatment exhibited improved survival (p=0.0025) of up to 30 months following treatment. Ibudilast was generally safe and tolerable when administered with riluzole. While we acknowledge ALS-1201's single-site risk and the potential confounding effect of riluzole combination, our positive view is primarily driven by the favorable ALSFRS-R outcomes. Notably, prior late-stage ALS trials have shown that investigational therapies often struggle to sustain long-term functional ALSFRS-R improvements and meet clinical endpoints. We expect the ongoing Phase 2b/3 COMBAT-ALS trial to validate prior efficacy (functional and survival) and safety signals in a larger population in the longer-term (12 months), and inform next steps for ibudilast, including potential progression to regulatory filing or a confirmatory pivotal trial. Enrollment in COMBAT-ALS has been completed with a total of 234 randomized patients, and topline data are anticipated by YE26. While COMBAT-ALS does not incorporate prespecified biomarker endpoints, the ongoing NIH-supported SEANOBI Expanded Access Program (EAP) aims to generate meaningful neurofilament light chain (NfL) and clinical outcome (ALSFRS-R) data from the real-world ALS population. We believe that its broad, genotype-agnostic, and multi-modal approach, together with Phase 2 evidence of favorable safety and efficacy, support the clinical and regulatory advancement of ibudilast as a novel treatment to delay ALS disease progression.

Advancing dual-asset pipeline; focused on ALS data. MediciNova remains focused on its core pipeline assets, including ibudilast (MN-166), currently in Phase 2b/3 studies for amyotrophic lateral sclerosis (ALS), and tipelukast (MN-001), which completed Phase 2 evaluation for the treatment of hypertriglyceridemia (HTG) and nonalcoholic fatty liver disease (NAFLD) in patients with type 2 diabetes (T2D). As a reminder, Ibudilast demonstrated stabilization or improvement in several functional ALS scores and prolonged survival in Phase 2 studies. The ongoing Phase 2b/3 COMBAT-ALS trial aims to validate ibudilast's longer-term efficacy in a larger ALS population, and inform next regulatory/clinical steps (topline data by YE26). For tipelukast, following preclinical data demonstrating significant anti-fibrotic effects, and clinically meaningful triglyceride (TG) reductions in NAFLD patients with HTG, which were greater in those with concomitant T2D, results from the Phase 2 NATG-202 trial pointed toward potential broader metabolic effects that warrant evaluation in a larger trial (pending program guidance). Current cash position should provide runway through ibudilast (Phase 2b/3) readout and, if results are positive, the initiation of a later-stage trial. See additional details from both programs in our initiation report: *A Novo Approach to Multi-Target Medicine; Initiating at Buy and $10 PT*.

Broad indication optionality strengthens strategic collaboration prospects for ibudilast. MediciNova structures its pipeline around internally-funded core programs (strategic priority; ALS and HTG/NAFLD/T2D) and a capital-efficient, non-core portfolio characterized by a differentiated "platform-in-a-molecule" approach focused on ibudilast. The likelihood of ibudilast's multi-indication potential is reinforced by its multi-modal MoA and extensive human safety data pre- and post-approval in Japan and South Korea. This model enables focused investment in priority indications (ALS) while expanding the clinical reach of ibudilast through external collaborations. We view the non-core pipeline as a meaningful value driver for the company, offering: 1) capital-efficient development with minimal internal R&D spend; 2) diversified clinical risk for ibudilast beyond ALS; and 3) a strategic pathway to potential partnerships for late-stage development and commercialization. Collectively, this framework enhances both downside protection and long-term optionality across indications, in our belief. Non-core programs include clinical development in progressive multiple sclerosis (MS; Phase 2b completed), substance dependence (Phase 2 ongoing in methamphetamine dependence), chemotherapy-induced peripheral neuropathy (CPIN; Phase 2b ongoing), degenerative cervical myelopathy (DCM; Phase 3 ongoing), glioblastoma (Phase 1/2 completed), acute respiratory distress syndrome (ARDS; Phase 2 completed), and Long COVID (Phase 2/3 ongoing). On top of ALS, the FDA granted Fast Track designations to ibudilast for the treatment of progressive MS and methamphetamine dependence. With respect to the lead, non-core MS program, ibudilast showed ability to significantly reduce the rate of whole-brain atrophy (48% reduction) vs. placebo (p=0.04) in these patients. Ibudilast also demonstrated a 26% reduction (HR=0.74) in the risk of confirmed disability progression, with the greatest benefits observed in secondary progressive MS patients without relapse (46% risk reduction; HR=0.538). MediciNova finalized the design of a future registrational trial, and the progressive MS program is Phase 3-ready pending potential collaborators and/or partnerships.

Valuation and Risks. We maintain our Buy rating and $10 price target. Our valuation is based on our clinical net present value (NPV) model, which allows us to flex multiple assumptions affecting a drug's profile. We currently value MediciNova based on the contribution of its core clinical-stage assets, ibudilast (MN-166) in ALS (15% PoS; 33% contribution) and tipelukast (MN-001) for the treatment of HTG and NAFLD due to T2D (20% PoS; 67% contribution) in the U.S. Moving forward, we believe significant upside potential exists based on: (1) attaining higher market penetration than currently projected in the above-mentioned indications; (2) augmenting projected chances of success based on the progress of the clinical candidates; (3) adding additional commercial geographies; and (4) progress of non-core programs through licensing deals and/or collaboration

agreements. Risk factors that could impede the stock from reaching our PT include failed or inconclusive clinical trials, the inability of the company to secure adequate funding to progress its drugs through the development pathway or the occurrence of dilutive capital raises.

($ in millions except per share data)

Profit & Loss	2022A	2023A	2024A	2025A	2026E	2027E	2028E	2029	2030
Licensing and R&D revenue	0.0	0.0	0.0	0.4	0.9	1.0	1.2	1.5	1.5
Milestone revenue	0.0	1.0	0.0	0.0	0.0	0.0	0.0	1.0	2.0
Product and Royalties	0.0	0.0	0.0	0.0	0.0	0.0	60.0	185.0	305.0
Other revenues	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.0	2.0
Revenues	**0.0**	**1.0**	**0.0**	**0.4**	**0.9**	**1.0**	**61.2**	**188.5**	**310.5**
CoGS	0.0	0.0	0.0	0.0	0.7	0.9	5.2	7.9	11.3
Gross Profit	**0.0**	**1.0**	**0.0**	**0.4**	**0.3**	**0.2**	**56.0**	**180.6**	**299.2**
Gross margin	*0%*	*100%*	*0%*	*100%*	*28%*	*15%*	*92%*	*96%*	*96%*
G&A	5.5	5.2	5.5	6.2	7.1	11.4	20.0	36.0	43.2
R&D	9.1	5.7	7.2	7.2	8.5	15.8	20.5	25.6	30.7
Other op ex	0.0	0.0	0.0	0.4	0.0	0.0	0.0	1.0	2.0
EBIT	**(14.6)**	**(9.9)**	**(12.7)**	**(13.3)**	**(15.4)**	**(27.0)**	**15.5**	**118.0**	**223.3**
EBIT margin	*nm*	*nm*	*nm*	*nm*	*nm*	*nm*	*25%*	*63%*	*72%*
Depreciation	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Amortization Intangibles	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
EBITDA	**(14.6)**	**(9.9)**	**(12.7)**	**(13.3)**	**(15.4)**	**(27.0)**	**15.5**	**118.0**	**223.3**
EBITDA margin	*nm*	*nm*	*nm*	*nm*	*nm*	*nm*	*25%*	*63%*	*72%*
Non operating expenses	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Net Interest Income/Other	0.6	1.3	1.6	1.3	1.5	1.6	1.7	2.1	2.4
Interest expense	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.2	0.3
EBT	**(14.1)**	**(8.6)**	**(11.0)**	**(12.0)**	**(13.9)**	**(25.4)**	**17.2**	**119.9**	**225.4**
EBT margin	*nm*	*nm*	*nm*	*nm*	*nm*	*nm*	*28%*	*64%*	*73%*
Provision for taxes	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	27.1
Net Income	**(14.1)**	**(8.6)**	**(11.0)**	**(12.0)**	**(13.9)**	**(25.4)**	**17.2**	**119.9**	**225.4**
Participation of preferred stock	0.0	0.0	0.0	0.0	0.0	0.0	0.0	1.0	2.0
Net Income to common	**(14.1)**	**(8.6)**	**(11.0)**	**(12.0)**	**(13.9)**	**(25.4)**	**17.2**	**120.9**	**200.4**
net margin	*nm*	*nm*	*nm*	*nm*	*nm*	*nm*	*28%*	*64%*	*65%*
Number of shares - basic	49.0	49.0	49.0	49.1	50.3	56.9	60.0	61.0	63.6
Number of shares - diluted	49.0	49.0	49.0	49.1	50.3	56.9	62.5	64.6	66.9
EPS - basic	**(0.29)**	**(0.17)**	**(0.23)**	**(0.24)**	**(0.28)**	**(0.45)**	**0.29**	**1.98**	**3.15**
EPS - diluted	**(0.29)**	**(0.17)**	**(0.23)**	**(0.24)**	**(0.28)**	**(0.45)**	**0.28**	**1.87**	**3.00**

Source: SEC filings and H.C. Wainwright estimates.

Lander Egaña Gorroño, Ph.D. legana@hcwco.com

Quarterly P&L

	Q1'25A	Q2'25A	H1'25A	Q3'25A	9M'25A	Q4'25A	FY'25A	Q1'26A	Q2'26A	H1'26A	Q3'26E	9M'26E	Q4'26E	FY'26E
Licensing and R&D revenue	0.00	0.13	0.13	0.12	0.26	0.15	0.4	0.19	0.46	0.65	0.15	0.80	0.10	0.9
Milestone revenue	0.00	0.00	0.00	0.00	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.0
Product and Royalties	0.00	0.00	0.00	0.00	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.0
Other revenues	0.00	0.00	0.00	0.00	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.0
Revenues	**0.00**	**0.13**	**0.13**	**0.12**	**0.26**	**0.15**	**0.4**	**0.19**	**0.46**	**0.65**	**0.15**	**0.80**	**0.10**	**0.9**
CoGS	0.00	0.12	0.12	0.12	0.23	-0.23	0.0	0.17	0.14	0.31	0.16	0.47	0.18	0.7
Gross Profit	**0.00**	**0.02**	**0.02**	**0.01**	**0.03**	**0.38**	**0.4**	**0.02**	**0.32**	**0.33**	**-0.01**	**0.32**	**-0.07**	**0.3**
Gross margin	*nm*	*nm*	*nm*	*nm*	*nm*	*nm*	*100%*	*nm*	*nm*	*nm*	*nm*	*nm*	*nm*	*28%*
G&A	1.36	1.44	2.80	1.81	4.61	1.55	6.2	1.59	1.83	3.42	1.85	5.27	1.87	7.1
R&D	1.84	2.19	4.03	1.58	5.61	1.54	7.2	1.26	1.03	2.29	1.57	3.86	4.65	8.5
Other op ex	0.00	0.00	0.00	0.00	0.00	0.38	0.4	0.00	0.00	0.00	0.00	0.00	0.00	0.0
EBITDA	**(3.2)**	**(3.6)**	**(6.8)**	**(3.4)**	**(10.2)**	**(3.1)**	**(13.3)**	**(2.8)**	**(2.5)**	**(5.4)**	**(3.4)**	**(8.8)**	**(6.6)**	**(15.4)**
EBITDA margin							*nm*							*nm*
Non operating expenses	0.00	0.00	0.00	0.00	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.0
Net Interest Income/Other	0.34	0.33	0.66	0.33	0.99	0.30	1.3	0.25	0.23	0.48	0.42	0.90	0.60	1.5
Interest expense	0.00	0.00	0.00	0.00	0.00	0.00	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.0
EBT	**(2.9)**	**(3.3)**	**(6.1)**	**(3.1)**	**(9.2)**	**(2.8)**	**(12.0)**	**(2.6)**	**(2.3)**	**(4.9)**	**(3.0)**	**(7.9)**	**(6.0)**	**(13.9)**
EBT margin							*nm*							*nm*
Provision for taxes	0.00	0.00	0.00	0.00	0.00	0.01	0.0	0.00	0.00	0.00	0.00	0.00	0.00	0.0
Participation of preferred stock							0.0							0.0
Net Income to common	**(2.86)**	**(3.3)**	**(6.1)**	**(3.1)**	**(9.2)**	**(2.8)**	**(12.0)**	**(2.59)**	**(2.3)**	**(4.9)**	**(3.0)**	**(7.9)**	**(6.0)**	**(13.9)**
net margin							*nm*							*nm*
NoSH basic	49.05	49.05	49.05	49.05	8.34	49.12	49.06	49.22	49.22	49.22	50.45	8.34	52.43	50.33
NoSH diluted	49.05	49.05	49.05	49.05	8.34	49.12	49.06	49.22	49.22	49.22	50.45	8.34	52.43	50.33
EPS - basic	**(0.06)**	**(0.07)**	**(0.13)**	**(0.06)**	**(1.10)**	**(0.06)**	**(0.24)**	**(0.05)**	**(0.05)**	**(0.10)**	**(0.06)**	**(0.95)**	**(0.11)**	**(0.28)**
EPS - diluted	**(0.06)**	**(0.07)**	**(0.13)**	**(0.06)**	**(1.10)**	**(0.06)**	**(0.24)**	**(0.05)**	**(0.05)**	**(0.10)**	**(0.06)**	**(0.95)**	**(0.11)**	**(0.28)**

Source: SEC filings and H.C. Wainwright estimates.

Lander Egaña Gorroño, Ph.D. legana@hcwco.com

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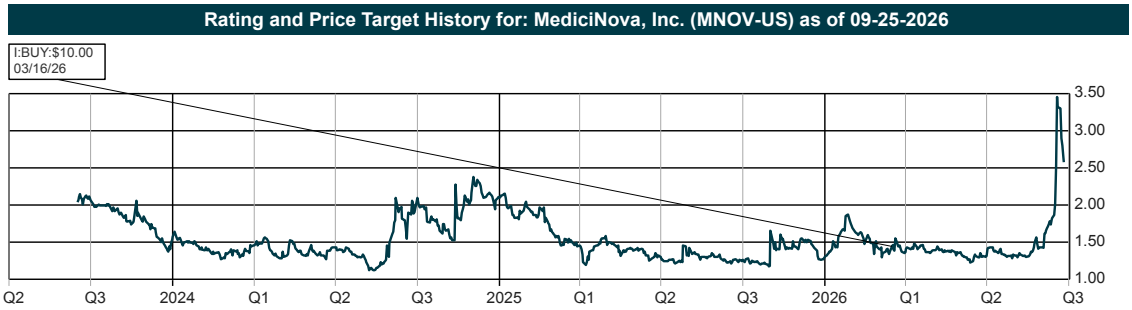

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			IB Service/Past 12 Months	
Ratings	Count	Percent	Count	Percent
Buy	545	84.50%	158	28.99%
Neutral	50	7.75%	12	24.00%
Sell	2	0.31%	0	0.00%
Under Review	48	7.44%	18	37.50%

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