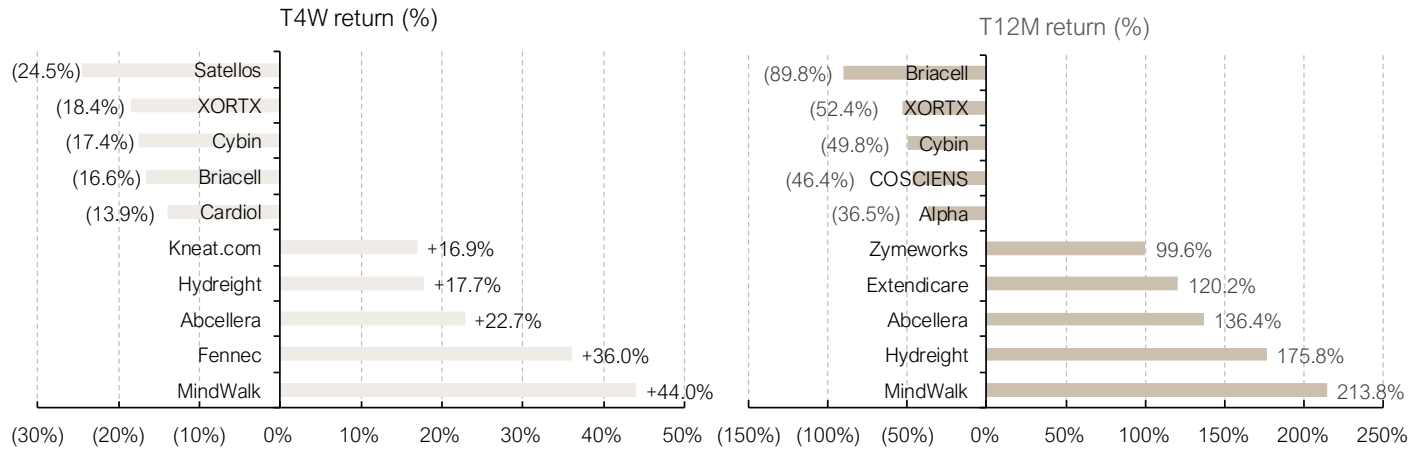
Core Highlights of the Week

Top Movers

Exhibit 1. Top Healthcare/Biotechnology Movers for the Trailing Four-Week & YTD Periods



Source: Leede Financial, Refinitiv

Updates From Our Healthcare Universe

- **Servier acquires Satellos peer firm Edgewise on attractive terms.** Earlier this week, France-based private specialty pharmaceutical giant Servier acquired Edgewise Therapeutics (EWTX-Q, NR) in a deal valuing the firm at US$1.55B in upfront cash to shareholders but with potential for up to another US$1.1B in downstream regulatory & commercial milestones. Edgewise has a few early-stage cardiometabolic-focused assets in its development pipeline but it is clear from the press release announcing the transaction that Servier's primary interests lie in Edgewise's myosin ATPase-inhibiting small-molecule drug sevasemten (EDG-5506) for which Phase II/III testing is already well underway in both Duchenne muscular dystrophy (DMD) & Beckers muscular dystrophy (BMD).

    - As we published in our original report on ON-based Phase II-stage DMD-focused SAT-3247 developer Satellos Biosciences (MSCL-T, Spec Buy, PT US$16.00), Edgewise Therapeutics & sevasemten were clearly identified as clinical-stage entities that we expected to track during Satellos' own Phase II activities in the 51-patient pediatric DMD-focused BASECAMP trial & in the 30-patient adult DMD-focused TRAILHEAD trial. Curiously, Satellos itself in its most recent Apr/26 investor presentation identifies many DMD drug development peers as suitable comparables for the firm, unsurprisingly featuring the US$12.0B recent acquisition of RNA-based oligonucleotide developer Avidity Biosciences by Novartis (NVS-NY, NR) in Feb/26, but not Edgewise even though the firm already reported positive Phase II data from the 76-patient LYNX trial, originally presented at the World Muscle Society in early FQ425.

    - Edgewise is simultaneously focused on Becker muscular dystrophy (a form of disease where dystrophin is still synthesized in muscle fibers but its function is diminished in some way) for which the 244-patient Phase II CANYON trial is ongoing (final efficacy data on muscle physiology & biomarker data are expected later this year). Satellos is not itself focused on Becker muscular dystrophy & our MSCL model does not ascribe value to this muscular dystrophy form.

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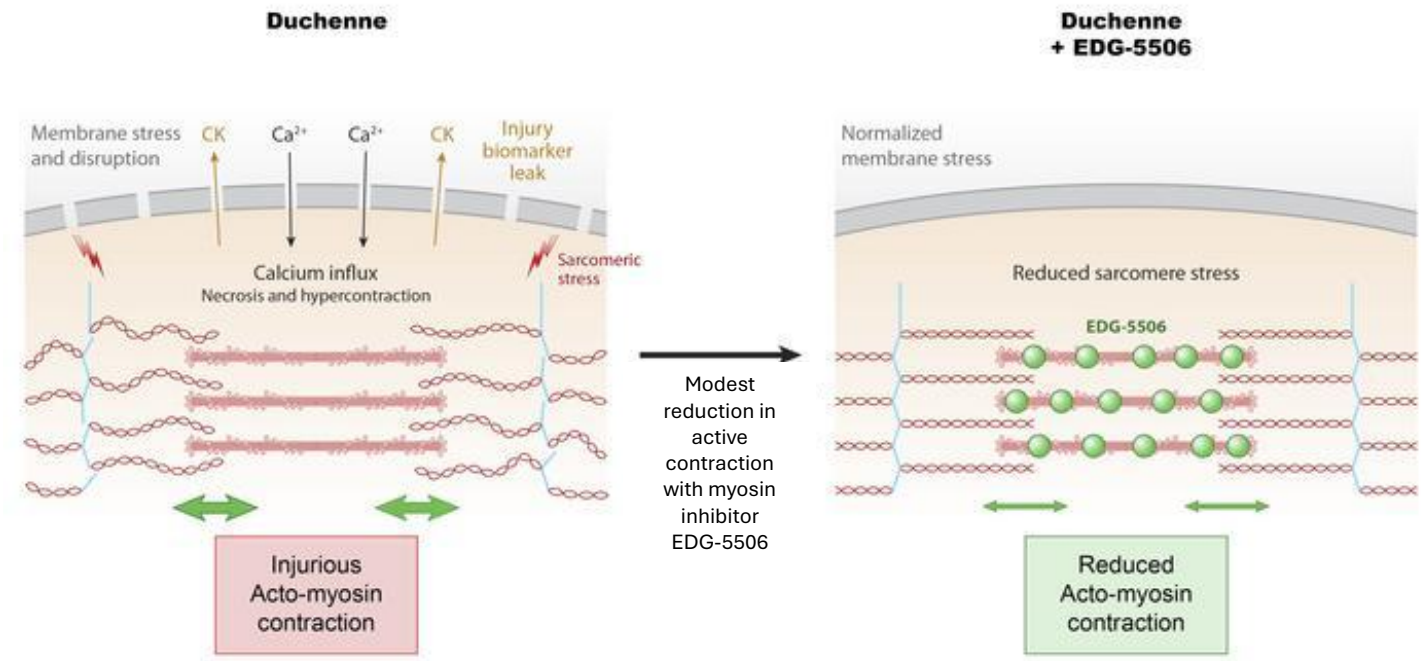
- We found it interesting then & we still do that the drug worked most effectively at one of the lower doses tested in the trial, as we show in Exhibit 3. Though Edgewise has published many sevasemten/EDG-5506 studies as conference proceedings, the firm has only two peer-reviewed study in the medical literature so far, a proof-of-concept preclinical pharmacology study published in 2023 in the *Journal of Clinical Oncology* & a description of a 97-patient Phase I/II trial in 2025 in the journal *Muscle & Nerve* that was actually focused on patients with Becker muscular dystrophy & not Duchenne muscular dystrophy, the latter of which is Satellos' sole target market for SAT-3247 to which we ascribe value in our MSCL model.

- As indicated above, sevasemten/EDG-5506 had distinctive pharmacology to the AAK1-inhibiting activity of SAT-3247 & so even if both molecules were successfully developed through Phase III DMD testing to approval, there is certainly potential for the drugs to be administered either in combination or sequentially in DMD patients. Semasemten/EDG-5506 modulates fast muscle fiber contraction by inhibiting a specific enzyme in muscle tissue called fast myosin ATPase. As background, there are two types of muscle fibers in human muscle, so-called fast muscle fibers (types IIa & IId) & slow muscle fibers (type I) & it is fast muscle fibers that are more susceptible to contraction-induced injury in DMD patients. Such injury manifests in multiple forms, including through muscle inflammation & degeneration, replacement of damaged fast muscle fibers with fat & fibrotic tissue, & ultimately a loss in muscle function.

Exhibit 2. Mechanism Of Action Originally Proposed For EDG-5506/Semasemten



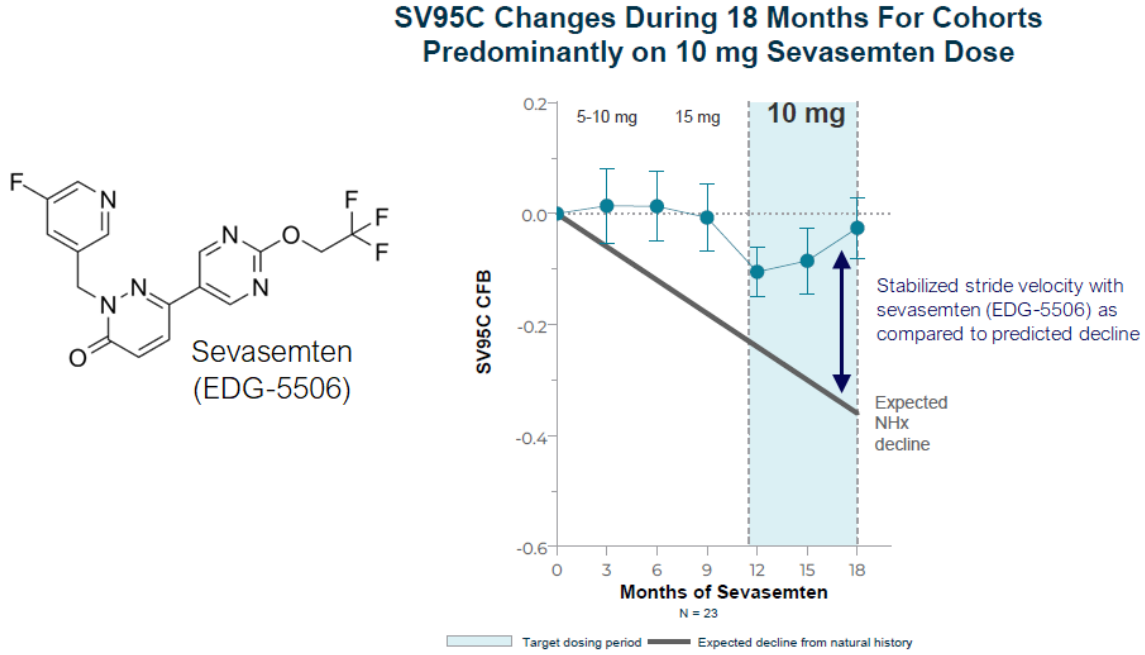
Source: Adapted from *Journal of Clinical Oncology (2023). Vol. 133, pp. e153837-153851*

- Absence of functional dystrophin of course kickstarts this process & restoration of dystrophin function by stimulating new dystrophin (or microdystrophin as a compromise) is the pharmacology through which most RNA-based therapies function. Alternative FDA-approved small-molecule drugs like Italfarmaco's (private) histone deacetylase (HDAC) inhibitor Duvyzat/givinostat or anti-inflammatory drugs Santhera Pharmaceuticals (SANN-SW, NR; also Health Canada-approved in Oct/25 & sold domestically by private specialty pharmaceutical firm ON-based Kye Pharmaceuticals) Agamree/vamorolone or PTC Therapeutics' (PTCT-Q, NR) Emflaza/deflazacort target secondary symptoms of disease without directly mitigating dystrophin physiology.

- Clearly the Biogen (BIIB-Q, NR)-Avidity transaction & now the Servier-Edgewise transaction establish reference valuations for Satellos if/when it generates an unambiguously positive Phase II efficacy signal from its ongoing TRAILHEAD & BASECAMP DMD trials, with both trials already reporting some encouraging (but not yet definitive) efficacy signals on improved muscle physiology in a handful of enrolled subjects. The schedule of imminent clinical

milestones for SAT-3247 in the aforementioned trials is well-populated with updates on both trials scheduled for FQ226-to-FQ426, including an update on BASECAMP progress expected before quarter-end. We are maintaining our Speculative Buy rating & one-year PT of US$16.00 on MSCL.

Exhibit 3. Edgewise's Fluorinated Nitrogen Heterocycle-Based Small-Molecule Drug Sevasemten (EDG-5506) Interestingly Showed Greatest Efficacy On Stride Velocity At the Lowest Dose Tested In the 76-Patient LYNX Trial



Source: Adapted from poster presentation at the *30th Annual International Congress of the World Muscle Society (Oct/25)*

- **Abivax reports Phase III ABTECT maintenance data - best-in-class ulcerative colitis efficacy but with emerging malignancy concerns – relevance to Eupraxia & its pending clinical programs with EP-104GI.** France-based drug developer Abivax SA (ABVX-PAR, NR) reported topline results from the Phase III ABTECT maintenance trial evaluating obefazimod, an orally-active once-daily small-molecule first-in-class miR-124 enhancer, in moderate-to-severe active ulcerative colitis. miRNA-124 is a well-characterized non-coding RNA sequence that regulates gene expression in the brain & the immune system, with the latter activity being notably relevant to gastrointestinal pathologies.
  - We provide commentary on the trial below, with relevance to our coverage universe uniquely flowing through Eupraxia Pharmaceuticals (EPRX-Q, Buy, PT US$15.50) & its emerging interests in deploying its DiffuSphere-based extended-release fluticasone propionate formulation EP-104GI in other gastrointestinal disorders beyond its current focus on esophageal disease (eosinophilic esophagitis in ongoing Phase II testing & imminently, esophageal strictures).
  - The trial re-randomized 580 clinical responders from the 8-week ABTECT induction studies to placebo (N=192), 25mg (N=193), or 50mg (N=195) for 44 weeks. Both active doses met the primary endpoint: placebo-adjusted clinical remission at Week 44 was 39.3% for 25mg (50.8% vs. 10.4% placebo, with extremely low p-value of <0.0001) and 40.3% for 50mg (51.3% vs. 10.4%; p-value also low at <0.0001). All key secondary endpoints were also met, including endoscopic remission (41.5% and 47.7% for 25mg and 50mg vs. 9.9% placebo), HEMI, corticosteroid-free remission, and sustained clinical remission.
  - The efficacy headline positions obefazimod at or near best-in-class for moderately to severely active ulcerative colitis. AbbVie's (ABBV-NY, NR) Rinvoq (upadacitinib 30mg, JAK inhibitor) at ~39% placebo-adjusted clinical remission after 52 weeks of maintenance is the only comparable figure, with Pfizer's (PFE-NY, NR) Velsipity (etrasimod, S1P modulator) next at ~25%. AbbVie's Skyrizi (risankizumab, IL-23p19), approved for UC in mid-2024, delivered placebo-adjusted maintenance remission of 14-16% in the COMMAND trial, though with a 25.1% placebo rate that reflects the withdrawal re-randomization design and complicates direct comparison.

- The IL-23 class is generally favored for its cleaner long-term safety profile, making the relevant framing one of efficacy-safety tradeoff rather than efficacy alone. On endoscopic remission, obefazimod's 47.7% (50mg) and 41.5% (25mg) at 44 weeks appear to exceed any published maintenance figure in UC, including upadacitinib 30mg at 25.9% in U-ACHIEVE (52 weeks). Cross-trial caveats around patient populations, trial design, and placebo rates apply throughout.

- Obefazimod is a quinoline-based small molecule that selectively enhances expression of microRNA-124 (miR-124) in immune cells. miR-124 is a physiological negative regulator of inflammation, acting primarily through post-transcriptional suppression of STAT3, a transcription factor central to Th17 differentiation, pro-inflammatory cytokine production, and macrophage activation. In UC, miR-124 expression is markedly downregulated in colonic tissue relative to healthy controls (Koukos & coworkers, as published in 2013 in *Gastroenterology*), and pharmacological restoration of miR-124 via obefazimod has been shown to reduce multiple pro-inflammatory markers and return cytokine profiles toward homeostasis (Tazi & coworkers, published in 2021 in *Drug Discovery Today*; also Apolit & coworkers, published in 2023 in *Clinical Translational Gastroenterology*). The mechanism is distinct from broad immunosuppression: rather than blocking entire cytokine signaling cascades (as JAK inhibitors do), obefazimod modulates a single upstream microRNA.

- The drug's safety profile, specifically malignancy risk, sent the stock tumbling in early Jun/26 following the after-hours data release. In the 50mg arm, seven malignancy events were observed over 44 weeks. Three observations of cancer emergence (one of prostate, breast, & colorectal cancer, with the latter known to emerge at higher frequency in ulcerative colitis patients anyway) were reported, as were four episodes of non-melanoma skin cancer (NMSC; two were basal cell carcinoma & two others were squamous cell carcinoma). By contrast, the 25mg arm had one diagnosis of squamous cell carcinoma while the placebo arm has one diagnosis of basal cell carcinoma. and no non-NMSC cases; placebo had 1 NMSC (BCC) and no non-NMSC cases. The dose-dependent imbalance in oncology-based side effects (if one can call cancer emergence a 'side effect') was likely the motivation for capital markets concern.

- ABVX share price declined from US$130/shr to an intraday low of US$72, corresponding to a US$4.5-to-US$5.0B decline in market value from a US$10.0B pre-data market value threshold, before re-equilibrating at a share value of US$105/shr in subsequent trading sessions. Investigators assessed all three non-NMSC cases as unrelated to treatment, with no organ-specific clustering. The NMSC cases occurred in patients with a mean age of 62 versus 42 for the overall trial population, and one had a prior history of skin cancer. NMSC is conventionally excluded from malignancy endpoints across oncology and I&I safety reporting; it is extremely common in the general population, heavily confounded by cumulative UV exposure, age, and skin type, and including it would obscure more clinically meaningful signals from visceral or hematologic malignancies. That said, the dose-dependent pattern in NMSC mirrors the non-NMSC imbalance and cannot be dismissed on convention alone.

- Mechanistically at odds with this signal, miR-124 has been extensively characterized as a tumor suppressor across multiple cancer types. In colorectal cancer, miR-124 is significantly downregulated relative to adjacent normal tissue, and reconstitution suppresses STAT3 expression, induces apoptosis, and reduces tumor growth in vitro and in xenograft models (Hatziapostolou & coworkers, published in 2013 in *PLoS One*). In glioma, miR-124 upregulation inhibits STAT3 signaling, reverses tumor-mediated immunosuppression of T-cell proliferation, reduces regulatory T-cell induction, and prolongs survival in murine models (Wei & coworkers, published in 2013 in the journal *Cancer Research*).

- In terms of the clinical safety database, the Phase IIb OLE (described by Sands & coworkers in 2025 in the *Journal of Crohns & Colitis*) treated 217 patients with obefazimod 50mg QD for up to 96 weeks, with a 75.6% completion rate (164/217). One meningioma, judged unrelated to treatment, was reported across the entire 96-week study period. Using a rough estimate of ~340 patient-years of exposure, that translates to approximately 0.3/100 PY for total malignancy. In the Phase III maintenance trial, the 50mg arm provides a much shorter exposure window (~165 PY, estimating ~195 patients across 44 weeks). The 3 non-NMSC events in this arm yield a point estimate of roughly 1.8/100 PY, and including NMSC reaches approximately 4.2/100 PY. These are point estimates with extremely wide confidence intervals given the small event counts (for 3 events in ~165 PY, the 95% Poisson CI spans roughly 0.4 to 5.3/100 PY), so definitive rate comparison is premature at this number of patients.

- For some context on the established bar for malignancy risks, in the U-ACTIVATE long-term extension of upadacitinib (Rinvoq) in ulcerative colitis (described in 2025 in the journal *Lancet Gastroenterology & Hepatology*), malignancy excluding NMSC was reported at 1.1/100 patient-year for the 15mg dose and 0.3/100 patient-year for 30mg, NMSC at 0/100 patient-year and 1.2/100 patient-year, respectively. Rinvoq carries a class-wide FDA black box warning for

malignancy, major adverse cardiovascular events, and venous thromboembolism. In the tofacitinib UC program (Olivera et al., J Crohns Colitis, 2020), malignancy excluding NMSC occurred at 0.7/100 patient-year [95% CI 0.3-1.2] across ~1,600 patient-year of exposure, with NMSC at the same rate.

- As stated above, we believe that Abivax's ulcerative colitis data are relevant to Eupraxia, for which more comprehensive clinical testing in gastroesophageal disorders beyond those already undergoing testing is on the horizon. Locally delivered corticosteroids with minimal systemic absorption sit at the opposite end of the immunomodulatory safety spectrum. In a GI landscape where novel-mechanism-of-action biologics and small molecules pose malignancy, cardiovascular, and thromboembolic signals, locally acting anti-inflammatory agents are increasingly differentiated from systemically-delivered anti-inflammatory agents based solely on their PK profile & not on their underlying pharmacology. New obefazimod data may sharpen that contrast for investors weighing risk-reward across distinct gastrointestinal disease-targeted modalities.

Other Significant Clinical Trial Updates With Relevance To Our Coverage Universe

- **Takeda reports positive clinical data for interleukin-2-based bispecific mAb, with relevance to Medicenna's own modified interleukin-2 formulation MDNA11.** Japan-based pharma giant Takeda (4502-JP, NR) reported encouraging Phase II data from a 136-patient Phase II non-small cell lung cancer trial at the annual ASCO clinical oncology meeting in Chicago this week, testing its anti-PD1 mAb-interleukin 2 bispecific fusion protein IBI363 (also called TAK-928 in Takeda's own pipeline) that is part of the firm's long-standing biologics development alliance with China-based Innovent Biologics (1801-HK, NR). by being at least partially based on the cytokine interleukin-2, data are clearly relevant to other interleukin-2 formulations that are targeting cancer-based disorders. The interleukin-2 component of IBI363 is engineered to enhance affinity for one of the three interleukin-2 receptor subunits, the alpha subunit, while mitigating interleukin-2 binding to its beta- or gamma-subunits of the trimeric receptor.

  - The list of such firms includes Werewolf Therapeutics' (WOLF-Q, NR) WTX-124, undergoing testing in a 150-patient Phase I/II solid tumor trial that coincidentally is combining the cytokine with an independent anti-PD1 mAb in pembrolizumab/Keytruda (final two-year survival data later this year), & of course ON-based Medicenna's (MDNA-T, NR) own modified albumin-fusion-interleukin-2 formulation MDNA11, undergoing testing in a 115-patient Phase I/II solid tumor trial (the ABILITY-1 trial) for which interim tumor response/survival data have been reported a few times, with notable but not exclusive impact on melanoma response rates. In ABILITY-1, MDNA11 is combined with an anti-PD1 mAb (pembrolizumab/Keytruda) & so has clear read-through to Takeda/Innovent's IBI363 & its bispecific pharmacology.

  - MDNA11 is also combined with pembrolizumab/ Keytruda in some ABILITY-1 patients; final data are also expected later this year. With emphasis on MDNA11, we were interested to see that Takeda/Innovent designed IBI363 so that it maintained its affinity for the interleukin-2 receptor's alpha subunit, while as shown in Exhibit 4, Medicenna's MDNA11 was genetically modified to mitigate its binding to this subunit & to enhance its binding affinity for the receptor's beta-subunit.

  - Takeda/Innovent's IBI363 lung cancer data were indeed encouraging, with median progression free survival (PFS) for non-small cell lung cancer patients treated at the highest of two test IBI363 dosage strengths of 10.1 months, median overall survival (OS) of 18.2 months, & with nearly half of high-dose patients surviving for at least two-years (47.8%, so fifteen of thirty-one enrolled lung cancer patients in that study arm). None of these patients harbored any known mutations in the epidermal growth factor receptor (EGFr).

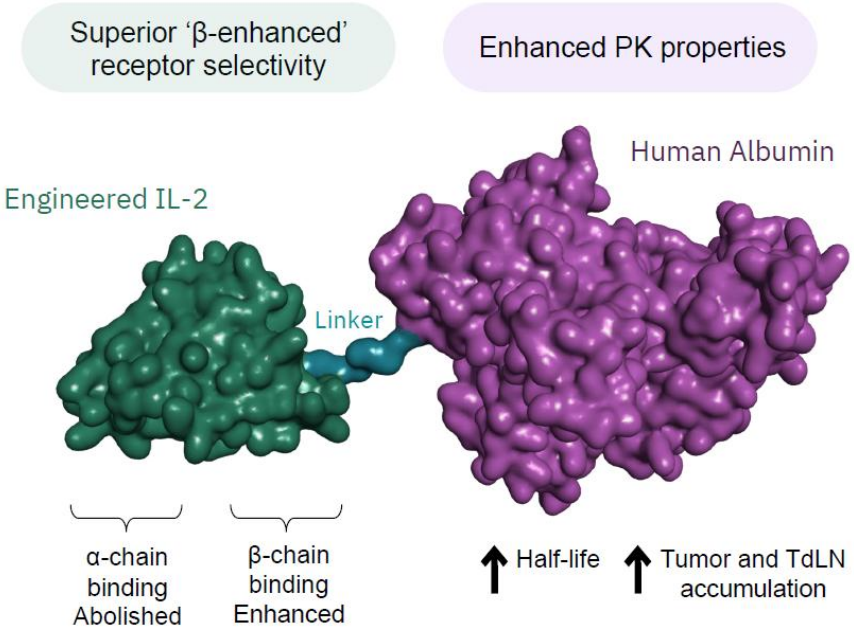
  - In a separate component of the trial testing 58 patients who did harbor EGFr mutations known to exacerbate lung cancer progression, high-dose IBI363-treated patients experienced slightly inferior but still directionally positive responses as compared to those patients described above, with median PFS-OS-two-year survival rate of 4.2 months, 15.2 months & 42.7%, respectively. Ironically, patients with mutated EGFr with a smoking history performed better, achieving median OS of 23.4 months & so anachronistically improving survival while enduring the lung cancer diagnosis that their smoking plausibly caused.

  - Coincidentally, Innovent entered into a multi-product development alliance with CT-based pharma giant Pfizer (PFE-NY, NR) in late May/26 in which up to twelve antibody-drug conjugated therapies are expected to be developed under the milestone-laden US$10.5B alliance, excluding downstream royalty revenue on any FDA/EMA-approved partnered assets that achieve commercial status, with US$650M of deal economics constitutes in upfront cash. None of the

partnered assets were specifically identified, as is conventional for omnibus alliances like this, but qualitative details shared made it clear that pipeline partnered assets will have 'differentiated immune-engaging' features, which the Takeda-partnered IBI363 certainly does with its interleukin-2 moiety.

- Innovent's pipeline independent of partnership status is both diversified & deep, & not exclusively focused on oncology by the way, with at least four clinical/commercial-stage targeting ophthalmologic indications (three are VEGF-targeted & thus likely targeting wet age-related macular degeneration or perhaps diabetic macular edema, the other is teprotumumab/Sycume, targeting insulin growth factor receptor-1 receptor & thyroid eye disease), autoimmune disorders (Crohn's disease, plaque psoriasis, rheumatoid arthritis, to name three), or cardiometabolic indications (mostly focused on serum cholesterol or serum glucose regulation).
- But shifting back to IBI363 specifically, the anti-PD1 mAb-interleukin-2 conjugated biologic is already undergoing testing in combination with the microtubule binding small-molecule docetaxel/Taxotere in a 600-patient Phase III non-small cell lung cancer trial (the MarsLight-11 trial) for which response rate/PFS/OS will constitute key endpoints for the trial. Other Phase II/III oncology studies focused on gastric cancer, melanoma, ovarian cancer or lymphoma are independently ongoing.

Exhibit 4. Medicenna's Own Albumin-Fusion Modified Interleukin-2-Based Biologic, MDNA11



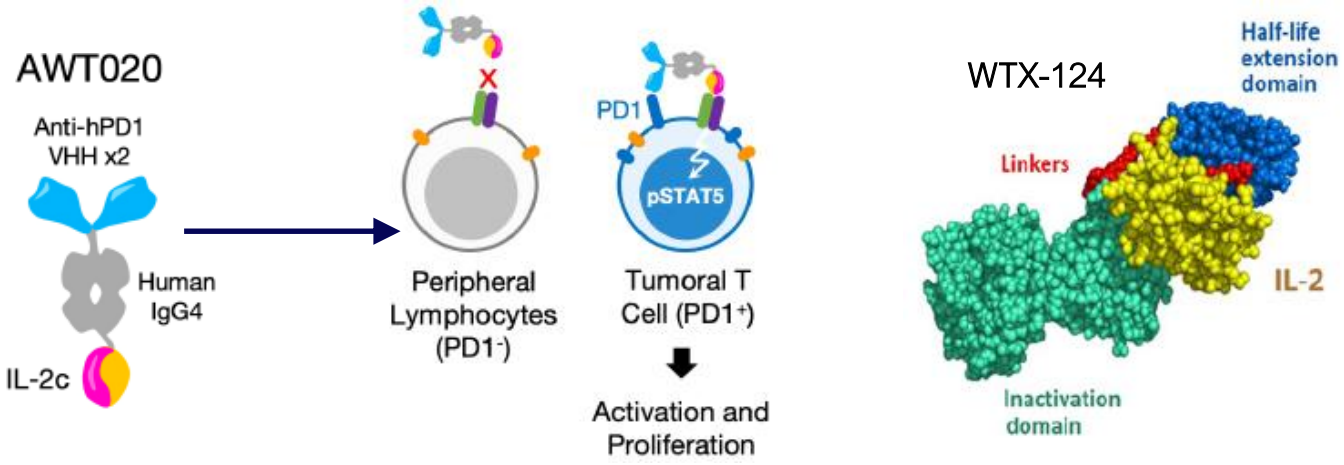
Source: Adapted from Medicenna presentation at *ESMO-IO Congress 2025* in Dec/25

- Notably, Merck's (MRK-NY, NR) multi-blockbuster anti-PD1 mAb pembrolizumab/Keytruda is itself already FDA-approved for treating metastatic non-squamous non-small cell lung cancer, in this case when combined with the folate derivative pemetrexed/Alimta & platinum-containing drugs (usually oxaliplatin/Eloxatin or carboplatin/Paraplatin), based on long-ago published data from the 607-patient Phase III KEYNOTE-189 trial (recently five-year survival data were summarized in a 2023 paper in the *Journal of Clinical Oncology*). Additionally, pembrolizumab/Keytruda is also approved for use in combination with carboplatin/Paraplatin & paclitaxel/Taxol or albumin nanoparticle-based paclitaxel (Abraxane) in treating squamous non-small cell lung cancer, as documented in the 558-patient Phase III KEYNOTE-407 trial (five-year survival data were separately summarized for this trial also in 2023 in the same journal).
- There are a few other interleukin-based bispecific targeted anti-cancer agents at various stages of development that we could identify in the medical literature & these include AWT020, an anti-PD1 nanobody fused with a modified interleukin-2 variation for which binding to the alpha subunit of the interleukin-2 receptor is mitigated (thus minimizing activation of regulatory T-cells) while binding to the beta- & gamma-subunits of the receptor still transpire but with lower affinity. This bispecific biologic was developed by CA-based private firm Anwita Biosciences & it is actually quite well-

characterized in the medical literature already, including in a review article published in 2025 by Anwita's research team in *Frontiers in Immunology* & in a separate functional characterization study published in Oct/25 in *International Journal of Biological Macromolecules*.

- An abstract was published at least year's ASCO meeting that described interim data from the firm's ongoing 60-patient Phase II Australia-based solid tumor trial, indicating therein that two of twelve evaluable subjects – both with thyroid cancer of variable severity – achieved partial responses, while six other patients exhibited disease stabilization or some measure of tumor response, with tumor size reduction of between 5%-to-24%. The study is still actively recruiting patients, as is a separate 214-patient Phase II solid tumor trial, in this case overseen by partner Shanghai-based Junshi Bioscience (1877-HK, NR) & enrolling patients in China-based hospitals.

Exhibit 5. Anwita Biosciences Anti-PD1-binding & Interleukin-2 Receptor-Binding Bispecific Anticancer Agent AWT020 & Werewolf's Interleukin-2-Based Indukine WTX-124



Source: Adapted from *Frontiers In Immunology (2025). Vol. 16, pp. 1537466-1537480; Cancer Immunology Research (2022). Vol. 10, pp. 581-596.*

- An alternative bispecific anticancer agent that we identified is BP2402, a fusion protein comprised of an anti-PD-L1 mAb & an anti-interleukin 8 mAb that is undergoing preclinical testing in triple negative breast cancer, as described in an Oct/25 paper published by China-based academic researchers & Jiangsu Kanion Pharmaceutical Co (600557-SHA, NR). Data in that paper nicely showed in a mouse model of disease that BP2402 could impede tumor growth to a greater degree than achievable by an anti-PD-L1 mAb alone (using Roche's [ROG-SW, NR] atezolizumab/Tecentriq) while stimulating T-cell infiltration of target tumors & impeding multiple signalling pathways known to propagate tumor growth, specifically VEGF-stimulated new blood vessel growth & JAK1-based inflammatory pathways to name two. BP2402 is not yet undergoing formal Phase II clinical testing, at least not to any stage of advancement that is indicated in the US NIH's clinical database.

- And lastly, another novel anti-PD-L1 mAb-based bispecific anti-cancer agent is acasunlimab (also called GEN1046), a fusion protein originally developed in partnership with BioNTech (BNTX-Q, NR) & Netherlands-based Genmab BV (GMAB-CPH, NR) that combines PD-L1-binding using Genmab's DuoBody platform with the T-cell surface receptor 4-1BB (a tumor necrosis factor analog that is also called CD137); acasunlimab is already characterized in the medical literature both in a 2022 paper in the journal *Cancer Discovery* & in a 2025 paper in the *Journal for ImmunoTherapy of Cancer*, showing in a MC38 mouse colorectal cancer model described in both papers that acasunlimab stimulated accumulation of tumor-specific cytotoxic T-cells within & near the tumor microenvironment. In parallel, acasunlimab stimulated Interleukin-2 signalling pathways in the same way that interleukin-2 itself presumably would.

- The mAb is of interest to us only from a mechanistic perspective since Genmab announced in its 2025 annual report that it will be discontinuing acasumlimab clinical testing. That said, Genmab does have three ongoing Phase II acasunlimab trials that may generate clinical data before they are terminated, two of which are focused on lung cancer (the 191-patient ABBIL1TY NSCLC-06 trial [data in late 2028] & a separate 125-patient Phase II trial on pace to

generate data later this year), with a 429-patient Phase I/II solid tumor safety/PK trial also on pace to generate data later this year. Genmab's suite of abstracts presented at the 2026 ASCO meeting this week were more focused on alternative bispecific DuoBody-based constructs, including FDA-approved CD3/CD30-dual-binding mAb Epkinly/epicoritamab & its clinical-stage EGFr-LGr5-targeted mAb petosem-tamab/GEN1158.

- But returning to Medicenna, the firm presented its own clinical update on MDNA11, featuring its intention to move forward with its Phase II 80-patient melanoma-focused NEO-CYT trial, with the four-arm trial comparing MDNA11 in combination with two other FDA-approved melanoma biologics in Bristol Myers Squibb's (BMY-NY, NR) anti-PD1 mAb nivolumab/Opdivo or its anti-CTLA-4 mAb ipilimumab/Yervoy, or both. An optional fifth arm could incorporate Roche/Genentech's (ROG-SW, NR) interleukin-6 receptor-targeted mAb tocilizumab/Actemra.
- The trial is not yet described in the US NIH's clinical database so we do not yet have any parameters to consider on timelines to data, but we do know that MDNA11 & one or more of the aforementioned melanoma therapies will be administered before surgery & primary endpoint will be response rate-based (major pathologic response, defined as <10% of viable tumor still visible during surgery). An MDNA11 clinical update focused on new patient outcomes from ABILITY-1 & on patient enrollment for NEO-CYT are expected in the next quarter or two.

- **Pemazyre (pemigatinib) shows evidence for first-line indication in the Phase III FIGHT-302 trial, with relevance to Knight Therapeutics & its Latin American marketing rights for the drug.** Results from the Phase III FIGHT-302 trial were presented at the 2026 ASCO meeting this week, representing the first randomized dataset evaluating a fibroblast growth factor receptor (FGFR) kinase inhibitor as first-line therapy in cholangiocarcinoma (CCA; a form of bile duct cancer). The trial compared pemigatinib to traditional chemotherapies, the nucleoside analog gemcitabine in combination with the platinum-containing drug cisplatin, in patients with untreated, unresectable or metastatic disease while harboring FGFR2 gene rearrangements that disrupt normal regulation of the protein.
  - Although enrollment stopped early (167 patients were randomized but with 432 patients initially targeted at initiation), the results were statistically positive on the primary endpoint - median PFS was 8.3 months for pemigatinib-treated patients as compared to 6.8 months for patients treated with gemcitabine/cisplatin, a difference that was deemed highly significant even with the smaller number of enrolled subjects in comparison to expected enrollment threshold. Additionally, there was a higher response rate (47% vs. 16%) & a much longer duration of response (14.2 months vs. 6.3 months) observed for pemigatinib-treated patients. Interestingly, overall survival did not improve (24.4 months vs. 25.0 months), but one explanation for this is that alternative FGFR inhibitors (including but not limited to J&J's [JNJ-NY, NR] urothelial carcinoma-targeted kinase inhibitor erdafitinib/Balversa) may have been available to control patients at the time that their disease progressed.
  - Pemigatinib is currently approved only as a second-line therapy; it received accelerated FDA approval in Ap/20 & conditional Health Canada approval in September 2021, both restricted to previously treated patients. QC-based specialty pharmaceutical firm Knight Therapeutics (GUD-T, NR) launched Pemazyre in Brazil & Mexico in Q4 2025 & Argentina in Q1 2026 under this second-line label. The commercial significance of a potential first-line label expansion is straightforward - the attrition from first- to second-line in advanced CCA is significant, with literature suggesting only 15–25% of patients maintain adequate performance status to receive second-line therapy (documented in a 2013 paper in *European Journal of Cancer* & a separate 2022 paper in *Liver International*).
  - With FGFR2 rearrangements present in roughly 10–16% of CCA patients, a first-line indication would capture the full pool of biomarker-positive patients at diagnosis rather than the fraction of patients who survive chemotherapy in good enough condition to receive targeted therapy. This analysis implies that a 4-6x expansion in the addressable patient population is achievable across Knight's Latin American markets. This product may contribute modestly to Knight's consolidated F2026 revenue expectations, especially when considering recently-augmented Canadian Rx sales expectations as driven by the Paladin Pharma re-acquisition, but still, pemigatinib Phase III data could drive a meaningful source of incremental optionality on a product franchise that is still in its early-launch phase across the region.
- **Hydreight reports FQ126 financial data.** BC-based digital health & wellness services provider Hydreight Technologies (NURS-V, NR) reported FQ126 financial results for the March-end quarter that were certainly up substantially & on all key metrics from FQ125 data, with adjusted revenue/gross margin/EBITDA in the quarter of $27.6M/$5.2M/$3.3M representing a seismic upward shift from $6.5M/$$2.0M/$0.2M in the corresponding FQ125 period. For added context, Hydreight's full-

year F2025 data were almost matched by the same corresponding data just in FQ126 alone, with F2025 adjusted revenue/gross margin/EBITDA of $43.6M/$9.4M/$2.5M itself dominated by FQ425 data of $16.9M/$2.9M/$1.6M that along with FQ126 data shows us that the firm is experiencing dramatic operating momentum just in the last two financial periods.

- We do not officially cover Hydreight but we have for some time been featuring the firm as the strongest performing stock within its peer group of healthcare IT & specialty healthcare firms, generating trailing two-year return of >1,788% that could be sustained at or near that relative return at least for another quarter or so – NURS.V shares experienced substantial appreciation during the Oct/24-to-Dec/24 period, during which NURS.V established a new floor from which we monitor comparative returns on the stock. NURS.V share value certainly generated a few trough levels since then, most notably during Mar/25 & again in Mar/26, presumably in anticipation of pending year-end financial data expectations.

- Interestingly, as we show in Exhibit 9, Hydreight is not trading at overly-aggressive profitability multiples, with current share price corresponding to 7.5x EV-to-F2027 consensus EBITDA forecast 10.4x & price-to-F2027 consensus EPS forecast. We will be interested to see if Hydreight can sustain revenue/EBITDA growth trajectory generated in recent periods & if more aggressive multiples could be ascribed to its share value in parallel.

- **NervGen provides clinical update on its NVG-291 spinal cord injury program, to which capital markets respond with acute caution.** BC-based central nervous system-focused peptide drug developer NervGen (NGEN-Q, NR) provided an update on its Phase I/II CONNECT SCI study testing its peptide drug NVG-291 as an agent for reversing neural damage in spinal cord injured patients.

  - The NVG-291 update was not overly quantitative but what data was shared certainly seemed at least directionally positive in our view, with NVG-291-treated patients experiencing improved gait quality as measured by the well-established Global Statistical Test (all ten evaluable NVG-291-treated patients were classified as responders, whereas only one in ten placebo patients were categorized as such). The firm asserted in its press release announcing the CONECT SCI update that it believes data are sufficiently positive to advance into a pivotal Phase III chronic tetraplegia trial (the RESTORE trial), for which FDA consultation has already transpired & for which enrollment is expected to commence by mid-year.

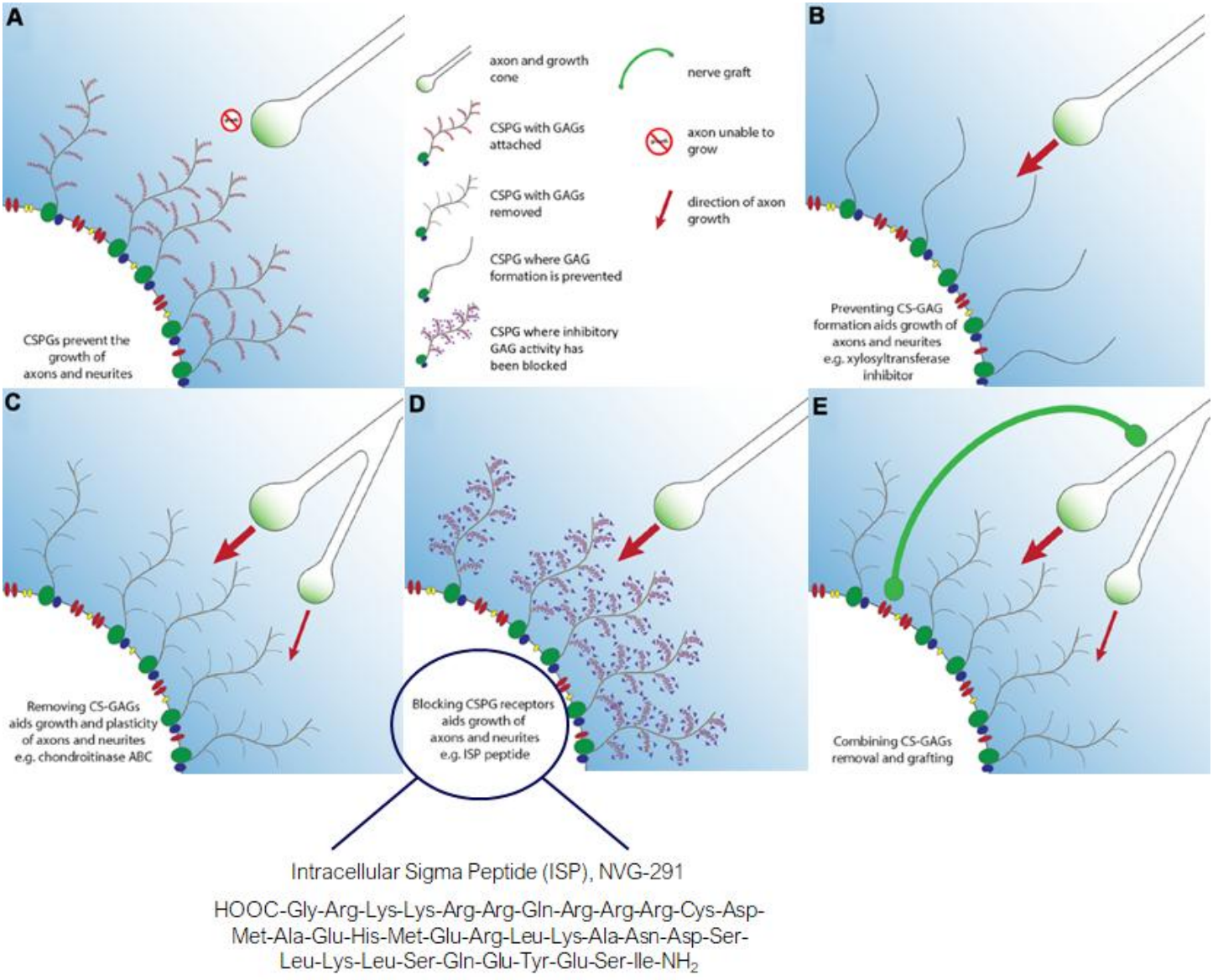
  - We have long believed that a single agent was unlikely to engender truly curative restoration-of-motor-function activity in spinal cord injury, but that bias is based on the historic refractoriness of spinal cord injury to any pharmacologic repair & not any mechanistic limitations in NVG-291 or the foundational science published by Case Western Reserve University researchers (as led by the now-deceased neuroscientist Jerry Silver). The drug is based on the concept that spinal cord healing is impeded by the deposition post-injury of a biopolymer called chondroitin sulfate proteoglycan (CSPG), which is actually a mechanism that the body evolved over time to mitigate any further neural damage to the site of injury but which unfortunately impedes any regenerative healing that the body might otherwise have been able to trigger.

  - The team at Case Western Reserve University discovered several years ago that a separate cellular receptor protein called protein tyrosine phosphatase sigma (PTPα) that is found both in the brain & spinal cord regulates CSPG deposition; accordingly, by impeding PTPα action in some way, it is thought that the body can initiate alternative regenerative mechanisms that CSPG was inhibiting. NVG-291 accomplishes this goal – the drug is a 35-amino acid unmodified peptide that is based on a peptide sequence that the Case Western Reserve University researchers characterized years ago called intracellular sigma peptide (ISP) that regenerated locomotor & sensory control in test animals, as published in a 2018 edition of the *Journal of Neuroscience* & in 2015 in the journal *Nature*, among other publications. The prospects for mitigating spinal cord injury through impeding PTPα still seems well-supported by the medical literature in our view & recent NGEN share price trajectory thus seems incongruous with the CONNECT SCI update just provided.

  - The firm did consummate a US$60M equity offering earlier in May/26 at discounted economics relative to trailing share price valuation, but pricing an equity offering in this way is not unusual & it does mitigate financial risk inherent in NVG-291 clinical testing for which Phase III activities are on the horizon. As we show in Exhibit 6, as originally described by Case Western Reserve University researchers in a 2018 review published in Physiological Reviews, the mechanism by which NVG-291 could impede scar formation through the PTPα pathway is well-established & thus provides a credible pharmacologic strategy for enabling neural regeneration in spinal cord injury – there are any number of ways in which

NVG-291 could underperform in future Phase III trials (magnitude/duration of motor/sensory recovery could be more modest than desired, compensatory mechanisms for scar formation could emerge over time, to name two), but scientific rationale is not one of them, in our view.

Exhibit 6. Proposed Interrelationship Between Glial Scars (Chondroitin Sulfate Proteoglycan) & Protein Tyrosine Phosphatase Sigma. The Interaction Can Be Impeded By NVG-291, Facilitating Nerve Regeneration In The Process

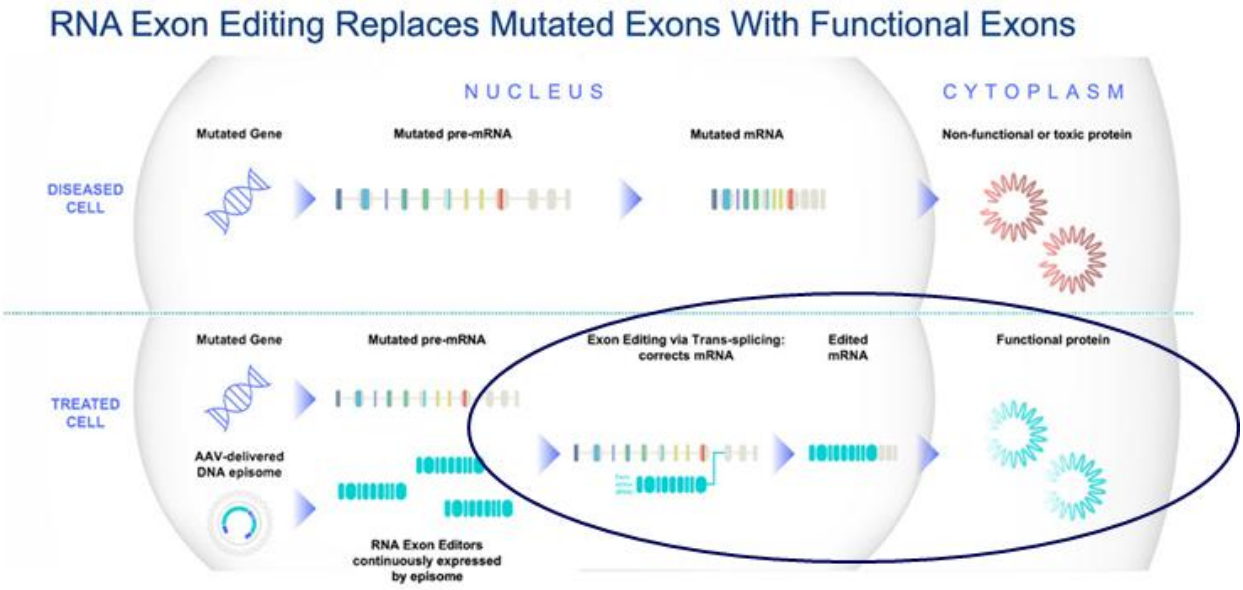


Source: Adapted from *Physiological Review (2018). Vol. 98, pp. 881-917; Experimental Neurol (2018). Vol. 309, pp. 148-159*

- **Eli Lilly consummates another blockbuster drug development alliance, this time in targeting kidney disease.** IN-based pharma giant Eli Lilly (LLY-NY, NR) announced a new relationship with private MA-based Ascidian Therapeutics in a deal that could be worth up to US$1.9B in upfront capital & downstream milestone payments to Ascidian, ostensibly for rights to co-develop Ascidian's RNA exon-editing platform for targeting genetic kidney disorders. Ascidian's technology creates biologics as DNA constructs (delivered either in an adenovirus vector or lipid nanoparticles) that are then converted inside target cells into mutation-free exon-only RNA constructs that are specific for the kidney-residing mutated protein that the technology seeks to transiently repair. Mechanistically, Ascidian's technology does not alter disease-associated mutated DNA sequences as a conventional gene therapy might but rather is able to snip out mutation-harboring exons in mRNA after it is formed, with the resulting exon-adjusted mRNA now able to translate functional rather than dysfunctional protein.

- The partners did not specify which genetic kidney diseases could be targeted under the alliance, but there are several plausible candidate indications, including polycystic kidney disease (fluid-filled cysts that grow in the kidneys & cause organ failure over time, caused by mutations in the PKD1 or PKD2 genes encoding either polycystin 1 or polycystin 2), Alport syndrome (an inherited pathology caused by mutations in genes encoding type IV collagen, leading to splitting of basement membranes in the kidney glomerulus) or perhaps nephrogenic diabetes insipidus (caused either by mutations in the arginine vasopressin receptor V2 [AVPR2] gene or the aquaporin 2 [AQP2] gene, the latter serving as a water channel protein that regulates water reabsorption & thus urine concentration).

Exhibit 7. Ascidian's Partnered RNA Exon-Editing Technology Mitigates Disease-Associated DNA Mutations At The Level Of The mRNA Constructs That Mutated DNA Sequences Encode, Not At The Level Of The Mutation Itself



Source: Adapted from *Molecular Therapy: Nucleic Acids (2024). Vol. 35, pp. 102331-102343*

- Other plausible genetic kidney diseases include VonLippel-Lindau disease (caused by mutations in the gene encoding VHL protein, a protein turnover-regulating E3 ubiquitin ligase that if mutated leads to unregulated formation of benign tumors in the kidney & elsewhere), Bartters syndrome (caused by mutations in the gene encoding a furosemide/Lasix-sensitive sodium-potassium cotransport protein found in the so-called Henley's loop where it facilitates water/salt recover from urine) & Fabry disease (caused by mutations in the gene encoding α-galactosidaseA, leading to buildup of sphingolipids in blood vessels in the kidneys & elsewhere; recombinant forms of the enzyme are approved as replacement therapies, including Takeda's [4502-JP, NR] agalsidase alfa/Replagal & Sanofi's [SNY-NY, NR] agalsidase beta/Fabrazyme but they do not reverse the underlying mutations causing disease), among others. The genetics of chronic kidney diseases is nicely described in a Aug/24 review published in the *New England Journal of Medicine*.
- Interestingly, Ascidian was already partnered with Swiss pharma giant Roche (ROG-SW, NR) in a US$1.84B alliance (US$42M upfront) focused on neurological disorders, as announced in Jun/24. So far, no formal clinical trials appear to have commenced under this alliance. Independently, Ascidian is advancing its nucleic acid-based exon-editing drug ACDN-01 in the 15-patient STELLAR trial & the 50-patient STARPATH trials targeting patients with retinopathy (Stargardt disease, a loss of central vision in children, similar symptomatically to age-related macular degeneration that manifests more commonly in elderly patients) caused by mutations in the ABCA4 transporter protein. One-year visual acuity data are expected from both trials during F2030. ABCA4 if found in photoreceptors in the retina, serving as a transporter for retinoid by-products that could be toxic to the eye, such as the derivative N-retinylidene-PE or all-trans-retinal aldehyde.

- **Eli Lilly continued its partnership momentum by acquiring rights to Hanmi's GLP-2 formulation.** Eli Lilly's second blockbuster alliance this week was with Korean firm Hanmi Pharmaceutical (128940-KRX, NR) in a US$1.2B deal that provides Eli Lilly with rights to Hanmi's novel glucagon-like peptide 2 (GLP-2) analog drug HM15912/sonefpeglutide, a long-acting form of

the GLP-2 protein that has long been FDA-approved in alternative forms (FDA-approved in Dec/12 by Takeda [4502-JP, NR] as teduglutide/Gattex, originally developed by legacy Canadian drug developer Allelix Biopharmaceuticals) for treating a GI disorder called short bowel syndrome. As an aside, Allelix eventually became NPS Pharmaceuticals through a FQ399 merger, with NPS thereafter being acquired by UK-based specialty pharmaceutical firm Shire plc in FQ115 & then Shire being acquired by Takeda in FQ119.

- Gattex itself is a slightly modified GLP-2, with an alanine residue replaced with a slightly smaller glycine residue at a key position in GLP-2's naturally-occurring amino acid sequence; in so doing, this reduces the activity of the enzyme that hydrolyzes/recycles GLP-2 in the body, an enzyme called dipeptidyl peptidase that itself is the target of selected blood glucose-modulating therapies like Merck's (MRK-NY, NR) sitagliptin/Januvia. GLP-2's pharmacology is known to be relevant to intestinal tissue regeneration, hence its initial approval in targeting pathologies related to this activity. Crohn's disease is a well-documented risk factor for developing short bowel syndrome over time. Takeda's F2025 GLP-2 sales, either as Gattex in the US or as Revestive in Europe, were essentially flat sequentially at ¥145.7B (US$903M), about ¥105.8B/US$707M of which were US sales. Accordingly, the GLP-2/short bowel syndrome medical market seems to be able to accommodate at least one other competitor, especially when considering that Gattex/Revestive lost its pediatric indication exclusivity last month (the reference patent in the US FDA Orange Book expired in mid-May/26).

- The deal provides Hanmi with US$75M in upfront cash, with the balance of deal economics predictably ascribed to achieving various clinical/regulatory/commercial milestones on sonefpeglutide development & cumulative sales. Sonefpeglutide's pharmacology is based on Hanmi's LAPSCOVERY platform that confers long-acting pharmacokinetics to peptide drugs, usually achieving once-monthly dosing with subcutaneous injection. An 18-patient Phase I/II sonefpeglutide/short bowel syndrome proof-of-concept trial is already underway, with six-month PK/safety data expected during F2028; a Phase I safety study & a Phase I/II kidney disease trial have both been completed already.

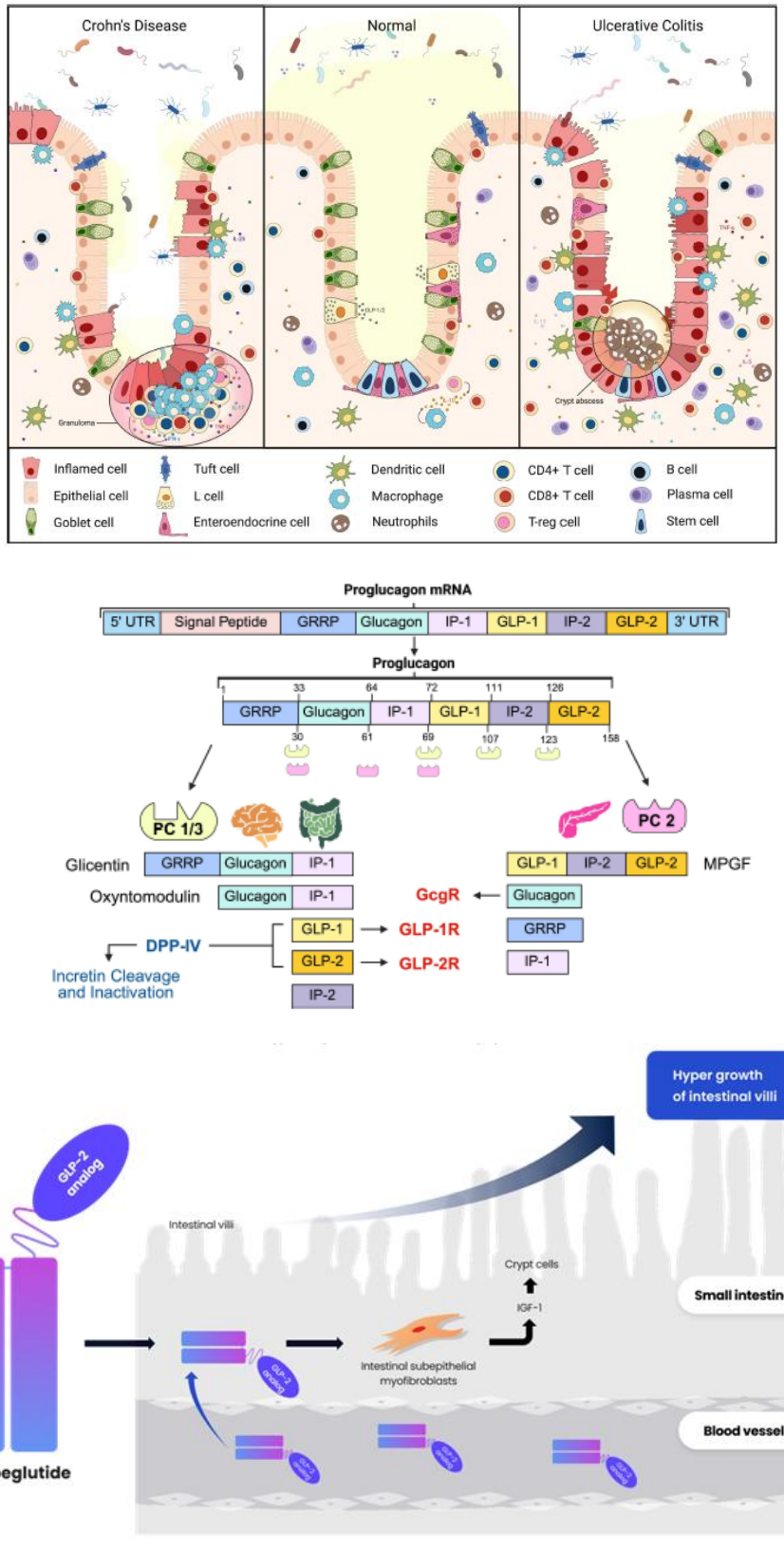
- Though sonefpeglutide has the word fragment 'peg' in its name, LAPSCOVERY is actually based on fusing non-glycosylated Fc regions of antibodies to the active protein-based drug using a non-peptide flexible chemical linker, in this case GLP-2. At least one other protein in Hanmi's pipeline, the glucagon/gastric inhibitory peptide (GIP)-GLP-1 triple agonist HM15211/efocipegtrutide, has been modified with this platform & is independently undergoing Phase II testing in a 215-patient MASH (metabolic dysfunction-associated steatohepatitis, for which one-year efficacy data is coincidentally expected later this year).

- It is interesting to us to observe that Eli Lilly clearly has expertise in how to convert GLP-1 into a long-acting formulation through its experience with tirzepatide (Zepbound or Mounjaro) for which GLP-1 is modified by linking gastric inhibitory peptide (GIP) to GLP-1 through a substituted lysine residue, to which the fatty acid 1,20-eicosanedioic acid is covalently linked, & then with a non-naturally-occurring acid (aminoisobutyric acid) substituted in two places within the GIP part of the molecule to confer added resistance to protease degradation. Though we have no direct evidence of this, we presume that Eli Lilly attempted a similar modification of GLP-2 before shifting its priorities to Hanmi's technology.

- As background, GLP-2 is a 33-amino acid peptide secreted from intestinal L-cells in similar proportions to GLP-1 production by the same type & indeed both peptide hormones are clipped from the same proglucagon precursor produced in that intestinal cell type (Exhibit 8). GLP-1 is quite clearly the more commercially relevant hormone with all of the FDA-approved formulations that target type II diabetes & weight loss, but GLP-2 has its own development history that is no less rigorously described in the medical literature. GLP-2 interacts with its own unique receptor appropriately named the GLP-2 receptor (GLP-2R) that is found in the gastrointestinal tract, the pancreas & throughout the central nervous system, so unsurprisingly those are the three organ systems where it exerts its pharmacologic action, as it does with short bowel syndrome.

- GLP-2R is not found in the cell type lining the inner intestinal wall (so-called enterocytes) but it is found in a category of cell types just underneath that cell layer (so-called myofibroblasts & enteroendocrine cells) & so its physiological action is more localized (paracrine signalling) than are other hormones that swim through the blood stream to more remote parts of the body (endocrine signalling). GLP-2 binding to its GLP-2R has been shown to stimulate localized release of at least two well-known growth factors – keratinocyte growth factor (KGF) & insulin-like growth factor-1 (IGF-1) – that themselves exert localized effects near the cell types that release GLP-2 itself, so mostly in the small intestine. Short bowel syndrome is a disease of the small intestine, arising from actual tissue loss or damage to the organ.

Exhibit 8. Cellular Pathways That Give Rise To GLP-2 & Its Receptor In The Body



Source: Adapted from *Frontiers In Gastroenterology (2026). Vol. 5, pp. 1790420-1790430 (upper two panels); Hanmi Pharmaceuticals (lower panel)*

Capital Markets Summary

Exhibit 9. EBITDA Or EPS-Positive Canadian Healthcare Stocks

Company	Filing Curr.	Sym.	Shrs Out. (M)	Share Price 4-Jun	Mkt Cap (M)	Mkt Cap (C$M)	Ent. Value (M)	Ent. Value (C$M)	EV/EBITDA (T12M)	EV/EBITDA FY1	EV/EBITDA FY2	Price/Earnings (T12M)	Price/Earnings FY1	Price/Earnings FY2
Profitable Canadian healthcare firms - specialty services [2,4]														
DRI Healthcare Trust	CAD	DHT.UN	55.0	$18.50	1,018	1,018	1,576	1,576	6.8x	6.8x	6.7x	NA	7.7x	7.4x
Jamieson Wellness	CAD	JWEL	41.5	$34.84	1,445	1,445	1,923	1,923	12.0x	10.8x	9.6x	19.7x	16.3x	14.0x
K-Bro Linen	CAD	KBL	13.0	$41.65	541	541	826	826	7.5x	7.6x	7.3x	26.3x	21.1x	17.1x
Medical Facilities [1]	CAD	DR	17.4	$12.59	219	305	338	470	5.6x	5.9x	5.9x	14.8x	6.2x	18.8x
Microbix Biosystems	CAD	MBX	137.8	$0.27	37	37	36	36	NA	NA	NA	NA	NA	NA
Savaria	CAD	SIS	71.9	$27.97	2,012	2,012	2,187	2,187	11.5x	10.7x	9.8x	25.3x	20.2x	18.0x
Profitable Canadian healthcare firms - specialty pharmaceuticals development/sales [2]														
Aurinia Pharma	USD	AUPH	128.6	$15.67	2,015	2,801	1,701	2,364	8.5x	NA	NA	6.9x	16.3x	12.7x
Bausch Health	USD	BHC	373.5	$5.01	1,871	2,600	30,936	42,989	6.2x	5.9x	6.1x	NA	1.2x	1.2x
BioSyent	CAD	RX	11.6	$13.83	161	161	155	155	12.0x	9.7x	8.5x	17.3x	14.7x	12.3x
Cipher Pharma [1]	CAD	CPH	25.4	$12.02	305	424	416	578	15.3x	13.7x	11.7x	9.9x	13.4x	11.5x
HLS Therapeutics [1]	CAD	HLS	31.3	$3.19	100	139	183	254	10.9x	9.1x	7.7x	NA	NA	NA
Knight Therapeutics	CAD	GUD	98.3	$8.20	806	806	733	733	8.4x	9.3x	8.9x	NA	44.6x	39.0x
Medexus Pharma [1]	CAD	MDP	32.0	$2.81	90	125	140	194	10.4x	8.8x	6.3x	NA	NA	10.2x
Profitable Canadian healthcare firms - eldercare services or infrastructure developers														
CareRx	CAD	CRRX	63.5	$3.24	206	206	269	269	8.7x	7.4x	6.5x	7.5x	27.0x	14.3x
Chartwell Retirement	CAD	CSH.UN	324.2	$20.34	6,594	6,594	9,296	9,296	21.5x	18.1x	16.3x	NA	NA	49.6x
Extendicare	CAD	EXE	94.8	$30.71	2,912	2,912	2,916	2,916	15.1x	11.7x	10.3x	22.3x	23.3x	21.0x
Vital Infrastructure	CAD	VITL.UN	250.0	$5.37	1,343	1,343	2,702	2,702	11.8x	13.0x	12.8x	NA	NA	NA
Nova Leap Health	CAD	NLH	87.3	$0.35	31	31	32	32	11.0x	NA	NA	NA	NA	NA
Sienna Senior Living	CAD	SIA	106.1	$20.94	2,222	2,222	3,476	3,476	22.4x	17.4x	15.3x	44.5x	34.9x	30.3x
Profitable Canadian healthcare firms - medical equipment distribution/sales [3]														
Covalon Technologies	CAD	COV	27.6	$1.97	54	54	40	40	42.8x	10.4x	8.1x	33.1x	21.9x	16.4x
Viemed Healthcare	USD	VMD	38.3	$9.48	363	363	505	702	6.8x	5.5x	4.8x	24.5x	20.8x	15.5x
Profitable Canadian healthcare firms - healthcare IT or digital IT services firms														
Healwell AI	CAD	AIDX	295.6	$0.90	266	266	332	332	NA	44.4x	20.1x	NA	NA	NA
Hydreight	CAD	NURS	53.7	$5.20	279	279	272	272	NA	11.9x	7.5x	NA	17.6x	10.4x
Kneat.com	CAD	KSI	96.1	$5.49	528	733	503	503	NA	30.1x	19.4x	NA	NA	NA
Vitalhub	CAD	VHI	63.3	$7.46	472	656	353	353	13.3x	10.2x	8.6x	NA	34.9x	24.6x
Well Health	CAD	WELL	255.4	$4.88	1,246	1,246	1,965	1,965	9.1x	10.8x	9.9x	46.3x	21.1x	25.5x
Average									**12.6x**	**12.6x**	**9.9x**	**23.0x**	**20.2x**	**18.5x**
Recently-acquired Canadian healthcare firms														
Andlauer	***CAD***	***AND***	***39.2***	***$54.97***	***2,152***	***2,152***	***2,165***	***2,165***	***13.4x***	***NA***	***NA***	***32.0x***	***NA***	***NA***
Dentalcorp Holdings	***CAD***	***DNTL***	***192.0***	***$11.00***	***2,112***	***2,112***	***3,112***	***3,112***	***10.9x***	***NA***	***NA***	***NA***	***NA***	***NA***
Quipt Home Medical	***USD***	***QIPT***	***44.5***	***$3.65***	***162***	***223***	***235***	***323***	***5.4x***	***NA***	***NA***	***2.1x***	***NA***	***NA***
Theratechnologies	***CAD***	***TH***	***46.0***	***$4.47***	***206***	***206***	***238***	***238***	***12.3x***	***NA***	***NA***	***NA***	***NA***	***NA***

[1] Share price converted to USD for stocks reporting financial data in USD but for which share value is reported in CAD; price refers to prior day close, EV calculations based on cash/LT debt reported in most recent quarter

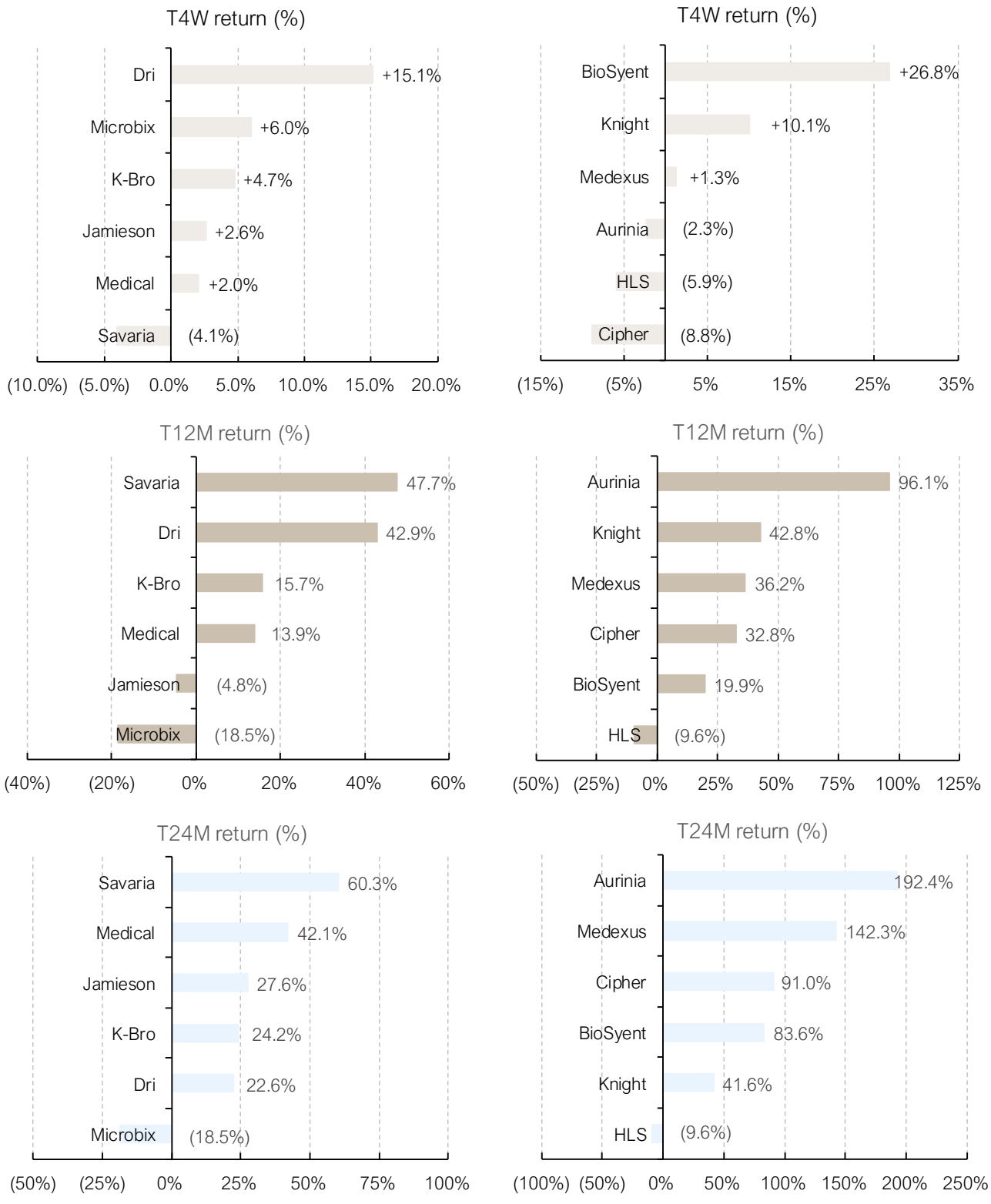
[2] Legacy specialty pharmaceutical firm & coverage stock Theratechnologies (TH-T, THTX-Q) was acquired in Sept/25 by CB Biotechnology/Future Pak for cumulative consideration of US$4.20/shr; Andlauer's acquisition by UPS (UPS-NY, NR) is closed as of Nov/25

[3] Quipt Home Medical was bid to be acquired by Kingswood Capital & Forager Capital for US$3.65/shr in Dec/25, transaction closed in Mar/26

[4] Dentalcorp Holdings was acquired by US private equity firm GRCR LLC in Sept/25 for an EV of C$3.3B (market value C$2.1B); transaction closed in Jan/26

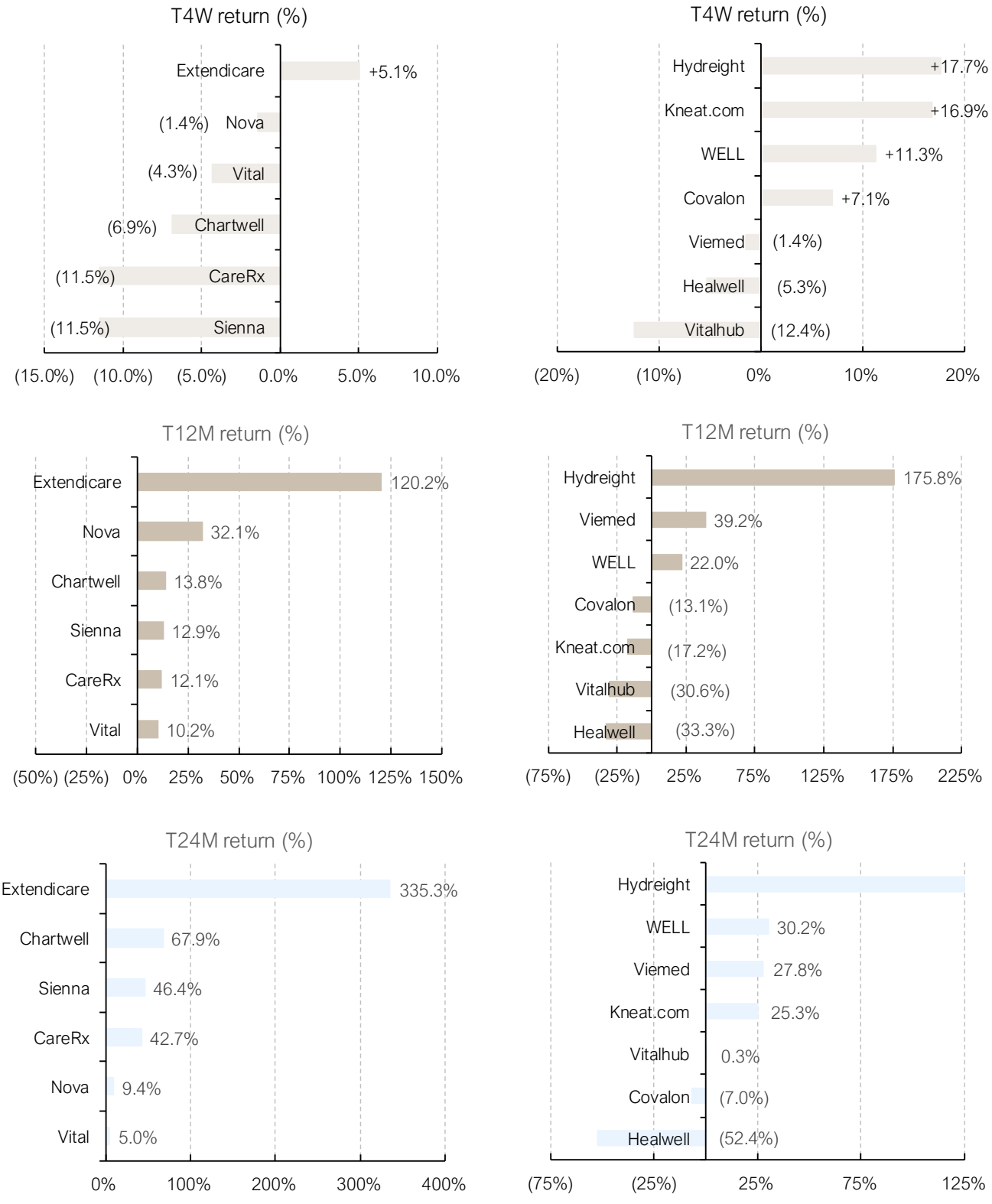
Source: Refinitiv, company reports, Leede Financial

Exhibit 10. Trailing Four-Week, One-Year & Two-Year Relative Share Price Performance For EBITDA/EPS-Positive Canadian Healthcare Equities – Specialty Services & Specialty Pharmaceutical Firms



Source: Refinitiv, company reports, Leede Financial

Exhibit 11. Trailing Four-Week, One-Year & Two-Year Relative Share Price Performance For EBITDA/EPS-Positive Canadian Healthcare Equities – Eldercare Services & Medical Technology Distribution/Healthcare IT Services



Source: Refinitiv, company reports, Leede Financial *(Hydreight [NURS-V, NR] T24M return 1,789%)*

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Speculative Buy	The security is considered a BUY but carries an above-average level of risk.
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RECOMMENDATION	NO. OF COMPANIES	%
Buy	9	60%
Speculative Buy	4	26%
Hold	1	7%
Sell	-	-
Tender	-	-
Under Review	1	7%

Historical Target Price

Appili Therapeutics | APLI-TSXV	None
Cardiol Therapeutics | CRDL-TSX, NASDAQ	None
CareRx | CRRX-TSX	None
Cipher Pharmaceuticals | CPH-TSX	None
Eupraxia Pharmaceuticals | EPRX-TSX, NASDAQ	None
Extendicare | EXE-TSX	None
K-Bro Linen | KBL-TSX	4
Medexus Pharmaceuticals | MDP-TSX	None
Medical Facilities | DR-TSX	None
Nanalysis Scientific | NSCI-TSXV	None
Oncolytics Biotech | ONCY-NASDAQ	None
Perimeter Medical Imaging | PINK-TSXV	None
Profound Medical | PRN-TSX, PROF-NASDAQ	None
ProMIS Neurosciences | PMN-NASDAQ	2
Satellos Biosciences | MSCL-TSX	2