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Biodexa Pharmaceuticals, PLC (BDRX-NASDAQ)

BDRX: Initiating Coverage of Biodexa Pharmaceuticals; A Deep Value Oncology Platform Built Around Two High-Conviction Areas

Based on our probability-adjusted DCF model that takes into account potential future revenues for MTX240 and MTX230, BDRX is valued at $7.00 per ADS. This model is highly dependent upon the continued clinical success of both compounds and will be adjusted accordingly based on future results.

Current Price (03/30/26)	$0.66
Valuation	**$7.00**

OUTLOOK

We are initiating coverage of Biodexa Pharmaceuticals PLC (BDRX) with a valuation of $7.00. Biodexa is a clinical-stage GI oncology company with two core assets: MTX240 and MTX230. MTX240 offers a novel approach to treating gastrointestinal stromal tumors (GIST) while MTX230 is in a Phase 3 study for the prevention of familial adenomatous polyposis (FAP). We anticipate the IND approval for MTX240 in the fourth quarter of 2026 and initiation of a Phase 1b/2a study later in the same quarter, with a dose escalation read out possible in the third quarter of 2027.

SUMMARY DATA

52-Week High	**$17.70**
52-Week Low	**$0.66**
One-Year Return (%)	**-94.88**
Beta	**1.02**
Average Daily Volume (sh)	**74,343**
Shares Outstanding (mil)	**3**
Market Capitalization ($mil)	**$2**
Short Interest Ratio (days)	**N/A**
Institutional Ownership (%)	**18**
Insider Ownership (%)	**0**
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	**N/A**
P/E using 2027 Estimate	**N/A**

Risk Level	**High**
Type of Stock	**Small-Value**
Industry	**N/A**

ZACKS ESTIMATES

Revenue
(in millions of £)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2024	0.0 A	0.0 A	0.0 A	0.0 A	0.0 A
2025	0.0 A	0.0 A	0.0 E	0.0 E	0.0 E
2026					0.0 E
2027					0.0 E

Earnings per Share (£)

	Q1 (Mar)	Q2 (Jun)	Q3 (Sep)	Q4 (Dec)	Year (Dec)
2024	-0.000 A	-0.001 A	-0.000 A	-0.001 A	-0.001 A
2025	-0.0001 A	-0.0001 A	-0.0001 E	-0.0001 E	-0.0001 E
2026					-0.0001 E
2027					-0.0001 E

WHAT'S NEW

Initiating Coverage

Source: Biodexa Pharmaceuticals, PLC

We are initiating coverage of Biodexa Pharmaceuticals, PLC (BDRX) with a valuation of $7.00. Biodexa is a clinical-stage GI oncology company focused on developing differentiated therapies for rare and difficult-to-treat cancers. The company's strategy centers on advancing scientifically validated mechanisms with the potential to address clear unmet medical needs, particularly in settings where existing therapies provide only transient benefit or have permanent life-altering impact on quality of life. Biodexa's pipeline is anchored by two core assets: MTX240, a novel molecular glue therapy targeting the PDE3A–SLFN12 axis with potential applications in gastrointestinal stromal tumors (GIST) and other malignancies, and MTX230 (eRAPA), a reformulated version of the mTOR inhibitor rapamycin that is currently in a registrational Phase 3 clinical trial for the treatment of familial adenomatous polyposis (FAP).

We believe Biodexa represents a compelling deep value opportunity, with its current valuation failing to fully reflect the scientific rationale and clinical potential of its lead programs. While MTX230 provides a late-stage, de-risked asset with a clear regulatory pathway, MTX240 introduces a first-in-class mechanism that could offer meaningful differentiation in the treatment of GIST. Together, these programs create a balanced pipeline with both near-term catalysts and longer-term value inflection points.

MTX240 Offers a Differentiated MOA for GIST
MTX240 is designed to exploit a novel tumor-selective cell death pathway involving PDE3A and SLFN12 that operates independently of traditional kinase signaling. This molecular glue mechanism enables selective induction of apoptosis only in cancer cells expressing both targets, offering a differentiated approach to treating tumors that have developed resistance to standard therapies. If validated clinically, MTX240 could establish a new therapeutic class for the treatment of GIST.

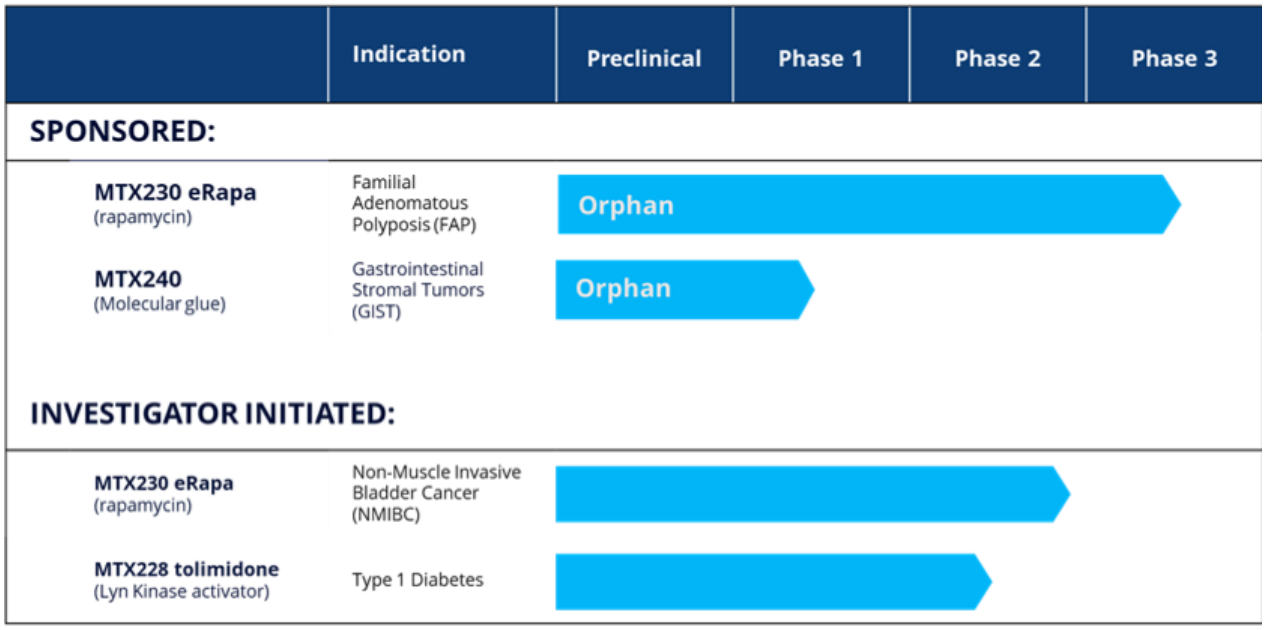
Significant Unmet Need in GIST Supports Opportunity for Novel Therapies
Despite multiple approved tyrosine kinase inhibitors, most patients with GIST ultimately develop resistance, resulting in limited durability of response across later lines of therapy. Current treatments primarily target KIT and PDGFRA mutations, but the heterogeneity of resistance mechanism creates an opportunity for therapies with alternative modes of action. MTX240's kinase-independent mechanism positions it as a potentially valuable option in this treatment landscape.

MTX230 (eRAPA) Provides Late-Stage Exposure to a High-Value Orphan Indication
MTX230 is being evaluated in a registrational Phase 3 trial for familial adenomatous polyposis, a rare genetic condition with a near-certain progression to colorectal cancer if left untreated. Current management relies heavily on prophylactic surgery, underscoring the need for effective pharmacologic interventions. By targeting mTOR signaling downstream of APC mutations, eRAPA has the potential to modify disease progression and delay and maybe even prevent the need for life-altering surgery.

INVESTMENT THESIS

Biodexa Pharmaceuticals, PLC (BDRX) is a clinical-stage biopharmaceutical company developing two core oncology assets, MTX240 and MTX230. MTX240 is a first-in-class molecular glue therapy that functions by complexing and stabilizing SLFN12 with PDE3A, with potential applicability in gastrointestinal stromal tumors (GIST) and other cancers. MTX230 (eRAPA) is a reformulation of the mTOR inhibitor rapamycin that is currently in a registrational Phase 3 clinical trial for the delay or prevention of surgery in patients suffering from familial adenomatous polyposis (FAP), a rare genetic cancer predisposition syndrome. Together, these programs provide a combination of early-stage mechanistic innovation and late-stage clinical validation, a profile that is relatively uncommon among micro-cap oncology companies.

We believe Biodexa represents a deep value oncology platform, where the current enterprise value appears to reflect limited credit for its pipeline assets, particularly MTX240. In our view, the company's investment case is underpinned by a) a novel and increasingly validated molecular glue mechanism with potential to address kinase-resistant cancer, and b) a late-stage orphan indication program targeting a well-defined genetic disease with high unmet needs. As MTX240 advances into clinical development and MTX230 progresses through Phase 3, we believe the company has multiple opportunities for value inflection.

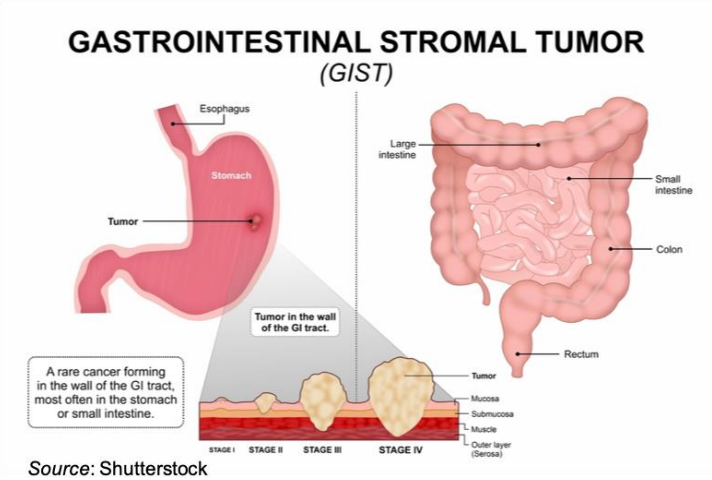


Source: Biodexa Pharmaceuticals, PLC

MTX240

Gastrointestinal Stromal Tumors

Gastrointestinal stromal tumors (GIST) are the most common mesenchymal tumors of the gastrointestinal tract, originating from the interstitial cells of Cajal (Corless *et al.*, 2011). GISTs occur all along the gastrointestinal tract but occur most commonly in the stomach (50-60%) and the small intestine (30-35%). Approximately 40% of GISTs that are localized at the time of detection will become malignant while 10-20% of GIST patients will present with overt metastases.



Source: Shutterstock

While relatively rare, age-adjusted incidence is seven cases per million in the U.S. and E.U. (Tran *et al.*, 2005), GISTs are the most common single type of sarcoma. They can arise at any age, however >80% are reported in individuals older than 50 years with a median of 63 years (Joensuu *et al.*, 2011).

Molecularly, GIST pathogenesis is driven predominantly by activating mutations in receptor tyrosine kinases. Approximately 75-80% of GIST harbor mutations in *KIT* (CD117), typically in the domain encoded by exon 11 (Hirota *et al.*, 1998). For the 20-25% of GISTs that do not have KIT mutations, approximately one-third have mutations in *PDGFRA* in domains homologous to those in *KIT* (Heinrich *et al.*, 2003). These mutations lead to constitutive activation of downstream signaling pathways, including RAS/RAF/MAPK, PI3K/AKT, and mTOR, promoting uncontrolled proliferation and survival.

A subset of GIST, often referred to as 'wild-type GIST', lack *KIT* or *PDGFRA* mutations and instead exhibit alternative oncogenic drivers such as succinate dehydrogenase (SDH) deficiency, BRAF mutations, or NF1-associated signaling abnormalities (Kinoshita *et al.*, 2004). The lack of SDH activity can lead to increased growth signaling through insulin-like growth factor receptor 1 (IGF1R) and vascular endothelial growth factor receptor 2 (VEGFR2) (Celestino *et al.*, 2013).

Clinically, localized GIST tumors can be managed with surgery, however advanced or metastatic GIST require systemic therapy. Despite advances in targeted therapy, long-term disease control remains challenging, with most patients developing resistance and progressive disease.

Limitations of the Current GIST Treatment Paradigm

The treatment of advanced GIST has been transformed by the development of targeted tyrosine kinase inhibitors (TKIs), beginning with imatinib, which demonstrated unprecedented efficacy in the disease (Blanke *et al.*, 2008). Imatinib achieves response rates of approximately 50-70% and median progress-free survival (PFS) of approximately 18-24 months in patients with *KIT*-mutant tumors. However, resistance inevitably develops, typically within 2-3 years of treatment initiation.

Mechanistically, resistance arises through secondary mutations in the KIT kinase domain, often affecting ATP-binding or activation loop regions, which reduces drug binding affinity. These mutations can occur heterogeneously within different tumor clones in the same patient, resulting in polyclonal resistance that is difficult to overcome with single-agent therapy (Antonescu *et al.*, 2005).

Subsequent lines of therapy (e.g., sunitinib, regorafenib, and ripretinib) have been developed to address specific resistance mutations, but their efficacy is progressively diminished. Median PFS declines to approximately 6-8 months with sunitinib (Demetri *et al.*, 2006), 4-5 months with regorafenib (Demetri *et al.*, 2013), and 6 months with ripretinib in later-line settings (Blay *et al.*, 2020). These agents provide incremental benefit but do not fundamentally alter the trajectory of the disease.

COMPOUND	KIT						PDGFR	PDGFR D842V	WILD TYPE		
	Primary		ATP-binding pocket		Activation Loop						
	exon 9	exon 11	exon 13	exon 14	exon 17	exon 18			SDH insufficient	BRAF V600E	NF1
APPROVED:											
Imatinib	✓	✓	✓				✓	✗	✗	✗	✗
Sunitinib	✓	✓	✓	✓			✓	✗	✗	✗	✗
Regorafenib					✓	✓	✓	✗	?	✓	?
Ripretinib	✓	✓	✓	✓	✓	✓	✓	✗	✗	✗	✗
Avapritinib					✓	✓	✓	✓	✗	✗	✗
DEVELOPMENT:											
Bezuclastinib					✓	✓	✗	✗	✗	✗	✗
IDRX-42	✓	✓	✓	✓	✓	✓	✗	✗	✗	✗	✗
MTX240	✓	✓	✓	✓	✓	✓	✓	✓	✗	✓	✓

Source: Biodexa Pharmaceuticals, PLC

A key limitation of this therapeutic paradigm is its reliance on continued inhibition of kinase signaling, which remains vulnerable to mutation-driven escape mechanisms. As tumors accumulate additional genetic alterations, they may activate compensatory pathways or develop structural changes that render kinase inhibitors ineffective. This has led to growing interest in therapeutic strategies that bypass kinase dependence altogether, targeting alternative vulnerabilities within cancer cells.

Expanding the Druggable Landscape with Molecular Glue Therapeutics

Molecular glue therapeutics represent a paradigm shift in drug discovery, enabling pharmacologic modulation of protein function through induced binding rather than direct enzymatic inhibition. Historically, small-molecule drug development has focused on proteins with well-defined binding pockets, such as enzymes or receptors. However, a large portion of the proteome, particularly scaffolding proteins and transcription factors, has been considered "undruggable" due to the absence of such features. Molecular glues overcome this limitation by stabilizing transient or non-existent protein–protein interactions, thereby reprogramming cellular pathways.

The clinical success of immunomodulatory drugs (IMiDs), such as lenalidomide, demonstrated that small molecules can induce selective degradation of target proteins by recruiting them to E3 ubiquitin ligases (Krönke *et al*., 2014). More recent work has expanded this concept to include molecular glues that do not necessarily induce degradation but instead trigger functional protein complexes that activate cytotoxic pathways.

This emerging class of therapeutics is particularly attractive in oncology, where it offers the potential to target previously inaccessible pathways, overcome resistance mechanisms, and achieve high levels of selectivity based on tumor-specific protein expression patterns.

The PDE3A-SLFN12 Tumor Suppression Axis

The interaction between phosphodiesterase 3A (PDE3A) and Schlafen 12 (SLFN12) represents one of the most compelling examples of a non-degradative molecular glue mechanism in cancer biology. PDE3A is an enzyme that regulates intracellular levels of cyclic nucleotides, including cAMP and cGMP, and plays important roles in cardiovascular physiology (Lugnier, 2006). SLFN12, a member of the Schlafen protein family, is implicated in regulation of cell growth and differentiation, though its precise biological functions have only recently been elucidated.

A 2016 study demonstrated that certain small molecules can bind PDE3A and induce the formation of a stable ternary complex with SLFN12, resulting in tumor-selective cytotoxicity (de Waal *et al*., 2016). Importantly, this effect was not mediated by inhibition of PDE3A enzymatic activity but rather by allosteric modulation that promotes protein-protein interactions. Subsequent structural and biochemical studies

revealed that these compounds effectively act as molecular bridges, creating a binding interface that stabilizes SLFN12 association with PDE3A (Li *et al*., 2019). This interaction leads to activation of SLFN12's RNase activity, resulting in inhibition of global protein translation and induction of apoptosis. A critical feature of this mechanism is its dependence on co-expression of PDE3A and SLFN12, which appears to define a subset of tumors that are highly sensitive to these agents. This creates a potential opportunity for biomarker-driven patient selection, enhancing the likelihood of clinical success.

Extensive preclinical studies have validated the PDE3A–SLFN12 axis as a therapeutic target (Chen *et al*., 2021). Chemical screening approaches identified compounds capable of inducing selective cytotoxicity in cancer cell lines expressing both proteins, with minimal effects on normal cells lacking this expression profile (Yan *et al*., 2022). Functional studies demonstrated that genetic knockdown of either PDE3A or SLFN12 abolishes drug-induced cytotoxicity, confirming that both proteins are required for activity. Conversely, forced expression of SLFN12 in resistant cell lines confers sensitivity, further supporting its role as a critical mediator of the response.

Importantly, molecular glue compounds targeting this pathway have demonstrated activity in multiple tumor types, including models of kinase inhibitor-resistant cancers. In the context of GIST, where resistance to TKIs is a major clinical challenge, this mechanism offers a compelling strategy to bypass canonical oncogenic signaling. Additionally, the correlation between target expression and drug sensitivity suggests that companion diagnostic strategies could be developed to identify patients most likely to benefit, potentially improving response rates and clinical outcomes.

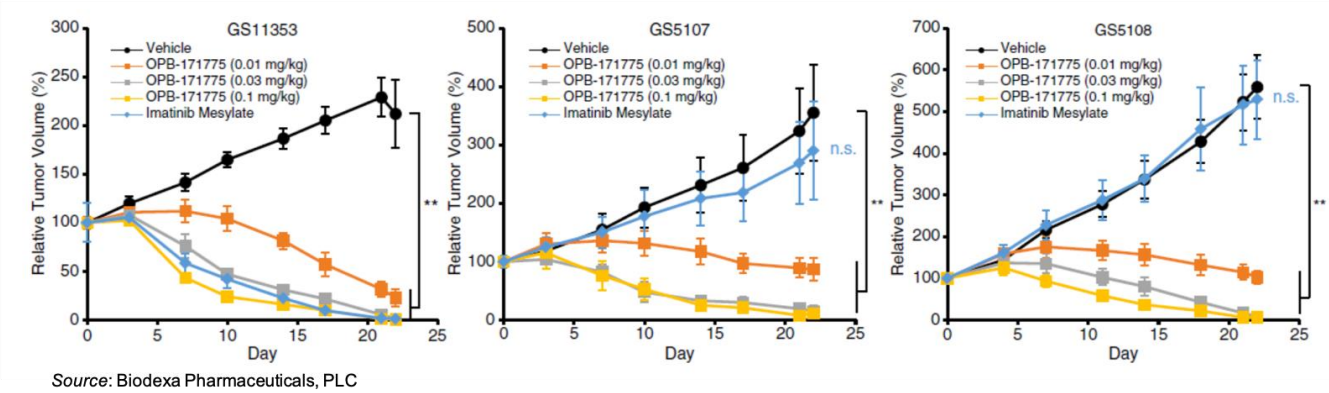
Preclinical Efficacy of MTX-240 in GIST Models

MTX-240 (formerly OPB-171775) was in-licensed from Otsuka by Biodexa in February 2026. The compound is supported by a robust preclinical dataset that not only validates its mechanism of action but also addresses resistance to TKIs and molecular heterogeneity, two key clinical limitations of GIST therapies. The translational strength of this dataset significantly de-risks early clinical development relative to typical Phase 1 oncology assets.

Activity Independent of *KIT* Mutation Status

A report by Takaki *et al*. evaluated OPB-171775 across a series of patient-derived xenograft (PDX) models of GIST, including both TKI-sensitive and TKI-resistant tumors harboring diverse KIT mutation profiles (Takaki *et al*., 2024). A key finding from the study was that antitumor activity was observed irrespective of KIT mutation status, including models containing secondary resistance mutations in KIT exons 13 and 17, which are mutations known to confer resistance to imatinib and other TKIs. Additional findings from the study included:

- While imatinib had limited efficacy in resistant PDX models, OPB-171775 (MTX240) produced consistent tumor regression across all tested models
- Efficacy was maintained across heterogeneous mutational backgrounds, suggesting independence from traditional oncogenic drivers
- Dose-dependent tumor regression was observed with once-daily oral administration in multiple mouse PDX models, which is shown in the figure below. GS11353 contained *KIT* mutations in exon 11, thus it was still inhibited by imatinib. In contrast, GS5107 and GS5108, which contain *KIT* mutations in both exon 13 and 17, and in both exon 11 and exon 17, respectively, were both resistant to imatinib while OPB-171775 (MTX240) still exhibited dose-dependent tumor regression.



Source: Biodexa Pharmaceuticals, PLC

These findings are particularly important in the context of advanced GIST, where polyclonal resistance mechanisms limit the durability of kinase inhibition strategies. MTX240's ability to bypass this strategy positions it as a potential therapy across multiple lines of treatment, including heavily pre-treated patients.

Potency and Selectivity with Nanomolar Activity

Across a large panel of cancer cell lines (~500+), OPB-171775 (MTX240) demonstrated highly potent antiproliferative activity, with IC50 values in the sub-nanomolar to low nanomolar range in sensitive models. Critically, sensitivity was not random but strongly associated with co-expression of PDE3A and SLFN12, establishing a clear biomarker-defined population:

- Cell lines expressing both PDE3A and SLFN12 showed marked sensitivity
- Knockdown of either gene abrogated drug activity
- Forced expression of PDE3A or SLFN12 conferred sensitivity in otherwise resistant models

Genome-wide CRISPR screening further validated this relationship, identifying PDE3A and SLFN12 as top determinants of drug response, reinforcing the mechanistic specificity of the compound. This level of genetically validated target dependency is important from a clinical development perspective as it could potentially allow for rational patient selection, development of companion diagnostics, and the potential for higher response rates in biomarker-enriched populations.

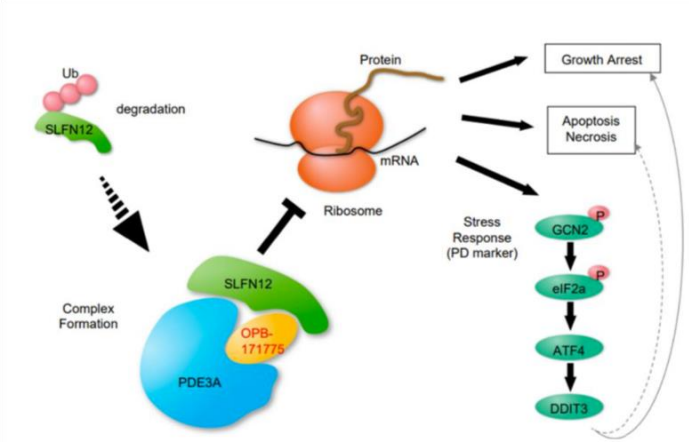
Mechanism of Action

The mechanism of action for OPB-171775 (MTX240) was extensively studied and appears to exert its anti-proliferative effects through the following:

Induced PDE3A-SLFN12 Complex Formation: The drug binds to PDE3A and induces stable complex formation with SLFN12, increasing SLFN12 protein stability without altering transcription.

Activation of SLFN12 RNase Activity: The interaction between PDE3A and SLFN12 activates SLFN12's RNase function, leading to degradation of tRNA species and inhibition of global protein synthesis.

Ribosomal Stress and GCN2 Pathway Activation: The inhibition of protein synthesis induces ribosomal stress, which activates the GCN2 (general control non-derepressible 2)



Source: Biodexa Pharmaceuticals, PLC

pathway. That pathway is a key sensor of amino acid deprivation and translational stress. Gene expression analyses showed enrichment of pathways related to ER stress and apoptotic signaling while pharmacologic inhibition of GCN2 attenuated OPB-171775's activity, thus confirming pathway dependence.

Induction of Apoptosis and Necrosis: The ultimate outcome is robust induction of both apoptotic and necrotic cell death, accompanied by cell cycle arrest. Importantly, this mechanism is independent of kinase signaling, thus reinforcing the use of OPB-171775 in TKI-resistant disease.

In Vivo Pharmacology

The *in vivo* studies performed with OPB-171775 (MTX240) showed a number of positive outcomes, including not just tumor suppression but complete regression of tumors in some GIST PDX models with daily oral dosing, activity across both imatinib-sensitive and imatinib-resistant tumors, and no significant body weight loss or overt toxicity observed at therapeutically active doses, indicating that the drug was well tolerated. This combination of potency, oral bioavailability, and tolerability supports advancement into clinical testing without the common issues associated with highly toxic agents.

Synergy with TKIs

Lastly, a very compelling result from the dataset was the demonstration of additive or synergistic activity of OPB-171775 when combined with imatinib. Using the GS11353 PDX model, treatment with OPB-171775 combined with imatinib showed total tumor regression in all six mice that were tested. These results suggest that MTX240 could be positioned not only as a later-line therapy but also as part of a combination regimen in earlier lines, potentially delaying or preventing resistance.

Clinical Plan for MTX240

The preclinical dataset fully supports advancing MTX240 into clinical testing. The company is planning to begin with a Phase 1b/2a dose escalation study in patients with advanced malignant solid tumors to establish safety, tolerability, and pharmacokinetics/pharmacodynamics (PK/PD) for dose optimization. Biodexa will be starting with once-weekly dosing at 0.2, 0.25, 0.3, and 0.35 mg. The dose expansion cohort will focus on GIST patients with demonstrated resistance to imatinib and sunitinib that express both SLFN12 and PDE3A, with outcomes centered around tolerability and efficacy (e.g., RECIST/tumor shrinkage, PFS, OS). We anticipate IND clearance for the study in the fourth quarter of 2026, dosing of the first patient in the same quarter, and topline results from the dose escalation portion of the study in the third quarter of 2027.

Market Opportunity

The treatment of GIST represents a rare but well-defined oncology market characterized by high unmet need in later lines of therapy and a treatment strategy that is dominated by sequential use of TKIs. There are approximately 4,000-6,000 new cases per year in the U.S. and approximately 10,000-12,000 across the E.U. (ACS; Medscape). Due to improved survival associated with targeted therapies, prevalence is meaningfully higher than incidence, with approximately 12-15 cases per 100,000 population in developed markets (Soreide *et al*., 2016).

The commercial opportunity in GIST is not defined solely by incidence but rather by treatment duration and line progression, as patients often cycle through multiple therapies over several years. Approximately 10-30% of patients present with unresectable or metastatic disease at diagnosis, and a significant portion of initially resected patients eventually relapse and require systemic treatment.

The current treatment paradigm is centered around sequential lines of kinase inhibition, beginning with imatinib and progressing through second-, third-, and fourth-line TKIs. While first-line therapy captures

the largest population, later lines represent a particularly attractive commercial segment due to: 1) high unmet need; 2) limited competition; and 3) premium oncology pricing.

The GIST market is heavily concentrated among TKIs targeting KIT and PDGFRA mutations. However, the clinical limitations of this approach, particularly the emergence of polyclonal resistance mechanisms, have created a structurally attractive opportunity for therapies with non-kinase-based mechanisms of action.

MTX240 is uniquely positioned to address the post-TKI treatment landscape, where therapeutic options are limited and outcomes are poor. Given its mutation-agnostic mechanism, the drug has the potential to capture patients across multiple lines of therapy, including those who have exhausted all approved kinase inhibitors. In addition, the demonstrated synergy with imatinib in preclinical models suggests potential expansion into earlier lines, which would significantly increase the addressable market.

MTX230

MTX230 (eRAPA) is a re-formulated version of Rapamune® (rapamycin) and represents a late-stage, mechanistically validated approach to chemoprevention in a genetically defined cancer predisposition syndrome. The reformulated version of rapamycin offers improved pharmacokinetics and bioavailability and targets dysregulated cellular proliferation downstream of APC loss, which is a foundational driver of disease pathogenesis. Unlike MTX240, which is positioned as a first-in-class oncology therapeutic, MTX230 leverages decades of biological insight into mTOR signaling, intestinal tumorigenesis, and rapamycin pharmacology, but applies this knowledge through a differentiated formulation and dosing strategy designed specifically for chronic use in FAP patients.

Familial Adenomatous Polyposis

Familial adenomatous polyposis (FAP) is a autosomal dominant cancer predisposition syndrome caused by germline mutations in the adenomatous polyposis coli (APC) tumor suppressor gene (Half *et al.*, 2009). Loss of APC function leads to constitutive activation of the Wnt/β-catenin signaling pathway, resulting in uncontrolled proliferation of intestinal epithelial cells and the formation of hundreds to thousands of adenomatous polyps throughout the gastrointestinal tract. Over time, these polyps accumulate additional genetic alterations, ultimately progressing to colorectal carcinoma. Historically, the lifetime risk of colorectal cancer in untreated FAP patients approaches 100%, often by the fourth decade of life (Kinzler *et al.*, 1996).

Current management of FAP is largely surgical, with most patients undergoing prophylactic colectomy or proctocolectomy in early adulthood to mitigate cancer risk. While effective in reducing colorectal cancer incidence, these procedures are associated with significant long-term morbidity, including altered bowel function, nutritional complications, and reduced quality of life. In addition, surgery does not eliminate the risk of neoplasia in the duodenum, stomach, or residual rectal tissue, necessitating lifelong surveillance and repeated interventions (Peterson *et al.*, 1991). Despite the severity and well-characterized biology of the disease, no pharmacologic therapies are currently approved that meaningfully alter its natural history, underscoring a significant unmet medical need.

mTOR signaling Drives FAP Pathogenesis

The mechanistic target of rapamycin (mTOR) is a master regulatory of cell growth, metabolism, and protein synthesis, integrating signals from nutrients, growth factors, and cellular energy status. In the context of *APC* loss, aberrant Wnt signaling leads to activation of downstream pathways that converge on mTOR complex 1 (mTORC1), promoting expansion of intestinal stem cells and accelerating adenoma formation (Laplante *et al.*, 2012; Saxton *et al.*, 2017).

Importantly, mTOR activation has been observed in both preclinical models and human adenomatous tissue, suggesting that it represents a critical downstream effector of tumorigenesis in FAP.

Pharmacologic inhibition of mTOR, therefore, offers a strategy to suppress the proliferative drive induced by APC mutations without directly targeting the Wnt pathway, which has historically proven difficult to modulate therapeutically. This concept of targeting a convergent downstream node is particularly attractive in genetically defined diseases such as FAP, where upstream mutations are ubiquitous but downstream signaling pathways are more tractable.

Preclinical Validation of Rapamycin in APC-Driven Tumorigenesis

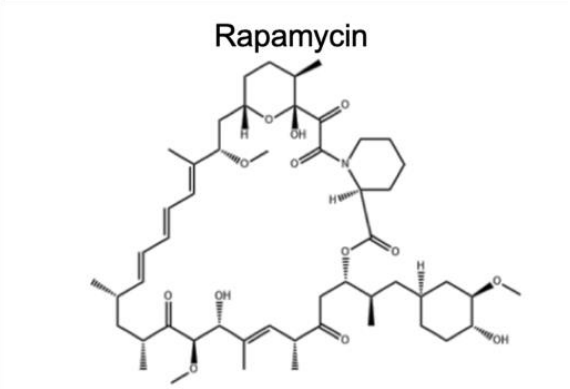
The preclinical foundation for MTX230 derives from its use in genetically engineered mouse models. The $APC^{Min/+}$ mouse model recapitulates key features of human FAP, including spontaneous intestinal adenoma formation (McCart *et al.*, 2008). Multiple preclinical studies have shown that treating $APC^{Min/+}$ mice with rapamycin has a positive effect on lifespan while decreasing intestinal neoplasia formation:

- A 2009 study showed that treating $APC^{Min/+}$ mice with rapamycin had a dramatic effect on long-term survival. Untreated mice lost weight, experienced intestinal bleeding, and succumbed to multiple neoplasia by 22.3±1.4 weeks, while mice treated with rapamycin maintained stable weight and survived long-term (39.6±3.4 weeks), with more than 30% surviving >1 year. This was accompanied by a reduction in abnormalities in colonic electrolyte transport that is typical in $APC^{Min/+}$ mice (Koehl *et al.*, 2010).

- A 2014 study examined the effect of enterically targeted rapamycin (eRAPA; the same formulation used in MTX230) on $APC^{Min/+}$ mice. The results showed that eRAPA improved survival of $APC^{Min/+}$ mice in a dose-dependent manner and that most $APC^{Min/+}$ mice fed 42 parts per million eRAPA lived beyond the median life span reported for wild-type syngeneic mice. Chronic eRAPA exposure did not cause detrimental effects in mouse models of cancer, infection, or autoimmunity, suggesting that chronic rapamycin exposure does not suppress normal immunity (Hasty *et al.*, 2014).

Differentiated Formulation of Rapamycin in eRAPA

MTX230 (eRAPA) builds upon the clinical success of rapamycin by addressing key limitations associated with traditional rapamycin formulations. Although rapamycin is a well-characterized mTOR inhibitor with established clinical use as an immunosuppressant, its application in oncology and chemoprevention has been constrained by poor and variable bioavailability, systemic toxicity, and challenges associated with chronic dosing. eRAPA is designed as an encapsulated, enteric-coated formulation that enhances delivery to the gastrointestinal tract while reducing systemic exposure. This approach is particularly well suited for FAP, where the disease is localized to the intestinal epithelium and long-term treatment is required. The drug was in-licensed by Biodexa from Emtora Biosciences with no development milestones, double-digit approval milestones, and low double-digit royalties. It has patent protection through 2035. It has received Orphan Drug Designation in the E.U., which provides 10 years of market exclusivity, and Fast Track and Orphan Designations from the U.S. FDA.



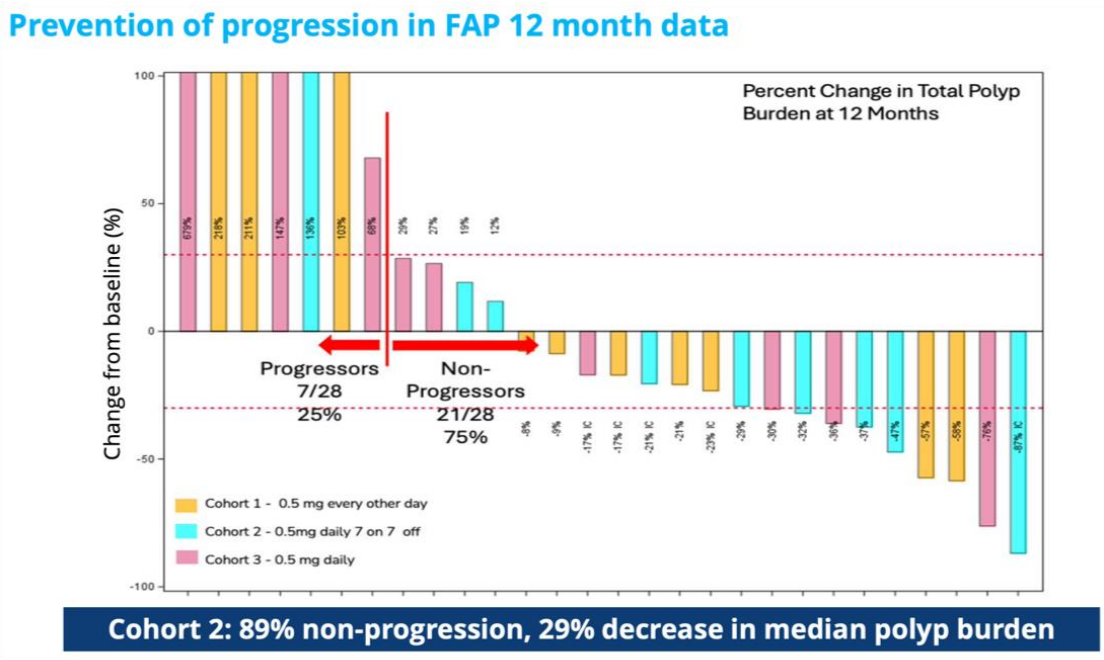
Source: Biodexa Pharmaceuticals, PLC

By improving pharmacokinetic consistency and potentially limiting off-target effects, eRAPA enables a chronic dosing strategy that would be difficult to achieve with conventional rapyamycin. This is a critical consideration in FAP, where the goal is not short-term tumor regression but long-term suppression of polyp formation and progression, potentially over many years. The ability to safely administer an mTOR inhibitor in this context represents a meaningful advancement in the therapeutic strategy for this disease.

Phase 2 Study of eRAPA

Biodexa previously announced positive results from a Phase 2 clinical trial of MTX230 (NCT04230499). The open-label study was conducted at seven U.S. sites in 30 adult patients with a median age of 43 years with intact colon (n=6) or post-colectomy and ileo-rectal anastomosis and at least 10 adenomas in the rectal remnant (n=24). The patients were enrolled into one of three dosing cohorts of 10 patients each for 12 months: 0.5 mg every other day (Cohort 1); 0.5 mg daily every other week (Cohort 2); or 0.5 mg daily (Cohort 3). The primary endpoints of the study were safety and tolerability and percent change from baseline in polyp burden at six months, as measured by the aggregate of all polyp diameters, however patients continued to receive treatment for 12 months (secondary endpoint).

The results of the trial showed that 21/28 (75%) of patients were deemed to be non-progressors at 12 months with a median reduction in polyp burden of 17%. In Cohort 2, 8/9 (89%) of patients were deemed to be non-progressors at 12 months with a median reduction in polyp burden of 29%. In regards to safety, there were four related Grade 3 or higher and one related Serious Adverse Event reported during the trial along with a 95% compliance rate at 12 months. One patient was removed from the trial due to non-compliance. A summary of the polyp burden for the patients is given in the figure below. While the magnitude of polyp reduction was modest relative to traditional oncology therapies, we believe it is important to remember the goals of FAP treatment, which is slowing disease progression and delaying the need for surgical intervention, two goals which we believe could be achieved based on this data.



Source: Biodexa Pharmaceuticals, PLC

Phase 3 SERENTA Trial

In June 2025, Biodexa initiated the Phase 3 SERENTA trial to build upon the findings from the Phase 2 study. It is a double blind, placebo controlled trial that will enroll 168 high-risk patients with germline or phenotypic FAP diagnosis (NCT06950385). Patients are randomized 2:1 drug/placebo to evaluate the safety and efficacy of MT240. The primary outcome of the trial is PFS as determined by a composite clinical progression measure between the following four conditions: Surgery/Meets Criteria for Surgery, Advancement of Spigelman Stage, Diagnosis of high-grade dysplasia or cancer, or death by any cause. The endpoints were agreed to by the U.S. FDA in a Type C meeting. The first patient was enrolled in August 2025 and there will be an interim analysis after 25 PFS events (projected to be in 1Q28) and the database will be locked after 75 PFS events (projected to be in 4Q29). The trial is being partly funded by a $20 million grant from the Cancer Prevention and Research Institute of Texas (CPRIT).

Market Opportunity

FAP is a classic orphan disease market, characterized by a well-defined genetic etiology, a small but identifiable patient population, and a near-certain progression to malignancy in the absence of treatment. The prevalence of FAP is estimated at approximately 1 in 10,000 individuals in the U.S. (approximately 30,000-35,000 patients) and approximately 1 in 20,000 individuals in the E.U. (approximately 35,000-40,000 patients).

Despite its rarity, FAP represents a uniquely attractive commercial opportunity due to several structural features of the market. First, patients are typically diagnosed at a young age and require lifelong disease management, resulting in a large prevalent population relative to annual incidence. Second, the disease course is highly predictable, with nearly all patients developing colorectal cancer if untreated, creating a strong rationale for early and sustained therapeutic intervention.

There are currently no approved pharmacologic therapies that alter the natural history of FAP, despite decades of investigation into agents such as NSAIDs and COX-2 inhibitors. While these therapies have demonstrated modest reductions in polyp burden, their effects have been insufficient to replace surgery or meaningfully delay disease progression.

The absence of an effective drug therapy creates a first-mover advantage for MTX230. As a potential first-in-class agent targeting a validated biological pathway, eRAPA could establish a new standard of care in FAP, particularly if it demonstrates the ability to delay surgical intervention or reduce polyp progression.

Even under conservative assumptions that include moderate penetration and pricing consistent with orphan oncology/preventative therapies, the FAP market supports a peak revenue opportunity in the several hundred million to low billion-dollar range. Importantly, this estimate does not take into account broader cancer prevention strategies, for example the non-muscle invasive bladder cancer (NMIBC) or prostate cancer indications that the drug is being evaluated in through small clinical trials.

Non-Orphan Indications

MTX230 is also being analyzed in clinical trials for non-orphan indications:

- An open label, single center investigator-initiated Phase 1 study was conducted with 14 low risk prostate cancer patients who had Gleason scores of ≤ 7. The patients were treated for three months and observed for an additional three months. The primary endpoints were safety and Phase 2 dose selection with secondary endpoints examining immunological response, quality of life and disease progression. The topline data showed that the drug was safe and well tolerated, had consistent predictable pharmacokinetics, and there was no disease progression seen during the study.

- A Phase 2 trial in non-muscle invasive bladder cancer (NMIBC) is currently being conducted at two centers and is being supported by a $2.8 million grant from the National Cancer Institute. The trial enrolled 166 NMIBC patients that had a diagnosis within the last 90 days and no prior BCG treatment. Patients are being treated for 12 months or until relapse, which ever occurs first. The primary endpoints are safety and tolerability along with relapse-free survival after 12 months. Secondary endpoints include immunological response, quality of life, and cognitive outcomes. Topline data is expected in mid-2026.

MTX228

MTX228 (tolimidone, previously known as MLR-1023) represents a mechanistically distinct approach to the treatment of type 1 diabetes (T1D), focused on preservation and regeneration of pancreatic β-cell

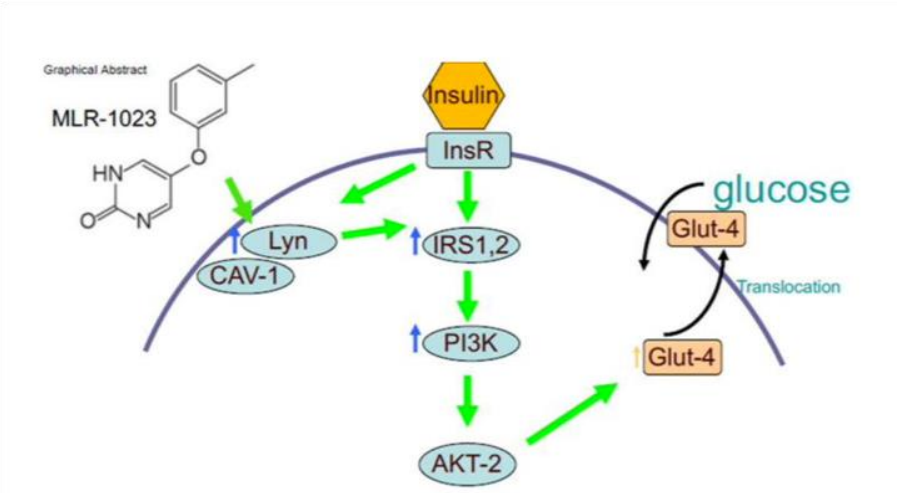
function rather than replacement of insulin. While not a core value driver in our model, the program introduces an additional layer of optionality for Biodexa, particularly given the large commercial market and differentiated biological rationale.

Mechanism of Action

MTX228 is a small-molecule activator of Lyn kinase, a member of the Src family of tyrosine kinases that plays a role in intracellular signaling downstream of insulin receptors and other growth pathways. Initially investigated as a gastric ulcer therapy, the compound was later repurposed following the discovery that Lyn kinase is involved in insulin signaling and glucose homeostasis (Lee *et al*., 2020).

Preclinical and biochemical studies indicate that Lyn activation can enhance insulin signaling efficiency, leading to improved glucose utilization in peripheral tissues. In this context, MTX228 has been shown to act independently of classical insulin sensitization pathways such as PPARγ, suggesting a differentiated mechanism relative to existing antidiabetic agents (Saporito *et al*., 2012).

In pancreatic β-cells, insulin receptor signaling and downstream pathways such as PI3K/AKT are known to influence cell survival, proliferation, and functional insulin secretion (Kulkarni *et al*., 2004). While Lyn is not the sole regulator of these processes, its role within this signaling network provides a biologically plausible rationale for investigating whether pharmacologic activation could contribute to preservation of β-cell function under stress conditions.



Source: Biodexa Pharmaceuticals, PLC

Clinical Data for MTX228

MTX228 has been evaluated in multiple clinical studies, first as a potential treatment for gastric ulcers (Lipinski *et al*., 2020) and later as a treatment for type 2 diabetes (T2D) (Lee *et al*., 2020). These studies provided a meaningful base of human safety and pharmacodynamic data, showing that the drug has excellent safety and bioavailability.

In the T2D studies, MTX228 demonstrated:

- Statistically significant improvements in postprandial glucose levels
- Evidence of insulin sensitization independent of PPARγ activation
- A generally favorable safety and tolerability profile across dosing regimens

Notably, dose-response analyses suggested that lower doses (e.g., 100 mg once or twice daily) may provide optimal metabolic effects, however this finding will need to be verified in the current study.

Phase 2a Study in T1D

MTX228 is currently being evaluated in a Phase 2a clinical trial in adults with T1D who retain measurable endogenous insulin production (NCT06474598). The study is designed to assess whether pharmacologic activation of Lyn can influence β-cell function in early or residual-stage T1D, where intervention may be most feasible. Multiple dosing regimens will be tested as it is unclear what the optimal dose for T1D patients will be. The primary endpoints of the study are change in C-peptide (a biomarker of endogenous insulin production) AUC during mixed-meal tolerance testing (MMTT) and dose selection for a Phase 2b trial. Once a dose is selected, the company is planning to initiate a Phase 2b trial with the same primary outcome following treatment of up to six months.

Financials and Capital Structure

In September 2025, Biodexa announced interim results for the six month period ending June 30, 2025. As expected, the company did not report any revenues for the first half of 2025 or 2024. R&D expenses in the first half of 2025 were £1.67 million compared to £2.19 million in the first half of 2024. The decrease was primarily due to a decrease in spending on the MAGIC-G1 rGBM study partially offset by an increase in spending on MTX230. Administrative costs in the first half of 2025 were £2.38 million compared to £2.08 million in the first half of 2024. The increase was primarily due to an increase in the foreign exchange charge partially offset by a reduction in transaction related costs.

Total cash burn for the first half of 2025 was £3.30 million and the company exited the first half of 2025 with approximately £4.04 million. In December 2025, the company announced a $10 million public offering of i) 157,000 American Depository Share (ADS) units, with each unit consisting of one ADS, two series L warrants, each to purchase one ADS, and ii) 2,891,781 pre-funded units, with each pre-funded unit consisting of one pre-funded warrant to purchase one ADS and two series L warrants. The combined public offering price of each ADS unit was $3.28. The Series L warrants will expire on the five-year anniversary of the date of issuance and were immediately exercisable at an exercise price of $3.28 per ADS.

As of March 23, 2026, Biodexa had 3,241,568 ADS outstanding (each ADS representing 100,000 of the company's ordinary shares) along with 489,000 pre-funded warrants, 6,097,562 Series L warrants, 182,525 other warrants, and 3,130 stock options for a fully diluted ADS count of 10,013,785. On March 18, 2025 the company announced a ratio change on its American Depository Receipts (ADR) from 1:100,000 to 1:500,000, which will become effective on Apr. 6, 2026.

Risks to Consider

In addition to the risk factors listed below, investors are encouraged to read the company's 20-F filing that discusses additional risk factors.

Development Risk: MTX240 is based on a novel molecular glue mechanism that, while compelling in preclinical models, introduces meaningful translational uncertainty. The relationship between PDE3A/SLFN12 expression and clinical response has not yet been established in patients, and there is a risk that biomarker-defined sensitivity observed *in vitro* may not translate into meaningful clinical efficacy. In addition, molecular glue therapeutics represent a relatively new class of drugs, and there is limited precedent for regulatory approval in solid tumors using this mechanism outside of specific hematologic contexts.

Clinical Risk: Biodexa's valuation is highly dependent on the successful development of its two lead assets, MTX240 and MTX230. As a result, negative clinical outcomes in either program could have a disproportionate impact on the company's valuation. MTX240, in particular, remains in early-stage development, where historical probabilities of success are low despite strong preclinical rationale. While the PDE3A–SLFN12 mechanism is well-supported biologically, it has not yet been clinically validated, and there is a risk that early trials may fail to demonstrate sufficient efficacy or tolerability in humans.

Similarly, although MTX230 benefits from a more advanced stage of development and a well-characterized mechanism, Phase 3 studies in rare diseases remain subject to variability in endpoints, patient heterogeneity, and trial execution.

Commercial Risk: Even if approved, MTX230 may face challenges in clinical adoption, particularly given its positioning as a chronic therapy in a preventative setting. Physicians may be cautious in adopting long-term pharmacologic treatment in a population historically managed with surgery and surveillance, especially if the magnitude of benefit is perceived as incremental. In addition, payer dynamics in orphan diseases, while generally favorable, may still require demonstration of clear cost-effectiveness relative to existing standards of care. For MTX240, commercial uptake will depend on its ability to demonstrate clear differentiation from existing TKIs, particularly in later-line settings where multiple therapies are already available.

Financial Risk: As a clinical-stage biotechnology company, Biodexa will require additional capital to fund ongoing and future clinical trials, including the Phase 3 program for MTX230 and early-stage development of MTX240. Given the company's current size and stage, future financing will likely involve equity issuance, resulting in shareholder dilution. Market conditions, clinical data, and broader biotech sentiment may impact the company's ability to raise capital on favorable terms. Delays in clinical development or unfavorable data could further exacerbate financing risk.

MANAGEMENT PROFILES

Stephen Stamp – Chief Executive Officer
Mr. Stamp is an experienced public company CFO and has held senior positions in a number of significant healthcare companies including as Group Finance Director of Shire plc, Chief Operating Officer of Xanodyne Pharmaceuticals Inc and most recently with AIM quoted Ergomed plc, where he served initially as CFO before becoming CEO. Mr Stamp qualified as a Chartered Accountant at KPMG and is a member of the Institute of Chartered Accountants.

Fiona Sharp – Chief Financial Officer
Ms. Sharp is a qualified accountant and has held senior finance positions within the PR and advertising industry, including Group Finance Director of Chime Communications Group. Fiona is a Fellow of the Chartered Association of Certified Accountants.

Gary Shangold – Chief Medical Officer
Dr. Shangold is a board-certified Obstetrician/Gynecologist and Reproductive Endocrinologist who is newly-appointed as Chief Medical Officer for Biodexa Pharmaceuticals PLC. His career in the pharmaceutical industry spans more than 30 years, including senior positions in Clinical Research, Product Development, Regulatory Affairs, and general management. He has played key roles in the development and/or registration of more than ten approved products, including having led the team at Johnson & Johnson which developed the world's first transdermal contraceptive patch. Before joining the pharma industry, Dr. Shangold spent a decade in academia, on the faculty of The University of Chicago Pritzker School of Medicine, and then later at the Massachusetts General Hospital and the Harvard School of Medicine. He has previously served as President of the American Academy of Pharmaceutical Physicians, and as Chair of the Association of Clinical Research Professionals, a 14,000-member nonprofit dedicated to excellence and professionalism in clinical research globally.

Steve Ellul – Chief Business Officer
A chemist by training, Mr. Ellul has spent more than 30 years in commercially focused roles across multiple sectors of the pharmaceutical industry. He has led commercial teams at drug delivery organizations, including Eurand and Bespak, and has held senior roles at specialty pharma companies, Elan, Shire and Theravance Biopharma.

VALUATION

We are initiating coverage of Biodexa Pharmaceuticals, PLC (BDRX) with a valuation of $7.00. Biodexa is a clinical-stage GI oncology company focused on developing differentiated therapies for rare and difficult-to-treat cancers though its two lead assets, MTX240 and MTX230. MTX240 is a first-in-class molecular glue therapy that functions by complexing and stabilizing SLFN12 with PDE3A, with potential applicability in gastrointestinal stromal tumors (GIST) and other cancers. MTX230 (eRAPA) is a reformulation of the mTOR inhibitor rapamycin that is currently in a registrational Phase 3 clinical trial for the delay or prevention of surgery in patients suffering from familial adenomatous polyposis (FAP), a rare genetic cancer predisposition syndrome.

MTX240

MTX240 belongs to a class of compounds know as molecular glue therapeutics, which represent a paradigm shift in drug discovery and potentially enable pharmacologic modulation of protein function through induced binding rather than direct enzymatic inhibition. It is being developed as a therapy for GIST, a relatively rare mesenchymal tumor type of the gastrointestinal tract that is driven predominantly by activating mutations in receptor tyrosine kinases. The drug was in-licensed from Otsuka by Biodexa in February 2026. It is supported by a robust preclinical dataset that not only validates its mechanism of action but also addresses resistance to tyrosine kinase inhibitors (TKIs) and molecular heterogeneity, two key clinical limitations of GIST therapies. The company is planning to begin with a Phase 1b/2a dose escalation study in patients with advanced malignant solid tumors to establish safety, tolerability, and pharmacokinetics/pharmacodynamics (PK/PD) for dose optimization. We anticipate IND clearance for the study in the fourth quarter of 2026, dosing of the first patient in the same quarter, and topline results from the dose escalation portion of the study in the third quarter of 2027.

MTX230

MTX230 (eRAPA) is a re-formulated version of Rapamune® (rapamycin) and represents a late-stage, mechanistically validated approach to chemoprevention in a genetically defined cancer predisposition syndrome. The reformulated version of rapamycin offers improved pharmacokinetics and bioavailability and targets dysregulated cellular proliferation downstream of the loss of the adenomatous polyposis coli (APC) tumor suppressor gene, which is a foundational driver of disease pathogenesis. It is being developed for the treatment of familial adenomatous polyposis (FAP), an autosomal dominant cancer predisposition syndrome. Activation of the mechanistic target of rapamycin (mTOR) pathway, which is a master regulator of cell growth, metabolism, and protein synthesis, has been observed in both preclinical models and human adenomatous tissue, suggesting that it represents a critical downstream effector of tumorigenesis in FAP.

Although rapamycin is a well-characterized mTOR inhibitor with established clinical use as an immunosuppressant, its application in oncology and chemoprevention has been constrained by poor and variable bioavailability, systemic toxicity, and challenges associated with chronic dosing. eRAPA is designed as an encapsulated, enteric-coated formulation that enhances delivery to the gastrointestinal tract while reducing systemic exposure. This approach is particularly well suited for FAP, where the disease is localized to the intestinal epithelium and long-term treatment is required.

In June 2025, Biodexa initiated the Phase 3 SERENTA trial to build upon the findings from the Phase 2 study. It is a double blind, placebo controlled trial that will enroll 168 high-risk patients with germline or phenotypic FAP diagnosis. The primary outcome of the trial is PFS as determined by a composite clinical progression measure between the following four conditions: Surgery/Meets Criteria for Surgery, Advancement of Spigelman Stage, Diagnosis of high-grade dysplasia or cancer, or death by any cause. The endpoints were agreed to by the U.S. FDA in a Type C meeting. The first patient was enrolled in

August 2025 and there will be an interim analysis after 25 PFS events (projected to be in 1Q28) and the database will be locked after 75 PFS events (projected to be in 4Q29). The trial is being partly funded by a $20 million grant from the Cancer Prevention and Research Institute of Texas (CPRIT).

Valuation

We value Biodexa using a probability-adjusted discounted cash flow model that takes into account potential future revenues for MTX240 and MTX230. While not currently a part of the model, MTX228 may offer additional upside to our valuation in the future.

MTX240: In the U.S., we assume a treatable population of approximately 5,000 patients, with MTX240 achieving close to 55% peak market share in later-line settings. At an annual price of $200,000, this yields peak revenues in the U.S. of approximately $600 million. In Europe, we estimate a treatable population of approximately 11,000 patients. Applying a 45% peak market share and a yearly price of $150,000, we derive peak E.U. revenue of approximately $550 million. Combined, these assumptions result in total peak global revenue for MTX240 of approximately $1.15 billion, which would position MTX240 as a potential blockbuster asset. We model for MTX240 to launch in 2032 with a 7-year ramp to peak sales and for the company to receive a 12% royalty on net sales from a commercialization partner. We apply a 15% discount rate and a 20% probability of approval, which yields an NPV of $27 million.

While derived from a traditional DCF model, recent transactions and market precedents in GIST and rare oncology provide important context for potential upside:

- The approximately $1 billion acquisition of IDRx by GlaxoSmithKline underscores the strategic value of differentiated GIST assets, even at relatively early stages of development. Notably, IDRx's program is focused on next-generation KIT inhibitors, whereas MTX240 represents a mechanistically different approach targeting the PDE3A-SLFN12 axis and potentially overcoming kinase resistance.

- Cogent Biosciences experienced an approximately $2 billion increase in market capitalization following the release of positive data from the Phase 3 PEAK trial for imatinib-resistant GIST, thus showing the sensitivity of valuations for GIST companies on clinical efficacy. This outcome suggests that even early positive signals for MTX240 could drive disproportionate valuation inflection relative to its current implied value.

MTX230: In the U.S., we assume a treatable FAP population of approximately 33,000 patients. Applying a 27% peak market share and annual pricing of $150,000, we derive peak U.S. revenue of approximately $1.5 billion. In the E.U., we estimate a treatable population of approximately 25,000 patients, with 22% peak market penetration and pricing of $75,000 annually, resulting in peak E.U. revenue of approximately $460 million. Combined, these assumptions yield total peak revenue of almost $2 billion, positioning MTX230 as a multi-billion-dollar commercial opportunity within a rare disease setting due to the combination of chronic treatment, high unmet need, and favorable orphan drug pricing. We model for MTX230 to launch in 2031 with a 7-year ramp to peak sales and for the company to receive a 12% royalty on net sales from a commercialization partner. We apply a 15% discount rate and a 50% probability of approval, which yields an NPV of $126 million.

Similarly to MTX240, while this valuation is derived from a DCF model, the following external benchmarks in rare disease and mTOR-targeted therapies provide important context:

- The current approximately $1.4 billion market cap of Palvella Therapeutics, driven by a Phase 3 topical rapamycin program in microcystic lymphatic malformations, a disease with comparable incidence to FAP, highlights the premium valuation investors assign to late-stage rare disease assets with validated biology. Notably, MTX230 targets a broader systemic indication with a larger commercial opportunity, suggesting that its valuation could ultimately converge toward similar levels with positive Phase 3 results.

- An additional benchmark can be seen in the sale of Fyarro (an mTOR inhibitor) for approximately $100 million in an ultra-rare oncology indication (PEComa), which further supports the strategic value of rapamycin-based therapeutics, even in small patient populations. MTX230 benefits from a similar mechanism of action but is positioned in a chronic, preventative setting with significantly greater revenue potential.

Combining the NPVs for MTX240 and MTX230 with the current cash position ($5 million) and the potential cash from warrant exercises (approximately $34 million) leads to a net present value for Biodexa of $193 million. The company currently has 3.7 million ADSs and approximately 6.3 million warrants outstanding for a fully diluted ADS count of approximately 10 million. We add an additional 17 million ADSs to account for future dilution, which leads to a valuation of $7 per ADS.

PROJECTED FINANCIALS

Biodexa Pharmaceuticals Plc (in millions of £)	2024 A	1H A	2H E	2025 E	2026 E	2027 E
MTX230 - eRAPA	£0.0	£0.0	£0.0	£0.0	£0.0	£0.0
MTX240 - GIST	£0.0	£0.0	£0.0	£0.0	£0.0	£0.0
Grants & Collaborative Revenue	£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**
Research & Development	£5.4	£1.7	£1.8	£3.5	£5.0	£15.0
Administrative Costs	£3.8	£2.4	£2.4	£4.8	£7.0	£7.5
Other Expenses	£0.0	£0.0	£0.0	£0.0	£0.0	£0.0
Operating Income	-£9.2	-£4.0	-£4.2	-£8.2	-£12.0	-£22.5
Non-Operating Expenses (Net)	£3.2	£0.0	£0.1	£0.1	£0.0	£0.0
Pre-Tax Income	-£6.0	-£4.0	-£4.1	-£8.1	-£12.0	-£22.5
Income Taxes Paid	£0.3	£0.2	£0.0	£0.2	£0.0	£0.0
Net Income	**-£5.8**	**-£3.8**	**-£4.1**	**-£7.9**	**-£12.0**	**-£22.5**
Exchange Gain/Losses	£0.0	£0.0	£0.0	£0.0	£0.0	£0.0
Total Comprehensive Gain/Loss	**-£5.8**	**-£3.8**	**-£4.1**	**-£7.9**	**-£12.0**	**-£22.5**
Net Loss per Share	**-£0.00**	**-£0.00**	**-£0.00**	**-£0.00**	**-£0.00**	**-£0.00**
Basic Shares Outstanding	4952.8	61952.3	61952.3	61952.3	61955.0	61955.0

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

HISTORICAL STOCK PRICE

Source: Biodexa Pharmaceuticals, PLC

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