The InhiBET™ Platform

Bromodomain & End Terminal (BET) Inhibitors in Autoimmune Disease

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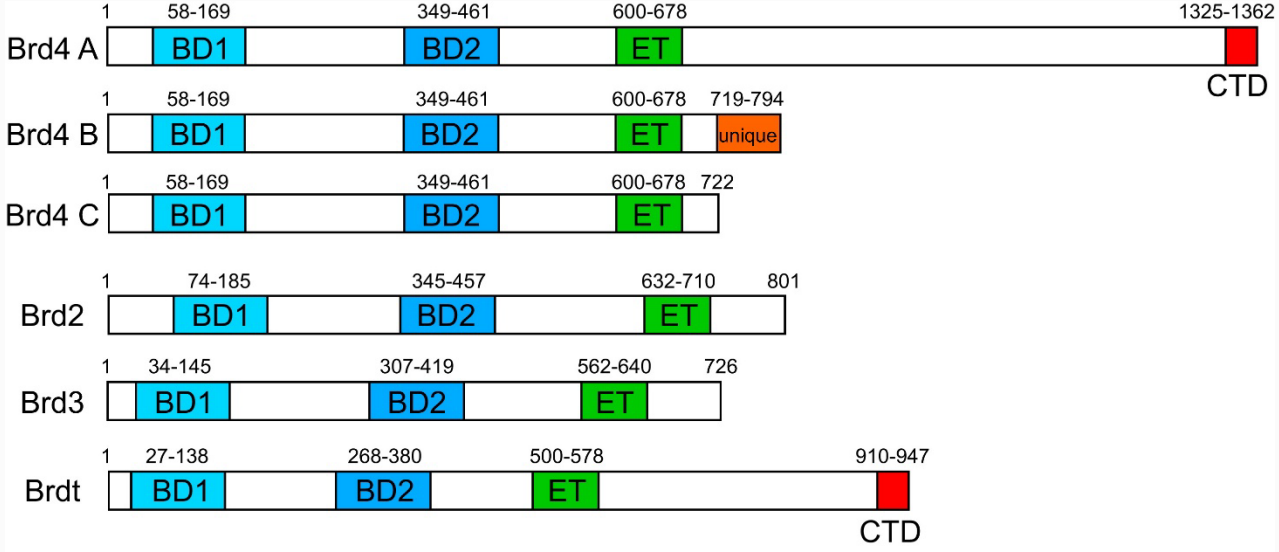
Bromodomain and end terminal proteins: Epigenetic enablers of transcription

Domain structure of human BET proteins



Functional binding domains

chromatin interaction

chromatin interaction

enzyme complex binding

interaction P-TEFb cyclin T1 cdk9

interaction P-TEFb cyclin T1 cdk9

- The bromodomain family (“Brd” or “BD”) is comprises of 4 members (BRD2-4, BRDT) and have two shared domains, bromodomain 1 and 2 (BD1 and BD2) with an extra-terminal domain
- Both BD1 and BD2 are recognized as druggable small molecule inhibitor targets
- Historically it had been difficult to design selective domain-specific inhibitors due to high amino acid homology between BD1 and BD2.

Shi & Vakoc, Mol. Cell 2014

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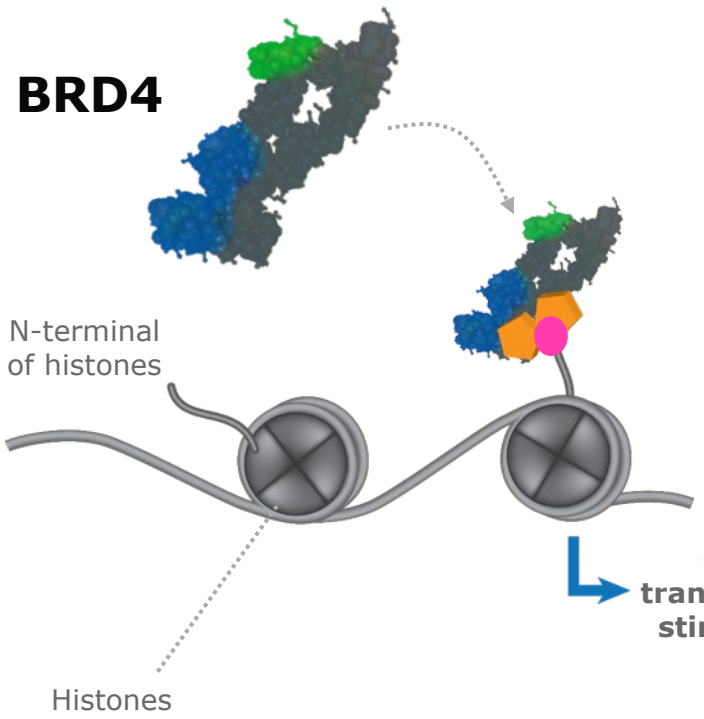
BET Proteins Play a Key Role
in the Regulation of Inflammatory and Oncogenic Genes involved in Several Diseases



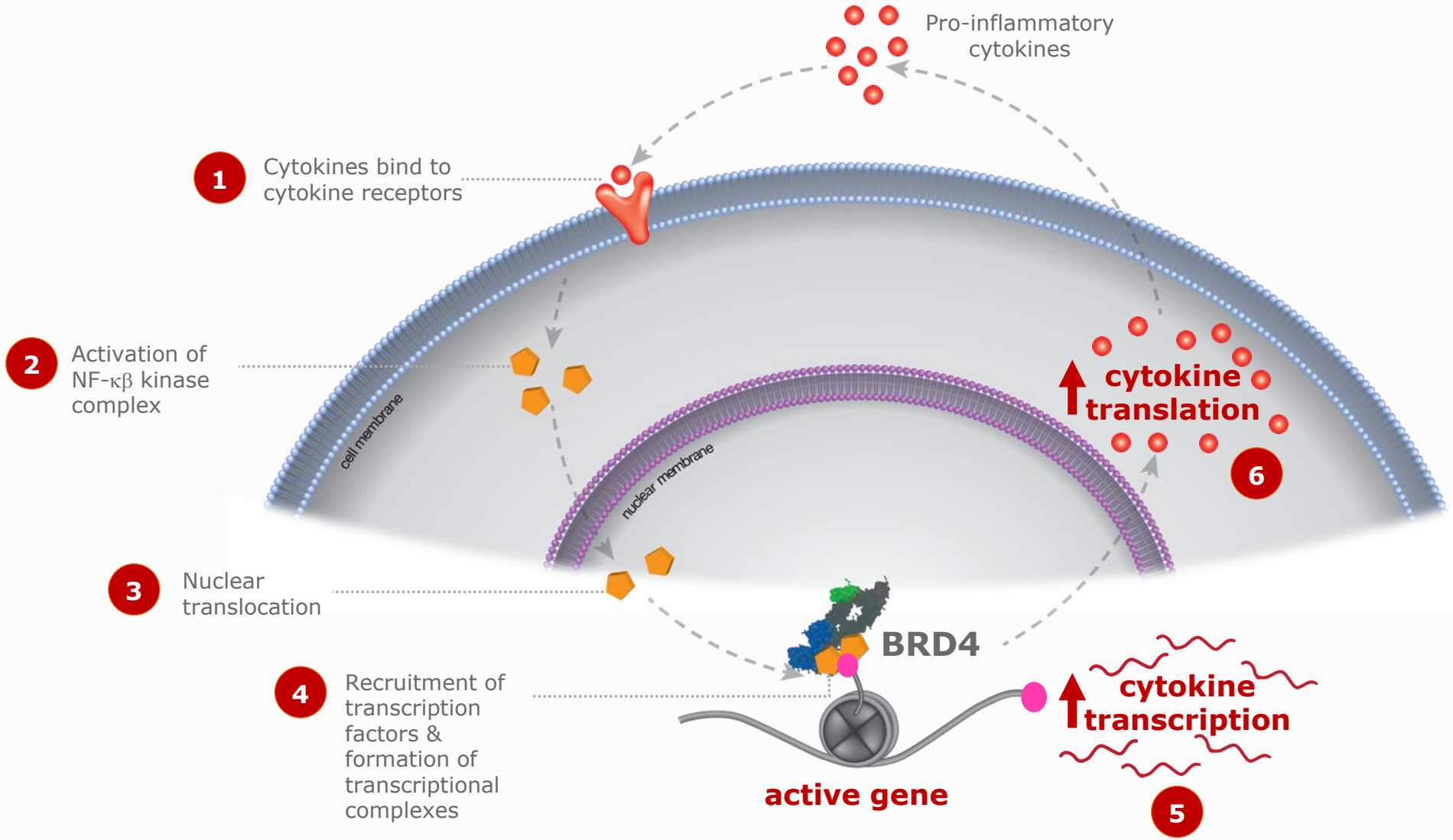
BET proteins bind to histones in chromatin by "reading" acetylated lysine motifs on N-terminus of histone tails and recruit co-regulatory complexes:

Pro-inflammatory genes, leading to:
- Increased cytokine expression that activate B&T cells
- An increase in autoimmune and cardiovascular diseases

Oncogenic genes, leading to:
- Increased cell proliferation/survival
- An increase in solid tumors and hematologic malignancies

How BET Proteins Influence Transcriptional Machinery

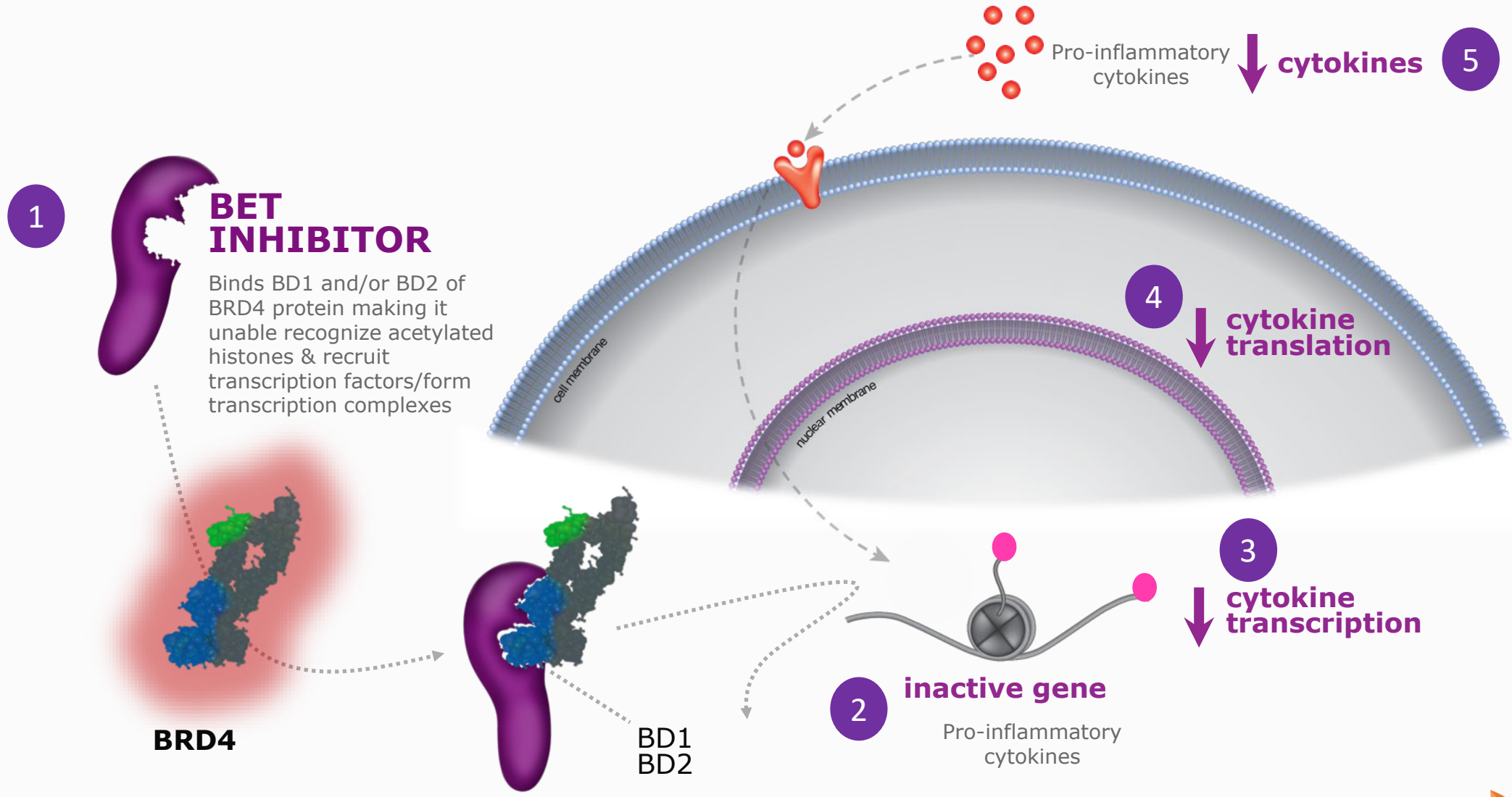
Role of BET proteins in Pro-Inflammatory Cytokine Production in Autoimmune Diseases



NF-κB = Nuclear Factor Kappa B

BET Inhibitors Block BD1/BD2 Binding

to Acetylated Lysines and Stall Pro-inflammatory Protein Transcription



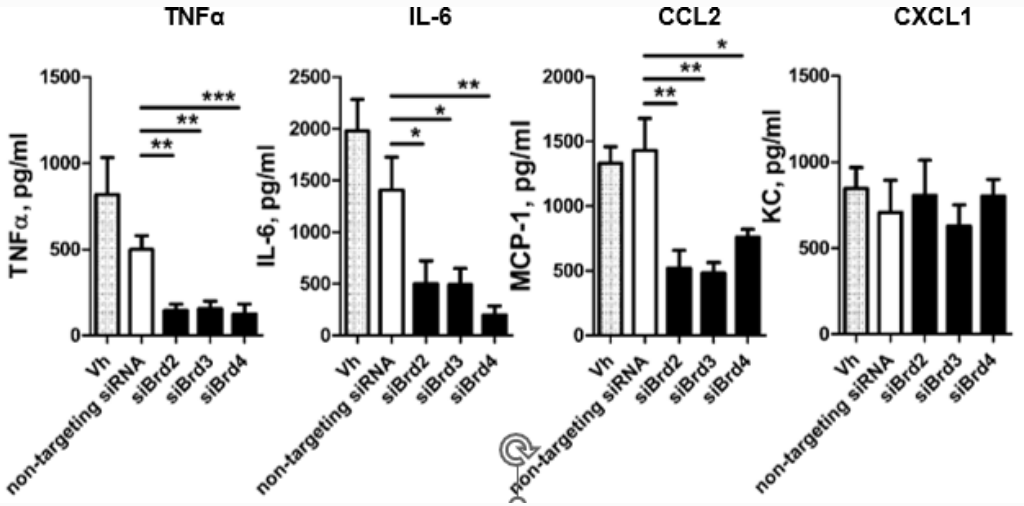
BET inhibitor biology – what have we learned?

Bromodomain functionality

- Each BRD bromodomain plays distinct roles transcription regulation – they don't do the same job
  - Steady-state gene expression primarily requires BD1.
  - Rapid increase of gene expression induced by inflammatory stimuli requires both BD1 and BD2.

General BET inhibitor effect on cytokine release

- BET inhibitors are not immuno-inhibitory "shotguns", not all cytokines are regulated by BETs. SAR is not intuitive.



BET inhibitor clinical safety

- BD2-selective BET inhibitors could have improved safety profiles with greater applicability outside of oncology due to less DLTs relating to reprotoxicity (BRDT target), GI stem cell toxicities and severe thrombocytopenias.

Drug design strategies for BET inhibitors

- Key for pan-BD BET inhibitors is to mitigate systemic exposure (tissue targeted administration routes/formulations, prodrug/soft drug delivery strategies
- For indications requiring systemic administration routes, focus on maximizing BD2 v's BD1 selectivity

Dawson et al, Science 2020
Belkina, Nikolajczyk & Denis, J. Immunol. 2013

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InhiBET™ BET Inhibitor Platform Overview

Small molecule pan-BD and BD2-selective BET inhibitors

VYN201

Locally administered Pan-BD BET inhibitor

Designed to address diseases involving multiple, diverse inflammatory cell signaling pathways with low systemic exposure

Broad BD activity:

- Low nM Kd against both BD1 and BD2 domains

Systemic toxicity mitigation:

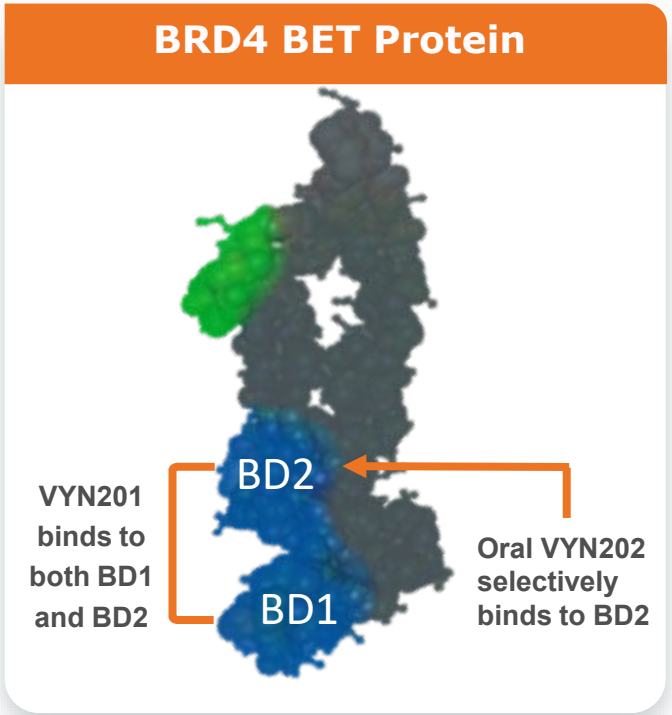
- Inflammatory tissue targeting, very high first pass metabolism

Competition:

- Many BET inhibitors in development bind to BD1 and BD2 and are orally delivered with significant DLTs

Potential Indications:

- Vitiligo
- Other indications benefiting from local application and "soft drug" approach



VYN202

Oral BD2-selective BET inhibitor

Designed to selectively bind to BD2 and is being developed for major immuno-inflammatory diseases

Focused BD activity:

- Low nM Kd against BD2 domain
- Highly selective inhibition of BD2 domain of the BRD4 protein (~1000-fold selectivity vs. BD1)
- Targeting improved safety profile compared to oral pan-BD BET inhibitors

Potential Indications:

- Immuno-inflammatory indications such as rheumatoid arthritis, systemic lupus erythematosus, ulcerative colitis/Crohn's and multiple sclerosis; additional potential in myeloproliferative neoplastic disorders

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VYN202
Orally available BD2 selective BET inhibitor

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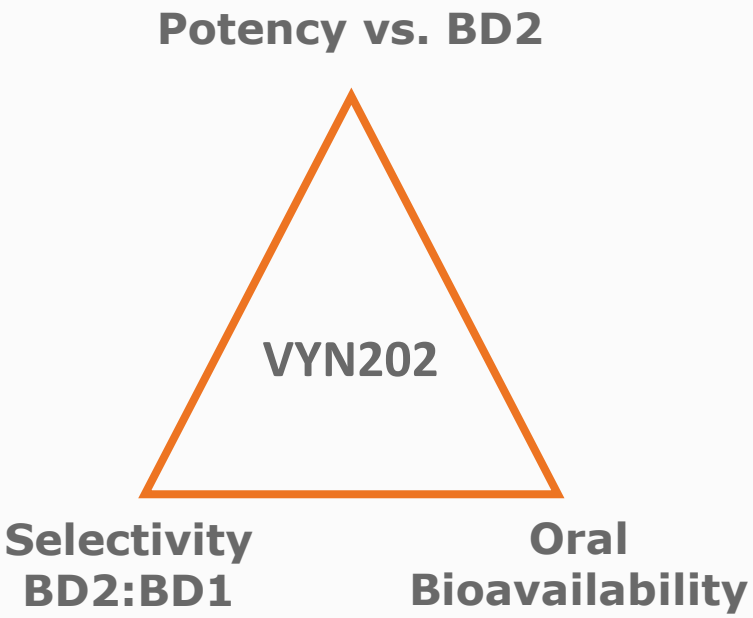
VYN202 Program Highlights & Molecular Profile

VYN202 is an oral BET inhibitor designed to selectively bind to BD2 and is being developed for major immuno-inflammatory diseases



Potential Target Market[1]:

- Immuno-inflammatory indications such as rheumatoid arthritis, systemic lupus erythematosus, ulcerative colitis/Crohn's and multiple sclerosis; additional potential in myeloproliferative neoplastic disorders[2]

Focused activity:

- Highly selective inhibition of BD2 domain of the BRD4 protein
- Targeting improved safety profile compared to oral pan-BD BET inhibitors

Targeted Near Term Milestones:

- Candidate Selection – 2022

1. Initial indication to be communicated following candidate selection, exercise of option, IND-enabling studies and completion of requisite pre-clinical evaluations
2. List included is not exhaustive of potential indications

VYN201
Locally administered pan-BD BET inhibitor

Non-clinical efficacy program

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VYN201 Pre-Clinical Efficacy Model Evaluations

Program designed to investigate the targeted administration of a "soft drug" pan-BD BET inhibitor to maximize local effect and minimize systemic exposure

Indication/area	Model	Administration Route	Status
Th17 autoimmune diseases	IMI-induction mouse model	Topical	Complete
Th2 autoimmune diseases	DNCB-induction mouse model	Topical	Complete
Fibrosis	Wound healing outcomes mouse model	Topical	Complete
Vitiligo	Reconstituted human epithelial skin TNFα/IFNγ induction model	Topical	Complete
Idiopathic pulmonary fibrosis	Bleomycin-induction mouse model	Intra-nasal	Complete
Rheumatoid arthritis	Intra-articular cytokine cocktail mouse model	Intra-articular	Complete
Macular degeneration	Choroidal neovascularization rat model	Intra-orbital/vitreous	Complete
Colitis (gut restricted)	DSS-induction mouse model	Oral	Complete
Oncology (AML/melanoma)	Human cell line screening and biomarker discovery	In-vitro	On-going

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VYN201
Psoriasis murine model (topical)

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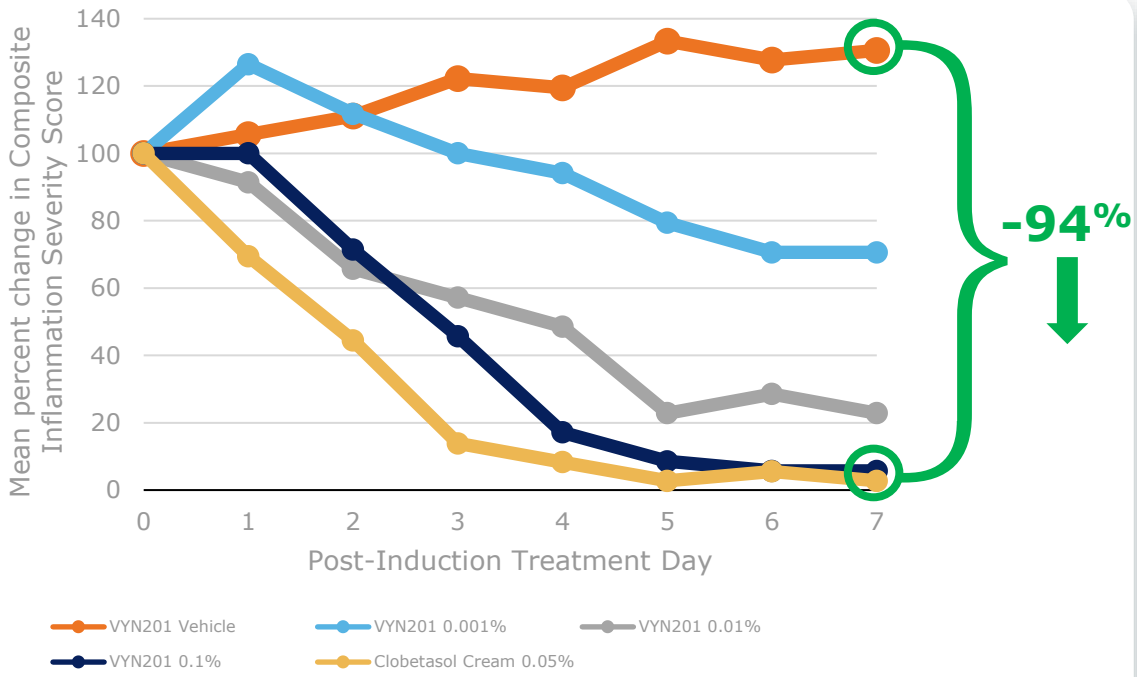
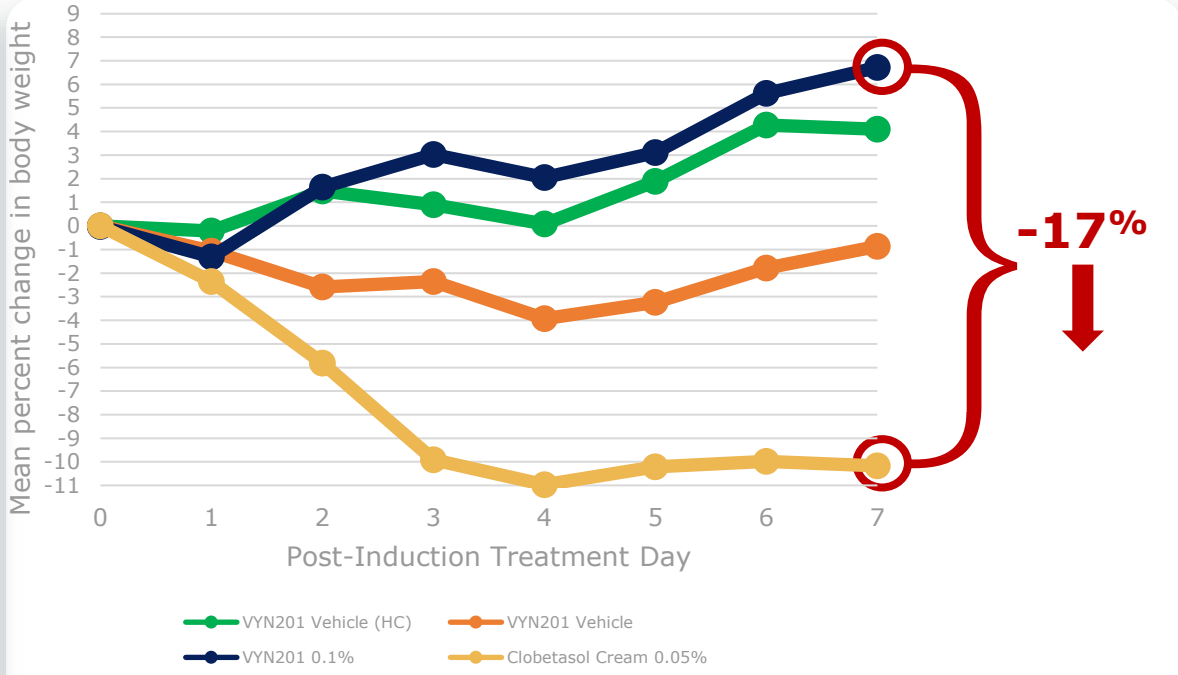
VYN201: Comparable Efficacy to Superpotent Steroid Clobetasol
in a TH17-Mediated Murine Inflammation Model; Potential for Greater Tolerability

Dorsal depilated BALB-C mice were dosed topically for 14 days with 100mg topical IMI cream (Day 1-7: induction phase, Day 8-14: treatment phase). N=4 animals were assigned to each treatment group with each group receiving 100mg of allocated treatment on Day 8-14 once daily



- Dose dependent response was observed over the VYN201 concentration range 0.001% to 0.1%
- There was a 94% reduction in composite score for VYN201 0.1% relative to vehicle control group at Day 7
- VYN201 0.1% had comparable efficacy to clobetasol propionate 0.05% cream



- Animals treated with VYN201 0.1% continued to gain body weight in a similar manner to healthy control group treated with vehicle
- Clobetasol cream 0.05% group had a 17% reduction in body weight compared to the VYN201 0.1% group at treatment day 7

IMI – Imiquimod.

*Composite Inflammation Severity Score is a composite mean score of erythema and peeling severity scored on a 4-point ordinal scale per domain (0=none, 1=mild, 2=moderate and 3=severe for a maximum score of 6), data expressed as a mean percentage change from initiation of treatment phase.

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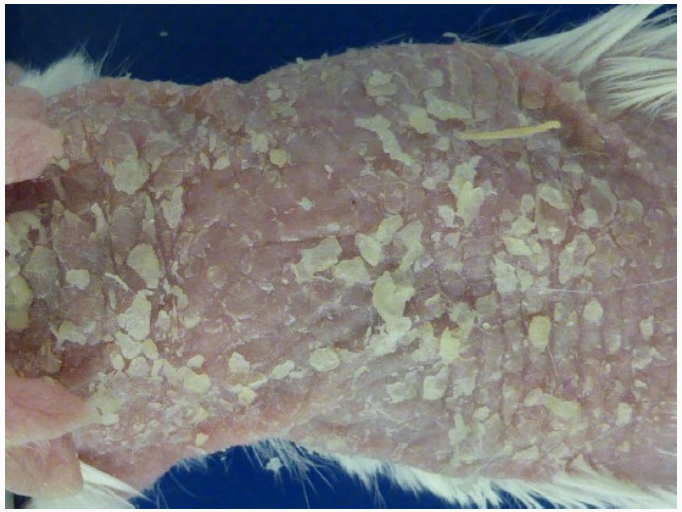
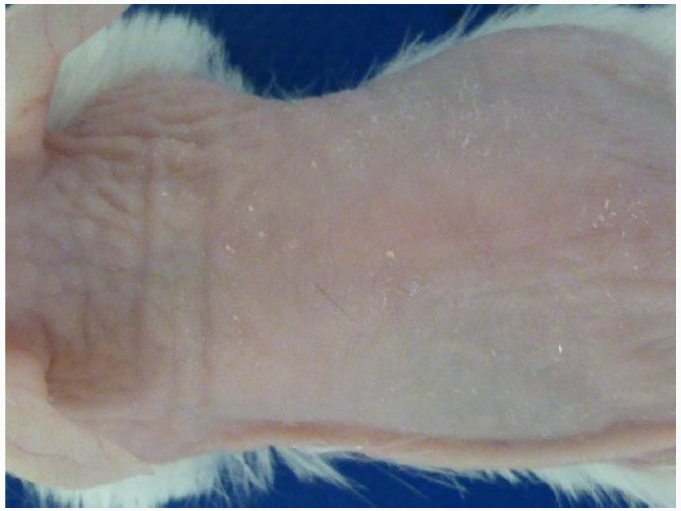
VYN201:
Normal Skin Physiology in TH17-Mediated Murine Inflammation Model Suggests VYN201 Well Tolerated (Day 7)



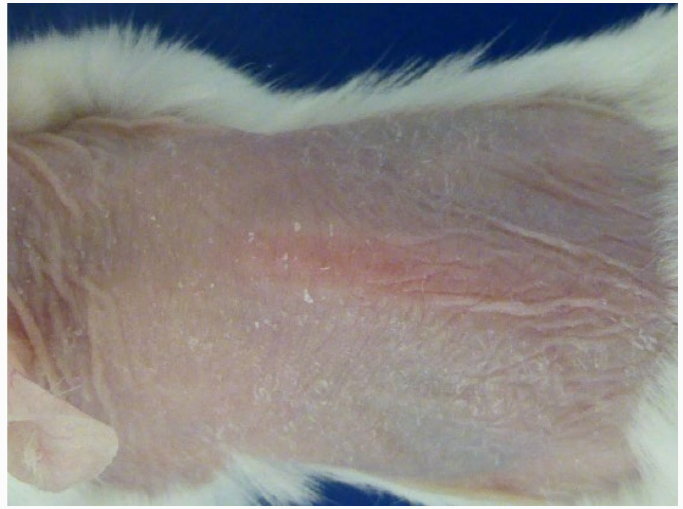
VYN201 Vehicle

- No appreciable improvement in clinical signs



VYN201 0.1%

- Substantial resolution of clinical signs
- Skin presents with normal physiology with no evidence of striae rubrae or atrophy
- No evidence suggestive of intolerance



Clobetasol Cream 0.05%

- Substantial resolution of clinical signs
- Significant evidence of dermal atrophy (clear presence of both rhytides/fine wrinkles and deep wrinkles)
- Marked dermal translucency and elastolysis

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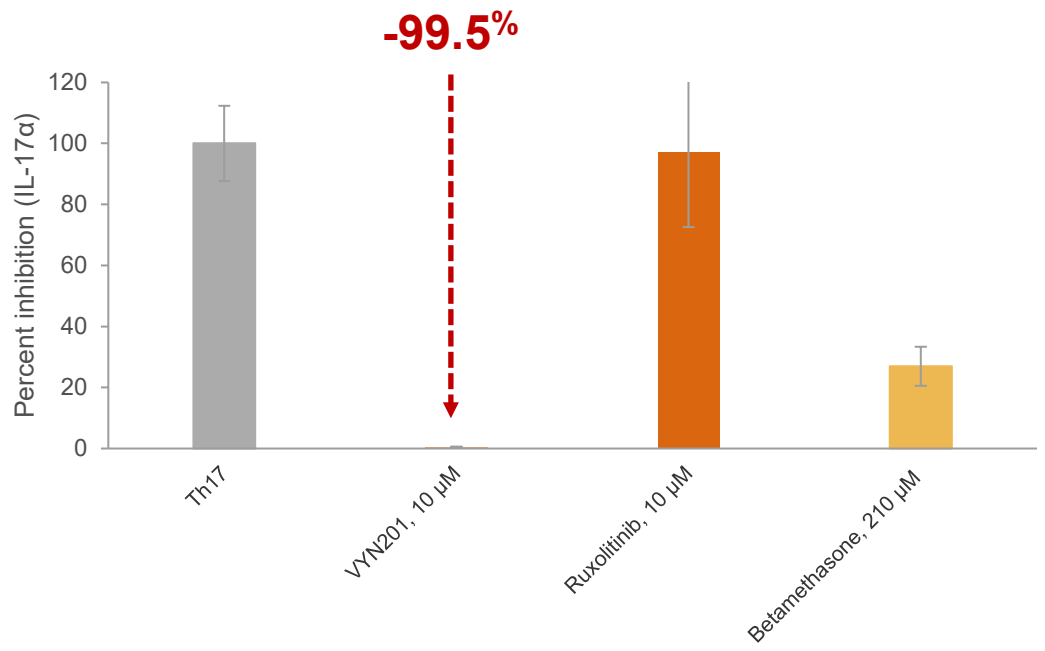
VYN201 Significantly Reduced Expression of Several Key Pro-Inflammatory Proteins Relevant to Th17-mediated Autoimmune Diseases in Human Tissue[1]



Interleukin 17-alpha

T-cells are polarized to Th1 and Th17 cells, the latter of which drives the production of IL17a which further upregulates the migratory action of pro-inflammatory cells and further inflammatory cell activation.

>95% Inhibition seen with assays for IL-36γ & LP-10

Interleukin 36-gamma

IL36γ is implicated in upregulating IL-17A signaling-related genes and so able to amplify keratinocyte inflammatory responses by promoting not only their own expression but also that of other cytokines related to Th17 signaling

CXC motif chemokine ligand 10 (LP-10)

An inflammatory cell chemoattractant secreted in response to interferon gamma. LP-10 is significantly overexpressed in many autoimmune diseases (>25-fold) vs. healthy skin[1]

1. Data on file. Results presented from qPCR analysis of processed and Th17-stimulated ex vivo human skin tissue based on a method derived from Garrett S.M., Zhao Q., and Feghali-Bostwick C. (2019) Induction of a Th17 phenotype in human skin – a mimic of dermal inflammatory diseases, *Methods and Protocols*, 2, 45

VYN201
Rheumatoid Arthritis murine model (intra-articular)

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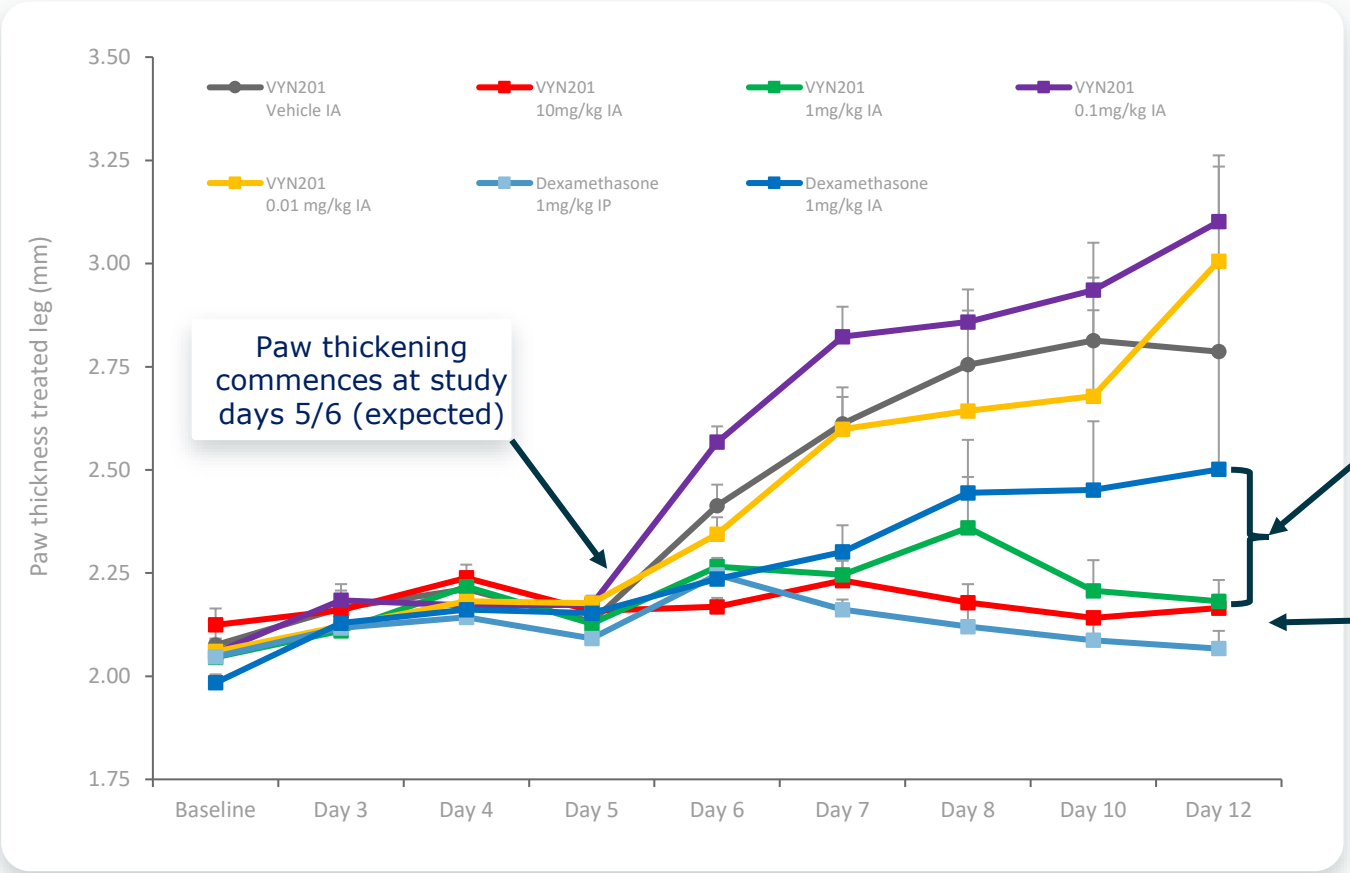
VYN201: CAIA Mouse Model of Arthritis – Paw Thickness

Marked inhibition of paw thickening/swelling

Inflammatory arthritis was induced in BALB/C mice using a mixture of four arthritogenic MAbs by IV injection at Day 0 and was further challenged with an LPS IV injection at Day 4 (N=7/treatment group). VYN201 treatment groups received 50µl intra-articular (IA) doses of VYN201 at 0, 0.01, 0.1, 1 or 10mg/kg on Days 0, 3, 6 and 9. Dexamethasone control animals received 50µl of 10mg/kg IA on Days 0, 3, 6 and 9 or 1mg/kg intraperitoneal (IP) on each treatment day (Day 0-11). Treatment response was evaluated based on an assessment of paw thickening/swelling.



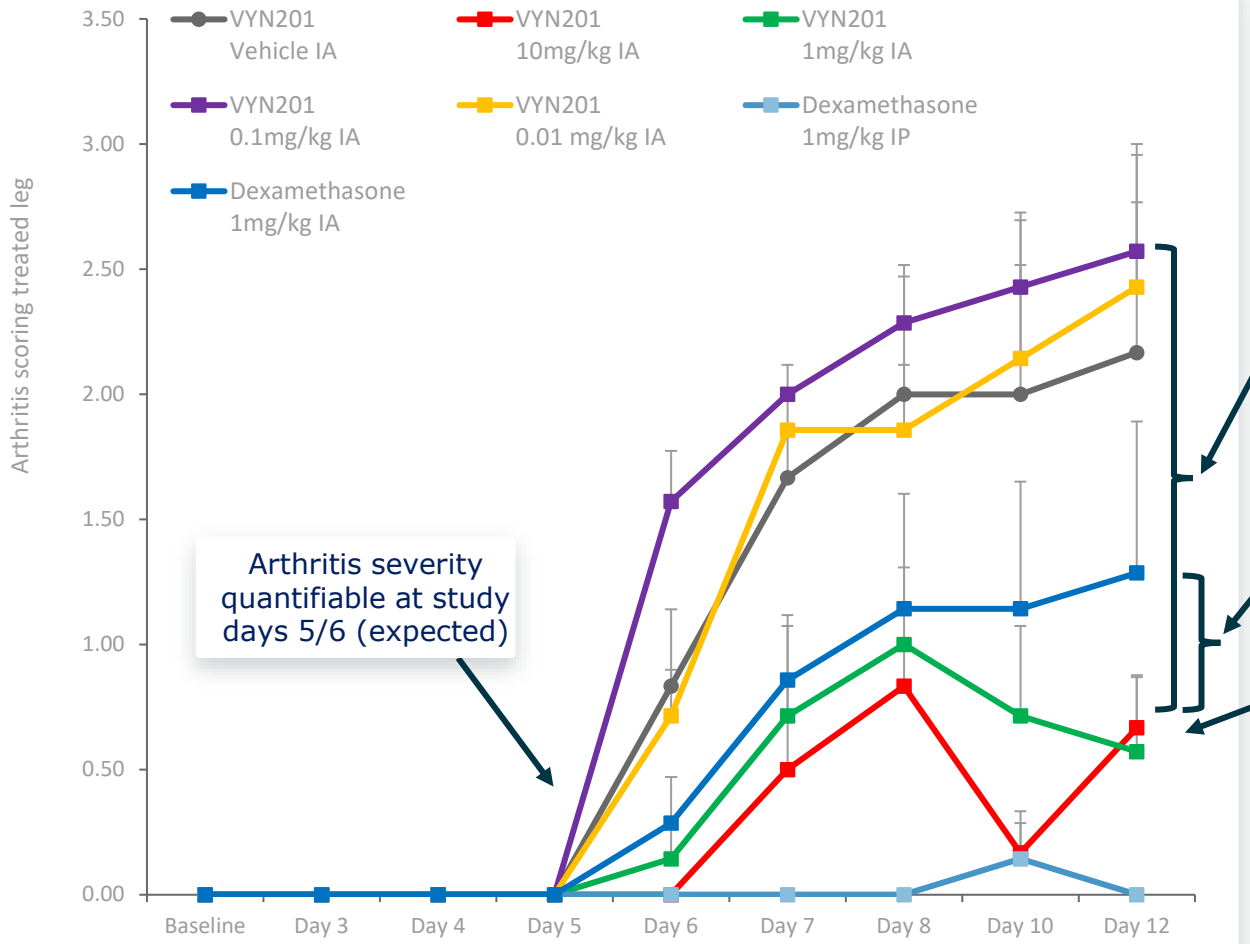
Both VYN201 1 & 10mg/kg superior to dexamethasone IA 10mg/kg

Marked inhibition of paw thickening for the VYN201 1 & 10mg/kg dose levels and in line with 1mg/kg dexamethasone systemic dose

CAIA = Collagen Antibody Induced Arthritis; IA = Intra-articular; IP = Intraperitoneal

VYN201: CAIA Mouse Model of Arthritis – Arthritis Score

Demonstrated dose dependent reduction in disease severity



Clear dose-response demonstrated between VYN201-treated groups

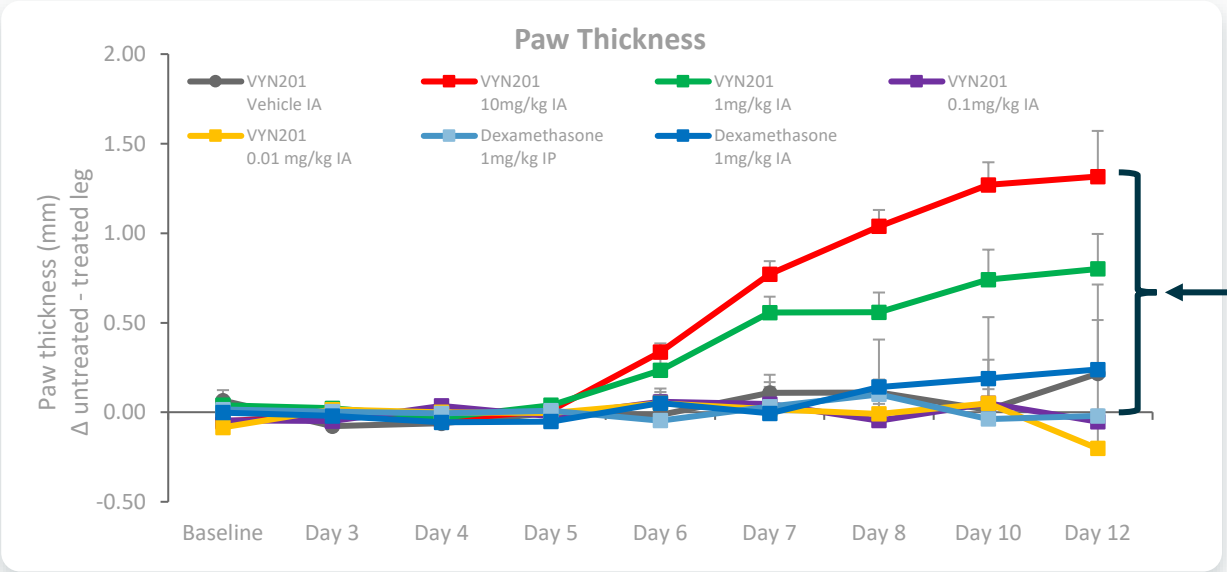
Both VYN201 1 & 10mg/kg superior to dexamethasone IA 10mg/kg

Marked inhibition of arthritis signs and symptoms for VYN201 at 1 & 10mg/kg dose levels with severity scores approaching "normal" (mean severity score <1)

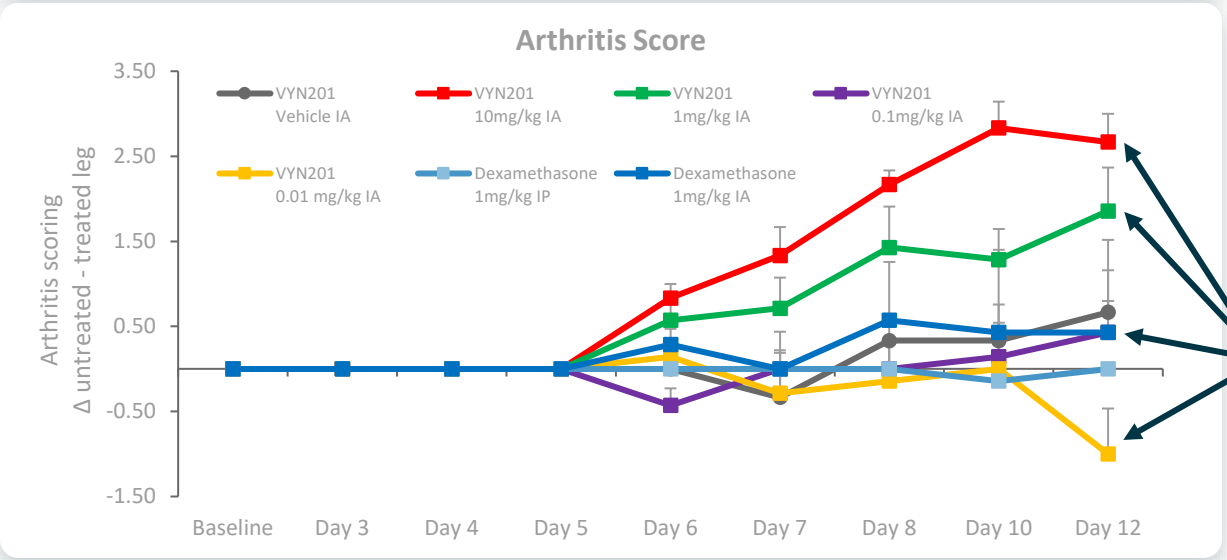
*Scoring based on a five-point composite severity scale of redness, swelling of the ankles and wrists, and paw thickness. Scoring ranges from 0 (normal) to 4 (extensive signs and symptoms of arthritis);
CAIA = Collagen Antibody Induced Arthritis; IA = Intra-articular; IP = Intraperitoneal

VYN201: CAIA Mouse Model of Arthritis – Systemic Impact

Demonstrated localized effect



VYN201 1 & 10mg/kg results demonstrated the highest localized effect in the treated limb when compared to the untreated limbs (largest delta between treated and untreated limbs)



Treatment effect for VYN201 treated animals was dose-dependent over the dose range 0.01 to 10mg/kg

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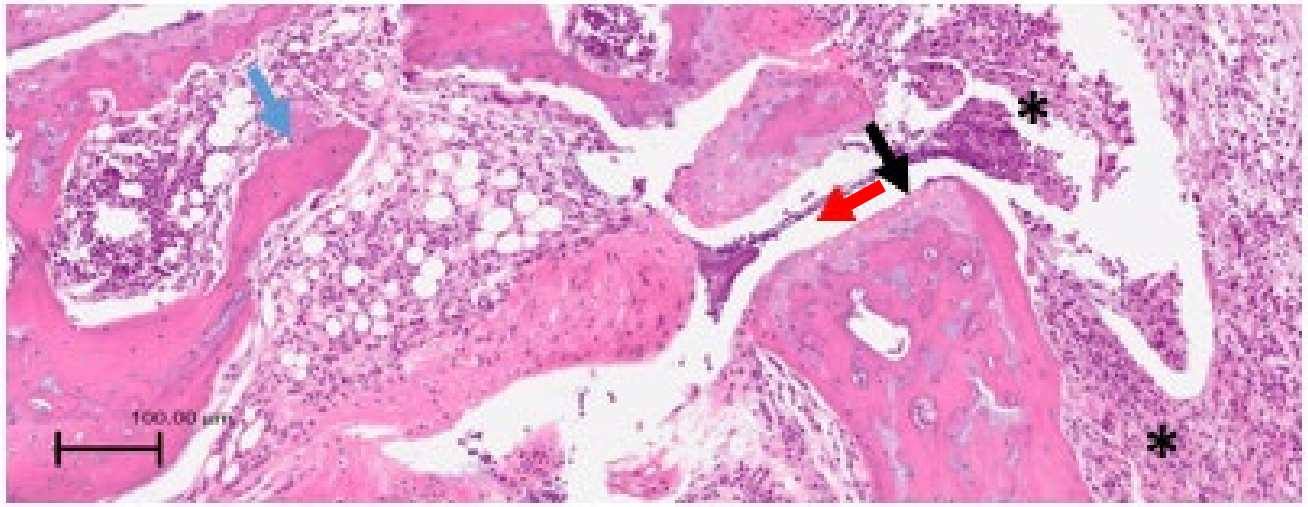
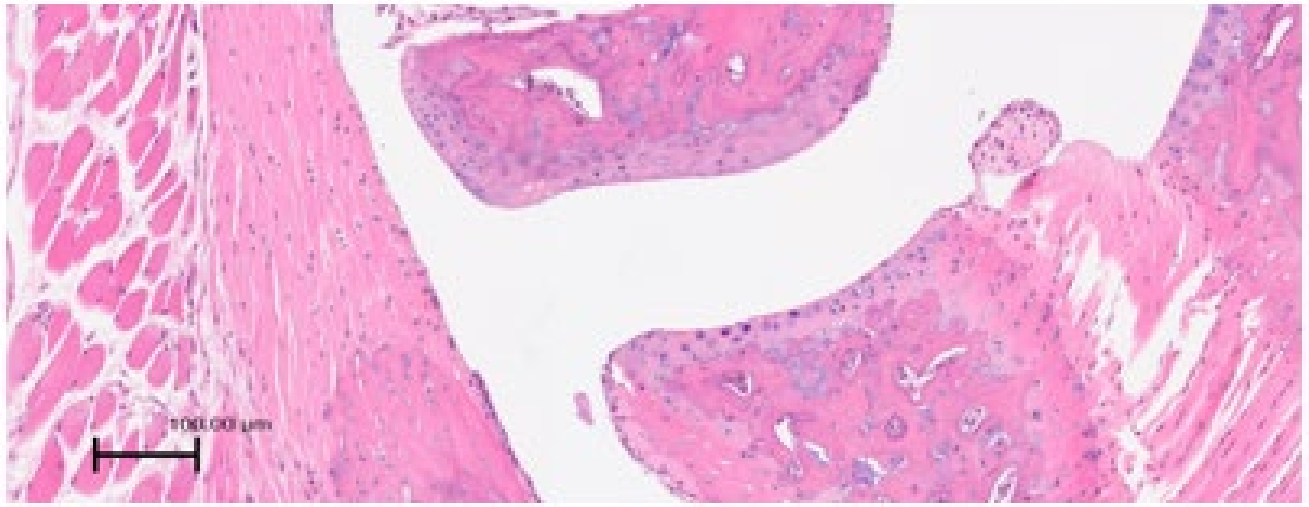
*Scoring based on a five-point composite severity scale of redness, swelling of the ankles and wrists, and paw thickness. Scoring ranges from 0 (normal) to 4 (extensive signs and symptoms of arthritis); CAIA = Collagen Antibody Induced Arthritis; IA = Intra-articular; IP = Intraperitoneal

VYN201: CAIA Mouse Model of Arthritis – Histopathology

Joint histopathology confirmed arthritis clinical scoring & local effect

Inflammation marked with asterisk; damage to bone marked with a blue arrow; inflammatory cells and cellular debris in the joint space marked with red arrow; damage to cartilage marked with black arrow; scale bar-100µm





VYN201 10mg/kg treatment group

Untreated right paw
Arthritis score 4 "severe"

Severe inflammation with destruction of both cartilage and bone.

Treated left paw
Arthritis score 0 "within normal limits"

No evidence of inflammatory cell infiltrate in the joint spaces.

VYN201
Anti-fibrosis/scarring murine model (topical)

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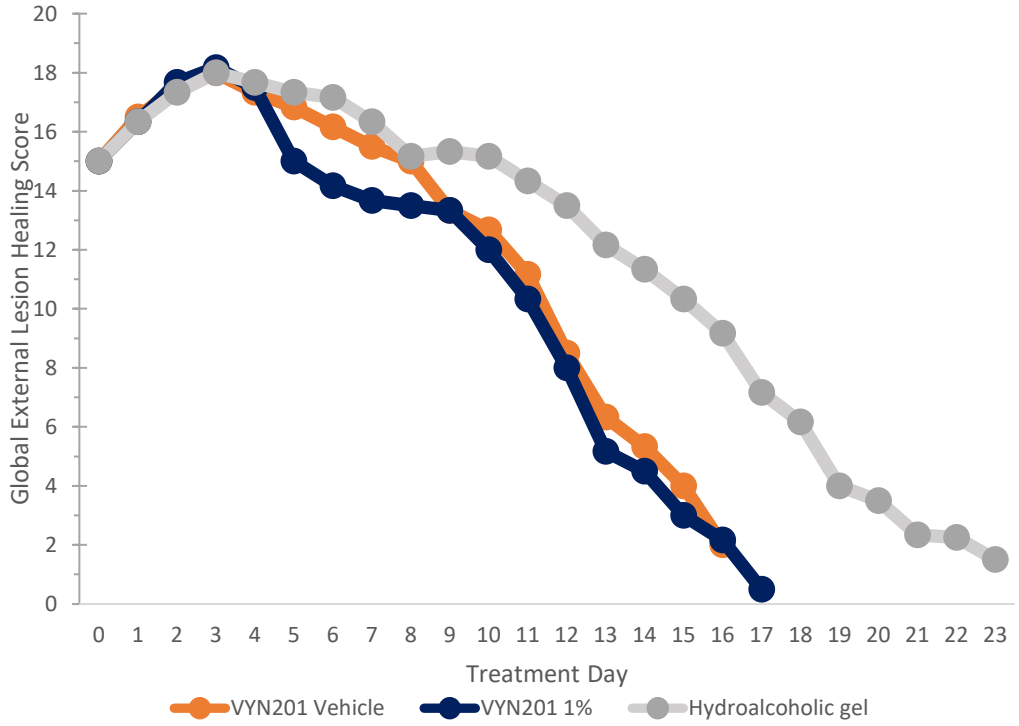
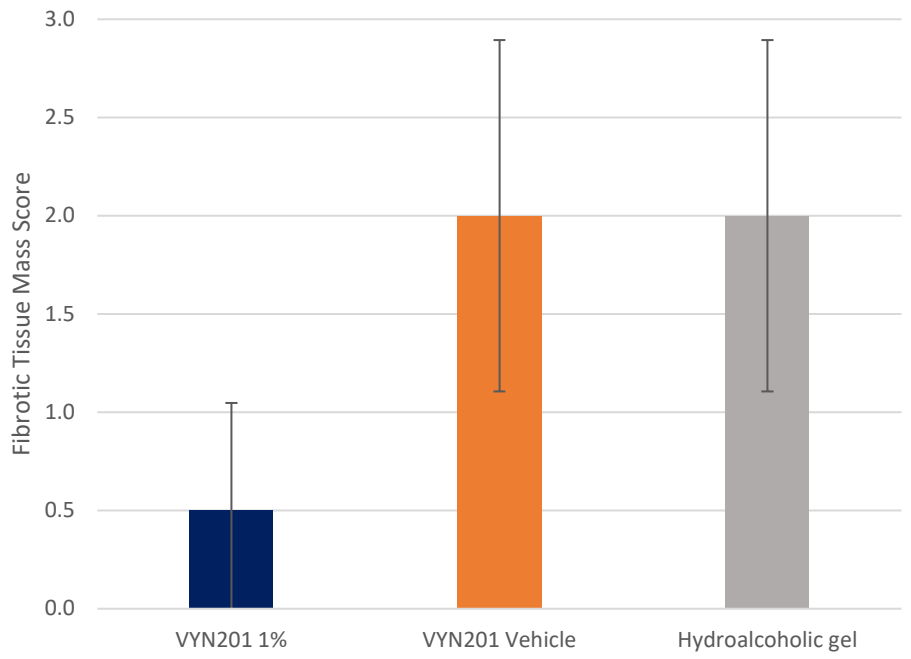
VYN201: Demonstrated Anti-Fibrotic Activity
without Delay in Healing Time in Murine Skin Healing Model

Female hairless mice (n=4/group) had two identical 10mm incisions made either side of the flank. Animals were topically dosed 1X daily with 100mg VYN201 vehicle, VYN201 1% or a hydroalcoholic gel* until each wound had completely healed



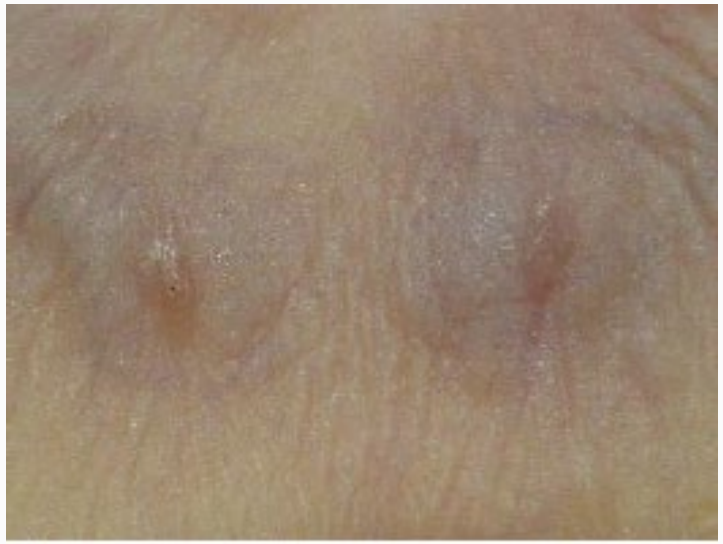
- Statistically significant difference ($p<0.05$) in composite global external healing score for VYN201 1% compared to Hydroalcoholic gel from Day 8
- Complete healing occurred for VYN201 1% and VYN201 vehicle approximately 5 days earlier compared to Hydroalcoholic gel (Mean day to heal:15.5 vs. 21 days)



- Animals treated with VYN201 1% had statistically significant less tissue mass/fibrosis compared to VYN201 vehicle or Hydroalcoholic gel, indicative of the known anti-fibrotic mechanism for BET inhibition (*P*<0.05 for VYN201 1% compared to VYN201 vehicle and Hydroalcoholic gel)

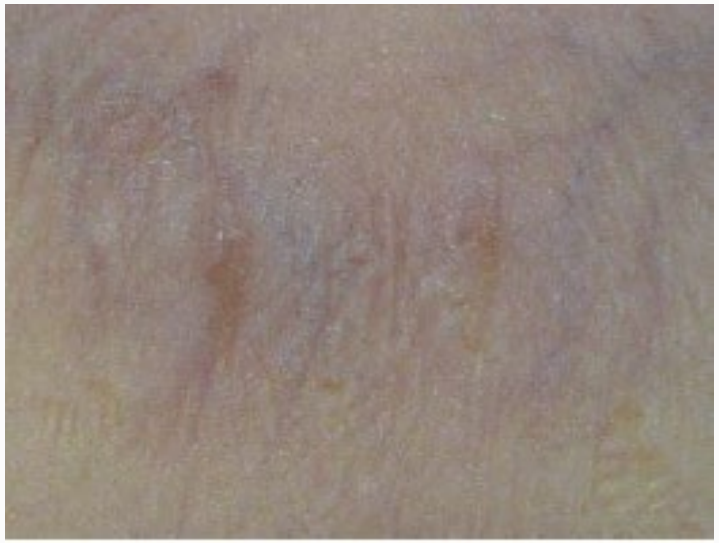
*A negative control known to delay wound healing
Global External Lesion Score is a composite severity score of lesion length, width, swelling and visibility
Fibrotic tissue mass is scored on a 4-point severity scale: 0=No tissue mass; 1=small tissue mass; 2=moderate tissue mass; 3=large tissue mass

VYN201: Little Evidence of Residual Swelling
and Macular Wound Appearance in Murine Skin Healing Model



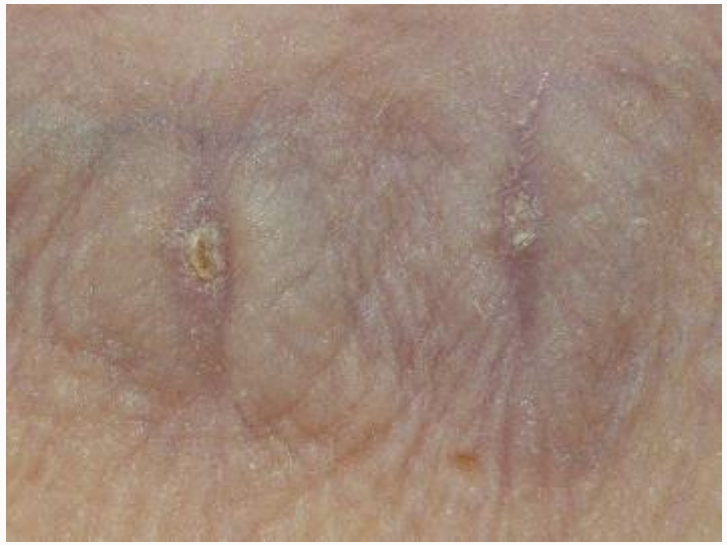
VYN201 Vehicle

- Still evidence of minor swelling around incision sites



VYN201 1%

- Little evidence of residual swelling
- Wound appears more macular in nature compared to VYN201 vehicle or the hydroalcoholic gel
- Incision sites appear less distinct and leave a more aesthetic outcome compared to other treatments



Hydroalcoholic gel

- Moderate swelling clearly evident at end of treatment
- Although healed, residual scabbing still remains
- Incision sites clearly visible

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VYN201
Idiopathic pulmonary fibrosis murine model (intra-nasal)

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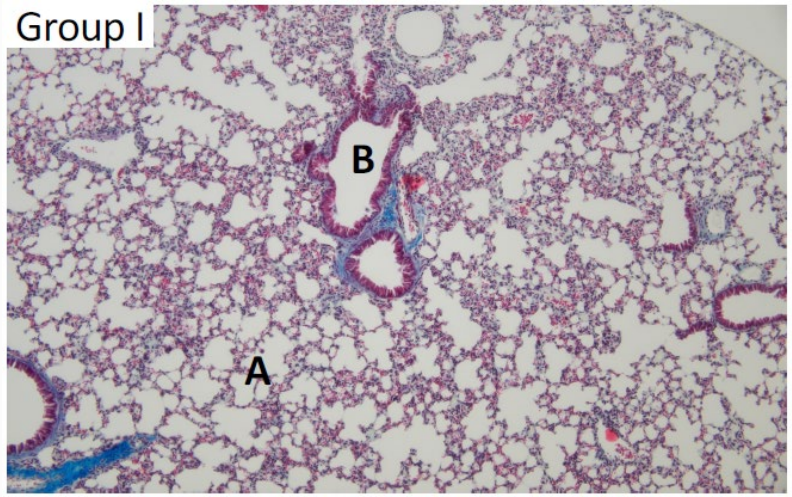
VYN201: Bleomycin-induced mouse model of idiopathic pulmonary fibrosis

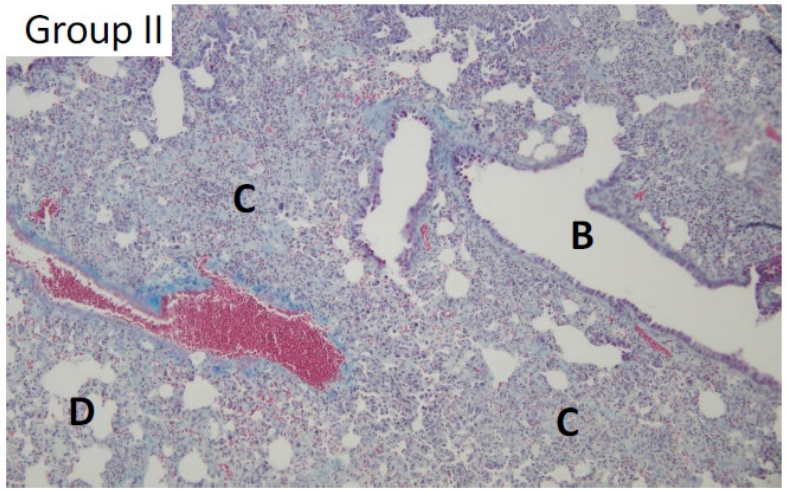
Histopathology Images

Lung fibrosis is induced in C57bl/6 mice using bleomycin at a dose of 4U/kg once daily by intranasal administration (N=10/treatment group). VYN201 treatment groups received nebulized, intra-nasal doses at 0, 0.06, 0.6 and 3mg/ml and bleomycin concomitantly for 21 days. A sham group received vehicle only.



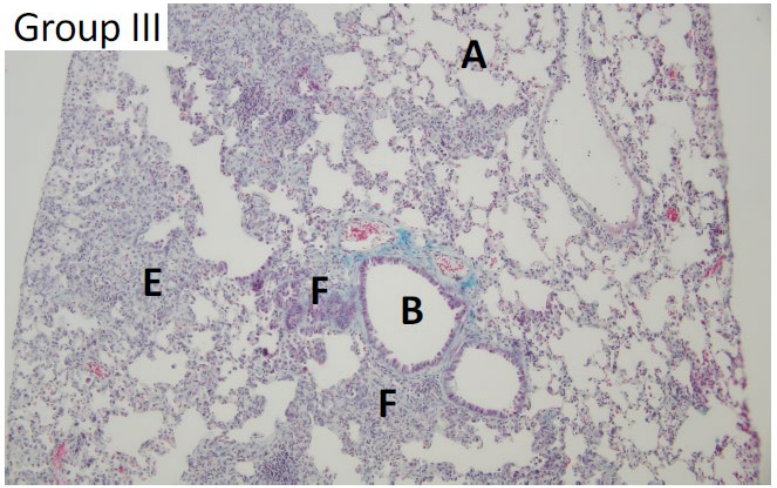
Sham control

A: Normal lung parenchyma with open airways and airspaces.
B: Conducting airway



Bleomycin control

B: Conducting airway
C: Fibrotic lesions with abundant cellularity
D: Sporadic airways



Bleomycin + 0.06mg/ml VYN201

A: Normal lung parenchyma with open airways and airspaces.
B: Conducting airway
E: Less severe localized fibrosis: parenchyma
F: Less severe localized fibrosis: airway

VYNE™ THERAPEUTICS

VYN201
Ex vivo human skin explant vitiligo model (topical)

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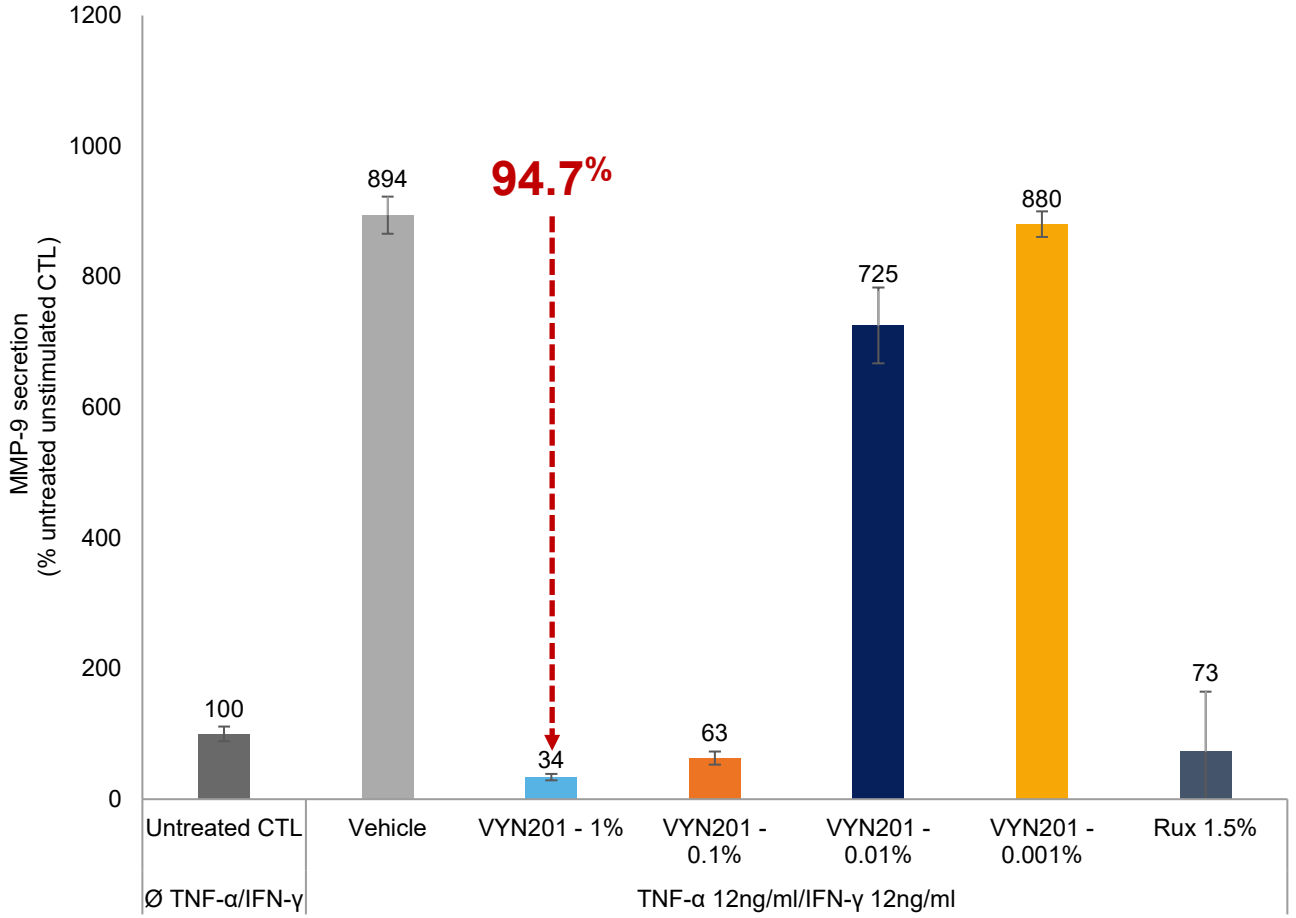
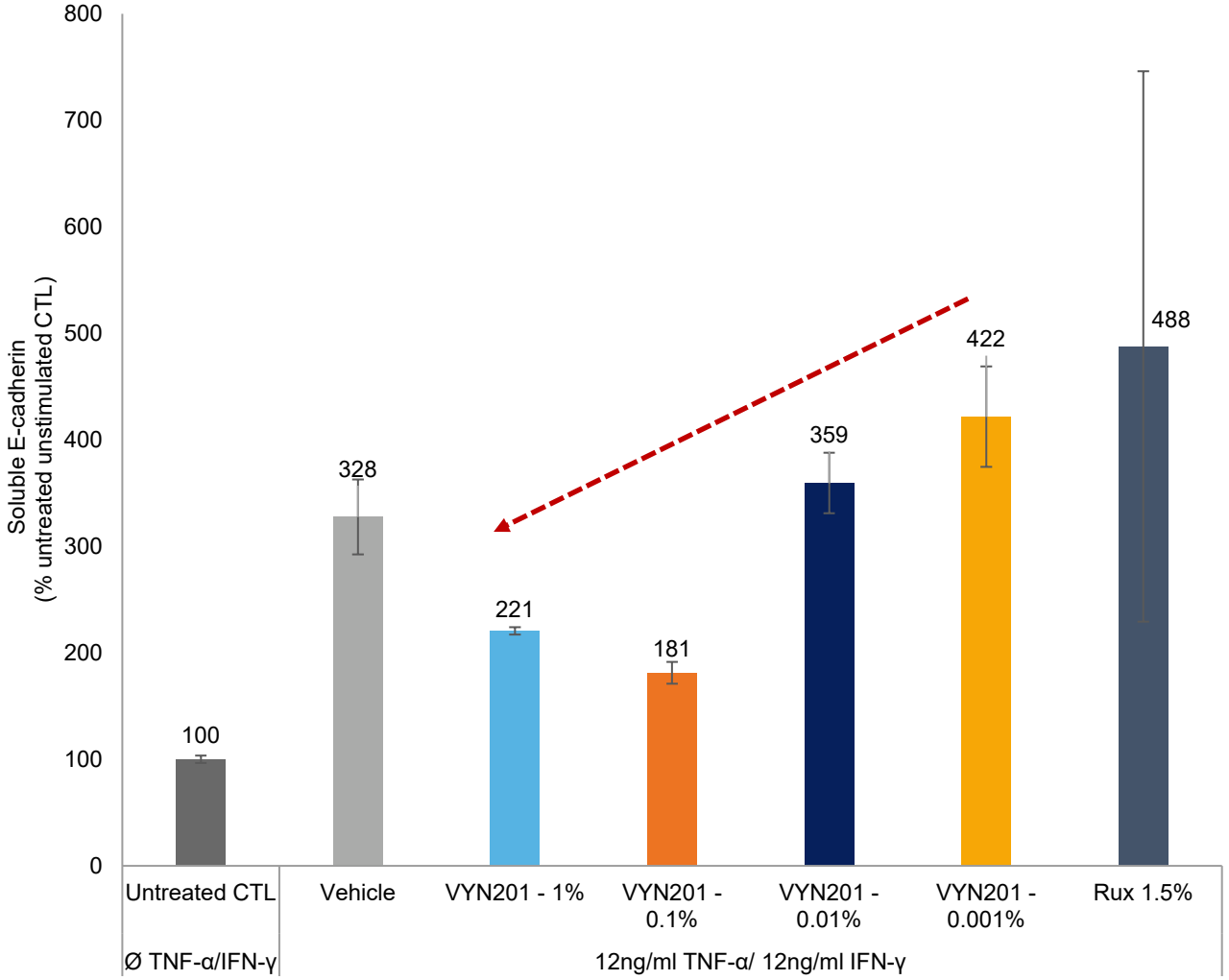
VYN201: Human Tissue Model of Vitiligo – Demonstrated Inhibition of MMP9

Reconstituted human epithelial (RHE) skin cultures were treated with a TNF-α and IFN-ɣ cytokine cocktail to induce a vitiligo phenotype melanocytorrhagy (loss of melanocyte), upregulation of MMP9 and soluble E-cadherin). 24hr prior to stimulation, RHE samples were topically treatment with VYN201 at varying concentrations and topical ruxolitinib 1.5% cream at 3 mg/cm^2



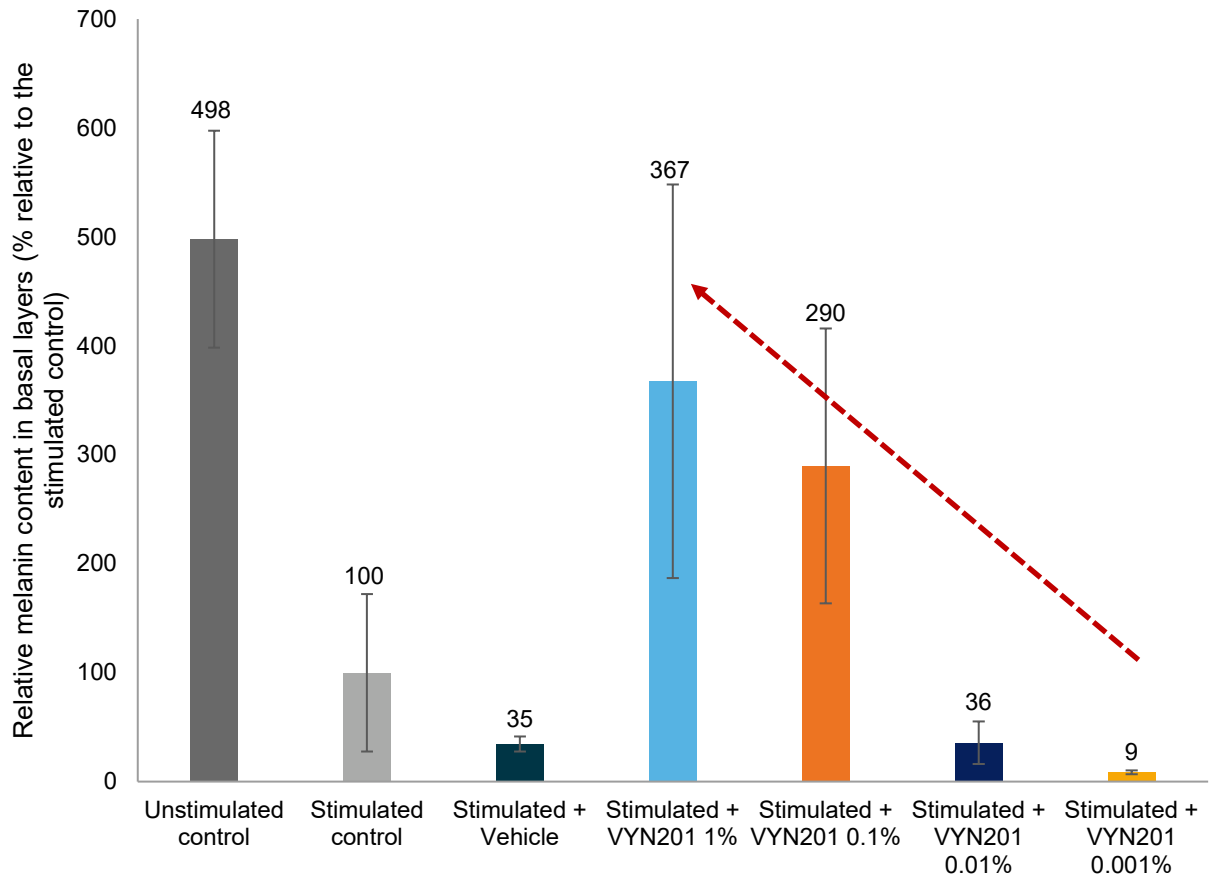
- Stimulated and vehicle treated RHE demonstrated a significant upregulation of MMP9, relative to unstimulated and untreated control
- VYN201 markedly reduced the expression of MMP9 in a dose-dependent manner with a maximal effect at the 1% concentration
- VYN201 1% reduced the secretion of MMP9 by 94.7%, relative to stimulated vehicle and numerically superior to ruxolitinib 1.5%

VYN201: Human Tissue Model of Vitiligo – Reduction of Soluble E-cadherin



- Stimulated and vehicle-treated RHE demonstrated a significant upregulation of soluble E-cadherin, relative to unstimulated control
- VYN201 affects a dose-dependent reduction in solubilized E-cadherin
- VYN201 was numerically superior to topical ruxolitinib cream 1.5%

VYN201: Human Tissue Model of Vitiligo – Effect on Melanocyte Retention

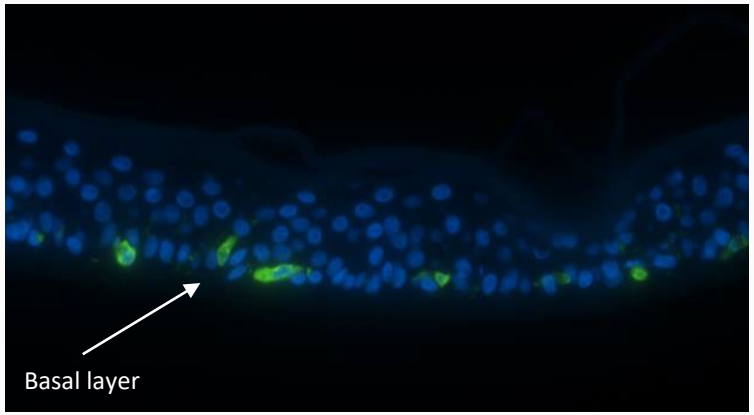


- Stimulated and vehicle-treated RHE demonstrated a significant loss in melanin content, relative to unstimulated control
- VYN201 substantially prevents the loss of melanin pigment in the basal layers of skin in a dose dependent manner
- Residual melanin levels for VYN201 1% was approximately 10-fold higher than vehicle, retaining approximately 75% of melanin relative to unstimulated control

Note: Melanin content in skin cultures treated with ruxolitinib 1.5% cream was immeasurable due to tissue intolerance to treatment

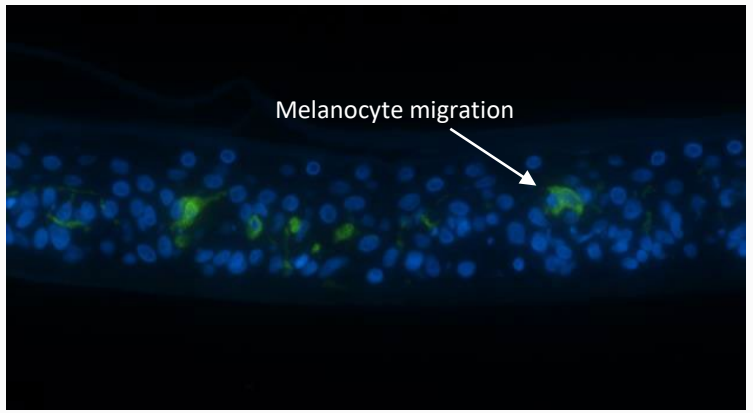
VYN201: Human Tissue Model of Vitiligo - Histology

Micrographic images of TRV immuno-stained induced RHE specimens demonstrating the preservation of melanocytes in the basal layer of samples treated with VYN201 1%



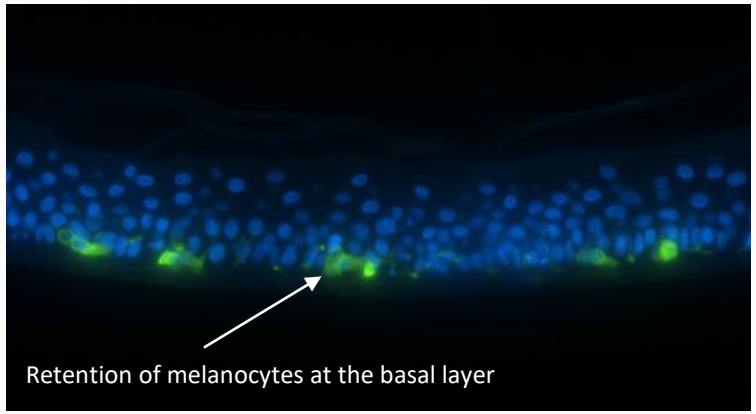
Unstimulated and untreated control

Melanocytes remain at or close to the basal layer implying that E-cadherin adhesion is still functional.



Stimulated and Vehicle treated

Clear evidence of melanocyte adhesion disruption, melanocyte detachment and migration through the skin.



Stimulated and VYN201 1% treated

VYN201 1% prevents detachment and subsequent loss of melanocytes from the basal layer implying that E-cadherin adhesion is still functional.

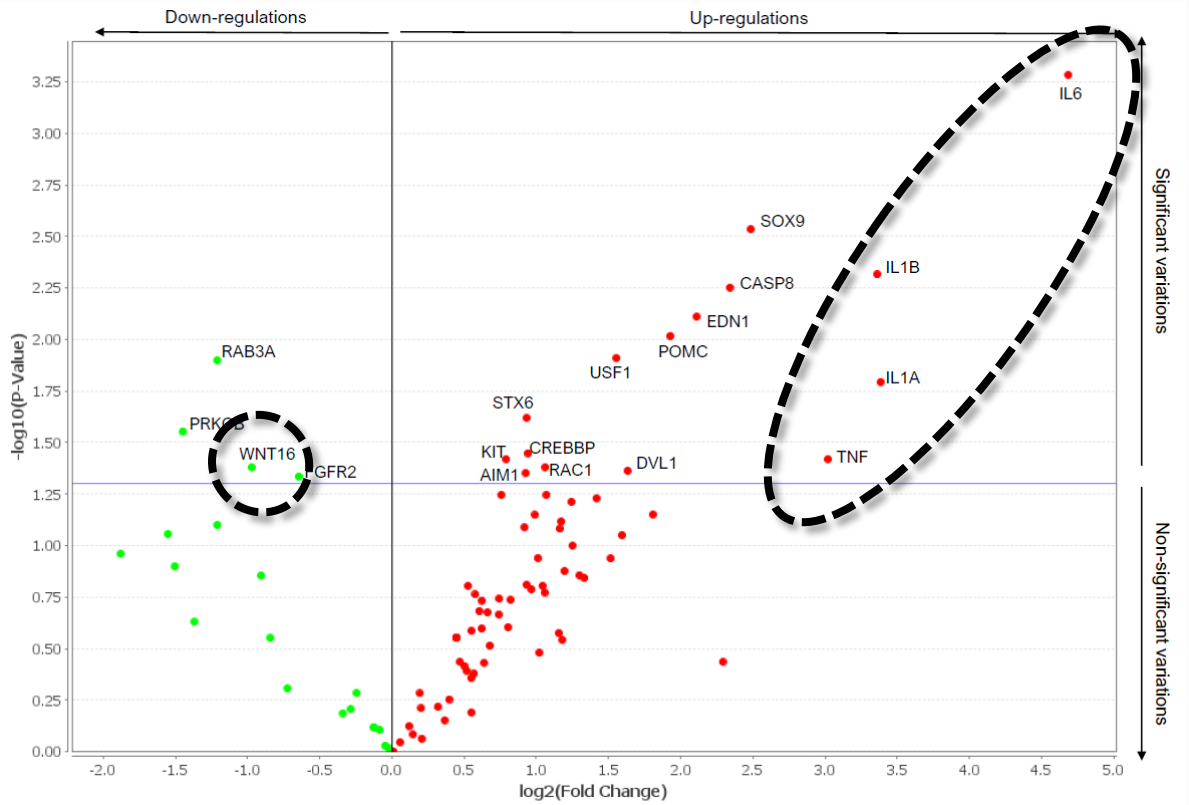
Keratinocytes (blue), melanocytes (green)

TRV: tyrosinase-related protein 1 (important enabler of melanogenesis)

VYN201: Human Tissue Model of Vitiligo – Gene regulation

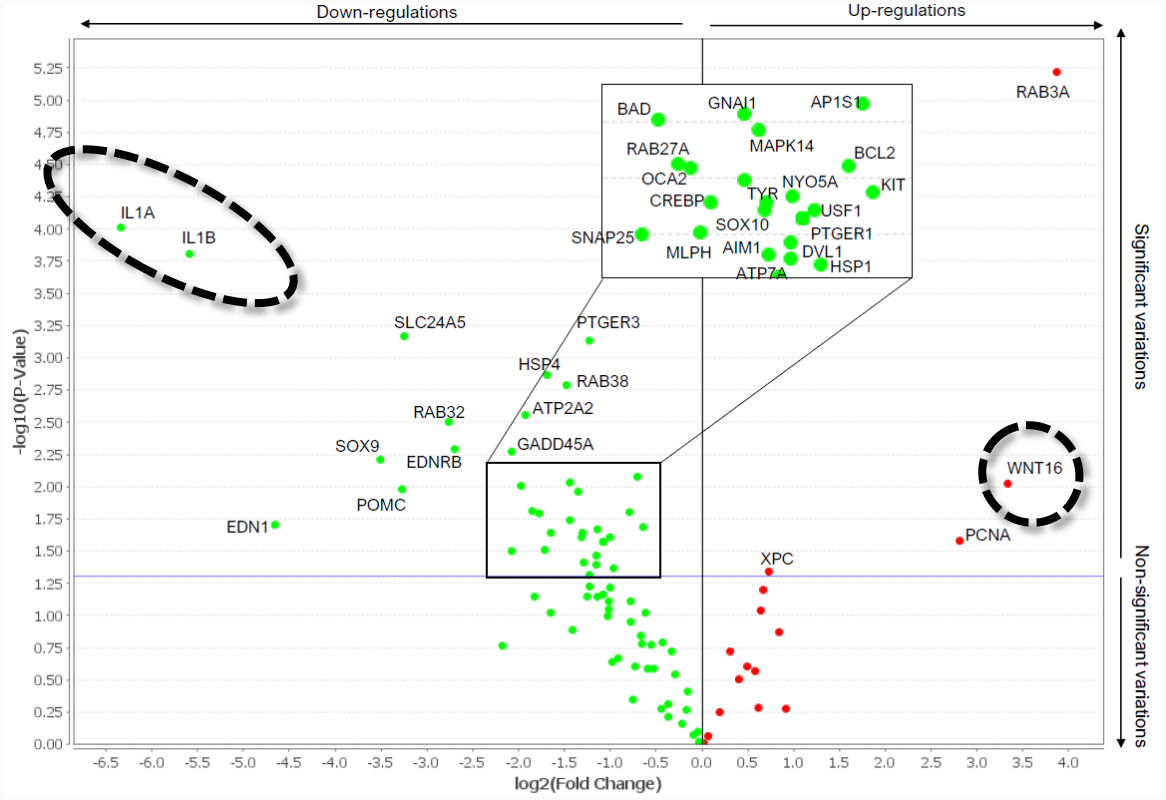
Transcriptome volcano plots of genes relevant to vitiligo (preliminary findings)

Stimulated control



Significant upregulation of cytokines IL6, IL1α and β and TNF

Stimulated and VYN201 1% treated



Significant downregulation of cytokines IL6, IL1α and IL1β and TNF and upregulation of the WNT pathway. IL6 and TNF below LLOQ of assay

- Preliminary data suggests that VYN201 upregulates the WNT pathway. Could be important in proactively supporting melanogenesis.

LLOQ: Lower Limit Of Quantification

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VYN201: First in Human Phase 1 Study Design in Non-Segmental Vitiligo*

Phase 1a/b design comprising of:

Phase 1a Part

A single ascending / multiple ascending dose cohort in healthy volunteer participants for up to 2 weeks of once daily treatment.

- Primary objective is to identify safe starting doses for vitiligo patients in Phase 1b portion of the study.
- Assessments will include TEAEs, pharmacokinetics and local skin tolerance assessments.

Phase 1b Part

Treatment on the face and target lesion(s) on trunk of non-segmental vitiligo patients for 8 weeks of once daily treatment.*

- Dose level(s) will be selected based on findings from the Phase 1a portion of the study.
- Assessments will include TEAEs, pharmacokinetics, local skin tolerance assessments, efficacy (F-VASI), biomarkers (from biopsy), photography.
- Pending additional supporting non-clinical information, dose duration is planned to be extended.

*Pending concurrence with US FDA.

Targeted Clinical Milestones through 2023

Driving Pipeline to Proof-of-Concept

Target	Candidate Selection	Preclinical	Clinical Trials	Near-Term Catalysts
FMX114 **(tofacitinib/fingolimod gel)** Mild-to-moderate Atopic Dermatitis	**Phase 1b/2a**			**Phase 1b: Completed** **Phase 2a: Enrollment Complete; TLR expected end of July/early Aug. 2022**
VYN201 Locally administered Pan-BD BET inhibitor	**Vitiligo (topical administration) IND-enabling studies underway**			**2H 2022: FPI Phase 1 for Vitiligo** **2023: Clinic-ready**
	Undisclosed indication (non-topical administration)			
VYN202 Oral BD2 BET inhibitor[1]	**Candidate Selection process underway**			**2022: Candidate Selection**

Exclusive Access to Library of NCE BET Inhibitors for Any Indication Worldwide

1. Initial indication for VYN202 to be communicated following candidate selection, exercise of option and completion of requisite pre-clinical evaluations

TLR = Top Line Results; FPI = First Patient In/Enrolled

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Thank you!

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