Technology Transfer Brief

KOREA UNIVERSITY SEJONG CAMPUS

A Heterocyclic (Oxazole & Oxadiazole) Based Compound Library Platform for CNS Drug Discovery and Subtype-Specific Pharmaceutical Development

Medicinal Chemistry Laboratory

College of Pharmacy, Korea University (Sejong Campus)

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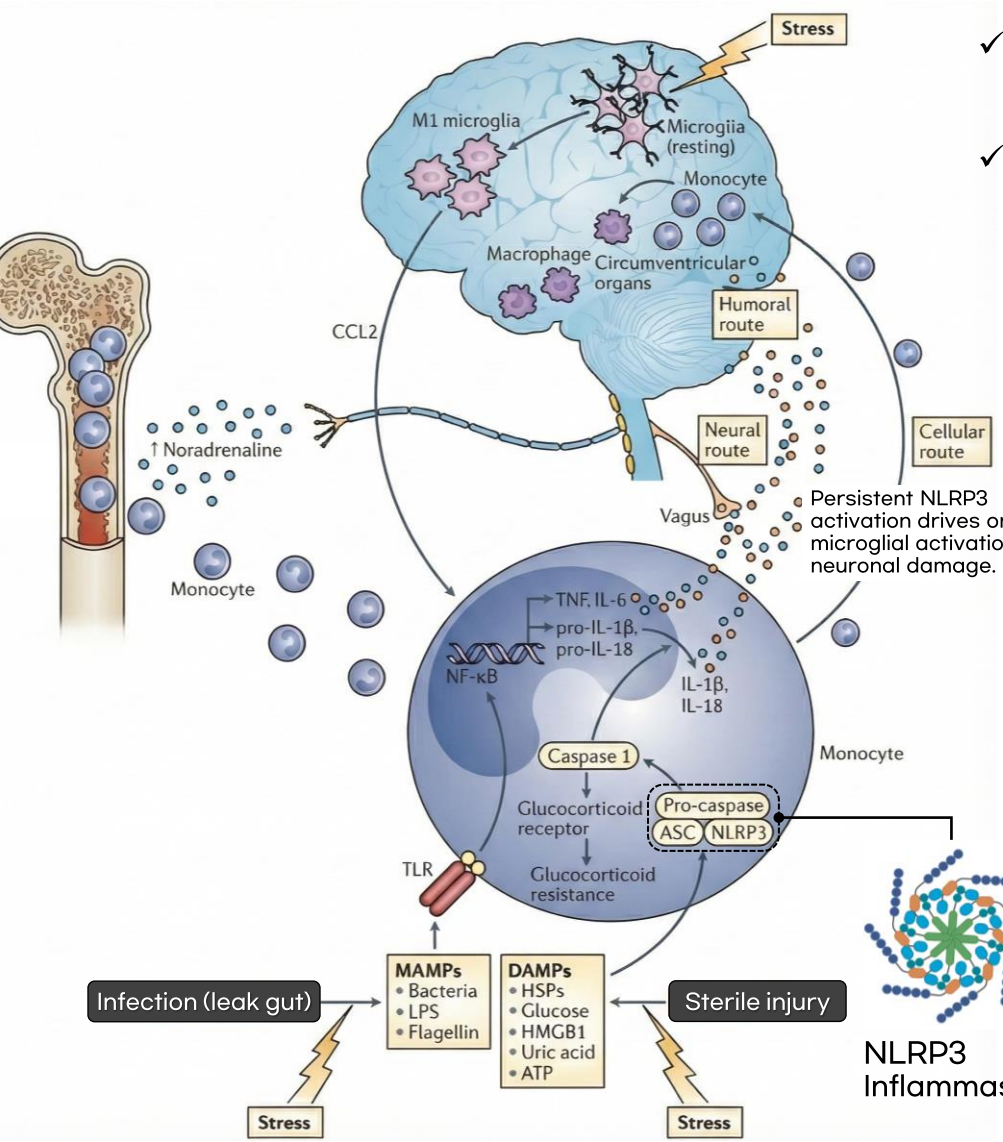
Technology Transfer Brief

A Heterocyclic (Oxazole & Oxadiazole) Based Compound Library Platform for CNS Drug Discovery and Subtype-Specific Pharmaceutical Development

Content

01 CNS Progression Is a Neuroinflammation Problem



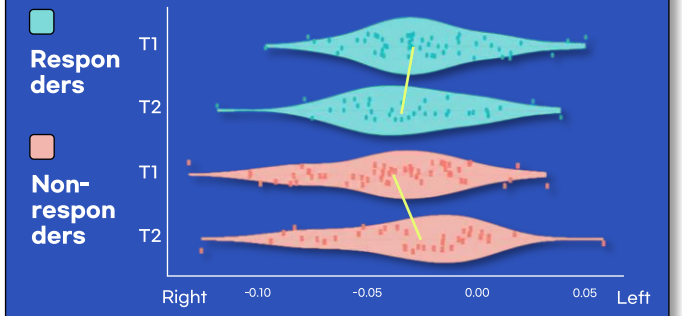
(*Source: Miller & Raison, NATURE REVIEWS IMMUNOLOGY, 2016)

✓ **NLRP3 inflammasome : key driver of neuroinflammation in CNS disorders.**
(*Depression, Parkinson's disease, Alzheimer's disease)

✓ **Need for NLRP3-targeted therapies in chronic neuroinflammatory conditions.**

- Symptom-focused drugs (e.g., SSRIs) fail to address underlying neuroinflammation.
- Urgent need for NLRP3-targeted disease-modifying therapies (DMTs) and effective combination strategies.

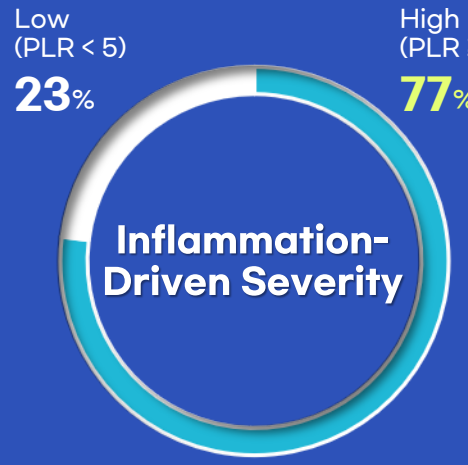
Structural Brain Recovery Fails in SSRI Non-Responders



Total Hippocampus Laterality Index

- Structural recovery after SSRI treatment is limited to responders, with persistent structural pathology in non-responders.
- Limitations of symptom-focused antidepressant therapy.

Inflammatory Burden in Moderate to Severe Depression



- High inflammatory burden in patients with severe depression.
- Need for combination therapeutic strategies in severe cases.

02 Technology Overview

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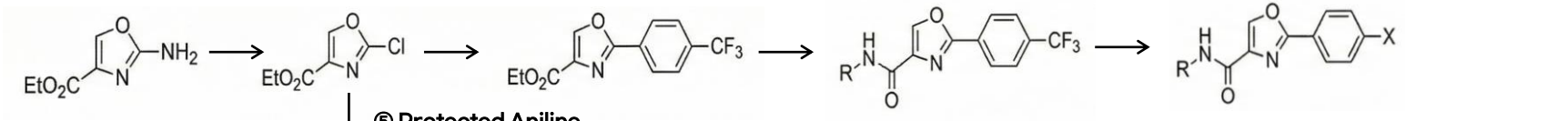
① Oxazole Core Activation

② Aryl Coupling to Oxazole Core

③ Ester-to-Acid Conversion

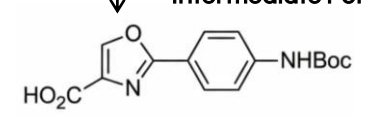
④ Amide Bond Formation



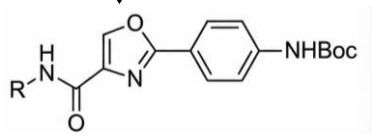
a. R=Cyclohexyl (KB2784)

b. R=Cyclopropylmethyl, X=SCH_3 (KB2790)
c. R=Cyclohexyl, X=$C(CH_3)_2$ (KB4048)
d. R=Cyclohexyl, X=$CH(CH_3)_2$ (KB4055)

⑤ Protected Aniline Intermediate Formation

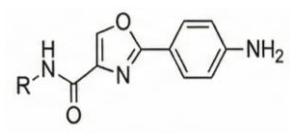


⑥ Deprotection to Aniline

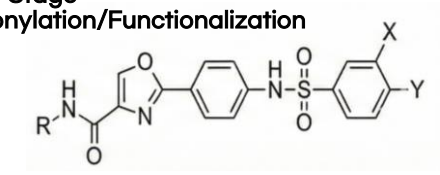


e. R=Cyclopentyl (KB4026)

⑦ Aryl Substituent Diversification (X-modulation)

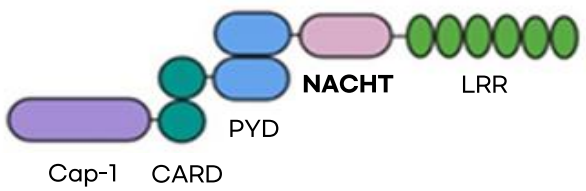


⑧ Late-Stage Sulfonylation/Functionalization

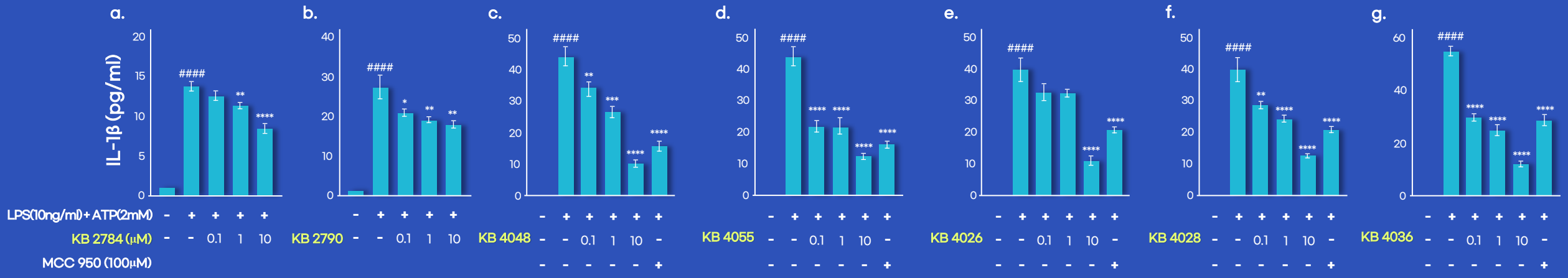


f. R=Cyclopentyl, X=H, Y=CH_3 (KB4028)
g. R=Cyclohexyl, X=H, Y=Cl (KB4036)

- ✓ Modular synthesis enables diverse **oxazole-based compounds**.
- ✓ **Dose-dependent inhibition** of IL-1β release is observed in an LPS/ATP-induced NLRP3 inflammasome model.



The inhibitory effect is mediated through interference with NACHT-dependent NLRP3 activation.



03 Key Features & Advantages

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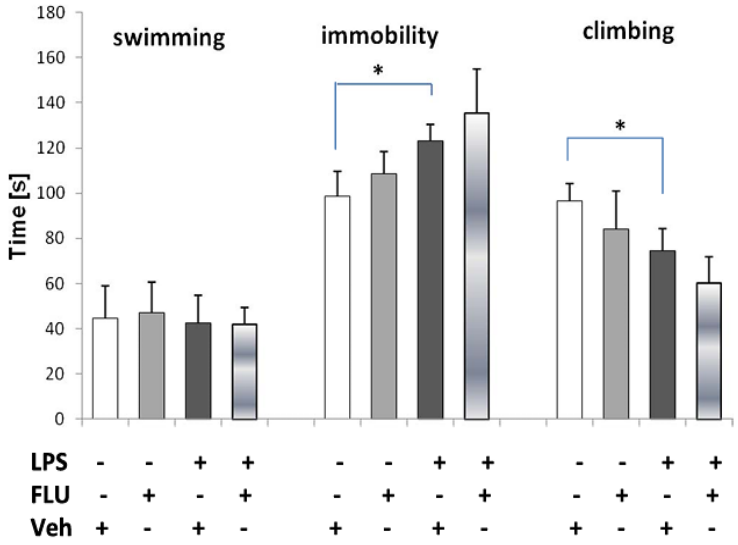
✓ **Inflammation-driven depression correction :** NLRP3 inflammasome modulation to suppress neuroinflammatory states.

✓ **Functional impact :** ① **IL-1β ↓**, ② **Microglia activation ↓**, ③ **Inflammation-induced depressive behaviors attenuated**

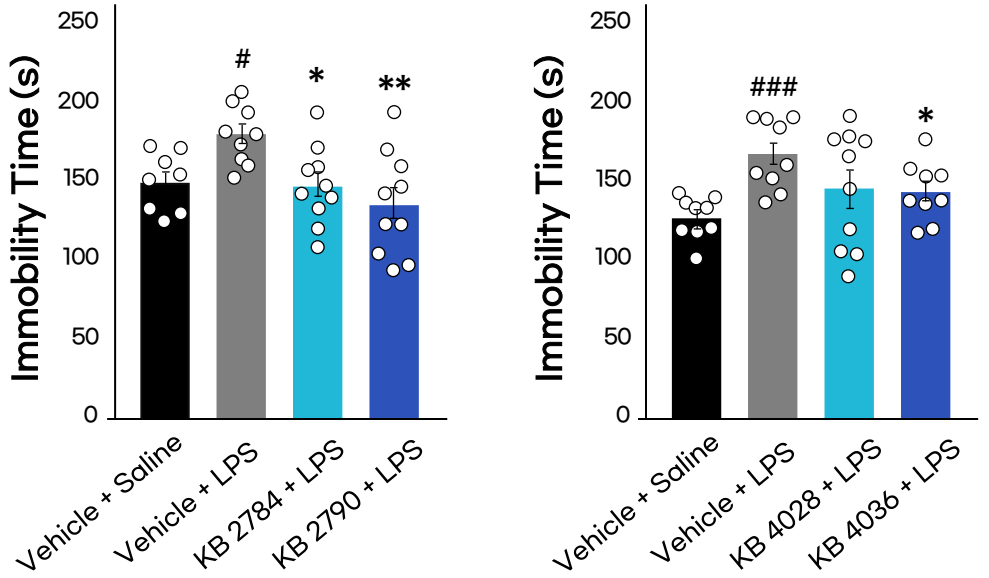


(*Source: W. Duda *et al.*, International Immunopharmacology, 2017)

- Selective serotonin reuptake inhibitors (**SSRIs**) target **monoaminergic pathways**, not inflammation-driven disease mechanisms..

  (e.g., fluoxetine, citalopram, paroxetine, sertraline)

- SSRIs fail to reverse LPS-induced immobility in inflammatory depression models.



✓ **Oxazole and oxadiazole-based compounds** (KB series) reduces **LPS-induced immobility**, indicating **antidepressant-like activity** in an inflammatory depression model.

✓ Consistent effects across multiple KB compounds support a **class-level effect** rather than an isolated hit.

① Mechanistic Mismatch

- LPS-induced depression is driven by neuroinflammation (NLRP3-IL-1β), not monoamine deficiency.
- Monoamine-targeting SSRIs show limited efficacy in this model.

② Inflammation-Induced Serotonergic Suppression

- Pro-inflammatory cytokines impair serotonin synthesis and receptor signaling.
- Increased serotonin availability does not translate into functional antidepressant effects.

③ Microglia-Driven Synaptic Dysfunction

- Activated microglia induce synaptic pruning and circuit dysfunction.
- Such circuit-level deficits are not corrected by SSRIs.

04 Strategic Business Opportunities

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- ✓ **Business Vision :** A platform-driven approach to CNS drug discovery, enabling subtype-specific pharmaceutical development through an oxazole/oxadiazole-based compound library.
- ✓ **Engagement Model :** Open to technology licensing and collaborative R&D to expand and validate CNS-focused chemical assets.

Collaborative development opportunities

❶ Compound library expansion

- Systematic expansion of an oxazole-based CNS compound library to define CNS-relevant chemical space and SAR.
- Establishing class-level pharmacological profiles beyond single lead development.

❷ CNS subtype-driven efficacy and mechanism validation

- Evaluation of oxazole analog series across CNS subtype-specific disease models.
- Linking pharmacological activity with disease-relevant phenotypes to de-risk target relevance..

❸ Translational efficacy and biomarker framework development

- Development of exploratory translational efficacy and biomarker readouts applicable across compound series.
- Providing a standardized foundation for CNS co-development programs.

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Medicinal Chemistry Lab
(College of Pharmacy)

Collaborative R&D

Subtype-driven preclinical co-development

IP license (Option/Exclusive)

Oxazole/oxadiazole-based CNS Compound Library

- Pharma (CNS Discovery)
- Biotech (CNS-focused)
- Discovery CRO / Research Platforms

Commercial License

Pharmaceuticals

- Pharma (CNS Development)
- Biopharma with IND capability
- Strategic licensing partners

Intellectual Property

- Title : NOVEL OXAZOLE-BASED COMPOUND AND COMPOSITION FOR PREVENTING, ALLEVIATING, OR TREATING INFLAMMATORY DISEASES COMPRISING SAME
- Patent : PCT/KR2024/017980, KR10-2024-0161913 (Date : Nov. 14, 2024)

CONTACT US

For inquiries regarding technology licensing, collaborative R&D, or partnership opportunities, please contact :

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- Email : **kimth@ibubiz.kr**

Partnering to unlock new business opportunities through innovation.

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