TECHNICAL BULLETIN

Zinpro® Zinc Lysine Glutamate Enhances Cellular Zinc Uptake Under Conditions That Limit Traditional Zinc Transport

Introduction

Zinc (Zn) uptake into intestinal cells depends on specific transport mechanisms within the intestinal epithelium. When these transport mechanisms are impaired, intracellular Zn delivery from conventional inorganic Zn sources may be reduced. This study evaluated whether Zinpro Zn lysine glutamate (ZnLG) could maintain intracellular Zn uptake under conditions where ZIP4-mediated Zn transport is compromised.

Study Objective

To compare intracellular Zn uptake from ZnLG and zinc chloride ($ZnCl_2$) under conditions where ZIP4-mediated Zn transport is impaired.

Experimental Models

- Caco-2 intestinal cell model
- Human enterocytes from a person with acrodermatitis enteropathica (AE)
- ZIP4-deficient enterocytes (impaired Zn transport model)

Zinc Forms Evaluated

- **$ZnCl_2$**: inorganic zinc chloride
- **Zinpro ZnLG**: zinc lysine glutamate

Zinc treatments delivered Zn at equivalent

Why ZIP4 Matters

ZIP4 is a primary transporter involved in intestinal Zn uptake. Reduced ZIP4 function can impair Zn absorption, making ZIP4-deficient cellular models useful for evaluating alternative Zn delivery mechanisms.

Key Findings

ZnLG Maintains Zn Delivery When ZIP4-Mediated Transport Is Impaired

ZnLG maintained intracellular Zn levels when ZIP4-mediated transport was impaired (Fig. 1 and 2). While $ZnCl_2$ uptake was reduced in ZIP4-deficient enterocytes, ZnLG continued to support intracellular Zn delivery.

- $ZnCl_2$ failed to significantly increase intracellular Zn in ZIP4-deficient enterocytes (Fig. 1)
- ZnLG continued to increase intracellular Zn levels despite impaired ZIP4-mediated transport (Fig. 1 and 2)
- ZnLG maintained Zn delivery more effectively than $ZnCl_2$ when ZIP4 function was impaired (Fig. 2)

⇒ **Results suggest ZnLG relies less heavily on ZIP4-mediated transport than $ZnCl_2$.**

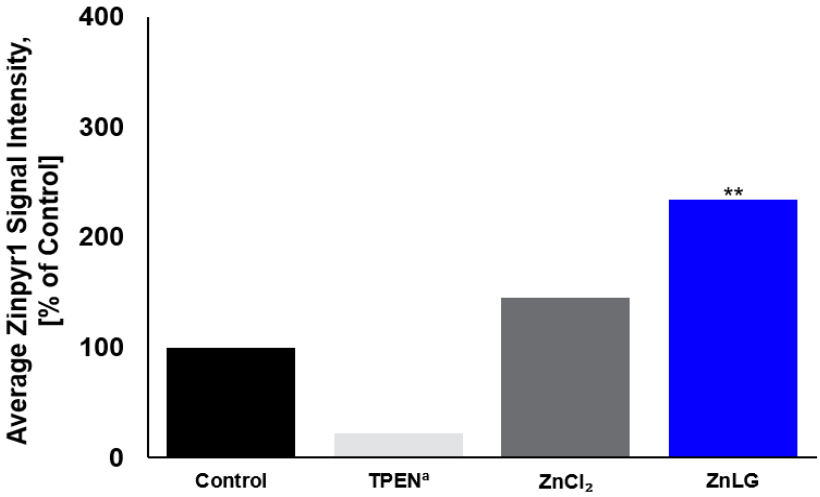


Figure 1: Cellular Zn Uptake in AE (ZIP4-deficient) Cells

ZnLG mitigates reduced Zn uptake in ZIP4-deficient human enterocytes.

[a]TPEN = Zn chelator used to reduce intracellular Zn levels and serve as Zn-depleted Control condition

** $P \leq 0.01$

Data extrapolated from chart in publication

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Why Use an AE Model?

Acrodermatitis enteropathica (AE) is a rare genetic disorder caused by impaired ZIP4-mediated zinc transport. AE-derived enterocytes provide a model for studying Zn uptake when conventional Zn transport pathways are compromised.

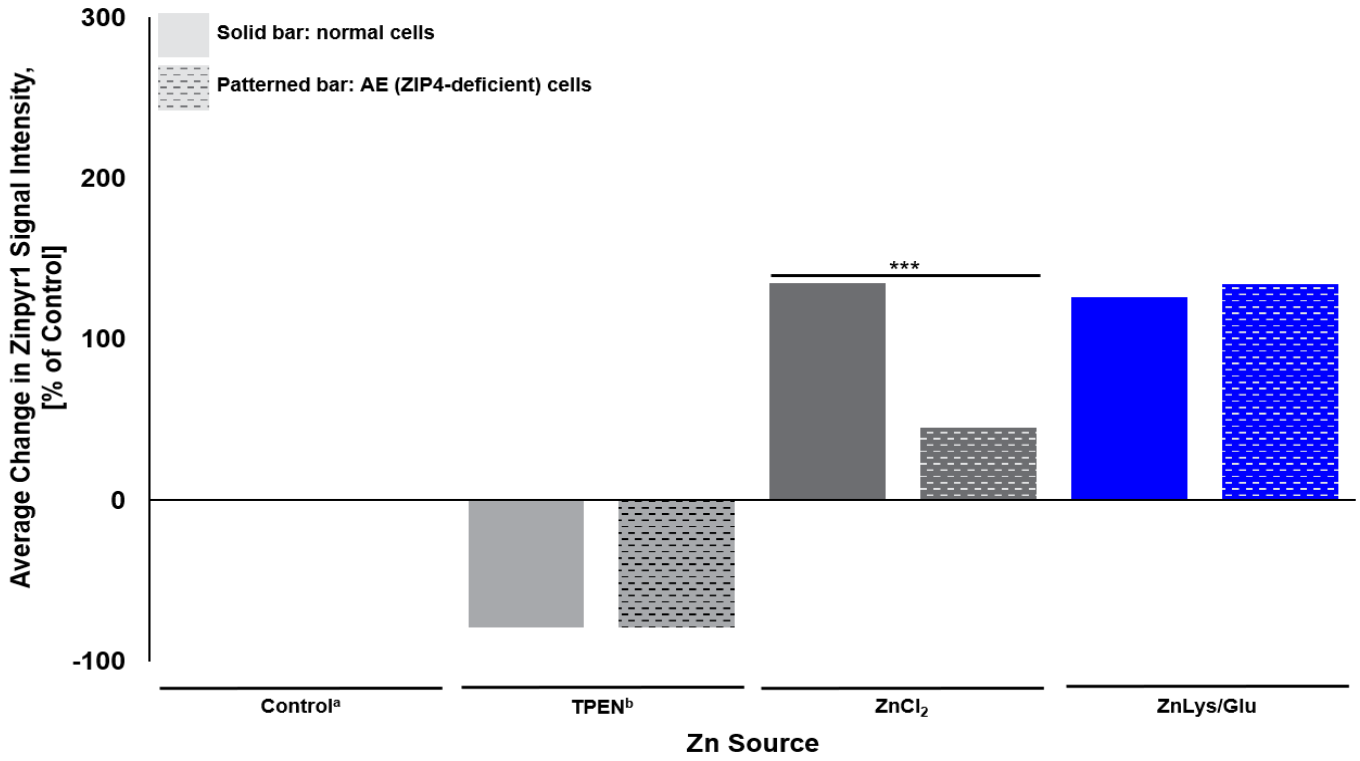


Figure 2: Comparative Zn Uptake in Normal and AE (ZIP4-deficient) Cells

ZnLG maintains intracellular Zn uptake more effectively than $ZnCl_2$ when ZIP4-mediated transport is impaired.

[a]Control represents the reference treatment (0% change); All values are expressed relative to Control

[b]TPEN = Zn chelator used to reduce intracellular Zn levels and serve as Zn-depleted Control condition

*** $P \leq 0.001$

Data extrapolated from charts in publication

Conclusion & Implication

ZnLG maintained intracellular Zn delivery in ZIP4-deficient cellular models where $ZnCl_2$ was less effective. These findings suggest that ZnLG may support Zn uptake under conditions that impair conventional Zn transport pathways.

ZnLG supported intracellular Zn delivery when ZIP4-mediated transport was impaired, demonstrating a more resilient approach to Zn delivery than $ZnCl_2$ in these cellular models.

Source: Sauer et al., 2017. Biometals. 30:643-661 **doi:**10.1007/s10534-017-0033-y

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