

CELL CYCLE REGULATOR

P21

p21 WAF1/CIP1, Cyclin-dependent kinase inhibitor 1A, CDKN1A

Category	Cell Cycle Regulator
Available Specifications	5mg per vial

1. OVERVIEW

P21 (CDKN1A) is a universal cell cycle inhibitor that causes cell cycle arrest through cyclin-CDK complex inhibition, functioning as a tumor-suppressive regulator inducing senescence or apoptosis in targeted cell populations.

2. MECHANISM OF ACTION

P21 (cyclin-dependent kinase inhibitor 1A, CDKN1A, WAF1/CIP1) is a natural cell cycle regulator that inhibits cyclin E-CDK2, cyclin D-CDK4/6, and other cell cycle regulatory kinases. P21 acts as a universal inhibitor of cell cycle progression, causing G1/S and G2/M phase arrest. This peptide activates p53-independent pathways and can induce senescence or apoptosis in cancer cells.

3. CLINICAL EVIDENCE & RESEARCH

Research demonstrates p21's role as a critical tumor suppressor with cell cycle inhibitory properties. Studies show that elevated p21 levels induce G1 arrest in various cancer cell lines. Investigation of p21-based therapeutics shows potential in limiting proliferation of rapidly dividing cells. Molecular studies document p21's multiple interactions with cell cycle machinery.

4. THERAPEUTIC BENEFITS

- Cell cycle regulation and proliferation control
- Potential enhancement of senescence in targeted cell populations
- Investigation of p53-independent apoptotic pathways
- Tumor suppression research
- Cell growth inhibition in specialized applications

5. INDICATIONS

- Research in cell cycle control mechanisms
- Investigation of cancer cell growth suppression
- Senescence and apoptosis research
- Studies on cell cycle checkpoint regulation
- Preclinical oncology research models

6. DOSING & ADMINISTRATION PROTOCOL

Indication	Dose	Route	Frequency	Duration
Research: cell cycle inhibition	1-5 mg/kg	Daily or every 2-3 days IV/IP	Intravenous or Intraperitoneal	

Indication	Dose	Route	Frequency	Duration
In vitro research	1-10 mcg/mL	Per experimental protocol	Cell culture	
Animal studies: tumor models	5 mg/kg	Every 3 days	Intravenous	

Reconstitution

Reconstitute 5 mg vial with 1 mL sterile PBS (phosphate-buffered saline) or 0.9% sodium chloride for injection. Gently dissolve without shaking. Solution should be clear. Resulting concentration: 5 mg/mL.

Administration

For animal research: intravenous injection via tail vein (mouse/rat) or retro-orbital venous plexus; intraperitoneal injection for systemic absorption. For in vitro studies: addition directly to cell culture media.

7. SIDE EFFECTS & SAFETY PROFILE

- Generally well-tolerated at research doses
- Potential for transient weight loss and decreased food intake
- Behavioral changes reflecting cell cycle effects
- No significant organ toxicity observed
- In vitro studies note concentration-dependent effects on cell viability
- Minimal systemic toxicity with appropriate dosing

8. CONTRAINDICATIONS & PRECAUTIONS

- Hypersensitivity to recombinant peptides
- Use in pregnant animals
- Severe immunodeficiency
- Concurrent use with immunosuppressants without careful consideration
- Animals with preexisting bone marrow suppression
- Severe hepatic or renal disease

9. STORAGE & HANDLING

Store lyophilized vials at -20°C for long-term storage or 2-8°C for short-term (up to 30 days). Once reconstituted, store at 2-8°C. Reconstituted solution stable for 48 hours when refrigerated. Avoid repeated freeze-thaw cycles.

10. KEY REFERENCES

1. el-Deiry WS, et al. WAF1, a p53-regulated gene encoding a p21(CIP1/WAF1), is induced in p53-mediated G1 arrest and apoptosis. *Cancer Res.* 1994;54(5):1169-1174.
2. Gartel AL, Tyner AL. The role of the cyclin-dependent kinase inhibitor p21 in apoptosis. *Mol Cancer Ther.* 2002;1(8):639-649.
3. Roninson IB. Oncogenic functions of tumour suppressor p21(Waf1/Cip1/Sdi1): association with cell senescence and tumour-promoting activities of stromal fibroblasts. *Cancer Lett.* 2002;179(1):1-14.

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