

Cellular Adaptations in Response to Stress

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Study guide – concise, organized, and ready for review

1. Why Cells Adapt

- **Homeostasis:** Cells must maintain internal stability despite external fluctuations.
 - **Survival:** Adaptations allow cells to survive, function, and proliferate under adverse conditions.
 - **Types of stress:** Physical (temperature, radiation), chemical (toxins, pH), biological (infection, inflammation), and metabolic (nutrient deprivation, hypoxia).
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2. Major Adaptive Strategies

Adaptation	Definition	Typical Triggers	Key Molecular Players	Outcome
Atrophy	Reduction in cell size, organ mass, and functional capacity	Disuse, denervation, decreased workload, loss of endocrine stimulation, aging	? protein synthesis, ? protein degradation (ubiquitin? proteasome, autophagy)	Conserves energy; reversible if stimulus removed
		Increased workload (e.g., muscle exercise, cardiac pressure overload), hormonal stimulation (e.g., estrogen)	? protein synthesis (mTOR, Akt), sarcomere addition (cardiac), ribosome biogenesis	
Hypertrophy	Increase in cell size (often with enhanced functional output)			Improves contractile force; may become maladaptive (e.g., heart failure)

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Hyperplasia	Rise in cell number via proliferation	Hormonal stimulation (estrogen ? endometrium), compensatory growth after loss (liver regeneration), chronic irritation (skin)	Growth factors (EGF, PDGF), cyclins/CDKs, MAPK/ERK pathway	Restores tissue mass; can be physiological or pathological (e.g., neoplasia)
Metaplasia	Reversible replacement of one differentiated cell type by another better suited to the new environment	Chronic irritation, inflammation, hormonal changes (e.g., smoking ? bronchial epithelium)	Transcription factors (SOX2, p63), stem/progenitor cell activation, BMP/Wnt signaling	Protective short-term; risk of dysplasia/carcinoma if stimulus persists
Dysplasia	Disordered growth with loss of normal orientation and maturation	Persistent irritants, carcinogens, chronic inflammation	Mutations in tumor ? suppressor genes (p53), altered cell cycle control, epigenetic changes	Premalignant lesion; may revert or progress to carcinoma
Anaplasia	Loss of differentiation; cells become primitive and pleomorphic	Advanced malignancy	Oncogene activation, loss of differentiation markers, genomic instability	Indicator of high-grade cancer; irreversible

3. Cellular Mechanisms Underlying Adaptation

3.1 Protein Turnover

- **Synthesis:** mTORC1 activation ? ribosome biogenesis, translation initiation.

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- **Degradation:**

- **Ubiquitin–Proteasome System (UPS):** Targets short-lived regulatory proteins.
- **Autophagy–Lysosome Pathway:** Engulfs organelles & protein aggregates, crucial for atrophy and stress clearance.

3.2 Signal Transduction Pathways

Pathway	Primary Stimuli	Main Effectors	Adaptive Result
PI3K/Akt/mTOR	Growth factors, nutrients	Akt, mTORC1, S6K	Promotes hypertrophy & hyperplasia (protein synthesis, cell growth)
AMPK	Low ATP/energy stress	AMPK?, TSC2	Inhibits mTOR, stimulates autophagy ? protects against energy depletion
**MAPK/ERK			