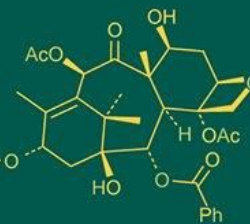
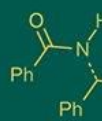
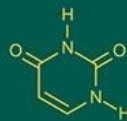
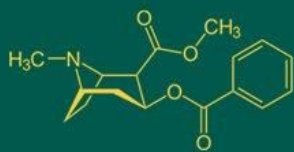


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Bacterial moonlighting proteins and their role in virulence: A review

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Abstract

Bacterial moonlighting proteins are multifunctional molecules that perform distinct physiological roles beyond their classical enzymatic or structural functions. These proteins challenge the traditional “one gene, one protein, one function” paradigm by exhibiting multiple, unrelated activities dependent on factors such as sub cellular localization, environmental cues, and protein interactions. Predominantly found among Glycolytic enzymes, metabolic enzymes, and molecular chaperones, bacterial moonlighting proteins contribute significantly to virulence by mediating adhesion, immune modulation, tissue invasion, and biofilm formation. Surface localization enables these proteins to bind host extracellular matrix components like fibronectin and plasminogen, facilitating colonization and penetration of host tissues. Additionally, they modulate host immune responses to promote bacterial persistence. Despite their conserved metabolic roles, these proteins possess structural and regulatory plasticity allowing functional switching in response to environmental stimuli. Their central involvement in bacterial pathogenicity positions them as promising targets for novel antimicrobial therapies. However, challenges such as functional diversity across species, homology with host proteins, and bacterial compensatory mechanisms complicate therapeutic development. A deeper understanding of the molecular mechanisms, regulation, and structural features of bacterial moonlighting proteins is essential to exploit their vulnerabilities effectively, offering potential pathways for innovative treatments against antibiotic-resistant infections.

Keywords: Moonlighting Proteins, Virulence, Glycolytic enzymes, Protein multifunctionality

1. Introduction

The traditional view of molecular biology, epitomized by Francis Crick’s Central Dogma, posits a linear flow of genetic information from DNA to RNA to protein, with an implicit assumption of “one gene, one protein, one function.” For decades, this dogma underpinned our understanding of cellular processes, protein specialization, and genetic regulation. However, accumulating evidence has challenged this paradigm, revealing that many proteins can perform more than a single, canonical function—a phenomenon referred to as “moonlighting.” The conceptual breakthrough came in 1988 when Joram Piatigorsky and colleagues observed that the duck lens crystalline protein was identical to the metabolic enzyme argininosuccinate lyase, introducing the notion of “gene sharing” (Piatigorsky *et al.*, 1988) [26].

Subsequently, the term “moonlighting” was appropriated from its colloquial meaning—holding a second job at night—to describe proteins that perform multiple, distinct functions, often in different cellular contexts or compartments. This concept has been further refined and popularized by Constance Jeffery, who has delineated the characteristics and boundaries of moonlighting proteins, distinguishing them from bifunctional or catalytically promiscuous enzymes (Jeffery, 1999; Jeffery, 2014) [13, 14]. The recognition of protein moonlighting has profound implications for our understanding of cellular complexity, evolution, and, notably, pathogen virulence.

Bacteria, in particular, have emerged as prolific exploiters of moonlighting proteins, co-opting essential metabolic enzymes and molecular chaperones for roles in adhesion, immune modulation, and tissue invasion. This capacity to “multitask” not only enhances bacterial fitness but also complicates the design of targeted therapies.

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This review critically examines the phenomenon of bacterial moonlighting proteins, focusing on their classification, mechanisms, and multifaceted contributions to virulence.

2. Defining Moonlighting Proteins

Protein moonlighting is defined as the ability of a single polypeptide to perform more than one physiologically relevant function, independent of gene fusions, alternative splicing, or covalent modifications (Jeffery, 2014) ^[14]. Jeffery (1999) ^[13] further clarifies that moonlighting functions must be biochemically and functionally distinct, and not merely variations of a single catalytic activity (e.g., catalyzing similar reactions with different substrates). Thus, moonlighting proteins are distinguished from bifunctional enzymes (which possess separate catalytic domains) and catalytically promiscuous enzymes (which catalyze multiple reactions at a single active site) (Moore *et al.*, 2004) ^[22].

2.1 Classification and Types of Enzymes

Moore *et al.* (2004) ^[22] classify enzymes based on the organization and multiplicity of functional sites:

- **Conventional enzymes:** Single catalytic domain, single function.
- **Catalytically promiscuous enzymes:** Single catalytic domain, multiple substrates but mechanistically similar reactions.
- **Bifunctional enzymes:** Two distinct catalytic domains, each with a separate function.

- **Moonlighting enzymes:** Single catalytic domain with an additional, structurally distinct functional domain, enabling a second, unrelated biological activity.

A defining feature of many moonlighting proteins is their “geographical” plasticity; their function often depends on sub cellular localization or environmental context (Barberis, 2021) ^[3]. For example, a protein may serve as a metabolic enzyme in the cytoplasm but act as an adhesin or signaling molecule when present on the cell surface.

2.2 Historical Context and Evolutionary Distribution

The existence of moonlighting proteins was first substantiated in 1988 when Piatigorsky *et al.* demonstrated that the duck lens crystalline protein, previously thought to be lens-specific, was identical to the metabolic enzyme argininosuccinate lyase (Piatigorsky *et al.*, 1988) ^[26]. While the term “moonlighting” was initially used by Hoffman-La-Roche to describe neuropeptides with unexpected immunological activities (Campbell & Scanes, 1995) ^[4], it was Jeffery (1999) ^[13] who provided a robust framework for the concept and its implications.

Moonlighting proteins are ubiquitous, found across all domains of life—animals, plants, fungi, protists, and prokaryotes (Arvizu-Rubio *et al.*, 2022) ^[1]. Notably, their prevalence in bacteria has garnered special attention due to their roles in pathogenesis and host-microbe interactions (Henderson, 2014) ^[8].

Table 1: Examples of Moonlighting Proteins across Kingdoms

Protein	Kingdoms	Functions
Aconitase	Animal- Human	- TCA cycle enzyme - Iron homeostasis
Presenillin	Plant- Physcomitrella patens	- γ - secretes - Cytoskeleton function
Aldolase	Yeast- Sacromyces cervices	- Glycolytic enzyme - V- ATPase assembly
Murl	Prokaryotes- Mycobacterium tuberculosis	- Glutamate recemase - DNA gyrase inhibitor

(Arvizu-Rubio *et al.*, 2022) ^[1]

2.3 Mechanisms and Regulation of Protein Moonlighting

Moonlighting functions typically arise from structural features that allow the protein to interact with distinct partners or undergo conformational changes. Regulatory mechanisms enabling functional switching include:

- **Sub cellular localization:** Translocation to the cell surface or secretion into the extracellular milieu exposes the protein to novel interaction partners (Barberis, 2021) ^[3].
- **Post-translational modifications:** Phosphorylation, acetylation, or redox changes can modulate binding affinities or functional outputs.
- **Ligand availability:** Variations in substrate, cofactor, or partner protein concentrations can trigger alternative activities.
- **Protein-protein interactions:** Assembly into different complexes can reveal or mask functional domains.
- **Environmental cues:** Changes in temperature, pH, or ionic strength can induce conformational shifts that favor moonlighting activities (Henderson & Martin, 2011) ^[9].

3. Moonlighting Proteins in Bacteria

3.1 Prevalence and Diversity

To date, moonlighting proteins have been identified in over 90 bacterial species, encompassing both pathogenic and commensal strains (Henderson, 2014) ^[8]. These proteins are not limited to one class or function; enzymes, cell surface receptors, ion channels, and chaperonin proteins can all function as moonlighters (Henderson & Martin, 2011) ^[9]. Within a single bacterial species, multiple moonlighting proteins may coexist, and conversely, a given moonlighting function can be performed by distinct proteins in different organisms.

3.2 Regulation of Moonlighting in Bacteria

Bacterial moonlighting proteins typically switch between functions via:

- Translocation to the cell surface or extracellular space
- Interaction with DNA or RNA
- Responsive changes to ligand or cofactor concentration
- Integration into distinct protein complexes or membrane associations
- Environmental stress or signaling cues

These regulatory strategies allow bacteria to rapidly adapt to changing environments or host defenses, conferring a significant evolutionary advantage.

3.3 Role of Moonlighting Proteins in Bacterial Virulence:

The capacity of moonlighting proteins to serve dual roles-supporting essential metabolism while also mediating virulence-makes them especially significant in pathogenic bacteria (Henderson & Martin, 2011) [9]. Their most common moonlighting virulence functions include adhesion to host tissues, modulation of immune cell activity, and facilitation of tissue invasion.

3.4 Types of Moonlighting Protein and their role in Virulence

3.4.1 Glycolytic Enzymes: Surface-Associated Glycolytic Enzymes

Glycolytic enzymes, which are universally conserved and essential for energy metabolism, are frequently found on the bacterial cell surface, where they acquire new, non-metabolic roles (Fothergill-Gilmore & Michels, 1993) [6]. Despite the lack of canonical secretion signals, these enzymes appear on the cell surface, possibly via lysis of dead bacteria or yet-unknown export mechanisms (Kim & Dang, 2005) [17]. Once surface-exposed, they function as adhesins, receptors, or modulators of host immunity.

Table 2: The moonlighting actions of bacterial glycolytic enzymes

Glycolytic enzyme	Moonlighting functions	Reference
Hexokinase	<i>E. coli</i> K1 Fim A promotes binding of VDAC1 to mitochondria that blocking cellular apoptosis	Soshan-Bermat <i>et al.</i> , 2009
Phosphofructokinase	Part of a RNA degradosome complex	Commichauet <i>et al.</i> , 2009
Aldolase	In <i>S. pneumoniae</i> a cell surface lactin which binds host cadherin receptor termed flamingo	Ling <i>et al.</i> , 2004
Triose phosphate isomerase	<i>S. aureus</i> enzyme act as adhesion for the fungus <i>C. neoformans</i> .	Ikeda <i>et al.</i> , 2007
Phosphoglycerate kinase	Plasminogen binding protein in oral streptococci.	Kinbby <i>et al.</i> , 2008
Phosphoglycerate mutase	Streptococcal cell surface plasminogen binding protein	Kinbby <i>et al.</i> , 2008
Pyruvate kinase	<i>L. lactis</i> cell surface protein that bind to yeast mannan.	Katakura <i>et al.</i> , 2010

Key Examples and Virulence Functions

- 1. Phosphoglucose Isomerase (PGI):** In the cytoplasm, PGI converts glucose-6-phosphate to fructose-6-phosphate. When secreted, it acts as neuroleukin, autocrine motility factor (AMF), differentiation and maturation mediator of myeloid cells (DMM), and neuropilin, contributing to cell migration and malignancy (Yavlovich *et al.*, 2007) [32].
- 2. Glyceraldehyde-3-Phosphate Dehydrogenase (GAPDH):** Cytoplasmic GAPDH catalyzes the conversion of glyceraldehyde-3-phosphate to 1,3-bisphosphoglycerate. On the bacterial surface, it acts as an adhesin, binding fibronectin, plasminogen, and cytoskeletal proteins (Pancholi & Fischetti, 1993) [25]. In *Streptococcus pneumoniae* and *S. pyogenes*, surface GAPDH facilitates invasion of endothelial and epithelial barriers (Attali *et al.*, 2008) [2]. In *Mycoplasma genitalium*, it promotes binding to mucin, and in pathogenic *E. coli* strains, it binds fibrinogen and plasminogen while exhibiting NAD-ribosylating activity. Furthermore, GAPDH can induce immunosuppressive IL-10 production in group B streptococci and modulate B lymphocyte activity in *Streptococcus agalactiae*.
- 3. Enolase:** Present in both prokaryotes and eukaryotes, enolase is found on the cell surface, where it binds plasminogen, facilitating tissue invasion. In *S. pneumoniae*, surface enolase-mediated plasminogen binding enables penetration of basement membranes, a critical step in the pathogenesis of meningitis (Pancholi, 2001) [24].

Functional Significance of Surface Glycolytic Enzymes

Surface localization of glycolytic enzymes may serve several purposes:

- ATP synthesis:** Local ATP production may fuel processes in the producing bacterium or its community (e.g., biofilm formation).

- Signaling:** Glycolytic intermediates or byproducts may act as signaling molecules, influencing host responses or bacterial communication (Mukai, 2009) [23].

- Immune evasion:** Binding host proteins such as plasminogen or fibronectin masks bacterial surfaces and disrupts immune recognition.

3.4.2 Other Moonlighting Bacterial Metabolic Enzymes

Beyond glycolysis, other central metabolic enzymes also play roles in virulence:

- Alcohol Acetaldehyde Dehydrogenase (LAP):** In *Listeria monocytogenes*, LAP binds the chaperonin Cpn60 on intestinal cell surfaces, facilitating colonization-a striking example of moonlighting protein-protein interactions (Santiago *et al.*, 1999) [29].
- Elongation Factor Tuf:** In *Pseudomonas aeruginosa*, Tuf binds host plasminogen and factor H, aiding in tissue invasion and resistance to complement-mediated phagocytosis.
- Glutamate Racemase (MurI):** In *M. tuberculosis*, MurI not only synthesizes D-glutamate for peptidoglycan but also inhibits DNA gyrase, affecting DNA topology and potentially modulating stress responses (Sengupta *et al.*, 2008) [30].
- cAMP Phosphodiesterase:** In *M. tuberculosis*, this enzyme controls cell wall permeability to cytotoxic agents, influencing drug resistance and persistence (Podobnik *et al.*, 2009) [28].

These examples underscore the ability of metabolic enzymes to influence cell wall integrity, immune evasion, and adaptation under hostile conditions.

3.4.3 Moonlighting of Molecular Chaperones and Protein Folding Catalysts

Molecular chaperones, such as the 60-, 70-, and 90-kDa heat shock proteins (Hsps), are another major class of bacterial

moonlighting proteins. These proteins, critical for protein folding and stress responses, also function as surface adhesins, immune modulators, and receptors (Pockley *et al.*, 2009) [27].

Key Examples and Virulence Functions

Role in Host-Pathogen Interactions

Hsp70 and Hsp90: These chaperones can act as receptors for bacterial lipopolysaccharide (LPS) and mediate interactions with host immune cells (Honda *et al.*, 2009) [11].

Cpn60 (GroEL):

- In *Helicobacter pylori*, Cpn60 acts as an immunodominant antigen, cell signal, and adhesin.
- In *Chlamydia pneumoniae*, it stimulates monocyte cytokine release and metalloproteinase production, contributing to inflammation.
- In *M. tuberculosis*, two Cpn60 proteins modulate macrophage signaling, granuloma formation, and osteoclast inhibition. Cpn60.2 binds CD43, facilitating macrophage uptake (Hickey *et al.*, 2010) [10].

Role in Immunomodulation

Bacterial chaperones can activate or inhibit mononuclear phagocyte cytokine synthesis, shaping the immune response. For example, mycobacterial Cpn60 promotes alternative macrophage activation, distinct from classical LPS-induced activation (Friedland *et al.*, 1993) [7]. This alternative activation may favor bacterial persistence by subverting effective immune clearance (Martinez *et al.*, 2009) [21].

3.4.4 Role in Adhesion and Invasion: The Centrality of Moonlighting Proteins

The most consistent theme among bacterial moonlighting proteins is their role in adhesion and invasion. By binding host extracellular matrix proteins (e.g., fibronectin, collagen, laminin) or recruiting host proteases (e.g., plasminogen), these proteins facilitate colonization, tissue penetration, and dissemination (Kainulainen & Korhonen, 2014) [15]. This multitasking not only enhances virulence but also enables immune evasion and persistence.

Table 3: Bacterial moonlighting proteins which act as adhesins and invasins

Enzymes/ Molecular chaperones	Organism	Adhesion/Invasion
Enolase	Yeast/gram +Ve bacteria	Laminin and Plasminogen
Pyruvate dehydrogenase	<i>Mycoplasma pneumoniae</i>	Fibronectin
Malate synthase of glyoxylate pathway	<i>M. Tuberculosis</i>	Fibronectin and Laminin
GAPDH	group A streptococcus	Fibronectin
Autolysins	<i>Staphylococcus aureus</i>	Fibronectin
6-phosphogluconate dehydrogenase	pneumococcal strains	Adhesin
Hsp60, Hsp70 and DnaK	Mycobacteria	Adhesin

3.5 Therapeutic Implications and Challenges

Given their central role in bacterial virulence, moonlighting proteins represent attractive targets for novel therapeutics, especially in the context of rising antibiotic resistance (Henderson, 2014) [8]. In principle, inhibitors targeting widely conserved moonlighting proteins, such as GAPDH, enolase, or Cpn60, could have broad-spectrum utility. However, several challenges complicate this strategy:

- **Functional Diversity:** Homologous moonlighting proteins may perform different moonlighting functions in different species due to divergent active sites or regulatory features (Henderson, 2014) [8].
- **Host Homology:** Many moonlighting proteins are highly conserved between bacteria and hosts, raising the risk of off-target effects.
- **Essentiality:** Targeting central metabolic enzymes may cause collateral damage, affecting bacterial viability but also host commensals.
- **Plasticity:** Bacteria may compensate for loss of one moonlighting function by upregulating alternative pathways or proteins.

Nevertheless, the unique structure-function relationships and surface exposure of many bacterial moonlighting proteins may offer opportunities for selective targeting, vaccine development, or immune modulation.

4. Conclusion

Protein moonlighting represents a fundamental strategy by which bacteria maximize functional output from limited genetic resources, responding dynamically to environmental cues and host defenses. Major metabolic enzymes and

molecular chaperones, once thought to be housekeepers, are now recognized as pivotal players in bacterial virulence, mediating adhesion, invasion, immune modulation, and survival.

The study of bacterial moonlighting proteins not only challenges traditional views of protein function but also reveals the evolutionary ingenuity of pathogens. As the search for new antimicrobial strategies intensifies, understanding the mechanisms, regulation, and diversity of moonlighting proteins will be essential for exploiting their vulnerabilities while minimizing harm to the host.

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