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Coenzyme A and Its Derivatives in Metabolic Dormancy: A Multifaceted Regulatory Network

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Abstract: Metabolic dormancy is a survival strategy that can be found in bacteria, fungi, plants, and hibernating animals, to survive under unfavorable conditions, such as nutrient shortage and oxidative stress. Coenzyme A (CoA), a central metabolic molecule, plays an important role during dormancy by integrating energy metabolism, redox homeostasis, and post-translational modifications. This essay discusses the different roles of CoA and its derivatives in metabolic dormancy, particularly in enzyme activity control, stress adaptation, and gene expression. By integrating energy metabolism, gene regulation, and redox balance, CoA functions as a metabolic switch that helps cells enter, maintain, and exit dormancy and as an antioxidant to defend against oxidative stress. Understanding these mechanisms may provide evidence of bacterial persistence, hibernation, and metabolic diseases, with potential applications in biotechnology and medicine.

Keywords: Coenzyme A Metabolism; Bioprocess Engineering; Metabolic Dormancy; Systems Biology; Oxidative Stress Response

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1. Introduction

Coenzyme A (CoA) is an important molecule found in a wide range of living cells. Coenzyme A (CoA) and its derivatives, such as acetyl-CoA, malonyl-CoA, and succinyl-CoA, participate in essential biochemical pathways, including carbohydrate metabolism, lipid metabolism, fatty acid oxidation, and protein modification through CoAlation[1]. Protein CoAlation is a post-translational modification (PTM), the thiol group of coenzyme A (CoA) covalently binds to the side chain of cysteine residues of proteins, forming a reversible disulfide bond which acts as an antioxidant in oxidative stress.

The biosynthetic pathway of CoA involves 5 enzymatic pathways. CoA is synthesized from pantothenic acid (vitamin B5), Adenosine triphosphate (ATP), and cysteine[2]. Pantothenate kinase (Pank) catalyzes the phosphorylation of pantothenate into 4′-phosphopantothenate; this reaction is a rate-limiting step due to the feedback inhibition of the product CoA and its other thioester derivative [2]. Exogenous substrates and hormones can influence the efficacy of CoA biosynthesis. The study has shown that substrates like glucose, pyruvate, fatty acid, and insulin can reduce the rate of CoA biosynthesis by inhibiting Pank[2].

Acetyl-CoA, acyl-CoA, and malonyl-CoA are key CoA thioester derivatives formed from distinct biochemical pathways with different metabolic functions. Acetyl-CoA can be synthesized from pyruvate catalyzed by the pyruvate dehydrogenase complex (PDH) in mitochondria or from citrate, linking carbohydrate and lipids metabolism to the

tricarboxylic acid (TCA) cycle[3,4]. Both reactions bind the acetyl group to the cysteine residue of CoA, generating a thioester bond[5]. Similarly, this thioester bond also exists in malonyl-CoA and acyl-CoA, where a malonyl group and an acyl group, respectively, are linked to the thiol (-SH) group of CoA. These CoA thioesters act as the central regulator of metabolism, allowing organisms to adapt to the varying nutrient conditions.

Metabolic dormancy is usually described as a reversible physiological state of organisms in adverse environmental conditions like unfavorable temperatures, pressure, pH value, oxidative stress, and nutrient shortage[6]. Such adaptation patterns can be observed in seed dormancy, bacterial sporulation, and mammals during hibernation, enabling them to survive under harsh conditions.

Dormancy is usually characterized by reduced metabolic activity, allowing organisms to conserve energy. Specifically, the key features include decreased respiration and biosynthesis, developmental arrest, and increased resistance to stress. During dormancy, species usually rely on stored nutrients such as fats, starch or glycogen, and proteins. Therefore, glycolysis, which uses glucose as a carbon source, is modulated during dormancy. In yeast, key glycolytic enzymes include glucokinase (Glk1) and phosphofructokinase (PFK1), which can transform into filamentous or punctate assemblies when glucose is absent[7,8,9]. Such conformational transition allows cells to regulate the enzyme activity to meet the metabolic demand. Specifically, Glk1, which catalyzes the phosphorylation of glucose to glucose-6-phosphate, is inhibited by filamentation in the presence of glucose[10]. This may indicate that Glk1 catalyzes the first step of glycolysis if glucose levels rise. Moreover, the energy level controls the two conformations of PFK1, the inhibited and activated forms. High-energy signals like ATP and citrate accumulation will result in their allosteric interaction with PFK1, locking PFK1 at the inhibited state. In contrast, low-energy signals like AMP and F6P activate glycolysis when energy is needed, balancing inhibition and activation[11].

2. CoA and Its Derivatives as a Key Regulator of Energy Conservation in Metabolic Dormancy

Efficient energy production is important for maintaining cell function. The universal energy currency, ATP, can be generated from two carbon sources: fatty acids and glucose. As shown in **Figure 1**, mitochondrial metabolism converts these fuels into ATP through sequential processes, including glycolysis, the tricarboxylic acid (TCA) cycle, β-oxidation of fatty acid, and oxidative phosphorylation. Decarboxylation and carboxylation of pyruvate produced from glycolysis generate acetyl-CoA and oxaloacetate (OAA) respectively. Moreover, acyl-CoA is synthesized from fatty acid and CoA by fatty acyl-CoA synthase. The acyl group is transported into the mitochondrial matrix as acylcarnitine, which is regenerated into Acyl-CoA for β-oxidation. The reduced equivalents produced from these pathways, including NADH and FADH2, donate electrons to the electron transport chain (ETC) and drive ATP synthesis through oxidative phosphorylation.

Acetyl-CoA is mainly produced from carbohydrates and fatty acids. Glucose is catalyzed into pyruvate through glycolysis. Pyruvate is converted into oxaloacetate (OAA) by pyruvate carboxylase (PC), which may enter the tricarboxylic acid (TCA) cycle$_2$ or be catalyzed back to phosphoenolpyruvate (PEP) by mitochondrial phosphoenolpyruvate carboxykinase (PECK-M) through gluconeogenesis. The acetyl group can also be transferred from pyruvate to CoA, generating acetyl-CoA by pyruvate dehydrogenase (PDH). The acyl group of fatty acid is transported to the matrix by carnitine palmitoyltransferase 1 (CPT1), carnitine-acylcarnitine translocase (CACT), and carnitine palmitoyltransferase 2 (CPT2). The acetyl-CoA enters the TCA cycle to generate NADH/FADH, which produces ATP in the electron transport chain (ETC). Acetyl-CoA carboxylase (ACC) catalyzes the acetyl-CoA into Malonyl-CoA in lipogenesis, which uses the citrate from the TCA cycle as the substrate. The figure was produced from bio render and was adapted from[12,13].

AMP kinase (AMPK) is a key energy sensor that responds to cellular ATP/AMP ratio fluctuations. The canonical regulation of AMPK involves activation when its Thr172 residue is phosphorylated by liver kinase B1 (LKB1). AMP binding to AMPK triggers three activation mechanisms. First, the direct binding of AMP to AMPK can cause allosteric activation of Thr172-phosphorylated AMPK[14]. Secondly, the phosphorylation action of LKB1 is promoted[15]. Moreover,

dephosphorylation catalyzed by phosphatases on Thr172 is inhibited by the direct binding of AMP. This system allows AMPK to sensitively monitor the fluctuation of the AMP to ATP ratio[16].

AMPK links fatty acid biosynthesis to the current energy status by inactivating ACC through phosphorylation. The study has shown that Ser79, Ser1200, and Ser1215 of human ACC1 are the critical sites where AMPK phosphorylates[17]. In contrast, Ser1157 in yeast ACC1 has been reported to be phosphorylated by AMPK-equivalent enzymes[18]. The ACC phosphorylation results in significant conformational changes that inhibit ACC activity, which decreases fatty acid synthesis and redirects acetyl-CoA toward energy production during starvation states. By contrast, in nutrient-rich states, the citrate generated from the TCA cycle enhances the activity of ACC by stabilizing active conformation by filament formation[19].

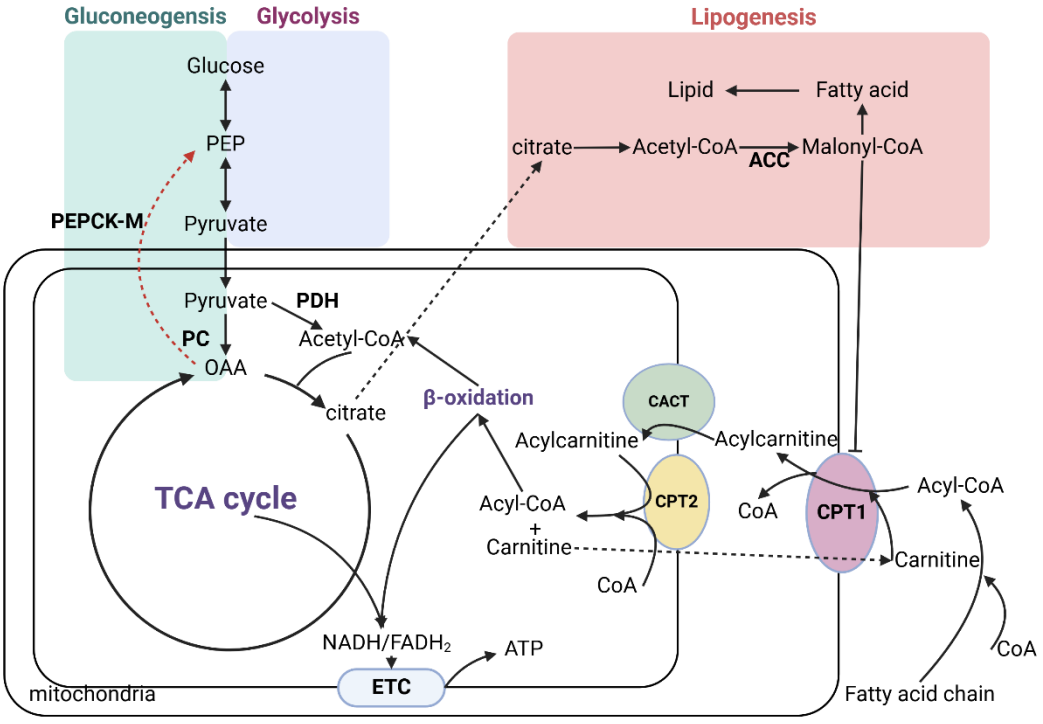

Figure 1. Mitochondrial metabolism and the CoA control sites

2.1. Allosteric Inhibitor Role of Malonyl-CoA

The regulation of ACC activity is essential for fatty acid metabolism, which controls the biosynthesis of lipids and regulates malonyl-CoA levels to prevent the suppression of fatty acid oxidation. The β-oxidation of fatty acids generally includes 2 steps (**Figure 1**): translocation and oxidation. The fatty acid is first converted to acyl-CoA by acyl-CoA synthase. The transportation of acyl groups into the mitochondrial matrix for β-oxidation is carried out by the carnitine shuttle system, including 3 key proteins [20]: carnitine palmitoyltransferase 1 (CPT1), carnitine-acylcarnitine translocase (CACT), and carnitine palmitoyltransferase 2 (CPT2). CPT1 is found on the external mitochondrial membrane of mitochondria; it catalyzes the transfer of the acyl group from acyl-CoA to carnitine, producing acylcarnitine and releasing CoA. The acylcarnitine is then transported into the mitochondrial matrix by CACT. CPT2 catalyzes a reverse reaction inside the mitochondria, the acyl group is reattached to CoA and therefore generates acyl-CoA[20]. This system transports the long-chain acyl groups across the membrane.

Malonyl-CoA is a key molecule that suppresses the β-oxidation of fatty acids by inhibiting the CPT1, which blocks the transport of the acyl group, which is the rate-limiting step of β-oxidation [20]. This process decreases the uptake of fatty acid from the cytosol, which ensures the cell favors lipid synthesis over fatty acid oxidation when nutrients and energy

are abundant. Conversely, during metabolic dormancy, when nutrients are deficient, AMPK phosphorylates and inhibits ACC, which lowers the concentration of malonyl-CoA and reduces the CPT1 inhibition. Increasing the level of fatty acid oxidation to generate ATP. The physiological study has shown the CPT1 inhibition by malonyl-CoA in CPT1c knockout (KO) mice, which have decreased body weight and food intake. In addition, CPT1c KO mice have shown increased susceptibility to high-fat diet-induced obesity, suggesting CPT1c may protect against fatty accumulation[21]. This energy use strategy dynamically controls lipids synthesis and degradation, optimizing energy use.

2.2. Allosteric Activator Role of Acetyl-CoA

Oxaloacetate (OAA) is a key metabolic intermediate involved in both the TCA cycle and gluconeogenesis (**Figure 1**). Pyruvate carboxylase catalyzes the carboxylation of pyruvate using bicarbonate ($HCO3^-$) as a CO2 donor to generate OAA, which is the first step of gluconeogenesis and provides OAA to sustain glucose synthesis, particularly in the liver and kidneys, where it plays a crucial role in maintaining blood glucose levels during fasting or metabolic dormancy. OAA is then reduced to malate to cross the mitochondrial membrane into the cytosol, where the malate is oxidized back to OAA. Decarboxylation and phosphorylation then occur to convert OAA to phosphoenolpyruvate (PEP) by phosphoenolpyruvate carboxykinase (PECK).

Certain tissues, such as the brain and red blood cells, are highly dependent on glucose for energy. The brain depends on glucose from gluconeogenesis, especially in the early stages of fasting before ketone bodies become a primary fuel source[22]. Red blood cells lack mitochondria; therefore, their energy production relies entirely on glycolysis. The pyruvate in gluconeogenesis may be synthesized from some non-carbohydrate precursors, including lactate and alanine, catalyzed by lactate dehydrogenase and alanine transaminase in the liver[12].

As a result, the activity of PC is tightly controlled to make sure that the gluconeogenesis adapts to the metabolic needs. Acetyl-CoA acts as an allosteric activator of PC, which binds to the regulatory site and causes conformational change [23]. The α4 quaternary structure of most PCs includes a biotin carboxylation domain in each subunit. This domain is responsible for catalyzing the ATP-dependent carboxylation of biotin, a necessary step for transferring the carboxyl group to pyruvate [23]. The catalytic efficiency of this domain is enhanced during acetyl-CoA binding. This may be because of the stabilization of the PC structure, which avoids the dissociation of PC into monomers or dimers[23]. A study carried out by Mayer et al.[24] suggests that in chicken, rat, and sheep livers, the tetrameric structure of the enzyme was much better preserved when acetyl-CoA was present during preparation for electron microscopy. Moreover, the structural study[24] also found that the PC tetramer exists in a more compact conformation when acetyl-CoA binds to it.

2.3. The Role of Acetylation in Gene Regulation and Metabolic Control

Acetylation is a reversible protein post-translational modification that occurs to regulate protein function and gene expression. The reaction involves binding the acetyl group to the lysine residues of the protein (**Figure 2**). This process is catalyzed by acetyltransferases such as lysine acetyltransferases (KATs) and histone acetyltransferases (HATs). The reverse reaction deacetylation removes the acetyl group, which is catalyzed by deacetylases, including histone deacetylases (HDACs) and lysine deacetylases (KDACs)[25]. Acetyl-CoA is the universal acetyl group donor, it has been proven that the acetyl-CoA concentration can affect the extent of acetylation in *Drosophila* S2 cells and in mammalian cells[26], where a decline in CoA levels corresponds with decreased acetylation of histones and tubulin.

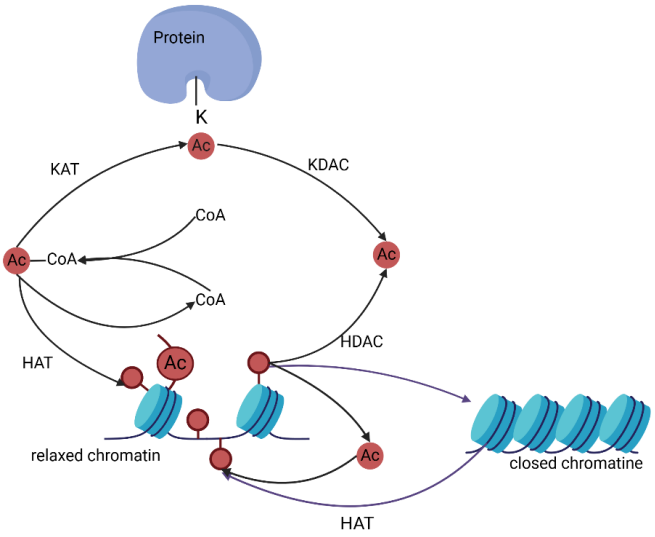

Figure 2. Acetylation and Deacetylation of Protein and Histone

The acetylation and deacetylation are catalyzed by acetyltransferases including lysine acetyltransferases (KATs) and histone acetyltransferases (HATs); and deacetylases such as histone deacetylases (HDACs) and lysine deacetylases (KDACs) respectively. Ac standard for Acetate. The figure was produced from biorender and was adapted from [25]

2.3.1. Gene Level Control by Histone Acetylation

Histone acetylation is a crucial PTM that was discovered about 60 years ago. The positively charged amino group on the side chain of lysine can interact strongly with negatively charged DNA, which stabilizes chromatin in a closed structure (**Figure 2**) [27]. HATs catalyze the binding of the acetyl group to the lysine residues, which neutralize its positive charge and reduce the interaction between histones and DNA molecules, resulting in a relaxed chromatin conformation available for transcription. Conversely, the deacetylation removes the acetyl groups returns chromatin to the closed structure, and inhibits transcription[27].

Histone acetylation provides the transcription controls for plant development and seed dormancy. In *Arabidopsis thaliana*, the expression of *DOG1* (DELAY OF GERMINATION 1) regulates the states of seed in maturation or dormancy, which involves dynamic histone modifications mediated by transcriptional repressors and histone deacetylases (**Figure 3**)[28]. The HIGH-LEVEL EXPRESSION OF SUGAR INDUCIBLE2 (HSI2) and HSI2-LIKE1 (HSL1) are the two transcriptional repressors that interact with the histone deacetylases HD2B and HD2A, forming a complex that directly inhibits the expression of *DOG1* during seed maturation by removing the acetyl group on histone H4 lysine 5 (H4K5ac) [28]. Conversely, when the histone acetylation level is high, *DOG1* remains activated and promotes seed dormancy[28].

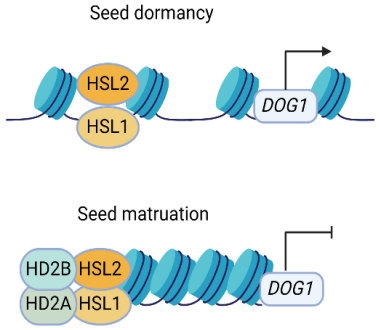

Figure 3. Epigenetic Regulation of *DOG1* Expression During Seed Dormancy and Maturation

Aberration: HIGH-LEVEL EXPRESSION OF SUGAR INDUCIBLE2 (HSI2); HSI2-LIKE1 (HSL1); DELAY OF GERMINATION 1 (DOG1) The figure was produced from biorender and was adapted from[27].

VERNALIZATION5/VIN3-LIKE 3 (VEL3) is a chromatin-associated protein that provides the maternal epigenetic control of seed dormancy, making sure that progeny adapt to environmental conditions experienced by the mother[29]. As a subunit of histone deacetylase complex, VEL3 combines with MULTICOPY SUPPRESSOR OF IRA1 (MSI1) and SIN3-ASSOCIATED PROTEIN 18 (SAP18) to decrease the acetylation level of histone. VEL3 also suppresses a key transcriptional regulator gene *ORESERA1* (ORE1), which will induce seed dormancy in the endosperm[29].

Overall, histone acetylation acts as a cell dormancy switch in gene expression level by controlling the transcription. Providing a survival mechanism for cells to adapt to the environment by deciding to enter the dormancy or development states.

2.3.2. Activity Control in Enzyme-Level Acetylation

Histone acetylation is widely known for its transcription regulation, but acetylation also directly affects enzyme activity. At the enzyme level, lysine acetylation regulates enzyme activity and interaction with metabolic pathways. Mass spectrometry analyses carried out by Wang, Q et al.[30], demonstrate that about 90% of the key enzymes involved in metabolism show acetylation in *Salmonella enterica*. Depending on the nutrient source of the *S. enterica* grows, the acetylation is different in cells, which is higher in glucose-source cells than in citrate-grown cells. The two distinct nutrient sources represent the different metabolic pathways to produce energy: glucose-based glycolysis and citrate-dependent gluconeogenesis.

The single forms of acetyltransferase and NAD-dependent deacetylase that exist in *S. enterica* are Pat and CobB respectively[31]. The two mutants generated in the study knock out Pat (Δpat) and CobB (ΔcobB) respectively[30], which tests the influences of the metabolic pathway. A pattern of increased glycolysis rate and decreased citrate utilization is observed in ΔcobB mutants. While an opposite growth pattern is found in Δpat mutants with lower acetylation levels[30]. This experiment evidence suggests that acetylation may promote glycolysis and inhibit gluconeogenesis and oxidative metabolism.

2.3.3. Acetylation in Regulating the Initiation of Autophagy

Autophagy is a highly conserved cellular degradation process that removes the damaged organelles and cytoplasmic proteins by the lysosome. During metabolic dormancy, autophagy is important to break down macromolecules to generate energy. The key steps of autophagy involve initiation, autophagosome formation, and fusion with the lysosome. Acetylation on the molecules in these processes helps activate or inactivate the signaling pathway.

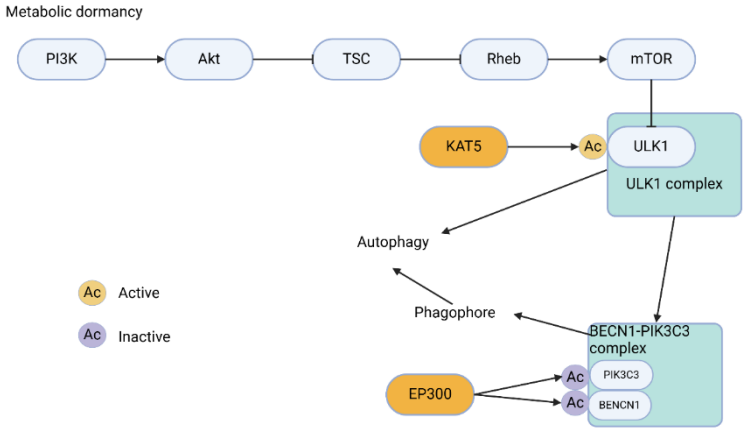

Figure 4. The activation step of autophagy through the PI3K-mTOR pathway.

Acetylation occurs on ULK1 catalyzed by KAT5 and activates autophagy, while acetylation catalyzed by EP300 at PIK3C3 and BENCN1 inhibits autophagy. The figure was produced from biorender and was adapted from[32,33]

When autophagy is initiated by the low mTOR (mammalian target of rapamycin kinase) signal during metabolic dormancy, the TOR complex activity is reduced at low-nutrient conditions including amino acid shortage, which leads to the activation of unc-51 like autophagy activating kinase 1 (ULK1) (**Figure 4**)[33]. The ULK1 complex stimulates another protein complex: BECN1-PIK3C3/VPS34 complex, which stands for beclin 1 and phosphatidylinositol 3-kinase catalytic subunit type 3, which is responsible for producing phosphatidylinositol-3-phosphate (PtdIns3P), facilitating the production of the phagophore[32], the early form of the autophagosome.

The lysine acetyltransferase 5 (KAT5) catalyzed the acetylation on the ULK1 at the residues K162 and K606, enhancing ULK1 activity and promoting autophagy (**Figure 4**). Interestingly, this activation is isolated to the mTOR-AMPK phosphorylation, meaning that it provides an alternative way for autophagy activation[32].

2.4. Antioxidant Role of Protein CoAlation

Oxidative stress is a state in a situation where the generation of reactive oxygen species (ROS) cannot balance with the cellular defense ability to neutralize or eliminate ROS. When oxidative stress occurs, excess ROS can damage cell components, including lipids, proteins, and DNA, leading to impaired function and potential cell death. This damage may result in a wide range of diseases, including aging, inflammation, cancer, cardiovascular diseases, and neurodegenerative disorders[34].

Protein CoAlation is a post-translational modification where the thiol group of coenzyme A (CoA) covalently binds to the thiol group of cysteine residues in proteins, forming a reversible thioester bond. Due to the slow oxidation rate of CoA, which is 4 times slower than glutathione (GSH) and 720 times slower than cysteine with a copper catalyst[35], protein CoAlation provides good protection against oxidative stress. Because of the location of cysteine usually at the catalytic site due to its hydrophobicity, CoA also helps maintain the activity of enzyme, by covalently forming the thioester bonds with the cysteine and glutathione[2].

2.4.1. Protein CoAlation in Bacteria Sporulation

Bacterial sporulation is a survival strategy of bacteria such as *Bacillus and Clostridium* species, forming highly resistant and dormant spores in harsh environments. They remain in metabolic dormancy until the conditions are favorable. Experimental results have shown that protein CoAlation is induced when the oxidizing agent, including H_2O_2, NaOCl, and diamide, is introduced and during carbon starvation. In addition, the CoAlation level increases according to the increase in the concentration of the oxidizing agent and the time length.

By comparing the CoA proportion in the developmental and dormant state of the spores, a high percentage of acetyl-CoA in growing spores, while only 1.5% of that is found in dormant state. In contrast, 43% of CoA in dormant spores are covalently linked to protein [36], which is rapidly degraded during spore germination, leaving only 2% of CoA bound to protein with the disulfide bond[36].

Key CoAlated proteins in *Bacillus subtilis* spores include alkyl hydroperoxide reductase AhpC, alcohol dehydrogenase AdhB, phosphopentosemutase Drm, and YneT which are related to redox regulation, metabolism, and stress responses. The reversible CoAlation of AhpC observed in S*taphylococcus aureus* occurs at the residue Cys168, which may protect its catalytic site, and the deCoAlation during germination might restore enzymatic function[37].

In general, the experiment results illustrate that protein CoAlation widely exists in dormant spores of *Bacillus* species. The difference in CoA/CoA thioester proportion between dormant and germinated spores indicates the adaptation to the environment, high acetyl-CoA level and activated enzymes are necessary for early cell growth. Whereas the high percentage coalated protein helps against oxidative stress.

3. Discussion and Summary

Coenzyme A is a key metabolic cofactor involved in energy production, enzyme activity regulation, and redox balance. Playing a crucial role in metabolic dormancy across bacteria, plants, and animals. This essay has discussed how CoA and its derivatives regulate energy conservation, oxidative stress resistance, and gene expression to enter or exit dormancy. Similar patterns of metabolic dormancy are observed across distinct species.

One of the functions of CoA in dormancy is controlling metabolic pathways to conserve energy. The further study in this area can provide metabolic evidence for oncology, which helps in understanding the influences of lipids metabolism and histone acetylation on cancer proliferation[38] and the survival mechanism of the dormant cancer cells[33].

Additionally, CoAlation acts as an antioxidant protection mechanism during dormancy. Binding the CoA to the side chain of cysteine residue protects it from irreversible oxidative damage. This modification may have potential applications in aging and age-related diseases, where oxidative stress and metabolic decline play a crucial role.

CoA derivatives also contribute to gene regulation by acetylation. In plants, histone acetylation and deacetylation regulate seed dormancy and germination. In bacteria, acetylation and deacetylation affect enzyme activity and transcription for the most efficient energy consumption, and control of autophagy.

Future research of the role of CoA in metabolic dormancy could explore areas like aging, metabolic regulation, and stress resistance. A deeper comprehensive of protein protection by CoAlation is needed to develop possible therapies and drug targets.

Disclosure statement

The author declares no conflict of interest.

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