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The Regulation of Lipid Digestion by Protein-Polysaccharide Complex Pickering Emulsions

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Abstract. Lipids are the core energy-supplying substances in the human body, and their rapid burst of hydrolysis and excessive absorption in the gastrointestinal tract are key pathological factors inducing chronic diseases such as obesity and fatty liver. Intervening in lipid degradation kinetics through food interface engineering has become an important strategy to regulate metabolic disorders. At present, traditional emulsions are prone to structural collapse in extreme digestive environments, while protein-polysaccharide complex Pickering emulsions exhibit great potential in controlling lipid release due to their excellent interfacial mechanical properties. However, the dynamic interfacial evolution of this system in complex in vivo environments and its targeted regulation mechanism on downstream metabolic pathways still lack systematic elucidation. In this paper, the microscopic self-assembly principle of polysaccharide-protein complex interfaces is comprehensively deconstructed, and its multidimensional regulation mechanism in the lipid digestion process is deeply analyzed. The results indicate that the complex system greatly weakens the adsorption efficiency of lipase by constructing a three-dimensional (3D) network framework with a high modulus, utilizing significant steric hindrance, an extremely high bile salt displacement energy barrier, and in situ microgelation characteristics. Consequently, the release mode of free fatty acids (FFAs) is successfully transformed into a stable and controllable sustained release. Given that current mechanistic demonstrations highly rely on in vitro static models, future research should prioritize combining in vivo in situ tracking technologies to map the interfacial fate under real dynamic gastrointestinal stress and deeply explore the interactive intervention mechanisms between specific polysaccharide structures and gut microbiota.

Keywords: Pickering Emulsions, Lipid Digestion, Protein-Polysaccharide Complexes, Targeted Delivery.

1. Introduction

Lipids are not only the core energy-supplying substances for the body but also key physical carriers for maintaining cell membrane integrity and delivering fat-soluble bioactive factors [1]. However, the efficient hydrolysis of lipids in the gastrointestinal tract and the excessive absorption of FFAs have been confirmed as the underlying pathological factors driving chronic metabolic syndromes

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such as obesity, hyperlipidemia, and metabolic dysfunction-associated steatotic liver disease (MASLD) [2]. In the continuous physiological environment of the digestive tract, precisely intervening in the interfacial degradation kinetics of triglycerides (TAGs) through physicochemical methods to reduce the peak release of FFAs has become an important non-pharmacological intervention strategy for preventing and regulating metabolic disorders [3].

Currently, common oil-in-water (O/W) lipid delivery systems in the food industry typically rely on a single type of protein or small-molecule surfactant to maintain stability. However, when encountering highly acidic gastric fluids rich in proteases, this monolayer interface is prone to rupture and structural collapse due to poor physical resistance [4,5]. As these damaged emulsions enter the small intestine, bile salts with strong surface activity can strip away the residual proteins on the oil droplet surface with an extremely low energy barrier. This allows the pancreatic lipase-colipase complex to directly attach to the naked lipids, causing rapid and explosive hydrolysis of fats [6,7]. This uncontrolled digestion process not only interferes with the body's normal satiety feedback but also highlights the fatal weakness of traditional monolayer emulsifiers in coping with complex physiological environments [8].

To solve this interface engineering problem, researchers have recently attempted to use protein and polysaccharide complexes to prepare high-strength Pickering emulsions [9]. Unlike conventional emulsifiers, amphiphilic molecules such as soy protein isolate (SPI) can self-assemble with macromolecular polysaccharides like bacterial cellulose (BC) and inulin. They intertwine at the O/W interface to form a dense 3D network, structurally similar to sturdy "reinforced concrete" [10,11]. This rigid interfacial skeleton can resist enzymatic degradation in gastric fluids to prevent droplet coalescence while exerting a strong steric hindrance effect in the intestine. Through this physical barrier, the adsorption efficiency of lipase at the interface is significantly weakened, thereby effectively delaying lipid digestion [1,12].

2. Construction and characterization of protein-polysaccharide complex pickering emulsions

2.1. Material basis and self-assembly principles of complex systems

The high surface energy state of traditional O/W emulsions makes them thermodynamically unstable. When a single protein (e.g., SPI, whey protein) is used as the interfacial layer near its isoelectric point (pI) or in a high ionic strength environment, the electrostatic repulsion of the interface significantly attenuates, easily inducing droplet coalescence and Ostwald ripening [4]. To break through this interfacial mechanical bottleneck, interface engineering introduces a non-covalent complexation mechanism between proteins and polysaccharides. Complex Pickering emulsions self-assemble to form a solid particle stabilization layer at the O/W interface through electrostatic attraction, hydrogen bonding, and hydrophobic interactions [9]. In this system, macromolecular polysaccharides (e.g., BC, inulin, or chitosan), with their high aspect ratios and rigid molecular chains, provide decisive steric hindrance for the interface; meanwhile, protein molecules provide the necessary amphiphilicity to effectively reduce the initial O/W interfacial tension [7,11].

2.2. Microstructure and mechanical characterization of the interface

Microscopic images from confocal laser scanning microscopy (CLSM) and scanning electron microscopy (SEM) intuitively show that macromolecular polysaccharides such as bacterial cellulose nanofibrils (BCNFs) typically intertwine and stack around the emulsion interface, building a 3D rigid network skeleton. At the same time, protein molecules like SPI embed into the voids or cross-

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linking nodes of this skeleton and bind firmly to it [10,11]. This complex physical cross-linking state not only significantly increases the thickness of the interfacial layer but also exponentially increases its viscoelastic modulus. Mechanically, this sturdy interfacial structure acts as a physical barrier, effectively preventing the internal droplets from colliding and coalescing due to thermal motion [5].

2.3. Core experimental data and environmental resistance indicators

When facing extreme acid-base environments, the complex system exhibits strong resistance to destruction. For example, in in vitro experiments of emulsions co-stabilized by BCNFs and SPI: since pH 4.5 is close to the pI of SPI, the absolute Zeta potential of ordinary monolayer SPI emulsions rapidly drops to near zero upon encountering this acidity. Without charge protection, the entire system quickly exhibits irreversible severe flocculation or even direct oil-water separation [10]. However, introducing structural polysaccharides into the interface changes the situation entirely. Emulsions with complex interfaces can maintain an absolute Zeta potential steadily above the safe line of >20 mV even under the same acidic conditions, providing sufficient electrostatic repulsion between colloidal particles to resist coalescence [10].

Additionally, this complex structure provides excellent encapsulation protection during gastric digestion. Tests in simulated gastric fluid (SGF) revealed that although the surroundings are full of highly active pepsin, the hard outer polysaccharide network acts as a physical shield, blocking most proteases and preventing them from accessing the internal SPI hydrolysis sites [12,13]. Continuous tracking by dynamic light scattering (DLS) instruments also confirms this: after two hours of simulated gastric digestion, the average size of the emulsion droplets hardly increases significantly. This means that before the droplets leave the stomach and enter the intestine, their outer structure remains basically intact, creating the necessary conditions for the slow release of lipids in the intestine [10].

3. Physiological indicators of lipid digestion and metabolic pathways

3.1. Physical and biochemical definition of lipid digestion

In the gastrointestinal hydrodynamic system of mammals, lipid digestion is defined as a continuous biochemical cascade degradation process highly dependent on multiphase interfaces. Its core physical reaction is the cleavage of ester bonds in dispersed dietary lipids (mainly TAGs) catalyzed by specific esterases, ultimately converting them into FFAs and monoglycerides (MAGs) that can be taken up by the intestinal epithelium [6]. The kinetic rate of this enzymatic reaction is directly limited by the effective specific surface area of the dispersed lipid phase in the aqueous continuous medium. Once the chyme enters the small intestinal phase, highly surface-active bile salt molecules competitively adsorb, sharply reducing the O/W interfacial tension and stripping away inhibitory peptides and primary metabolites from the interface [12]. This physical displacement allows the pancreatic lipase-colipase complex to overcome the energy barrier, irreversibly anchoring to the naked lipid droplet surface and triggering the "interfacial activation" effect, thereby driving the rapid enzymatic hydrolysis of lipid macromolecules [1,6].

3.2. Physical transfer of metabolic pathways and physiological purposes

From a physiological dimension, the main purpose of lipid digestion is to provide high-density energy reserves for body operation and to act as a key physical carrier mediating the transmembrane absorption of fat-soluble micronutrients (e.g., vitamins, plant polyphenols, and other functional

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factors) [1]. Regarding the actual absorption process in the intestine, newly hydrolyzed FFAs and MAGs are insoluble in water and cannot travel independently in the digestive juice. To overcome this transfer difficulty, they spontaneously aggregate with free bile salts and phospholipids in the intestinal lumen to assemble into mixed micelles at the nanoscale [8]. Next, pushed by Brownian motion, these tiny micelles cross the static water layer covering the outside of the intestinal epithelial cells. Once touching the cell surface, they enter the cell interior through simple passive diffusion or specific channel proteins on the membrane. After these lipid components successfully enter the cell, the endoplasmic reticulum re-esterifies them back into TAGs and further packages them into larger chylomicrons, finally secreting them into the mesenteric lymphatic vessels to truly join the systemic blood circulation [12].

3.3. Gut-liver axis metabolic feedback and pathological indicators

Normal lipid metabolism is fundamental to sustaining life, but once the rate of hydrolysis in the intestine gets out of control, FFAs in the blood will experience extreme peaks in a short time. In vivo animal experiments with a high-fat diet (HFD) clearly show that without a physical barrier to delay the digestion process, rapidly and excessively absorbed lipids will "deviate" and wrongly accumulate in non-adipose tissues like the liver, forming ectopic deposition [2]. The acute overload of intracellular FFAs induces severe endoplasmic reticulum stress and triggers continuous chronic inflammatory responses. From a pathophysiological perspective, these microscopic cellular damages are the core factors triggering central obesity, insulin resistance, and MASLD [2].

Furthermore, animal model data further indicate that the sudden massive influx of lipids into the intestine significantly disrupts the original gut microecological homeostasis. This alteration not only leads to a marked decline in the abundance of beneficial symbiotic bacteria but also severely impairs the ability of the microbiota to synthesize and secrete SCFAs [2]. As key signaling molecules maintaining the energy balance of the "gut-liver axis" and inhibiting hepatic de novo lipogenesis, a significant decrease in SCFA concentration will further worsen hepatic steatosis, thereby promoting an unbreakable vicious metabolic cycle [2]. Based on the above pathological mechanisms, utilizing complex Pickering systems in the proximal gastrointestinal tract to build degradation-resistant physical interfaces to flatten the FFA release rate has become an important intervention strategy to improve and reverse such metabolic disorders.

4. Regulatory mechanisms of lipid digestion

4.1. Steric hindrance and kinetic retardation

In colloidal kinetic models, the primary physical mechanism by which Pickering emulsions delay lipid hydrolysis stems from the significant steric hindrance generated by the high-density solid particle network [11]. Traditional monolayer protein interfaces are highly stretched in polar aqueous phases, typically only a few nanometers thick, and are easily penetrated by digestive enzymes. In contrast, protein-polysaccharide complex systems (e.g., self-assemblies of SPI with BC or inulin) can construct a dense viscoelastic shell ranging from tens to hundreds of nanometers thick around the O/W interface [7]. With this sturdy 3D barrier blocking the outside, lipases originally in the aqueous phase can hardly penetrate it, naturally making it difficult to access the internal lipid substrates. From the perspective of reaction kinetics, the dense arrangement of these particles at the interface greatly reduces the chance of effective collisions between lipases and lipids, directly raising the activation energy required for catalytic hydrolysis. Reflected in macroscopic test

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indicators, the adsorption rate of enzymes at the interface significantly slows down. Because of this, FFAs are not fully released "explosively" at once as in ordinary emulsions, but rather shift to a pattern similar to zero-order or first-order reactions, exhibiting a smooth, continuous, and controllable sustained release state [11].

4.2. Competitive adsorption and displacement energy barrier

When the chyme enters the duodenum, the real test begins. At this point, highly surface-active bile salts intervene in large quantities. For ordinary emulsions, bile salts only need to consume very low energy to easily "squeeze out" the proteins originally attached to the oil droplets, leaving the internal lipids completely unprotected [5]. However, complex Pickering emulsions can effectively resolve this interface reconstruction crisis thermodynamically; their secret lies in substantially increasing the detachment energy of surface particles [11].

Specifically, macromolecular polysaccharides (e.g., hydrophobically modified cellulose nanocrystals or chitin nanofibers), after non-covalent cross-linking with proteins, can form a high-strength interfacial network. This structure not only effectively locks the three-phase contact angle of the particles at the O/W interface but also significantly increases the mechanical work required for the complex particles to desorb from the interface [13]. Microscopic morphological scans from atomic force microscopy (AFM) and dynamic interfacial tension test data demonstrate that this rigid complex skeleton can effectively block the competitive penetration of bile salt molecules; even under Marangoni convection driven by interfacial tension gradients, this physical barrier can maintain high structural integrity without interfacial disintegration. In other words, because this structure resists the physical displacement action of bile salts, lipases lose the prerequisite of clearing interfacial inhibitors, thereby cutting off the pathway of rapid lipid enzymatic hydrolysis from the physical source [13].

4.3. Microgelation and physical entrapment

Not only does the structure of the microscopic interface provide protection, but the loosely adsorbed or free macromolecular polysaccharides in the complex system also exhibit macroscopic phase state changes when exposed to specific digestive tract microenvironments. Taking simulated intestinal fluid (SIF) as an example, free divalent cations (such as calcium and magnesium) and bile salts within it prompt the molecular chains of polyanionic polysaccharides like inulin and pectin to unfold and further undergo inter-ionic cross-linking. This interaction directly causes the entire system to experience in situ microgelation or secondary flocculation [7,14]. Through this phase reorganization, the free polysaccharides intertwine into a 3D gel network capable of covering multiple droplets. The unhydrolyzed lipid cores are thus deeply embedded inside these highly entangled polymer matrices. Constrained by this dense macroscopic network, the diffusion resistance of digestive enzymes in the external continuous phase significantly increases, and their speed of migration and penetration towards the lipid surface decreases substantially [14]. Meanwhile, the polysaccharide network itself carries negative charges and can bind large amounts of free bile salts and calcium ions through electrostatic interactions. This directly causes a significant decrease in the concentration of surface-active substances available to participate in emulsification and micelle formation in the intestine. Through the combined effects of substrate entrapment and cofactor depletion, the release amount of specific SCFAs and lipophilic molecules is further controlled [7].

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5. Application and translation of delivery systems

5.1. Functional foods and targeted delivery

Given the structural stability exhibited by complex Pickering emulsions during gastrointestinal digestion, this system has gradually been applied to the delivery research of high-value-added bioactive components. Taking a complex system composed of starch nanoparticles (SNPs) and chitin nanofibers (ChNFs) as an example, studies have shown that it can effectively encapsulate hydrophobic and highly light/heat-sensitive polyphenolic substances like curcumin [15]. The dense interfacial network provides a certain chemical protection for curcumin in the gastric fluid environment, reducing degradation and destruction by strong acids and proteases. Subsequently, in the small intestinal digestion stage, relying on the physical blocking effect of the interface, the transmembrane absorption rate of the active substances is improved. Relevant in vivo animal experiments have also confirmed that the encapsulated curcumin can follow the incompletely hydrolyzed lipids to reach the distal intestine. It can regulate the gut microbiota at the systemic metabolic level, promote the secretion of SCFAs, and thereby exert interventional effects on pathological indicators such as hepatic steatosis caused by an HFD [2,15].

5.2. Beverage formulation and rheological control

For the development of liquid nutritional products such as plant-based milk alternatives or foods for special medical purposes, controlling the suspension stability and rheological properties of the dispersion system is key to formulation design. Research has found that complex Pickering stabilizers can form a weak gel structure with shear-thinning characteristics in the continuous phase of the system. This not only increases the apparent viscosity of the liquid but also enhances the spatial repulsive force between droplets [1]. By changing the thermodynamic and kinetic states of the system, phase separation caused by gravity and Ostwald ripening phenomena are effectively suppressed [6]. Considering the retarding effect of this type of complex emulsion on lipid digestion, the food processing industry can utilize this characteristic to develop liquid food formulations with a low glycemic index (GI) or those that help stabilize blood lipid fluctuations for populations with special nutritional needs [11].

5.3. Industrial production and shelf-life verification

For lipid delivery systems to move from the laboratory to actual industrial production, they must possess the ability to adapt to complex processing environments, such as high-temperature treatment, mechanical shearing, and long storage periods. In an accelerated shelf-life study on egg yolk/fish oil complex emulsions, the addition of specific structural polysaccharides played a core role in maintaining system stability [3]. In the accelerated shelf-life test at 45 °C, the high-modulus polysaccharide-protein interfacial layer not only maintained the high convergence of the emulsion droplet size distribution but also acted as an excellent antioxidant physical barrier. This dense interfacial shell effectively delayed the diffusion rate of dissolved oxygen in the aqueous phase and blocked the contact pathways between pro-oxidants (e.g., transition metal ions) and internal lipids through the electrostatic shielding effect of the polysaccharide skeleton. Experimental data validated this physical barrier efficacy: within the first 4 days of continuous storage, the complex emulsion built with guar gum showed almost no significant generation of primary oxidation products (peroxides); at the end of the storage period (day 10), the content of propanal, a key secondary

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oxidation marker, was strictly suppressed at an extremely low level of 4.85 to 4.92 μg/g emulsion. This quantitative result confirmed the core role of the complex interface in slowing down the oxidation reaction rate and extending the storage life of functional lipids [3].

6. Conclusion

In summary, protein-polysaccharide complex Pickering emulsions effectively overcome the physical vulnerability of traditional monolayer emulsifiers in the gastrointestinal tract by constructing a high-modulus 3D network skeleton at the O/W interface. Relying on significant steric hindrance, an extremely high bile salt displacement energy barrier, and responsive microgelation characteristics in the intestine, this system substantially weakens the interfacial adsorption efficiency of lipase, successfully transforming the explosive hydrolysis of lipids into a controllable sustained release. The systematic exploration of this mechanism strongly echoes the original intervention intention of stabilizing lipid absorption through food physical structure engineering. The physical barrier regulation insights proposed from the perspective of interfacial colloids not only provide an underlying mechanistic explanation for the targeted delivery of bioactive factors but also offer reliable formulation design references for the industrial creation of novel functional foods and liquid beverages that stabilize blood lipids and regulate metabolism. However, current mechanistic demonstrations in this field still heavily rely on in vitro static simulations or basic animal models; verifications of interfacial evolution laws under real dynamic gastrointestinal stress in complex human bodies, as well as exact metabolic pathways, still have limitations. Future research should prioritize combining human clinical trials with in situ dynamic tracking technologies to accurately map the complete digestive fate of complex systems in vivo. Meanwhile, deeply exploring the targeted regulation mechanisms of specific polysaccharide configurations on gut microbiota will be a core direction driving the implementation of next-generation precise nutritional intervention systems.

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