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Review Article

Odland bodies: A Review

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***ABSTRACT*:**
Odland bodies, are small sub-cellular organelles of size 200-300nm.Odland bodies are otherwise known as lamellar bodies, membrane coating granules, lamellar granules and keratinosomes. They are present in the upper spinous and granular cell layers of the epithelium. They are made of glycolipids. They have lamellated internal structure. They are formed near the Golgi apparatus in the cell, migrating to the cytoplasm and discharging its contents into the intercellular space forming the permeability barrier. They act as vital multi-functional constituent of epithelium. They also act as processing and repository areas for lipids that contribute to the epithelial permeability barrier. They contain cathepsin D, kallikrein, proteases and other proteins including corneo-desmosins. Odland bodies play an important role in maintaining homeostasis as they are involved in epithelial permeability barrier function, formation of the cornified envelope, desquamation of keratinocytes, and in local anti-microbial immunity. Recent studies also reveal that they play a role in the local innate immune response as they contain beta 2 defensins, which are anti-microbial peptides with potent activity against candida and Gram-negative bacteria. This article reviews the structure, formation and functions of the Odland bodies.
Key words: Lamellar bodies, stratum granulosum, spinous cells, stratum spinosum.

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1. Introduction:

Lamellar bodies also known as Odland bodies, membrane coating granules, keratinosomes, corneocytes and cementosomes are tubular and/or ovoid-shaped membrane -bound secretory organelles of the epidermis. In the initial years of electron microscopy Selby (1957) first observed the lamellar bodies in the stratum granulosum layer of human epidermis but believed it to be small keratohyalin granules[1]. The same structures were also noticed by Horstman (1958) on rat food pad epidermis who interpreted it as virus particles [2]. Odland (1960) on his study found these granules that made their appearance in the upper spinous and lower granulosum layers but disappear in those cells in which keratohyalin granules advance and advocated that these granules were "attenuated mitochondria" [3]. Frei and Sheldon (1961) unlike Odland suggested to refer these granules found on mouse epidermis as "corpuscula "and believed them to be either viral substances or secretory granules[4].Matoltsy and Parakkal (1965) first identified the role of these granules in the formation of

permeability barrier that withstand the flow of solutes between the cells of stratum corneum and referred them as "membrane coating granules"[5].

2. Structure:

Odland bodies are sub-microscopic membrane limited ovoidal organelles which is made up of complex system of lamellar structures [6]. The discrete lamellar units consist of three parallel membranes parted from each other by a gap of 55 angstroms. Odland bodies are ovoid in shape, 200-300 nm in size, made of glycolipids and they are secretory organelles with a membrane bilayer as shown in the figure 1. They do not occur in the basal cell layer of the epithelium and are seen in upper stratum spinosal and granulosal layers. In keratinized epithelium Odland bodies are elongated and contain a series of parallel lamellae acts as effective barrier; keratohyalin granules are associated with tonofilaments. In non-keratinized epithelium Odland bodies are circular with amorphous core, forms comparatively less effective barrier; keratohyalin granules are not associated with tonofilaments.

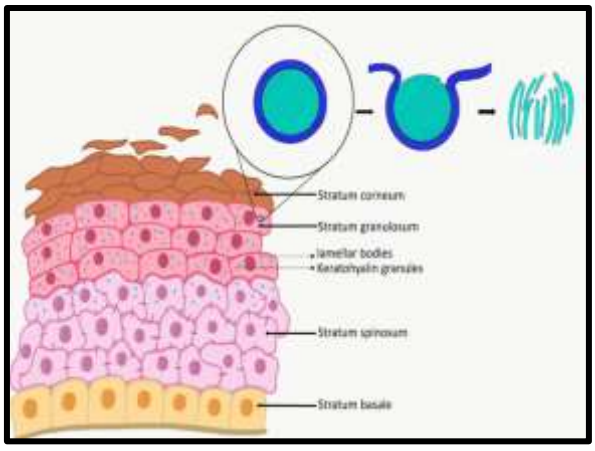

Figure 1: Layers of epidermis and the structure of Odland bodies.

3. Formation:

Odland bodies are formed as discrete granules in the upper stratum spinosum and in the stratum granulosum layers , most likely from the Golgi apparatus, and then move to the cell surface of the granular cell layer, fuse with the plasma membrane, and extrude their contents into the intercellular space forming permeability barrier(intercellular lamellar material) as shown in the figure 2. Damage to the permeability barrier induces rapid secretion of the contents of the Odland bodies in the outer granulosal cell layers resulting in a marked decrease in the number of Odland bodies (50-80%). Soon thereafter newly formed lamellar bodies appear in the stratum granulosum cells until permeability barrier function is restored [7]. Odland body synthesis is regulated by the ambient calcium gradient in the upper granular cell layer [8].

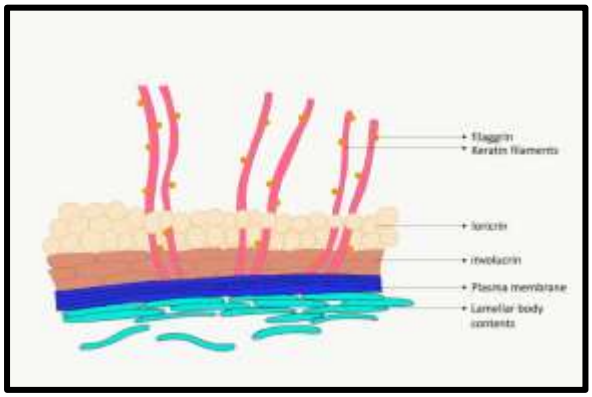

Figure 2: Formation of permeability barrier by the secreted lamellar body content.

4. Secretions and Functions of Odland bodies:

The structure of stratum corneum barrier is often referred as the brick and mortar model. The keratin filament is considered as the bricks and the lipid matrix as mortar. The lipids are the major component in the formation of the permeability barrier and they are found to be secreted by Odland bodies [9].

Over the years, a plethora of cytochemical, immune-electro microscopic investigations were carried on epidermal lamellar bodies which revealed the contents of lamellar bodies. Lamellar bodies contains-lipids, lipolytic enzymes, lysosomal enzymes and proteases which are essential for the formation of permeability barrier. The lipids seen in lamellar bodies are glucosylceramides, phospholipids, sphingomyelinase and cholesterol and they are polar in nature. In addition to these lipids, abundant enzymes are also found in lamellar bodies most of which are lipid hydrolases which converts the polar lipid precursors into non polar lipids extracellularly [10] as depicted on table 1.

Lipid precursor	Enzyme	Non polar lipid	functions
Glucosylceramides	β-glucocerebrosidase	Ceramide	Cohesion, antimicrobial, barrier function
Glycerophospholipid	phospholipase	FFA	
sphingomyelin	acidic sphingomyelinase	ceramide	

Table 1: List of lipid precursor, the enzymes that hydrolyze them into their corresponding non polar lipid derivative and their functions.

Other enzymes that are found in lamellar bodies are proteases such as cathepsin, glycosidase which are crucial for desquamation (listed in table 2). A few protease inhibitors are also found in lamellar bodies liked serine protease inhibitors which has an inhibitory effect on serine protease that holds the corneocytes together and thus helps in desquamation.

Enzymes	Function
Proteases	
Cathepsins	Desquamation
Glycosidases	Desquamation
Chymotryptic enzymes	Degrades corneo-desmosomes
Protease inhibitors	
Serine protease inhibitor	Desquamation
Cysteine protease inhibitor	Cytokine activation

Table 2: Proteases and protease inhibitors secreted by lamellar bodies and their function

The major role of the lamellar bodies is to provide lipids and lipolytic enzymes for the formation of permeability barrier but apart from they also play roles in keeping the stratum corneum layer well hydrated. Glycerol is formed from phospholipid found in lamellar bodies and this glycerol has a water holding capacity.

5. Diseases associated with Odland bodies:

Formation of permeability barriers requires Intercellular lipids – ceramides ,cholesterols and free fatty acids which are non-polar and are secreted from epidermal lamellar bodies at the stratum granulosum -stratum corneum interface .These non-polar lipids are either secreted directly or as polar lipid precursors which by action of several hydrolytic enzymes also secreted by Odland bodies gets converted to a less polar lipids .Disruption in metabolism and transport of these lipids will lead to formation of a dysfunctional permeability barrier.

5.1 Ichthyoses

"Ichthys" a Greek term refers to fish and ichthyoses also called "disorders of cornification" refers to disorders of skin that resembles scales of fish.

Ichthyoses is associated with Odland body secretions and defects

5.1.1. X linked ichthyosis

Cholesterol sulfate is formed and stored in the Odland bodies .The cholesterol sulfate are seen in the highest concentration in the stratum granulosum .Steroid sulfatase is microsomal enzymes that disulfates cholesterol sulfate to provide free cholesterols to the formation of the permeability barrier which prevents water loss and the enzymatic activity persists even in the stratum corneum which breaks the adhesion between the corneocytes which ultimately results in desquamation. Absence of the enzyme steroid sulfatase is seen in X linked ichthyosis and this leads to increased cholesterol sulfate in stratum corneum and alters desquamation which appears clinically as dry and scaly skin. [11,12]

5.1.2. Netherton syndrome

Netherton syndrome is a rare form of autosomal recessive form of ichthyosis occurs due to mutation in the SPINK5 gene which codes for the protein LEKT1 .LEKT1 is a serine protease inhibitor and in normal human epidermis this limits the action of serine protease especially in the layer stratum corneum and thus helps in breaking the connection between the corneocytes which will lead to desquamation. But in Netherton syndrome due to deficient serine peptidase inhibitors normal skin shedding i.e. desquamation does not occur which clinically will manifest as dry, flaky and red skin also called "ichthyosiform erythroderma" [13]

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5.1.3. Harlequin ichthyosis and lamellar ichthyosis

Harlequin ichthyosis is a congenital skin disorders caused due to mutations in ABCA12 gene. ABCA is a subclass of ABC transporter protein family which is required for the active transport of lipids across cell membranes. Functional impairment of ABAC12 gene due to mutation will lead to improper formation of Odland bodies. Clinically harlequin ichthyosis is manifested as armour like plates with fissure and ectropion (outward turning of eyelids),lamellar ichthyosis occurs due to partial dysfunction of ABCA12 gene caused by mutation, unlike harlequin ichthyosis ,lamellar ichthyosis manifests clinically only as dry and scaly skin. [14,15]

5.2 Gaucher's disease

Gaucher's disease is a rare disease caused by deficiency of lysosomal enzyme beta glucocerebrosidase. This enzyme catalyzes the hydrolyzation of glucoceramides to ceramides. Gaucher's disease subtype II is caused when mutation occurs in glucocerebrosidase gene which eventually leads to a decline in ceramide required for the formation of the epidermal barrier leading to a flawed barrier. Gaucher's type II disease is dermatologically manifested usually as dry and scaly skin but in recent years cases has been reported on Gaucher's disease which manifest as collodion skin [lamellar desquamation] [16,17]

5.3 Niemann-Pick disease

Niemann-Pick disease is an inherited lipid storage disorder which occurs due to deficiency of enzyme acidic sphingomyelinase, this enzyme

sphingomyelinase is essential for the conversion of sphingomyelin to ceramides which forms the permeability barrier. Based on the genetic cause and signs and symptoms Niemann-Pick disease is classified as type A, type, type C1 and type C2.mutations in the sphingomyelinase gene give rise to type A (fatal) and type B (non-fatal-thrive to adulthood) which eventually will exhibit as abnormal permeability barrier function, dermatological manifestation of this disease is rare but can present as fatty irregular patch on skin commonly called as "xanthoma" [18].

6. Conclusion:

The epidermal permeability barrier also known as the skin barrier is what keeps our body safe from many environmental insults and prevents loss of water from our body to environment. Odland bodies secretes lipids and enzymes which are vital for the formation of permeability barrier and any mutations on genes that plays a role in the formation of these lipids will lead to a substandard permeability barrier. Knowledge on Odland bodies are essential for physicians who treat various skin diseases.

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