Chapter 1

The Decomposition of Human Remains

A Biochemical Perspective

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1. Introduction

The end result of decomposition of humans is more intimately familiar and perhaps of greater interest to forensic pathologists than to any other group whose duties include the evaluation and investigation of postmortem remains on a routine basis. From such remains, the forensic pathologist may be asked to make an evaluation of the cause and manner of death and, perhaps, how long the body had been *in situ*. These determinations may be challenging, even for the experienced investigator, depending on the condition and location of the remains. The extent, pattern, and nature of decomposition in a specific circumstance may be of great significance and utility in the forensic investigation of a death. Conclusions and inferences drawn from the investigation can be the subject of scrutiny, consideration, and documentation.

Clearly, an understanding of the processes of decomposition can be of benefit for such purposes as estimation of the postmortem interval, recognition of postmortem artifacts, and in an overall evaluation of the death scene. For the forensic pathologist faced with the even more complex issues associated with partial remains, such as the lower extremity, knowledge of the mechanistic processes of decomposition may facilitate an understanding of the specific circumstances of the death in question.

Because the physical appearance and sequence of decomposition events has been extensively detailed and reviewed in the forensic literature, the focus of this chapter is to explore the biochemical reactions and processes that provide the ultimate basis for

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the visible (or otherwise detectable) evidence of the decomposition with which we regularly deal with in medicolegal contexts.

There are many macroscopic processes or events that can impact the fate of postmortem remains in an unpredictable and variable nature, such as physical disruption, scavenging, and deliberate or accidental burial. However, the common underlying sequence of biochemical events provides a basis for understanding decomposition as a logical progression of natural processes. It is anticipated that an enhanced understanding of the biochemistry of decomposition of human remains will be useful to some investigators, and facilitate the evaluation, and extraction of useful information from the seemingly unpredictable and chaotic processes lying at the center of a crime or death scene.

Life can be viewed in its essence, as an energy-utilizing sequence of interrelated chemical reactions whereby order and structure, of truly magnificent scope and complexity, is derived from an intricate and multifaceted process of breakdown, combination, synthesis, and modification or rebuilding of biomolecules from an otherwise random or partially organized collection of both simple and more complex available molecules, ions, and atoms. The maintenance of life—or more accurately, the maintenance of the underlying order, as reflected in molecular and macromolecular structures—requires the continued input and utilization of energy. The interlinked reactions of life comprise overall, an endothermic process both in total and on an individual basis. Clearly, the termination of a life results in the cessation of the flow of energy available to maintain, support, and reproduce the molecules, structures, and biochemical processes of the organism.

Organic biomolecules are—as considered from the temporal perspective of days, months, or years—extremely fragile and ethereal constructions, readily broken apart by relatively small amounts of thermal, electrochemical, or electromagnetic energy. Within living organisms, the molecules of life are primarily "reduced," in the chemical sense, in that they are hydrogen-rich and able to react with molecular oxygen either directly or via intermediate molecules as electron donors or "reducing agents." Indeed, a major form of energy storage in living cells is long-chain carbohydrate molecules. The energy potential of such molecules, which is released upon oxidation, is readily demonstrable in the burning wax of a candle, the fat stores of animals preparing for winter hibernation, and the utilization of geochemical (although essentially biological in origin) oil and its refinement product, gasoline.

Despite the reduced state of most biomolecules, our earthly environment provides an oxidizing atmosphere and (generally) oxygenated aqueous ecosystems. In such a system, the ultimate fate of organic molecules is the corresponding oxidation products of component atoms. Hydrogen yields water and other oxidized products such as hydrogen sulfide. Carbon ultimately yields carbon dioxide. Nitrogen and other elements yield corresponding oxide forms. The facility with which organic molecules are able to transfer single electrons to molecular oxygen provides the underlying basis for aerobic life, given that the energy of that transfer is captured and made available to the organism in the form of high-energy molecules, such as adenosine triphosphate (ATP).

The relative fragility of organic molecules necessitates active biological structures to maintain some degree of flux at the molecular level, in that the molecules from which such structures are comprised are always degrading (albeit at different rates). Damaged or degraded molecules are then constantly being replaced by newly synthesized (or otherwise

acquired) versions of the same molecule (with the rate of repair dependent to an extent on the health and age of the individual). Complex and elegant enzymatic systems exist to perform the functions of evaluation, repair, and/or replacement of biomolecules.

As a consequence of death, the molecules created by the organism that serve as the building blocks of its corporeal body, internal structures, and organs—both on the macro and micro scale—must, as noted above, ultimately undergo oxidation and degradation in an environment where the normal repair and replacement mechanisms are no longer operative. The process in total, whereby the structures and biomolecules of an organism become less organized or broken down—either by chemical or biochemical processes—and the constituent molecules and atoms are made available to new life, is referred to as "decomposition." The extent to which this process is dependent on the chemical nature of the surroundings is readily illustrated by the recovery of well-preserved specimens—both human and otherwise—from bogs (or, more accurately, peat excavations of ancient bogs). The subsurface ecosystem of a bog is severely oxygen depleted and acidic, resulting—for any unfortunate individual trapped therein—in a preservative entombment that precludes normal molecular oxidative degradation and will not support the aerobic life forms (nor many anaerobes) necessary for a complete decomposition process.

In contrast to the synthetic functions of the living organism, postmortem decomposition of the body is reflective of a collection of physical and degradative biochemical processes that will be situationally dependent for any given body. To an extent, the temporal sequence, and even occurrence, of particular decomposition events in a specific situation will be the result of the combined effects of environmental conditions and the physical setting of the body, as well as the physical actions associated with death. Similarly, the rate at which changes will occur—correlating with the physical state of the remains at any point—will also be a function of those circumstances. With the exception of physically disruptive processes, decomposition is essentially a biological and biochemical phenomenon, mediated by enzymes that are already present in the body, by digestive enzymes and the activities of exogenous flora and fauna colonizing the remains. All of the processes are driven by the stored chemical energy that the decomposing body represents. This chapter is devoted to understanding—in a general but mechanistic and biochemical sense—the nature of the decomposition processes to which remains of the lower extremity may be subjected.

2. *The Decomposition Sequence*

In the absence of physical disruption and in unfrozen, unpreserved tissue, the processes of decomposition follow a reasonably predictable pattern. The breakdown of body tissues consists primarily of two processes: autolysis and putrefaction. *Autolysis* is an aseptic phenomenon caused by the release and subsequent uncontrolled activity of intracellular enzymes that hydrolytically break down cellular constituents that may serve as catalytic substrates. The autolytic process sets the stage for the subsequent massive transformation of previously (more or less) solid tissue to gas, liquid, and salt products during the septic process of putrefaction.

In contrast to autolysis, *putrefaction* is the consequence of a "population explosion" of xenobiota in the body, as various organisms (both micro- and macrobiological

endogenous and invasive plants, animals, and fungi) compete for the available energy the decomposing tissues represent. Although the difference between autolysis and putrefaction may be relatively clear and definable on a molecular level, the distinction between the processes in an actively decaying corpse may be considerably less so, with both functions occurring to some extent simultaneously.

Although recognizing that there may be significant variations in the time course of the decompositional process within any specific corpse, generally five stages of decomposition have been identified in unpreserved bodies:

1. Fresh (~0–2 d): Begins at the moment of death and includes the autolytic processes. Usually relatively few changes are readily observable on a macrobiologic scale. This stage ends with the beginning of putrefaction. Generally, insect infestation and utilization of the corpse is not extensive at this stage.
2. Bloated (~2–6 d): Begins as the processes of putrefaction start to produce enough gaseous byproducts to start inflating the abdomen and other soft tissue. This activity reflects the geometric rates of growth of invasive and/or opportunistic organisms—primarily anaerobic bacteria—in the corpse. Insect activity may be significant during this stage and particularly enhanced as fluids start to seep from the body.
3. Decay (~5–11 d): Begins as the skin of the abdominal wall ruptures or is otherwise breached, thereby allowing the release of trapped putrefactive gases. This process may be facilitated by insect feeding or other scavenger activities. The body may appear moist and blackened. During this stage, a significant loss of soft-tissue mass will occur, primarily as a consequence of maggot feeding activity. Toward the end of the decay stage, the body will begin to dry and the insect population may shift somewhat toward scavenging and predatory beetles.
4. Postdecay (~10–25 d): The nature of the postdecay process is a function of the degree of moisture available in the immediate surroundings of the body. In dry climates, remains will be primarily bones, dried skin, and cartilaginous materials. In moist regions, byproducts of decomposition may remain in the vicinity of the body (e.g., in adjacent soil) for an extended period of time.
5. Dry state (~>25 d): This is generally defined as the stage at which only bones and hair remain, and no significant odor distinct from normal soil or forest duff is readily discernable.

2.1. Autolysis

Autolysis is the "self-digestion" of the cell. In the context of postmortem decomposition, it refers to the process by which catabolically active enzymes are able to act on cellular organelles and molecular components that would not normally serve as substrates. The release of these enzymes from their subcellular locations marks the beginning of an irreversible process that will eventually result in the complete reduction of the newly dead organism to the remnants of decomposition, available to serve as food for other life forms that can derive energy and nutrients from the decaying corpse. The potential for the autolytic, enzymatic breakdown of cellular biomolecules exists in every cell as a consequence of the presence of the biochemical machinery necessary to process nutrients, degrade toxic species, and recycle structural and functional molecules and, indeed, entire cellular organelles. That tissues differ in rates of autolytic processes can be understood in terms of differential enzyme complements and reflects the functional

distinctions of the tissues. Hence the liver, with a broad spectrum of highly active catabolic enzymes, undergoes rapid autolysis, whereas tissues with more limited biochemical activity, e.g., muscle, tend to degrade more slowly.

Interestingly, the essentially aseptic nature of the autolytic process can be understood on reflection that the "aging" process used to enhance the flavor and texture of certain meats and game produce a tenderized yet not microbially contaminated product. The operation of autolytic processes in a manner that is protected from either infection or other means of contamination is primarily responsible for the change in physical properties of the muscle mass.

Clearly some mechanism for isolating or segregating the activities of catabolic enzymes with generalized substrate capability is required in the cell, because the maintenance of intracellular structures is in the interest of the organism. The primary segregating elements for catabolic enzymes and processes within the cell are the lysosomes, peroxisomes, and, to a lesser extent, mitochondria. An understanding of autolysis and hence of the entire decomposition process requires familiarity with the enzymatic complements and mechanism for the release of enzymes from these subcellular organelles after the death of the organism *(1,2)*.

Subcompartmentalization of catabolic enzymes within the lysosomes, peroxisomes, and other subcellular vesicles protects the operating molecular machinery of the cell from the degradative potential of these enzymes. In addition to the limitation of activity by intracellular segregation, there is elegant additional protection of intracellular constituents. The activity of the lysosomal hydrolases is optimal at an acidic pH (~5.0), which is significantly distinct from the somewhat more basic pH (~7.2) of the surrounding cytosol.

With the death of the organism and the associated circulatory failure, there is a concomitant failure of oxygen transport and delivery to cells. Molecular oxygen serves as the terminal electron acceptor in the electron transport chain in biochemical reactions known collectively as oxidative phosphorylation. This sequence of connected reactions is the primary source of high-energy ATP molecules in the body, which in turn provide the energy for a multiplicity of cellular functions by hydrolysis of phosphate ester linkages *(3)*. A consequence of oxygen deprivation is the failure of oxidative phosphorylation to take place, causing a shift in cellular metabolism favoring anaerobic glycolysis, a fermentative process which acts to compensate for the energy deficit. Anaerobic glycolysis results in the conversion of glucose to pyruvate and eventually to lactate. The elevation in lactic and pyruvic acid levels in the cell causes the intracellular pH to decline and the intracellular buffering capacity to become quickly overwhelmed. As the glycolytic process continues, intracellular glucose is rapidly depleted, as is the glucose polysaccharide glycogen; thus, it eventually deprives the cell of even this limited resource of ATP production. Anaerobic glycolysis is less efficient than oxidative phosphorylation, in that it leaves an incompletely oxidized product (lactate) and it is further limited to the available substrate, endogenous glucose, and molecules that can be readily converted to glucose, e.g., glycogen *(3)*.

The consequences of the limitation in ATP synthesis are multifaceted and ultimately devastating for the cell. Many cellular transport mechanisms depend on ATP to provide the energy required to drive energetically unfavorable processes. Many nutrients and other molecules essential to the life of the cell are actively transported across

cellular membranes by mechanisms that require ATP hydrolysis. The cellular membrane potential, ranging from approx –10 mV to as much as –90 mV (depending on the tissue or cell type) is also maintained by the action of the sodium–potassium ATPase pump; termination of the activity of this structure allows intracellular sodium to accumulate while potassium diffuses out of the cell through permanent membrane ion channels *(4)*. As the membrane potential disappears, calcium ions enter the cell (a key indicator and perhaps contributor to the impending death of the cell). The intracellular accumulation of solute ions occurs concomitantly with cell swelling, a function of an increase in the intracellular water content, which is driven by osmotic pressure. Typical necrotic changes in the cell, such as vacuolization and dissociation of cellular organelles, further characterize the cellular demise. Of major significance for autolysis is the disruption of the lysosomal membrane consequential to intracellular acidification and ionic changes. The leakage of the lysosomal acid hydrolases into an acidic environment at a now near-optimal pH for hydrolytic activity facilitates the enzymatic breakdown of cellular components and membranes.

Lysosomes have a single limiting membrane, and the intravesicular pH is maintained at approx 5.0 (corresponding to the optimal pH for hydrolytic enzymes) by a membrane-bound hydrogen ion pump. Lysosomes typically contain a broad spectrum of enzymes capable of hydrolytically cleaving polysaccharides, proteins, nucleic acids, lipids, phosphoric acyl esters and sulfates (Tables 1 and 2). Lysosomal action is primarily mediated as a consequence of the fusing of a primary lysosome with an intracellular vesicle produced via phagocytosis (for extracellular materials) or by the analogous budding of an intracellular membrane (for intracellular materials). The fused product is referred to as a *digestive vacuole*, and it is in this protected environment that complex biomolecules are hydrolytically "de-constructed" *(5)*.

As previously noted, the capability of the cell to rapidly degrade molecules of significant size and complexity requires compartmentalization and segmentation of the process. The integrity of the lysosomal membrane also prevents the unwanted destruction of other intracellular components, the loss of which could have a negative impact on the viability of the cell. Clearly a loss of lysosomal membrane integrity can result in the appearance within the cytosol of a significant and indiscriminant hydrolytic function with the potential to damage cellular organelles, membranes, and other important biomolecules.

Peroxisomes function primarily in the breakdown of lipids, with long chain fatty acids (>20 CH_2 groups) processed essentially exclusively within these organelles. Medium-chain fatty acids (~10–20 CH_2 groups) may be degraded in either mitochondria or peroxisomes. Although the reactions of the mitochondria and peroxisome are similar in many respects, some significant differences serve to point out the functional distinctions. The mitochondrial oxidative phosphorylation process oxidizes flavin adenine dinucleotide, reduced $(FADH)_2$, to yield FAD, with $FADH_2$ regenerated by the oxidation of a fatty acyl CoA molecule. In contrast peroxisomal $FADH_2$ is generated by the same enzyme-catalyzed reaction but oxidized in the process of the reduction of molecular oxygen to hydrogen peroxide, a potentially cytotoxic molecule *(1)*.

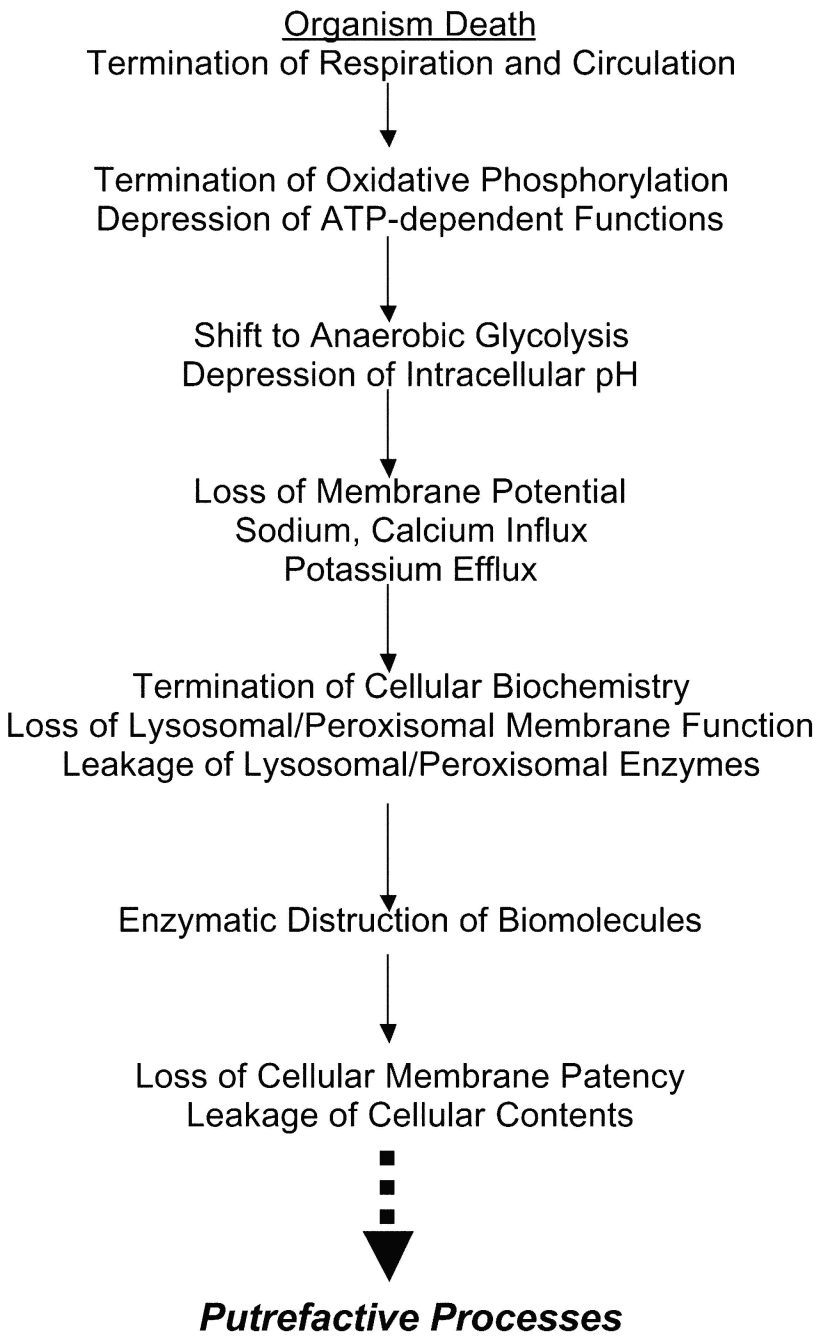
Peroxisomes contain significant quantities of the protective enzyme catalase, the activity of which breaks hydrogen peroxide down to water and oxygen. In the peroxisome, unsaturated fatty acyl CoA molecules are converted to the hydroxy analogs, which

Table 1
Lysosomal Enzyme Classes: Examples and Typical Substrates

Enzyme Type	Typical Substrate
Lipid Hydrolases:	
Lipases	Triacylglycerol esters
	Cholesterol esters
Esterases	Fatty acyl esters
Phospholipases	Phospholipids
Nucleic Acid Hydrolases:	
Ribonuclease	Ribonucleic acids
Deoxyribonuclease	Deoxyribonucleic acids
Phosphatases:	
Phosphatase	Phosphomonoesters
Phosphodiesterase	Phosphodiesters
Polysaccharide Hydrolases:	
α-*Glucosidase*	Glycogen
α-*Flucosidase*	Membrane fucose
β-*Galactosidase*	Galactosides
α-*Mannosidase*	Mannosides
β-*Glucuronidase*	Glucuronides
Hyaluronidase	Hyaluronic acid
	Chondroitin sulfates
Arylsulfatase	Organic sulfates
Lysozyme	Bacterial cell walls
Protein Hydrolases:	
Cathepsins	Proteins
Collagenase	Collagen
Elastase	Elastin
Peptidases	Peptides
Sulfatases:	
	Heparan sulfate
	Dermatan sulfate

are subsequently oxidized to their corresponding ketones by means of hydroxy fatty acyl dehydrogenase with the concomitant formation of NADH. In the absence of an active electron-transport chain and associated cellular synthetic processes, there is no metabolic "sink" for the reducing equivalents and nicotinamide adenine dinucleotide is exported to the cytosol. Therefore, peroxisomal catabolism of fatty acids represents a source of both acetyl CoA and reducing equivalents in the form of

Table 2
Autolytic Events: Cellular Death



nicotinamide adenine dinucleotide. Significant for the autolytic process, the enzymatic systems contained in the peroxisome represent the catabolic potential for fatty acids and also for the production of active oxygen species, e.g., hydrogen peroxide. The peroxisomal membrane suffers the same fate in the necrotic cell as the lysosome, with the leakage of its enzymatic machinery into the cytosol, where it becomes available to further catalyze the destruction of cellular components.

Failure of respiration and hence, cellular oxidative phosphorylation, is therefore the key trigger in the autolytic process. Termination of the availability of high-energy molecules that are routinely required to maintain the integrity of the cell, key cellular components and processes (e.g., membranes), synthetic capability, and ion and molecular pumps causes significant changes in the biochemical operation of the cell. This process ultimately leads to the lysis of intracellular organelles, of particular significance being lysosomes and peroxisomes, and the release of their constituent enzymes into the cytosol, where their catalytic actions can break down and destroy the very molecules that had previously served to define the living cell and functions in it.

2.2. Rigor Mortis

The postmortem depletion of cellular energy stores leading to autolysis also produces a well-recognized macro-scale phenomenon characterized by the stiffening of voluntary and involuntary muscles, known as rigor mortis. Mechanistically, this process is the result of association of the muscle proteins actin and myosin as intracellular pH decreases to less than approx 6.5 and calcium—normally sequestered in the sarcoplasmic reticulum (SR)—leaks into the cytosol as the SR membrane is compromised during autolysis. Cytosolic calcium then binds to troponin, causing a conformational change that results in the "unmasking" of myosin binding sites on the actin molecule. In living cells, the subsequent dissociation of the actin–myosin complex is promoted by ATP as part of the normal sequence of events that results in muscle contraction *(6)*. However, in the ATP-deficient postmortem environment in dead or dying cells, the actin–myosin complex remains until it is either denatured or enzymatically degraded *(7)*. The process causes the "death stiffness" of muscles, or rigor mortis. In contrast to active muscle contraction, there is no process of actin–myosin translocation in rigor mortis and, hence, no shortening of muscle fibers. Thus, rigor is characterized by muscles that are stiff but not contracted. Although rigor mortis occurs in the muscles, it is readily detectable only when the affected muscles are connected to central joints, such as the knees.

2.3. Livor Mortis

Lividity is a discoloration of the skin—generally to a dark purple—that results from the pooling of deoxygenated blood in the veins and capillary beds of the body as circulation fails. This process occurs in direct response to gravitational forces. The blood remains fluid after death as a consequence of the release of plasmin, a fibrinolytic enzyme, from the vasculature and serous surfaces. This process depletes the blood of fibrinogen, thereby eventually rendering the blood permanently incoagulable (~30–60 min after death, depending on the ambient temperature). Blood, by providing an ideal liquid growth medium, facilitates the rapid growth of xenobiotics during the putrefaction stage and serves as a conduit for the spread of microbes throughout the body.

The characteristic color of body surfaces during lividity is reminiscent of cyanosis, i.e., the bluish discoloration of skin, nail beds, and mucous membranes that develops in clinical settings as a consequence of inadequate oxygenation. In the postmortem environment, the continued kinetically driven dissociation of oxygen from the hemoglobin molecule continues, changing the absorption spectra of the blood and producing the characteristic and readily observable change in color *(7)*.

2.4. Putrefaction

The result of autolysis is the development of a slightly acidic, anaerobic, nutrient-rich environment, with significant degradation of biomolecules at the cellular level. In this fertile milieu, devoid of normally protective and defensive cells and barriers, the proliferation of both invasive and opportunistic endogenous micro-organisms can be rapid and extensive. Ultimately, bacterial growth can affect and transform all the tissues in the body, being limited only by environmental factors, such as temperature and humidity.

The decomposition processes that begins as bacteria proliferate results in the production of gases and other metabolic products. These consequences of microbial growth result in some of the characteristic color changes, bloating, and odor changes that are universally recognized as the hallmarks of a decaying body and are collectively referred to as *putrefaction.* On the molecular level, the actions of microbial degradation transform the complex biomolecules of the body into gases, liquids, and simple molecules. Putrefaction results in the complete (albeit gradual) loss of structural integrity and recognizability of tissues and, indeed, the ultimate reduction of those tissues into their component molecules, molecular fragments, and atoms.

The primary source for the opportunistic microbiological colonization in the decomposing body is the microbially rich environment of the gastrointestinal tract. These enteric micro-organisms can cross the failing membrane barriers, a process that is facilitated by the autolytic degradation of body tissues, and migrate and proliferate throughout the body. Hence, the pronounced impact of autolytic processes on the structural integrity of the cellular membranes and the end of the viability of regular "defensive" or protective cells (e.g. macrophages and neutrophils) as a function of pH changes and the loss of available oxygen, provides the basis for the population explosion the putrefactive period represents. Characteristic microbial species observed during putrefaction include various Bacilli and *Pseudomonas, Bacterioides fragilis, Eschericia coli, Clostridium perfringens, Proteus mirabilis, Staphylococcus epidermidis,* and *Staphylococcus faecalis (8).*

Although the primary source of anaerobes in decomposition processes is the intestinal tract, other organisms—such as those found in the respiratory tree—may be present and able to take advantage of the conditions for growth. Naturally, any significant antemortem infection (e.g., septicemia or pneumonia) will give the causative agent a "head start" on the putrefactive process and may therefore result in an unusual microbiological population, at least during the initial stages of decomposition. The rate of putrefaction will vary with temperature, which will affect primarily the rate of enzymatic activity, with acceleration occurring until temperatures become inconsistent with the maintenance of protein structures.

In the absence of a septic condition, putrefaction begins in the stomach and intestines. The gastric mucosa and intestines acquire a dark purple-brownish color as a

result of the release of heme compounds. The mucosal epithelium of the airways becomes deep red, and a hemolytic plum coloration may be noted in the myocardium and large blood vessels, again a result of the release of heme. Changes in organ structure are readily apparent, as seen in the thinning and softening of the myocardium. The liver develops a honeycomb pattern as a result of extensive gas formation. The brain similarly goes through a structural disintegration process that may proceed to complete liquefaction. The spleen becomes exceptionally soft and may extrude through its delineating capsule. The lungs become filled with and surrounded by fluid.

Putrefactive processes are generally first represented by the generation of a greenish color—a consequence of the formation and accumulation of sulphhemoglobin in the abdominal wall where it coincides with the large intestine. Eventually, the discoloration spreads over the entire abdominal wall and may extend over the entire body. Coincident with this color change is the appearance of the superficial veins of the skin as a pattern of lines often described as "marbling." As the process continues, the skin eventually acquires a dark pigmentation that may range from a red-tinged greenish color through purple to black.

The skin color changes are accompanied by structural disintegration of the tissue that results in the characteristic skin-slippage that accompanies the process of decomposition. Large sections of epidermis may be dislodged as a consequence of even a small amount of shear. The newly exposed basal layers appear moist and pinkish and may take on a yellow–tan parchment appearance when they dry. Blisters as large as 20 cm may develop. These blisters are generally filled with a dark fluid and gases of putrefaction and may be easily disrupted to expose a dermal surface similar to that seen as a result of skin-slip.

Putrefaction is often characterized by pronounced bloated, distended bodies as a consequence of the formation of gas in the stomach, intestine, and abdominal cavity. The gas, a consequence of microbial action, is composed of hydrogen sulfide, methane, carbon dioxide, ammonia, and hydrogen, and is responsible for the characteristic odor of putrefaction, along with low molecular-weight organic compounds, including mercaptans, indoles, and the aptly named cadaverine and putrescine. This gas invades all body tissues and causes a generalized swelling to occur, which is characteristically crepitant to palpation. The pressure generated by the evolution of this gas may contribute to the separation of necrotic tissue layers.

At the molecular level, it is the ability of the bacterial species to secrete enzymes into their immediate environment that provides both the basis for the delivery of nutrients back to the microbe, yet also results in the degradation of biomolecules in the vicinity of the organism. These *exoenzymes* are responsible for the significant denaturation and breakdown of proteins into their constituent amino acids, which may be taken up and utilized, or further catabolized by the microbial population. The gas that characterizes the putrefactive process arises as a direct consequence of protein breakdown. Sulfur-containing amino acids are readily reduced to yield hydrogen sulfide, which plays a significant role in the production of the greenish sulfhemoglobin pigmentation and the reaction with reduced (ferrous) iron (released from the iron transport protein transferrin or the iron storage protein ferritin) that produces a black precipitate of ferrous sulfide. Ornithine—a four-carbon diamine amino acid—and lysine, its five-carbon

analog, are readily decarboxylated to produce carbon dioxide, but more significantly for humans and "cadaver dogs" results in the production of the four-carbon "putrescine" and five-carbon "cadaverine," which are associated with a decomposing body.

2.5. Formation of Adipocere

Adipocere is a decomposition product of adipose tissue and may be an extensively formed a consequence of the putrefactive process in the presence of appropriate environmental conditions—generally high humidity or an aqueous environment coupled with relatively warm temperatures, conducive to the growth of putrefactive organisms. The formation of adipocere is a specific consequence of bacterial action, in which triacyl glycerols and other fatty esters are enzymatically hydrolyzed to produce both fatty acids and salts *(9,10)*. Other bacterial reactions that will subsequently affect the chemical composition and physical nature of the final product are hydrogenation, stereoisomerization, hydration, and dehydrogenation *(11)*. The physical nature of the material (increasingly hydrophobic as a consequence of the reactions noted above) limits additional breakdown and utilization of the high-energy molecules. The final nature of adipocere varies with the extent of hydration and the fatty acyl cation. Sodium salts produce a relatively soft material, whereas potassium salts are harder. Replacement of sodium with calcium creates an insoluble, somewhat brittle material *(7)*. Hence, the adipocere may vary from a grayish white, relatively soft, greasy substance to a crumbly, friable material as the water content is reduced *(9)*.

2.6. Mummification

Mummification (or dehydration of tissues) is neither a direct consequence of autolysis or putrefaction but is, in a sense, a competing process. As a function of environmental conditions, the rate at which water evaporates from the body or from exposed sections of the body can be rapid enough to reach a point where dehydration of individual tissues precludes the bacterial action of putrefaction. However, such tissues are subjected to slow oxidative processes that result in the characteristic darkening of the tissues. Internal organs in circumstances of mummification may be somewhat preserved, but usually have undergone some degree of autolysis and putrefactive changes because of the relatively protected and hydrated conditions. Clearly, conditions favoring the dehydration of bodies will facilitate mummification, but the effects of cold should not be discounted. When microbial activity is sufficiently slowed by temperature, the evaporation (or sublimation from frozen tissues) of water in a low-humidity environment may provide conditions for partial or complete mummification.

3. Factors Affecting Decomposition Processes

The dependence of the processes of decomposition on physical environmental factors, such as humidity and temperature, was noted previously in this chapter. However, the "contamination" or poisoning of the decomposing remains—either deliberately via an embalming process or accidentally by leaching of adjacent metal ions or the deposition of the body in a matrix that is unable to support microbial growth—can severely inhibit or effectively preclude any appreciable amount of decomposition.

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