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Inflammatory reaction - A posit to the double-edged sword

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Abstract

Response by inflammation is triggered by arrays of causes, which include disrupted cells, toxins, germs, and others. It underlies a wide variety of pathophysiological changes. Many aspects of inflammation as it relates to the pathology of various inflammations are very much understood. Yet the healthy roles of inflammation are widely unknown. Inflammation has a controversial role in health and its meanings are, a matter of viewpoint. It has critical roles in protecting organisms from pathogens and injurious substances likewise causing a driving variety of disease progression. On this ground the research aimed at prescribing the essential needs for effective regulations of inflammatory responses. Efficient control of the inflammatory process will avert a plethora of diseases. Articles used for this review were obtained using appropriate keywords on six electronic databases including nature, advantage, disadvantage, and immune response regarding inflammation and immunological response. Inflammation is self-perpetuating though no disease is caused by inflammation as it is not self-triggering. Additionally, the research did weigh up the merits alongside the demerit of inflammation to advocate for effective regulation of inflammation. Essentially, inflammation is a required mechanism in healthy and unhealthy status in humans hence there is a need for importunate reconsideration, exploring its therapeutic benefits.

Keywords: Inflammation; Disease; Regulation; Double-edge; Self-perpetuating

1. Introduction

Inflammation, a biological response to homeostatic perturbation [1], involves vascular and cellular components with diversities of clinical signs, which presents swelling, redness, heat, pain and functional impairment [2, 3]. The disease expression of the inflammation is centered on the imbalance of these five cardinal signs. This response is triggered by altered tissue in any form of injury. Fundamentally inflammation is tissue-damaging which progresses through the involvements of fluid, plasma proteins, and leucocyte to the inflamed cells [4]. The movement is aided by changes in the local vasculature and caused the vasodilation, in addition to intensified permeability in the vascular cells. All these give rise to much blood flow to the site [5]. The release of cytokines, noticeable alter in the number of leucocyte cells is the main feature of inflammation [6, 7]. This pervasive phenomenon operating during severe perturbations of homeostasis [8], is broadly characterized by nonspecific response to tissue malfunctioning so as to combat the disrupted cells. This multifarious biological response involves the interplay of biochemical molecules and is targeted at, protecting tissue from damage [2]. Effective regulation of response to inflammatory will reduce the severity due to inflammatory damage and the necessary homeostasis will be achieved.

The Inflammatory process is a normal, natural response by immunity. However, lingering inflammation destroys and may be linked with autoimmune ill-health. Research shows that hiked-in inflammation is the key force causing inflammation disease progression that results in organ disease [9, 10]. Chronic inflammation associates with rising and increase of various diseases [11].

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Failure to efficiently control the inflammatory process results undesirable outcome [12], hence the inflammatory reactions should be restricted to healing. Considering the vast array of disease of which prolong inflammation may precipitate and drive-cancer, stroke, diabetes, cardiovascular disease, gum disease, nephritis, bowel inflammatory disease, rheumatoid arthritis, lupus and polymyalgia rheumatic [13, 14, 15, 16]. Therefore, it is thoughtful to have a controlled inflammatory reaction rather that spontaneous reaction to injury. In contemporary 'revelations', chronic inflammation is factored in worsening conditions of diseases functions loss, among others (Fig. 2). Some literatures have considered the demerits of inflammation as unparalleled amid physiologic processes while others viewed it as necessity for the continuation of life process. Then inflammation has puzzling roles in health.

Of course, inflammation is not without merits. The acute form of inflammation can defend and heal of the body of disease. This acute inflammation is an amazing process, necessary for good health. The research work aims on the need to regulate inflammation, the knowledge of inflammation. The specific objectives were to advance for efficient regulation of inflammation using current issues on nature, advantages and disadvantages of inflammation.

1.1. Concept statement on inflammation

Inflammation is not self-triggered but self-perpetuating, having the capabilities of bittersweet connection and is highly a conserved process that appear crucial in defending organisms in the event of threat to organism. The living organisms engages inflammation as its initial defense process. Clearly, it is certain that we are aware of the inflammatory processes for futuristic modalities in treatment various health issues. Health caregivers should have fundamental knowledges on nature of cellular inflammation processes.

1.2. Nature of inflammation

Inflammation, a primary component of the process on the protection of the host on infection, injury, and wounds [17], is elicited by stimulus perceived as a threat to tissue homeostasis [18]. To counteract the insult eliciting threat, immunity dispatches series of events that open up in several stages [19]. Firstly, resident cells in the tissue of the innate immune system will alarm neutrophils in circulation, preceding the detection of the damaging insult. The movement to the perturb tissue at the site of the inflamed cells, will advance the draft of monocytes and increase the effectiveness of pro-inflammatory environment, promoting appropriate interaction at inflamed cell. In a particularly appropriate situation, apoptosis by neutrophils occurs after performing the required task at the site of inflammation, and subsequently, these apoptotic neutrophils are engulfed by macrophages. Removal of apoptotic neutrophils will promote a switch from pro-inflammatory to an anti-inflammatory cell phenotype [20], this is a basic required for macrophage exit through the lymphatic vessels favoring restoration of homeostasis of tissue.

The decisive stage in the concept is that resolution is a vigorous process with the desire to energetically suppress and end vigorous reaction of inflammation once its desired aim is attained. It is designated by five cardinal signs [21]: redness (resulting from increase flow of blood to the site of the inflamed area), swelling (resulting due to chemicals that cause blood vessels to leak protein-rich fluid into the tissues), heat (resulting from increase flow of blood alongside prostaglandin E_2 increase in the amount in the brain), pain (a defense mechanism to alert the body to move away from the site) and Loss of function ('Rigor', tissue destruction, pyrogen or dehydration). The imbalances of these signs are factored in the development of diseases. The components of inflammation are depicted by inflammatory triggers; the sensors; mediators and the effector tissues that are affected. The nature and extend of responses to inflammation are reliant on the feature of the inducer (parasite and others) and its perseverance [22]. A distinctive feature of inflammatory response is that harm to self cannot be avoided [8].

This all-purpose aggressive defense response (inflammation) towards disrupted tissue is used by immune systems [23], to combat interlopers [8]. Self-healing and disease progression in many metabolic diseases have inflammatory connotations [24]. Metabolic diseases are the drawback and are due to the self-perpetuating and the nonspecific nature of the inflammatory response to tissue injury. The researcher's train of thought presents inflammations as a natural, normal physiological response by the immune system to injury or infection. The researcher considers effective regulation of inflammation as a positive triumph for the body, inflammation is crucial in immunologic responses needs to be adjusted for a satisfactory outcome.

According to Noah *et al*. [8] and Medzhitov [1], microscopic organisms, physical and chemical agents, unfitting responses of immune cells, and dead tissues are among listed several factors that can stimulate and initiate inflammation. The responses are an initial effort to stop the damaging effect of the trigger on the normal function of the tissue [25]. The researchers perceived inflammation, as not a disease in itself but may have links to several diseases because it is the response to situations perceived as injurious to the body.

The primary nature of inflammation targets the primary inducer and vigorously removes the cause of the insult with the subsequent homeostatic restoration of tissue functions [1, 19]. As mentioned previously, the self-perpetuating nature of inflammation may depict the innocuous and malicious mischief in its perception. Extrapolating inflammatory reactions from literature searched, the double-edged nature of inflammation, stems from its self-perpetuating nature and non-direction response. Understanding the inflammatory cascade (Fig. 1) on how inflammation is initiated, perpetuated among others, coupled with the detailed inflammatory mechanism, will advance a better way to control inflammation. This will result in a positive impact on health.

The acute/self-resolving or chronic/non-resolving type of inflammation [26], (Fig. 3), involves the permeability of vascular membranes, aggressive exodus of blood components out and into the inflamed tissues [27]. Inflammation of acute type, lasting for a short period of reaction will mostly promote remedial of tissue malfunctioning: the infiltration of leukocytes to the injured site will get rid of the stimuli, and reparation of the tissue follows. Conversely, inflammation of chronic type is a long-term, uncontrolled and unfavorable response that engages vigorous inflammation, tissue damage, and efforts to repair tissue. This tenacious response of inflammation (Fig. 4) is linked to a plethora of life-debilitating conditions and its disease attendance, which are exemplified as allergic reaction, atherosclerotic conditions, cancer disease, and arthritis diseases, autoimmune disorders among others.

Generally, inflammatory phenomenon serving well-desired effect, could promote good outcome. Notwithstanding, issues may come up if the inflammatory reactions did halt, rather perpetuate and transformed to the chronic stage, will invariably affect negatively the health conditions in the body. Despite huge advances towards the knowledge in understanding the actual processes underlying inflammation, much has not been known of the role inflammation plays in regulating health.

Inflammation, when regulated properly, is theoretically useful in addressing different health situations. The exertion is anchored on the fact that there are higher serious chances of severe disease due to infections in individuals' deficient in the gene of primary components of inflammation, like neutropenia-a very small amount of neutrophils. Studies in a genetic defect in the genes that encode for proinflammatory cytokines and promotor of inflammation using mouse knock-out showed features of hyper susceptibility to infection [28]. However, substances that reduce inflammation may be beneficial. Cytokine (IL-10), having anti-inflammatory potentials, has a key role in limiting the immunological reactions pathogens and hence averting destructions to the cells of the host organism [29]. Of late, literature has reviewed that IL-10 and related cytokines can hasten the process of healing of tissue in damage due to inflammatory reaction [30].

The ability to logically control inflammation is regarded as the basis for its therapeutic value as distinct from mere reducing inflammation-which is in common practices.

1.3. Advantages of inflammation

From evolutionary advantage, some of the players of inflammatory cascade (phagocytosis and chemotaxis), used by lower organisms, later were co-opted as a defensive protocol in maintaining the integrity of higher organisms [31]. As several analysts have noted, the inflammatory system is crucial for survival [32]. It alarms the body's defensive mechanism of the existence of infection, hence putting the leucocyte into action. This results in a fast clearance infection [33]. Under a healthy situation, immunity has the potential to fight a sizable volume of pathogens, with favorable levels of inflammatory responses in clearing the infections from the affected host tissue. Resolution of inflammation happens very quickly under minimal destruction of the host. This is dependent on the tight control of cytokines. In the initiation of inflammation reactions, cytokines are paramount likewise hiking immune response.

Newer researches are providing, convincing shred of evidences on the healing power of inflammatory response to muscle tissue injury. A favorable amount of inflammation could initiate quality reparation of injury [34]. In this train of thought, *Mikkelsen et al.* [35] And Nunes-Silva [36], demonstrated that medicine to keep inflammation at check is able to blunt muscle growth if given in high doses.

In the furtherance of excess anti-inflammatory medication such as cortisone, studies have shown that, it slows wound healing [37]. This study is interrelated to the reason insulin-like growth factor and inflammatory cell components help the healing of wounds [38, 39]. In addition, factors (deficiency of leukocyte) that decrease the capacity for inflammatory defense will subsequently cause vulnerability to infection. Literature searched have shown that inflammation actually helps to heal damaged muscles [40].

Reducing inflammation through ice-cold water treatment among the sportspeople is no longer in invoke. Researches have shown that training adaptation using cold water treatment lowered proliferation of satellite cell [35]. Prolong effects results in reduce muscular enlargement and complexity of cell muscle fibers structure [41]. On the basis of conventional wisdom, inflammation may be adapted to suit the challenges in health.

Inflammation as previously mentioned in tissue reparation due to infection or otherwise malfunctioning tissue, sometimes seen as crucial parts of the regeneration of tissue and destruction of microbes [42], detoxification of toxins, clearing of infections [43], healing process and repair of damaged tissue [44]. This may be turning conventional wisdom on its head necessitating that inflammation must be largely controlled to encourage healing. The idea in these findings may suggest that existing and future therapies used to combat inflammation should be closely examined to ensure that the benefits of inflammation are not eliminated.

On the other hand, many relevant genes of immune disruption can give rise to unrestrained inflammation, showing that inflammation is rapidly repressed by gene regulating the productions of pro-inflammatory proteins so as to up-hold health when the stimuli of the inflammation are no longer their [18]. In the absence of efficient regulation, a disproportionate inflammatory response could have destructive effects, with resultant unintended harm.

Inflammation among other responses in the body must be efficient [45] through effective regulation [46]. The therapy here will be to control inflammation in order to achieve desired results and prevent lingering. If this agent cause of the inflammatory response is unremoved, or rather there is an obstruction in the repairing mechanism, the inflammation might result in an undesirable stage. Research justification hinges on how we can subtly alter inflammation at a later time point to explore the advantages of inflammation.

1.4. Disadvantages of inflammation

The compulsive character of aged tissues and most importantly in all age-related ill-health is severe inflammation. During aging, the body is weak in the resolution of inflammation. Licastro *et al.* [47] reported increased pro-inflammatory response with age. An increase in plasma level of IL-6 is associated with aging and IL-6 positively correlates with inflammation [48]. The mechanism is complicated and versatile [49]. Though humans are prone to more inflammations as we age, naturally the body has agents that modulate inflammation such as Nitric Oxide [50]. The worst case is those who are obese [51] because of the increased levels of pro-inflammation cytokines.

The development of inflammation unintended harm might rely on the inefficiency of host defense capability, late stimulation of adaptive immune system alongside poor specificity of the innate immune system [52]. That being said, there are conditions that cause exaggerated or abnormal inflammatory responses, some are exogenous while others are endogenous. It will not be very correct to assume that mortality due to infection is exclusively a result of pathogenic microbial exploitation. Although most of the literature searched showed collateral evidence suggesting mortality of host from the pathogen is most time rely on the level of unintended harm triggered by the inflammatory response from the host [53, 54]. Inflammation from the host is of paramount contribution to the pathology of the disease and in most instances, the harm from self-damage is more than that inflicted by the microbial pathogen [54].

Most neuronal destruction caused by bacterial meningitis and cerebral malaria is attributed to wild inflammation reactions [55]. Moreover a high amount of injury which happens from the trauma of brain injury comes from inflammatory responses rather than from direct mechanical damage. Furtherance to this, it is suggestive that brain inflammation preceding trauma is destructive rather than favorable. Traumatic injuries on the central nervous system are mostly fatal in humans and likewise, other species, in the absence of improved medical intervention [56]. However, peripheral tissues, injury to the central nervous system can cause irreparable damage because of limited regenerative potentials [56].

Previous works have shown that the inflammatory response has a key role in atherosclerosis development [57]. This formerly known bland lipid storage disease (atherosclerosis), actually has an ongoing inflammatory response undertone. Modern improvement through basic science has proven a basic role of an inflammatory process in facilitating each of the stages of this disease from beginning to progression and the thrombotic complications of atherosclerosis [58]. Mounting experimental evidence strongly supports the role of inflammatory mechanisms among others, in many facets of vascular disease [59]. Again, research has revealed the specific function of inflammation in disease like Alzheimer's disease [60], it facilitates serious and life-threatening complications in stroke patients [61] and a host of others.

Table 1 A case in points of inflammation contributing to pathogenesis in human disease

Disease	Mechanism
Allergy	Hypersensitivity of the immune system to typically harmless substances in the environment.
Alzheimer's disease	Brain cell inflammation, causing destruction of the tissues of brain.
Anaemia	Cytokines storm on erythropoietin production due to inflammation
Ankylosing spondylitis	Autoimmune reactions against joint surfaces due to inflammatory cytokines.
Asthmatic disease	Autoimmune reactions induced by cytokines against airway lining.
Autism	Autoimmune reactions due to inflammatory cytokines in the brain causing seizure of right hemisphere development
Arthritis	Destruction of joint cartilage and synovial fluid due to inflammatory cytokines.
Carpal Tunnel Syndrome	Chronic inflammation induced excessive muscle tension shortening tendons in the forearm and wrist compressing the nerves
Celiac	Severe immune mediated damages in the intestinal lining due to inflammation.
Congestive heart failure	Heart muscle wasting due to chronic inflammation.
Eczema	Chronic inflammation of the gut and liver with poor detoxification and often antibodies against Transglutaminase-3.
Fibromyalgia	Inflamed connective tissue often food allergy related and exacerbated by secondary nutritional and neurological imbalances.
Fibrosis	Traumatized tissue due to inflammatory cytokines attack
Gall Bladder Disease	Swelling of the bile duct or excess cholesterol produced in reaction to gut inflammation.
GERD	Inflammation of the oesophagus and digestive tract nearly always food sensitivity and pH driven due to inflammation.
Guillain-Barre	Nervous system autoimmune attack often triggered by autoimmune response to external stressors such as vaccinations.
Hashimoto's Thyroiditis	Antibodies triggered autoimmune attack against thyroid enzymes and proteins, originating from the gut.
Toxic Shock Syndrome/Scarlet Fever	Extreme inflammation Cytokine storm Oxidative damage
Kidney failure	Damage nephrons and tubules in the kidneys due to inflammatory cytokines which are restricted in circulation.
Lupus	Autoimmune attack against connective tissue due to inflammatory cytokines.
Multiple Sclerosis	Autoimmune reactions against myelin due to inflammatory cytokines.
Neuropathy	Autoimmune reactions against myelin and vascular and connective tissues due to inflammatory cytokines which irritate nerves.
Pancreatitis	Pancreatic cell injury due to inflammatory cytokines.
Psoriasis	Severe inflammation of the gut and liver with poor detoxification
Polymyalgia Rheumatica	Autoimmune reactions against muscles and connective tissue due to inflammatory cytokines.

Source: Adapted with modified from Margolis and Levin [53].

In a special situation, a good number of intracellular pathogens have adapted to reproduce inside immune cells. In the course inflammation process, cells of the immune system are employed and channeled to the infection site, which

accidentally spread with more transmission and spread of the pathogen [52]. Recent literature is suggestive of the inflammatory response of acute type as an enhancer in horizontal gene transfer among commensal and pathogenic flora in the gut which may encourage development and transmission of the antibiotic-resistant and virulence genes [61]. Unnecessary inflammation directs to expensive unintended harm, especially when unrestrained, may result in untold consequential pathology (Fig. 4).

From Table 1, it is arguable to note that in the absence of inflammatory process, the majority of diseases would be in nonexistence. The number of diseases with the mechanism showcases the relationship, inflammations have on diseases. This is why inflammation must be addressed.

Acute inflammatory process mighty be the led in the progress of the destruction of tissue following stimulus of harmful origin (e.g. bacteria) and it possibly can reduce the survival potentials rates of the organism. Furthermore, inflammation of chronic type has the capability of causing a plethora of diseases (Table 1). Hence inflammation is a normal tightly regulated mechanism of living things, failure in the regulation of inflammation will disturb the homeostatic conditions of the body. The attendant consequences range from subtle subclinical to serious life threatening-diseases (Table 1)

However, sometimes inflammation can cause further inflammation, in this process, becomes self-perpetuating and more inflammations are created in response to the existing inflammation. During the robust inflammatory reaction, there is the possibility of DNA repair machinery and cell-cycle checkpoints being halted [62], which may result in an increased mutation rate [62]. It is now known high-level inflammation contributes to cancer development [63, 64, 65]. In many human tumors, Infection-driven inflammation is the blame for the pathogenesis of many of these disease types.

The idea that the immune response drives the inflammation in the disease process is understood and that inflammatory reaction is part of the immunity that could be stimulated by any trigger has the capability to disturb tissue homeostasis [18]. Globally, coronary artery disease is the lead cause of mortality, and a major predisposition to the disease is inflammation as they have variants of genes that associate with inflammation. Though without inflammatory response, being present in the body, there might not be the presence of strokes, diabetes, cardiovascular diseases, and endless count of diseases, the catchphrase is 'controlled inflammatory'.

Inflammation is very important for normal repair processes to occur. The unregulated wild inflammation response type is unrequired and needless inflammation.

2. Mechanism of inflammation and probable therapeutic points.

The Inflammatory mechanism is a series of ordered, active, and changing reactions. It includes cellular and vascular events alongside specific secretions of hormones (Fig. 2).

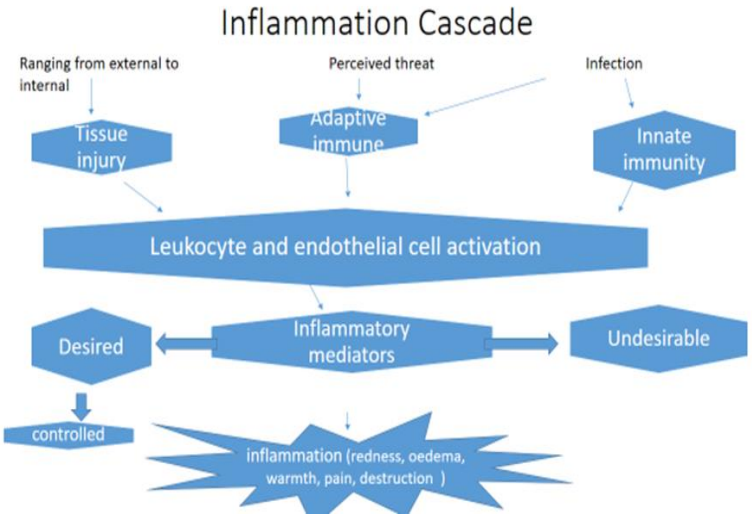

Figure 1 Simplified nature of inflammatory cascade

Vasodilation (expansion), exudation (oedema), emigration of cells, chemotaxis, and phagocytosis define the mechanism of inflammation. Inflammatory responses from inappropriate stimulation are the primary cause of several known medical conditions (Fig. 3 and Fig. 4). The main fundamental basic feature of inflammatory response is an increase in the temperature of the host, alongside loss in function (Fig. 1) and physical change in location of cells (leukocytes, monocytes, basophils, eosinophils, and neutrophils), plasma exodus to the site of inflammation [66].

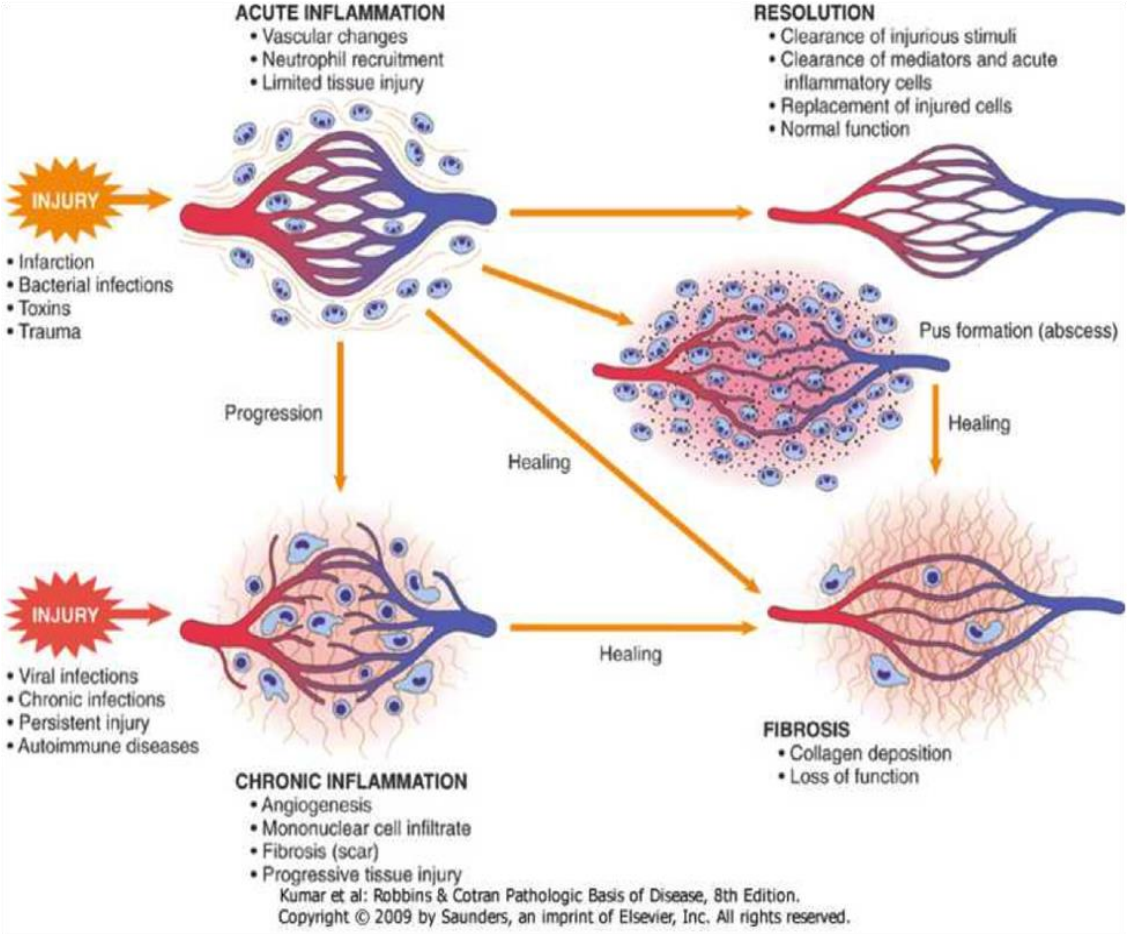

Figure 2 Morphologic patterns of inflammation source

Source: https://d1yboe6750e2cu.cloudfront.net/i/f439f61622223489ea9b13b3ec3a56ff58d8705e

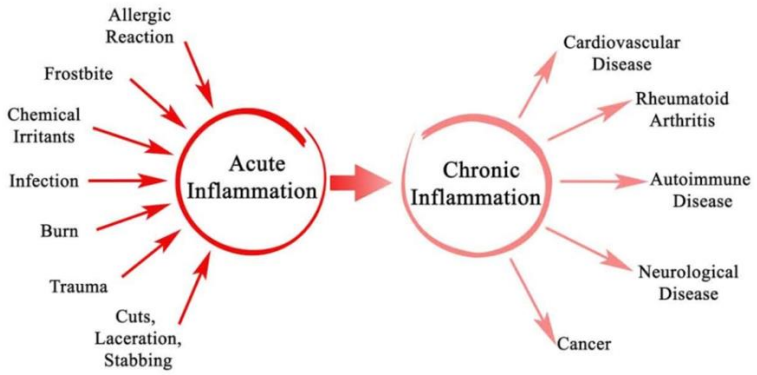

Figure 3 Inflammation becoming harmful in chronic inflammation

Source: https://www.nmamilife.com/a-z-health/chronic-v-s-acute-inflammation-know-the-difference/

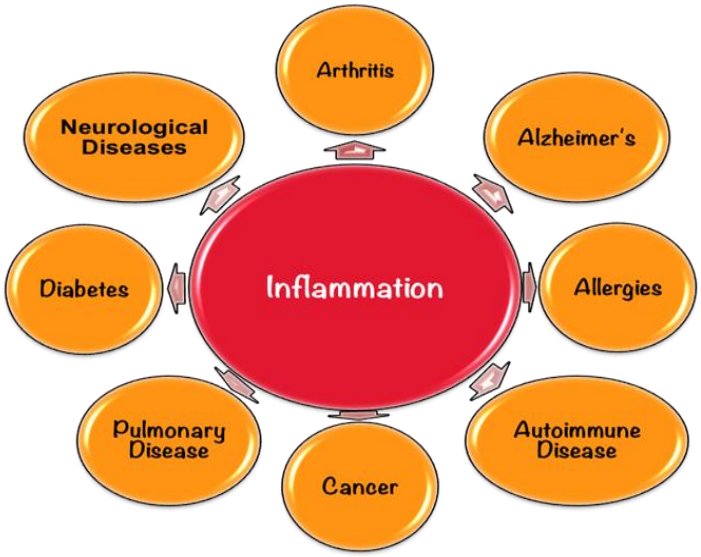

Figure 4 Inflammation at the root of most diseases

Source: https://images.app.goo.gl/7APFD77XmXfmZ9BW8
https://www.quora.com/If-inflammation-is-a-part-of-healing-why-do-we-try-to-stop-it-with-anti-inflammatory-drugs

Following the presence of inflammogen (allergens, Pathogens, toxins, frostbite, and burns) (Fig. 1 and Fig. 2), the resident immune cells (dendritic cells, macrophages, histiocytic, mast cells, and Kupffer cells) already present in tissue involved will initiate the inflammatory process. The cells are surface receptors known as pattern recognition receptors (PRRs) are capable of recognizing and binding molecules of two subclass: damage-associated molecular patterns (DAMPs). And pathogen-associated molecular patterns (PAMPs) [32, 67]. At the start of a burnt, infection, injuries, and other inflammogens, the cells are activated (PAMP or DAMP is been recognized by the PRRs) which follows the secretion mediators of inflammation that gives rise to inflammatory clinical signs (Fig. 1) [68].

The inflammatory response at the inflamed site is depicted through the initial rise the flow of blood towards the site of the injury due to an increase in vascular permeability [69] and the coordinated directional migration, with the careful build-up of various effector cells from the peripheral blood flow at the place of injury. The vasodilation, increase in blood flow and permeability of vessels, characterized the redness (*rubor*) and increased heat (*calor*) at the inflamed cell (Fig. 1). This vascular change (Fig. 2) will increase the permeability of the blood vessels, resulting in exudation (leakage) of plasma proteins (such as fibrin and immunoglobulins (antibodies), into inflamed tissue) and fluid into the inflamed tissue. These manifest as swelling (*tumor*) [70]. The increased tissue fluid would lead to an increase in lymphatic drainage. This will help to remove the damaging substance.

Among the released mediators like bradykinin would hike the feeling of pain (hyperalgesia). The molecules of the mediator, additionally change the blood vessels to allow the movement of cells, mainly macrophages, and neutrophils, (Fig. 2) out of the vessels of blood (extravasation) into the tissue. This moved tissue fluid has various antimicrobial mediators coming from the plasma. It includes antibodies, complement, and lysozyme that would promptly handle the inflammogens (Fig 1 and Fig 2). Proportions of the exuded tissue fluid are channeled to lymphatics regional lymph nodes, clearing microbes. The neutrophils exodus following chemotactic gradient pattern as created through the local cells to reach the injurious site. The functional loss called *functio laesa,* is presumably from neurological reflex in pain response. The migration of granulocytes to the inflammatory site will cause the neutralization and removal of inflammogen [2].

Cell-derived mediators alongside, many acellular biochemical events consisting of preformed plasma proteins work in parallel to start and propagate inflammation. They are the complement system activated by the coagulation and fibrinolysis systems activated by necrosis (a burn or a trauma) and bacteria [71]. After removal of inflammogen, resolution of inflammation is followed by the eventual return of tissue mononuclear cell members to the basal levels [72] (Fig 1 and Fig. 2). In this case, the reaction is acute and is constrained to the area of damaged tissue [73]. In some situations of acute inflammation, the quick employment of granulocytes (eosinophils, neutrophils, and basophils) towards the inflammation area may last longer without resolution. This unresolved inflammation (acute) may transform to chronic, thereby leading to many different types of chronic inflammatory diseases [74]. Atherosclerosis is

an example of a chronic disease that involves inflammatory mechanisms. Atherosclerosis, identified as a lipid-driven inflammatory disease is characterized by continuous immune activation [74] and unresolved inflammation [75].

Heightened inflammation may advance plaque rupture, causing ischemic disease [76, 77]. This inflammation of the vascular wall is the onset of atherogenic events and it involves lipoprotein accumulation, Tumor Necrosis Factor (TNF) α and leukocyte recruitment (especially monocytes and macrophages), and expression of proinflammatory cytokines, such as interleukin (IL)-1β [58, 76]. Failure to resolve this inflammatory response is the drive-in arteriosclerosis and beyond. Atherosclerosis underpins the pathological nature associated with many cardiovascular diseases, including but not limited to coronary artery disease, stroke, ischemic gangrene, and myocardial infarction [76]. Inflammatory markers may be predictive and associated with the causes, together with the consequences of the aforementioned inflammatory diseases [78].

Inflammation could potentially be halted by actively turning on/promoting a functional and effective resolution of a phenomenon [75] using a therapeutic approach to avert progress beyond arteriosclerosis. Additionally, proteases in inflammatory exudates [79] have roles in the inflammation response [80]. Proteolytic activities of these enzymes are vital therapeutic points for the regulation inflammation responses [81].

3. Conclusion

In the past, the only accepted treatment was medications that reduce Inflammation. At the moment these prescriptions are no longer scientifically or morally defensible. The discovery, a few years ago that inflammation is crucial for the life process is slowly leading to a radical change in how inflammation is viewed. The obvious roles of inflammation, in disease progression and healing, as well as the disadvantages and advantages of inflammation were discussed to drive a more rigorous approach to inflammation. From the discussions, inflammation plays both positive and negative roles in health. Evidently, this study has exposed the good, bad, and ugly of inflammation and proposing therapy for the control of inflammation. At present, Scholars' stance on inflammation is difficult to interpret or understand. On this, further explanations are required.

Compliance with ethical standards

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