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Anti-Inflammatory Therapies for Long Term auto Immune Diseases

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Review Article

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ABSTRACT

Some of continual illnesses like atherosclerosis, type 2 diabetes and Alzheimer's disorder, have a pathophysiologically essential provocative compound. In these chronic diseases, the suitable identification of the inflammatory stimulus is regularly unknown and, if recognized, is hard to expel. Along these lines, there is enthusiasm for restoratively focusing on the incendiary reaction. In spite of the fact that there has been accomplishment with inflammatory treatment in ceaseless ailments activated by essential inflammation or autoimmunity, there are extensive constraints. Specially, the inflammatory reaction is basic for survival. As an end result, redundancy, compensatory pathways and necessity narrow the danger: advantage ratio of anti-inflammatory drugs. however, new advances in understanding inflammatory signaling and its links to identification pathways, together with new medicine development, offer assure right here of translational biomedical examination.

INTRODUCTION

Enhanced comprehension of the incendiary reaction has prompted imperative advances inside the remedy of sicknesses with an essential imperfection in irritation control, for example, CAPS and in immune system impelled provocative maladies, especially seropositive RA and some other rheumatoid infections. Could these advances likewise be connected to different sorts of incessant ailments in which aggravation is an imperative main thrust? ^[1-15]. In constant immune system ailments, the instruments connecting the immune system trigger to the maladaptive provocative reaction are regularly preferable comprehended over in endless incendiary sicknesses with a non-autoimmune etiology ^{[1,6,15-28].}

Additionally, immune system illnesses are typically connected with extraordinary and frequently excruciating, side effects on an everyday premise, which tends to expand quiet acknowledgment of unfavorable impacts of treatment ^[29-32]. In spite of this elegance of illnesses, remedy isn't always best. The gainful reactions are variable, especially when mitigating treatment is begun after the malady has gotten to be set up; dependable reductions are not normal; and antagonistic impacts, especially in the range of traded off host resistance, can be generous ^[6].

These issues are liable to be considerably more maintained with complex and regularly slothful endless infection forms in which the essential trigger is concept to be an option that is aside from autoimmunity ^[33-40]. The difficulties in specializing in irritation in any limitless provocative illness (continual) lie in three properties which can be every day for tactics which are fundamental for evolutionary survival: redundancy, reimbursement and necessity ^[41,42]. Thus, infection is organized via many molecules and thus while the previous two challenges are effectively overcome. Infection is also a finely tuned method that has inborn sensors and enter pathways, accordingly hindrance of a primary a part of aggravation may just cause a compensatory proinflammatory reaction including another pathways ^[46].

At long last, the incendiary reaction is basic for host resistance and along these lines when the past two difficulties are effectively overcome, the risk: benefit profile is frequently unsatisfactory. With this background, the review of essential additives of the inflammatory response, with emphasis on those components that make a contribution to redundancy and compensation and which are goals of presently to be had drugs or have promise for destiny healing intervention ^[47-50].

TYPES OF INFLAMMATORY DISEASE

Chronic Inflammatory Diseases

This means long-run inflammation, a number of the chronic disease air respiratory disorder, chronic ulcer, infectious disease, autoimmune disease, chronic periodontal disease, inflammatory bowel disease and regional ileitis, Chronic inflammation, Chronic active infectious disease.

Acute Inflammatory Diseases

Starts rapidly and quickly gets the opportunity to be amazing, Some of Acute provocative affliction are Acute bronchitis, Infected ingrown toenail, Sore throat from a cool or flu, A scratch/cut on the skin, Exercise (especially exceptional planning), Acute a cracked supplement, Acute dermatitis, Acute tonsillitis, Acute infective meningitis and Acute sinusitis ^[51].

INFLAMMATION IS MAIN SOURCE OF MOST CHRONIC DISEASES

It's critical to understand that interminable aggravation is the wellspring of numerous if not most ailments, including tumor, stoutness and coronary illness, which basically makes it the main source of death in the US. While irritation is a superbly typical and valuable process that happens when your body's white platelets and chemicals shield you from outside intruders like microscopic organisms and infections, it prompts inconvenience when the incendiary reaction escapes hand. Our eating regimen has a considerable measure to do with this chain of occasions ^[52-61]. While among the most strong, ounce for ounce, herbs and flavors are absolutely by all account not the only mitigating fixings accessible. Various nourishments are understood for their mitigating properties and ensuring you're eating a wide assortment of them all the time can go far toward avoiding perpetual disease.

THE IMMUNE SYSTEM AND THE INFLAMMATORY RESPONSE

Numerous specialists now consider aggravation to be emerging from an insusceptible framework reaction that is crazy. When you come down with a bug or sprain your lower leg, your invulnerable framework switches into rigging. Disease or damage trigger a chain of occasions called the incendiary course ^[58]. The commonplace indications of typical irritation heat, torment, redness and swelling are the primary flags that your insusceptible framework is being called energetically. In a sensitive parity of give-and-take, aggravation starts when master incendiary hormones in your body get out for your white platelets to come and get out disease and harmed tissue ^[62]. These specialists are coordinated by similarly intense, firmly related calming mixes, which move in once the risk is killed to start the recuperating procedure. Intense aggravation that back and forth movements as required means a very much adjusted insusceptible framework. In any case, manifestations of aggravation that don't retreat are letting you know that the "on" switch to your insusceptible framework is trapped ^[62-65]. It's balanced on high caution notwithstanding when you aren't in up and coming peril. Now and again, what began as a solid component, such as building scar tissue or swelling, just won't stop.

CONCEPTS OF THE INFLAMMATORY RESPONSE

• At the tissue level, severe aggravation is defined via redness, warmness, pain and swelling, which result from local reactions of immune, vascular and parenchymal cells to infection or harm ^[66].

- At a signaling level, infection or tissue harm is at first detected by means of example acknowledgment receptors (PRRs) that understand pathogen- related molecular patterns (PAMPs) and/or harmassociated molecular patterns (DAMPs).
- At a cellular level, acute inflammatory reactions are defined by means of stamped temporal adjustments in amounts and traits of tissue resistant cells ^[67].

PRINCIPLES OF ANTI-INFLAMMATORY THERAPY IN CHRONIC AUTOIMMUNE INFLAMMATORY DISEASES

Chronic diseases related to associate in inflammatory component in some way evoked by Associate in nursing auto-immune method square measure the foremost commonplace illnesses of getting old and represent our greatest fitness threats (three). Those consist of maximum styles of cardiovascular sickness, type 2 diabetes and all neurodegenerative illnesses [68-75].

In every case, a non-autoimmune number one pathological method-as an instance, extra sub endothelial Apo lipoprotein B-containing lipoproteins, saturated fatty acids, or formation of macromolecule aggregates, respectively-results within the generation of DAMPs that square measure detected by PRRs ^[76]. Moreover, the inflammatory reaction itself ought to increase the meeting of ailment-particular DAMPs, leading to effective-comments loops that boost up the underlying pathogenic approach.

As an instance, the assessment of anti-inflammatory tablets for type 2 diabetes, compared with atherosclerosis and, particularly, Alzheimer's disorder, is extra possible in phrases of quit-factor analysis (fasting blood sugar, hemoglobin A1c and plasma insulin levels) and may not be as annoying in phrases of the necessity for early-level treatment ^[77-86].

Continuous inquires about to enlarge the advantage to-danger window of calming treatment in perpetual infections will require endeavors on various correlative fronts. To the degree that there is the possibility to expel the incendiary boost in these ailments, as there is in atherosclerosis (atherogenic lipoproteins) and heftiness (supplement overabundance), continuous endeavors around there are essential [87-93]. For instance, in atherosclerosis, there might be inventive restorative ways to deal with keep the maintenance of atherogenic lipoproteins notwithstanding bringing down plasma LDL^[44].

In standard, but these goals have been difficult to achieve even in which they are theoretically feasible and it isn't but feasible in different persistent sicknesses, which includes neurodegenerative sickness [94,95]. An alternative strategy to inhibiting inflammation might be to commandeer nature's personal anti-inflammatory mechanisms to set off a "dominant" program of resolution ^[96-100].

At long last, it is imperative to consider one should noninvasively screen the anti-inflammatory or proresolving activities of medications that objective the inflammatory segment of chronic infections. This is especially imperative in diseases like atherosclerosis where the real clinical end points themselves are postponed, sporadic and regularly destroying.

CONCLUSION

Finally concluded that the previous two decades have given an abundance of data on how maladaptive, nonresolving irritation drives a number of sizable persistent illnesses wherein contamination, important imperfections in infection direction, or autoimmunity aren't the critical pathophysiologic process. In spite of the reality that this learning can possibly open up giant opportunities for brand new therapeutic advances, the character of the inflammatory reaction as a complex system that is basic for typical physiology renders this promise difficult. New gaining knowledge of about inflammatory signaling, in particular inside the levels of endogenous homeostatic pathways and infection resolution, offers the guarantee to new healing alternatives that may correctly meet these demanding situations

REFERENCES

- 1. Fabrègue F, et al. Association of Inflammation and Possible Mild Cognitive Decline Measured by the Stroop Cognitive Function Test. J Alzheimers Dis Parkinsonism. 2016;6:237.
- 2. Mohamed MSA. Role of Genetic Testing in Lung Transplantation; Prediction of Inflammation. J Genet Syndr Gene Ther. 2016;7:298.
- Sugama S, et al. Effect of Chronic Stress in the Onset of Parkinson's Disease: Possible Role of Microglial Cells in Neuroinflammation. J Neurol Disord. 2015;S2:001.
- Acebo E, et al. Allergic contact dermatitis from Boswellia serrata extract in a naturopathic cream. Am J Contact Dermat. 2004;51:91–92.
- Sagor MAT, et al. Fresh Seed Supplementation of Syzygium Cumini Attenuated Oxidative Stress, Inflammation, Fibrosis, Iron Overload, Hepatic Dysfunction and Renal Injury in Acetaminophen Induced Rats. J Drug Metab Toxicol. 2016;7:208.
- Aguilar JL, et al. Anti-inflammatory activity of two different extracts of Uncaria tomentosa (Rubiaceae) J Ethnopharmacol. 2002;81:271–276.
- 7. Bigley NJ. M1 and M2 Myeloid Cells in Inflammation. J Cell Signal. 2016;1:e103.
- 8. Almekinders LC, Gilbert JA. Healing of experimental muscle strains and the effects of nonsteroidal anti-inflammatory medication. Am J Sports Med. 1986;14:303–308.
- 9. de la Monte SM, et al. Tobacco Smoke-Induced Hepatic Injury with Steatosis, Inflammation and Impairments in Insulin and Insulin-Like Growth Factor Signaling. J Clin Exp Pathol. 2016;6:269.
- 10. Trunov A, et al. Cytokines and Infertility Influence of Cytokines and Local Inflammation in Women of Reproductive Age with Infertility. J Cytokine Biol. 2016;1:102.

- 11. Jena PK, et al. Influence of Gut Microbiota on Inflammation and Pathogenesis of Sugar Rich Diet Induced Diabetes. Immunome Res. 2016;12:109.
- 12. Almekinders LC. Anti-inflammatory treatment of muscular injuries in sport. An update of recent studies. Sports Med. 1999;28:383–388.
- 13. Spahiu V, et al. Cytokines: Key Biomarkers in Elucidating the Pathogenesis of Inflammation. J Clin Cell Immunol. 2016;7:421.
- 14. Rivera-Rivera Y, et al. Impact of Depression and Inflammation on the Progression of HIV Disease. J Clin Cell Immunol. 2016;7:423.
- 15. Shrihari TG. Inflammation Related Cancer Highlights. J Carcinog Mutagen. 2016;7:269.
- Ernst E. Adulteration of Chinese herbal medicines with synthetic drugs: A systematic review. J Intern Med. 2002;252:107–113.
- 17. Ester PR, et al. Giant-Cell Arteritis: Immunopathogenic Mechanisms Involved in Vascular Inflammation and Remodeling. J Vasc. 2016;2:103.
- 18. Berezin A. The Neutrophil Extracellular Traps: The Missed Link between Microvascular Inflammation and Diabetes? Metabolomics. 2016;6:163.
- 19. Elmali N, et al. Effects of resveratrol in inflammatory arthritis.Inflammation. 2007;30:1-6.
- 20. Mitra S, et al. Screening of Novel Natural Product Derived Compounds for Drug Discovery in Inflammation. J Plant Biochem Physiol. 2016;3:159.
- 21. Demori I and Grasselli E. Stress-Related Weight Gain: Mechanisms Involving Feeding Behavior, Metabolism, Gut Microbiota and Inflammation. J Nutr Food Sci. 2016;6:457.
- 22. Ehrich EW, et al. Characterization of rofecoxib as a cyclooxygenase- 2 isoform inhibitor and demonstration of analgesia in the dental pain model.Clin Pharmacol Ther. 1999;65:336–347.
- 23. Yazdani Shaik BD and Conti P. Relationship between Vitamin C, Mast Cells and Inflammation. J Nutr Sci. 2016;6:456.
- 24. El-Shazly AA, et al. Therapeutic Effects of Extracts from Spirulina platensis versus Bevacizumab on Inflammation-Associated Corneal Neovascularization. J Med Surg Pathol. 2016;1:102.
- 25. Wang S, et al. Antiarthritic Effects of Daphne giraldii Nitsche (Thymelaeaceae) Mainly through Suppression of the Secondary Inflammation. Pharm Anal Chem Open Access. 2015; 1:107.
- Daviglus ML, et al. Fish consumption and the 30-year risk of fatal myocardial infarction. N Engl J Med. 1997;336:1046–1053.
- 27. Zapolska DD, et al. Trans Fatty Acids and Atherosclerosis-effects on Inflammation and Endothelial Function. J Nutr Food Sci. 2015;5:426.
- 28. Palestro CJ, et al. Neutrophil Trafficking in Pulmonary Inflammation: Monitoring Migration and Blockade with 111In-Labeled Leukocytes. J Pulm Respir Med. 2015;5:289.
- 29. Graham DJ. COX-2 Inhibitors, Other NSAIDs, and Cardiovascular Risk: The Seduction of Common Sense. JAMA. 2006;296:1653–166.

- Tashiro N, et al. Doxycycline attenuated Mycobacterium avium Induced Inflammation in Mice. Mycobact Dis. 2015;5:192.
- Jiangyan C, et al. Association among Systolic Blood Pressure Variation, Inflammation and Arterial Rigidity in Essential Hypertension. J Hypertens. 2015;4:207.
- 32. Curtis CL, et al. Pathologic indicators of degradation and inflammation in human osteoarthritic cartilage are abrogated by exposure to n-3 fatty acids.Arthritis Rheum. 2002;46:1544–1553.
- 33. Gnoni ML, et al. Sources for Inflammation and Accelerated Aging in Well Controlled HIV Patients on Antiretroviral Therapy. J Infect Dis Ther. 2015;3:239.
- 34. Curtis CL, et al. N-3 fatty acids specifically modulate catabolic factors involved in articular cartilage degradation. J Bio Chem. 2000;275:721–74.
- 35. Mainardi P, et al. From the Ancient Diets to the Recent Acquisitions on the Role of Brain Inflammation in Epilepsy, Are there Any Links?. J Neurol Neurophysiol. 2015;6:304.
- Mehling BM, et al. Evaluation of Immune response to Intravenously Administered Human Cord Blood Stem Cells in the Treatment of Symptoms Related to Chronic Inflammation. J Stem Cell Res Ther. 2015;5:297.
- 37. Ahmad HS, et al. Baseline Synovial Blood Flow Signals in Very Early Rheumatoid Arthritis is Associated with Joint Inflammation and Radiographic Joint Damage. Rheumatology. 2015;5:164.
- Curtis CL, et al. Biological basis for the benefit of nutraceutical supplementation in arthritis. Drug Discov Today. 2004;9:165–172.
- 39. Harris M. Is there a Role for Long Chain Omega-3 (N-3) Fatty Acids in Reversal of Malnutrition Inflammation Syndrome among Hemodialysis Patients?. Vitam Miner. 2015;3:e143.
- 40. Banerjee AK. Computation in Analyzing Inflammation: A General Perspective. Interdiscip J Microinflammation. 2015;2:130.
- 41. Clemett D, Goa KL. Celecoxib: A review of its use in osteoarthritis, rheumatoid arthritis and acute pain.Drugs. 2000;59:957–980.
- 42. Musba AT, et al. The Effect of Dexamethasone on the Dynamics of Inflammation, Cortisol and analgesia in Lower Limb Surgery. J Pain Relief. 2015;4:186.
- 43. Woldemeskel M. A Brief Review of Mast Cells in Microbial Infection (Inflammation) and Tumor-Associated Angiogenesis . J Clin Exp Pathol. 2015;5:e119.
- 44. Birandra KS, et al. Is Metabolic Activation of Topoisomerase II Poisons Important In The Mechanism Of Cytotoxicity?. J Drug Metab Toxicol. 2015;6:186.
- 45. Yarla NS, et al. Phospholipase A2: A Potential Therapeutic Target in Inflammation and Cancer (In silico, In vitro, In vivo and Clinical Approach). J Cancer Sci Ther. 2015;7:249-252.
- 46. Madu AJ, et al. The Role of Lipids in Inflammation: Review of the Evolving Pathogenesis of Sickle Cell Disease. Biol Med. 2015;7:244.
- 47. Claeson P, et al. Three non-phenolic diarylheptanoids with anti-inflammatory activity from Curcuma xanthorrhiza. Planta Med. 1993;59:451–454.

- 48. Bajic V, et al. Cyclin Dependent Kinase 11, Neuroinflammation and Alzheimer's Disease: A Review. J Clin Cell Immunol. 2015;6:305.
- 49. Huang Z. The Activity of Hyaluronan and Hyaluronidase PH20 in Inflammation-A Role by Reagent Contaminants?. J Clin Cell Immunol. 2015, 6:314.
- 50. Chung JM, et al. Effects of capsaicin applied to a peripheral nerve on the responses of primate spinothalamic tract cells. Brain Res. 1985;329:27–38.
- 51. Munari ACF and Cervera LFF. Inflammation, Metalloproteinases, Chronic Venous Disease and Sulodexide. J Cardiovasc Dis Diagn. 2015;3:203.
- 52. Faulkner J, et al. Inhibition of 12/15-Lipoxygenase Reduces Renal Inflammation and Injury in Streptozotocin-Induced Diabetic Mice. J Diabetes Metab. 2015;6:555.
- 53. Esgalhado M and Mafra D. Influence of Physical Exercise on Oxidative Stress and Inflammation in Hemodialysis Patients. J Mol Biomark Diagn. 2015;6:e120.
- 54. Chrubasik S, et al. Treatment of low back pain with a herbal or synthetic anti-rheumatic: A randomized controlled study. Willow bark extract for low back pain.Rheumatology. 2001;40:1388–1393.
- 55. Janssens H, et al. Analysis of Short and Long Term Variability of Nasal Mucus versus Breath Condensate Inflammation Markers in Healthy Individuals. J Pulm Respir Med. 2015;5:265.
- 56. Herbert C, et al. Immunoreactivity for Interleukin-33 in Allergic Airway Inflammation. J Cytol Histol. 2015;6:302.
- 57. Chrubasik S, et al. Treatment of low back pain exacerbations with willow bark extract: A randomized double blind study. Am J Med. 2000;9:9–14.
- 58. Jacob PS, et al. Use of Curcumin in Periodontal Inflammation. Microinflammation. 2014;1:114.
- 59. Bedouhene S, et al. Polyphenols Extracted from Olive Mill Wastewater Exert a Strong Antioxidant Effect in Human Neutrophils. Int J Waste Resources. 2014;4:161.
- 60. Mizejewski GJ. Alpha-Fetoprotein (AFP) and Inflammation: Is AFP an Acute and/or Chronic Phase Reactant?. J Hematol Thrombo Dis. 2015;3:191.
- Cho KJ, et al. Effects of bioflavonoids extracted from the bark of Pinus maritime on proinflammatory cytokine interleukin-1 production in lipopoysaccharide-stimulated RAW 264.7. Toxicol Appl Pharmacol. 2000;168:64–71.
- 62. Majeed S, et al. Dual Role of Inflammation in Prognosis and Prevention of Tuberculosis. J Clin Cell Immunol. 2015;6:298.
- 63. Rosales–Corral S, et al. Cannabinoids in Neuroinflammation, Oxidative Stress and Neuro Excitotoxicity. Pharm Anal Acta. 2015;6:346.
- 64. Caterina MJ, et al. Impaired nociception and pain sensation in mice lacking the capsaicin receptor. Science. 2000;288:306–313.
- 65. Lewis M and Merched AJ. Tumor-Associated Macrophages, Inflammation and Pathogenesis of Hepatocellular Carcinoma. J Mol Genet Med. 2014;8:132.

- 66. Altman R, et al. The Postprandial Effects of a Moderately High-Fat Meal on Lipid Profiles and Vascular Inflammation in Alzheimer's Disease Patients: A Pilot Study. J Gen Practice. 2014;2:186.
- 67. Patsouris D and Jeschk MG. Stress Induced Insulin Resistance in Regards to Cellular Organelles, Inflammasome and Inflammation and Lipids. Mol Biol. 2014;3:e114.
- Caterina MJ and Julius D. The vanilloid receptor: A molecular gateway to the pain pathway. Annu Rev Neurosci. 2001;24:487.
- 69. Simas TAM and Corvera S. The Roles of Adipose Tissue and Inflammation in Gestational Diabetes Mellitus. Intern Med. 2014;S6:010.
- 70. Fioranelli M and Roccia MG. Low Dose Interleukin-10 and Anti-IL-1 Antibody in Modulating Intestinal Inflammation. Interdiscip J Microinflammation. 2014;1:101.
- 71. Neelapu NRR, et al. Helicobacter Pylori Induced Gastric Inflammation, Ulcer and Cancer: A Pathogenesis Perspective. Microinflammation. 2013;1:113.
- 72. Calder PC. N-3 Polyunsaturated fatty acids, inflammation, and inflammatory diseases. Am J Clin Nutr.2006;83:1505S-1519.
- 73. Bloom HL, et al. Left Atrial Enlargement Correlates with Infl ammation and Oxidative Stress in Patients at High Risk for Atrial Fibrillation. J Clinic Experiment Cardiol. 2010;1:101.
- 74. Duong HQ, et al. Comparing Three Post- Op Regiments for Management of Inflammation Post Uncomplicated Cataract Surgery. "Are Steroids Really Necessary?". J Clinic Experiment Ophthalmol. 2011;2:163.
- 75. Burton TM. Monsanto arthritis-pain drug, Celebrex, surpasses Viagra's early sales success. The Wall Street Journal B: New York. 1999
- 76. Skopec R. Mechanism Linking Aggression Stress through Inflammation to Cancer. J Cancer Sci Ther. 2011; 3:134-139.
- 77. Kanwar JR, et al. Toll Like Receptors Play a Role in General Immunity, Eye Infection and Inflammation: Tlrs for Nanodelivery. J Clin Cell Immunol. 2012;2:114.
- 78. Heymann MC and Hofmann SR. Novel Inflammasomes and Type II Diabetes, Intestinal Inflammation and Psoriasis as Newly Inflammasome-Related Diseases. J Genet Syndr Gene Ther. 2011;S3:001.
- 79. Bernstein JE, et al. Treatment of chronic postherpetic neuralgia with topical capsaicin. A preliminary study. J Am Acad Dermatol. 1987;17:93–98.
- Bonvillain RW, et al. Battling Inflammation in Acute Lung Injury and Acute Respiratory Distress Syndrome: Stem Cell-Based Therapy Targeting the Root Cause of Acute Lung Injury. J Pulmonar Respirat Med. 2011;S2:001.
- Bengmark S. Curcumin, an atoxic antioxidant and natural NFk B, cyclooxygenase-2, lipooxygenase, and inducible nitric oxide synthase inhibitor: A shield against acute and chronic diseases. JPEN J Parenter Enteral Nutr. 2006;30:45–51.
- Arazi HC, et al. Soluble Thrombomodulin Levels are Related to Inflammation after Coronary Bypass Surgery. J Clinic Experiment Cardiol. 2011;2:165.

- Banno N, et al. Anti-inflammatory activities of the triterpene acids from the resin of Boswellia carteri. J Ethnopharmacol. 2006;107:249–253.
- 84. Nair PP, et al. Markers of Inflammation and Lineage on Exfoliated Colonic Cells In Pediatric Inflammatory Bowel Disease. J Gastrointest Dig Syst. 2016;S8:001.
- 85. Hotz-Behofsits C. Food and Inflammation: Role of Nutrition in Metabolic Syndrome, Diabetes and Cardiovascular Disease and The Complexity of The Search for A Culprit. Autacoids. 2016;1:e104.
- 86. Muhlestein JB, et al. Effect of the Chinese Drugs Nao Xintong and Dan Hong on Markers of Inflammation and the Lipid Profile in a Hypercholesterolemic Rabbit Model. J Clinic Experiment Cardiol, 2016;2:168.
- 87. Soetikno V, et al. Role of Protein Kinase C-MAPK, Oxidative Stress and Inflammation Pathways in Diabetic Nephropathy. J Nephrol Therapeutic. 2016;S2:001.
- 88. Banerjee M, et al. Modulation of inflammatory mediators by ibuprofen and curcumin treatment during chronic inflammation in rat. Immunopharmacol Immunotoxicol.2003;25:213–224.
- Morin C. New Omega-3 Derivatives Reduce Airway Inflammation and Prevent Rho-Kinase Activation in an Allergic Model of Asthma. J Aller Ther. 2012;S1:003.
- Riesenfeld E, et al. The Temporal Evolution of Airways Hyperresponsiveness and Inflammation. J Aller Ther. 2012;S1:005.
- 91. Badria FA, et al. Boswellia-curcumin preparation for treating knee osteoarthritis: A clinical evaluation. Alt Complement Ther. 2002;8:341–348.
- 92. Kim J and Wessling-Resnick M. The Role of Iron Metabolism in Lung Inflammation and Injury. J Aller Ther. 2015;S4:004.
- 93. Janssen-Heininger YMW, et al. Airway Hyperresponsiveness and Inflammation: Causation, Correlation, or No Relation? J Aller Ther. 2012;S1:008.
- 94. Campbell SC. Obesity, Intestinal Inflammation, and Antioxidant Bioavailability. J Nutr Food Sci. 2012;2:e102.
- Araujo CC, Leon LL. Biological activities of Curcuma longa L. Mem Inst Osawaldo Cruz. 2001;96:723– 728.
- 96. Sconocchia G. Innate Inflammation and Cancer: The Colorectal Carcinoma Paradox. Endocrinol Metabol Syndrome. 2012;1:e105.
- 97. Boswellia serrata. Altern Med Rev. 1998;3:306-307.
- 98. Camoretti-Mercado B, et al. S100A12 and the Airway Smooth Muscle: Beyond Inflammation and Constriction. J Aller Ther. 2012, S1:007.
- 99. Alfonseca-Silva E, et al. Good Protection but Excessive Pulmonary Inflammation in Balb/C Mice Vaccinated with Mycobacterium Bovis Mce-2A Mutant after Challenge with Homologous Strains. Mycobac Dis. 2012,2:111.
- Andersohn F, et al. Use of first- and second-generation cyclooxygenase-2-selective nonsteroidal antiinflammatory drugs and risk of acute myocardial infarction. Circulation. 2006;113:1950–1957.