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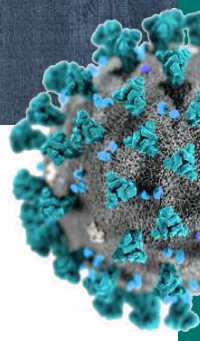
ACCESS BIOLOGY (CUTN)
2020:2;1-8.

ACCESS BIOLOGY

The quarterly official newsletter of the Department of Life Sciences
APRIL 2020



SPOTLIGHT:
COVID-19: Understanding the Game of Firing Cytokine Crackers

E M Shankar PhD FRSB FRCPath
Infection Biology Research Group, Department of Life Sciences
Central University of Tamil Nadu, Thiruvarur, India

SARS-CoV-2 responsible for coronavirus disease 19 originated in Wuhan reportedly spread to over 210 countries across the world represents a huge challenge to be overcome. The pathogenesis is attributed to excessive levels of proinflammatory cytokines in the body, especially in the lungs. In this section, the author coughs out his views on cytokine storm and COVID-19 pathogenesis.

This Issue

Editors Desk

Dear Readers,

As the world is fighting yet another miniscule SARS virus that has caused widespread devastation to humankind, efforts are on to keep-off the virus from spreading any further, and governments are scaling up measures to pump funds for boosting fast-track translational research efforts to promptly develop an effectual vaccine against SARS-CoV-2 that has already devoured millions of lives across the globe. Drug repurposing is being spruced up by scientists, and clinical trials are being scaled-up in an effort to render swift control of the pandemic. The current issue is devoted to covidology and other areas of life sciences, and we sincerely thank *Dr. Dinakar Challabathula* for shouldering this issue, and the entire CUTN fraternity for standing together in responding to COVID-19 at this trying time.

We sincerely hope that the current issue will keep everyone entertained and energetic as has been always!

Editorial Team
Access Biology

The nascent and abruptly devastating SARS-CoV-2 has launched a forcible and terrible attack on humankind eversince it's purported origin from a wet market in Wuhan, China, in September 2019, and in response, scientists have started to decode the Achille's heel of the virus from all quarters, from across the globe. The virus has already infected 9,47,3214 individuals, and has claimed 484249 lives as of 26th June 2020, as reported in the WHO Situation Reports Statistics (WHO, 2020). The virus continues to intensify it's war on the human community inflicting multi-organ failure, especially heralded by severe acute respiratory distress syndrome (ARDS) and intravascular coagulopathy engendering severe loss to human lives.

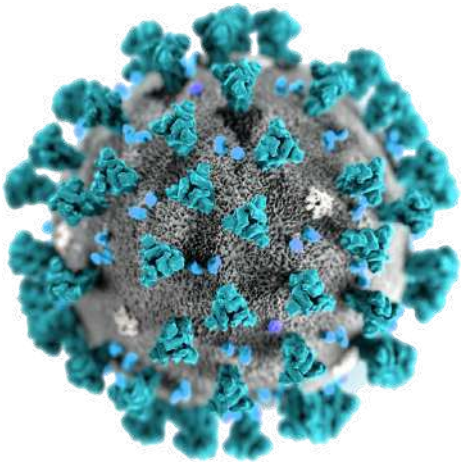
So, what is the problem with the virus? A virus is a virus, and has no other subsistence other than hijacking a susceptible cell! But, 'what's the hell going on' in the body? As soon as the virus takes charge of a susceptible cell, classically, the ciliated upper respiratory airway epithelial cell in the host that almost always expresses ACE2 or TMPRSS2 receptor, especially in the upper respiratory mucosa, it triggers a series of damaging mechanisms in the cell, which a clever virus seldom does! A shrewd virus does not permit a harboring cell to get killed because that indirectly is aimed at killing the host, which means that the virus is foolishly killing it's owner to jeopardise it's chances of survival in the infected individual! The virus appears to inflict damage, but the cell has a means to conceive and deliver SOS signals in the form of chemicals called DAMPS (danger-associated molecular patterns) into its surrounding. An infected cell upon sustaining damage releases DAMPs that can alert the patroling immune cells in the closer vicinity, which are known as inflammatory cells, the neutrophils and the airway macrophages (large cells that can engulf pathogens). These cells concertedly arrive at the site of SARS-CoV-2 presence in response to DAMPs relesed by the infected airway epithelial cells that depends on a concentration gradient. This means, the higher the concentration of DAMPs, the greater the turnover rates of inflammatory cells. Upon arrival at the sites of infection, inflammatory cells engage with the virally-infected cells using pattern recognition receptors (notably the RAGE receptor), which leads to activation of the inflammatory cells. On activation, the inflammatory cells release pro-inflammatory mediators called cytokines whose role is to communicate with several other inflammatory cells entailing their recruitment at the infection site, and their concurrent activation, which eventually magnifies the inflammatory cascade manifold culminating in a clinical condition collectively called as cytokine storm a.k.a. hypercytokinemia syndrome. The important proinflammatory cytokines released at the inflammed site initially happens to be by the upper respiratory epithelial and endothelial cells of capillaries, the former rich in cilia (hair-like fibrous structures that are meant to offer a barricade against viral adherence to the underlying mucosa). But, once infection has successfully manifested, the infected cells further release a battery of cytokines, especially IL-1b, IL-6, TNF-a, IL-8, IL-9, IL-13 etc. Of these, IL-9 is effective at recruiting highly potent inflammatory cells called mast cells (mastocytes) in the mucosa that are effective at magnifying inflammation.

Some of these proinflammatory cytokines, a.k.a endogenous pyrogens (chemicals causing rise in body temperature), travel to the brain where they arrive at a site called hypothalamus, whose posterior aspect has nuclei that nestles a row of thermoregulatory centres. Upon arrival, the cytokines 'tune' the thermoregulatory knobs to increase the body temperature (a.k.a. fever or pyrexia, which is normally an innate mechanism to alert the host that an infection has occurred). The cytokines direct the pituitary gland to release ACTH, a hormone that acts on the adrenal cortex to release corticosteroid hormones that in turn acts on the liver to release a bunch of inflammatory proteins called acute-phase proteins (complement, C-reactive proteins, fibrinogen, mannose-binding lectins, etc). Such proteins contribute significantly to the magnification of the exisiting cytokine storm that becomes too overwhelming. Concurrently, cellular remnants called platelets release clotting factors that necessitate intravascular coagulation (microthrombus formation within capillaries). The final phase of inflammation also includes several other inflammatory mediators such as type 1 interferons that adds to the severity of symptoms. Together, the exaggerated inflammation aggravates manifold leading to extensive swelling, heat, pain and redness, the cardinal signs of inflammation. In COVID-19, the mechanism is often a classical upper respiratory infection like for any other upper respiratory viral infections, excepting that COVID-19 engenders abortive changes in the upper respiratory tract that gradually spreads to the bronchi and alveoli, where alveolar macrophages (dust cells) spearhead the inflammatory cascade, leading to accumulation of pulmonary fluids. The pathology deteriorates further due to damage inflicted on type 2 pneumocytes that are normally responsible for producing chemicals called as surfactants (lipoproteins) that are necessary to lower the surface tension at the air-exchanging zones of the alveoli. Accumulation of fluid (edema) within the alveoli compromises O2-CO2 exchange at the thin capillary-alveoli interface entailing poor O2 tension in the circulation. This condition can end-up in tissue hypoxia (lack of oxygen) and elevation of CO2 (hypercapnia) in the blood circulation. This is when an individual requires mechanical ventilation as he/she suffers from shortness of breath (asphyxia).

The systemic inflammation prevailing in the body could also impact other visceral organs in the body, especially the heart, kidneys, liver and brain resulting in far-reaching consequences. Magnification of cytokine storm also gets aid from several other pathophysiological components, for instance intracellular sensors called inflammasomes (viz., NLRP3, AIM-2), STING, RIG-1, and cells such as natural killer (NK) cells. Cytokine storm remains the most widely accepted disease deterioration mechanism. While, COVID-19 severity is reportedly grave, a vast majority of infected population remain asymptomatic, and surprisingly the global mortality rates have remained relatively far lesser in third-world nations as compared to the developed world, which therefore, remains a conundrum! Does it has something to do with hygiene hypothesis, HLA/KIR haplotypes, gene alterations, life-style, food practices, BCG vaccination and so on? No answers yet; at least until we identify the correlates of protection! Bats and Malayan pangolins, the reservoir hosts of SARS-CoV-2 and several other insidious viruses, remain asymptomatic (means no cytokine storm in them), although might spread the viruses to human. Available evidence suggest that the inflammatory cascade in the mammals gets truncated largely owing to a 'silence-maintained' by host cells despite seeing a load of viruses. Interesting, isn't it? Biology is fascinating, and lets hope the underlying mechanisms of COVID-19 pathophysiology are unveiled promptly for a healthy tomorrow. (Image Courtesy: Business Insider, retrieved on 16th May 2020 from https://markets.businessinsider.com/news/stocks/visualizing-what-covid-19-does-to-your-body-1029083496

I think it's going to take a really, really long time to understand the mechanistic, biological basis of why some people get sicker than others.

-Angela Rasmussen, Columbia University



FURTHER READING

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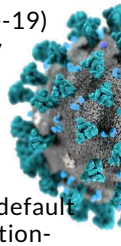
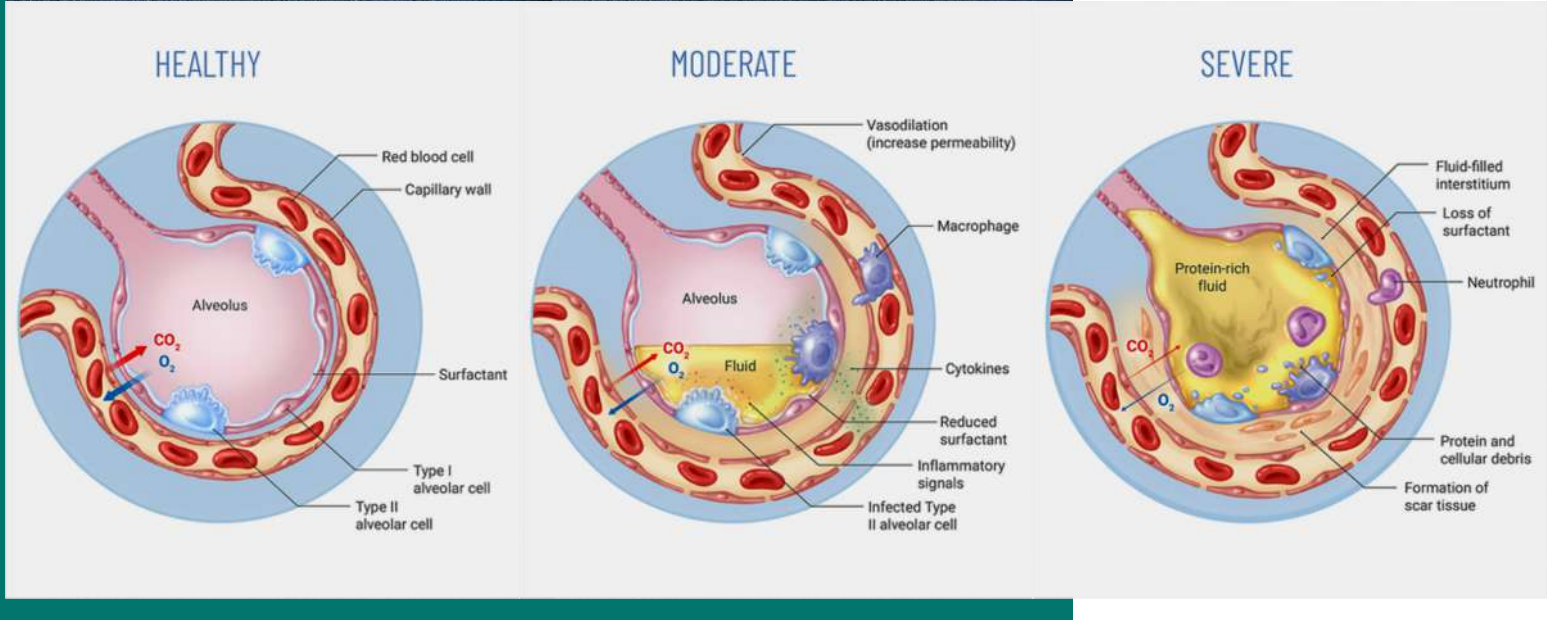


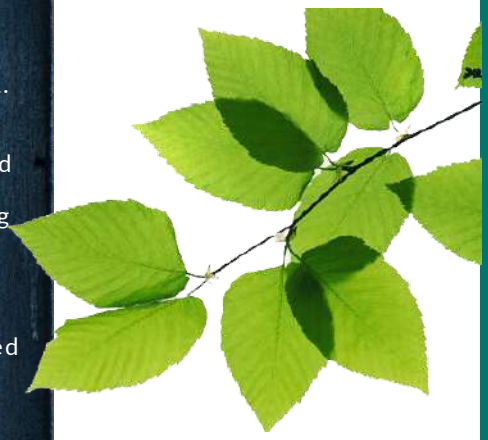


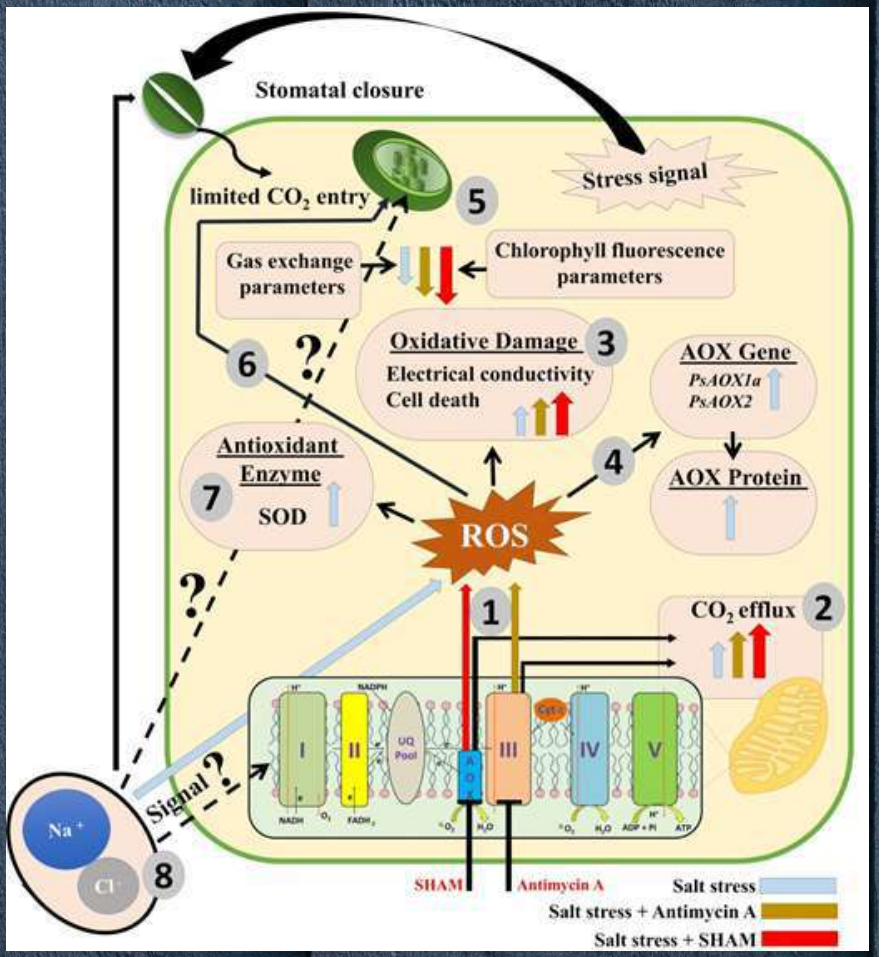
RESEARCH HIGHLIGHTS

Inter-organellar Cross-talk: Unravelling the Chloroplast-Mitochondrial Interdependency Conundrum

Dinakar Challabathula MSc PhD
Plant Molecular Stress Physiology Research Group, Department of Life Sciences, Central University of Tamil Nadu, Thiruvarur, India

Photosynthesis and respiration being the two most important metabolic processes associated with carbon and energy metabolism in higher plants interact with mutual benefits at biochemical and molecular levels. The Plant Molecular Stress Physiology research group of Department of Life Sciences has recently published a research article entitled *Cytochrome oxidase (COX) and alternative oxidase (AOX) pathways of mitochondrial electron transport chain are important for the photosynthetic performance of pea plants under salinity stress conditions* in the *Plant Physiology and Biochemistry* Journal. In the article, the importance of COX and AOX pathways of mitochondrial oxidative electron transport chain for photosynthetic performance of pea plants (*Pisum sativum* L. Pea Arkel cv) was analysed by using the inhibitors antimycin A (AA) and salicylhydroxamic acid (SHAM). While salinity stress in pea plants resulted in decreased CO2 assimilation rates, leaf stomatal conductance, transpiration and leaf intercellular CO2 concentration in a stress dependent manner, superimposition with AA and SHAM caused aggravation in their decrease along with enhanced damage to photosystem (PS) II suggesting the importance of mitochondrial oxidative electron transport for photosynthesis. Additionally, restriction of COX and AOX pathways caused cellular H2O2 and O2− accumulation and promoted cell death. Furthermore, increased expression of AOX1a and AOX2 transcripts along with increased AOX protein levels indicated up regulation of AOX pathway in pea leaves during salinity stress. The results suggested the beneficial role of COX and AOX pathways for optimal photosynthetic performance in pea leaves during salinity stress conditions.





GRAPHICAL ABSTRACT

Schematic representation of effects of restriction of COX and AOX pathways in a plant cell during salt stress.

For details please go to: https://www.sciencedirect.com/science/article/abs/pii/S0981942820302461

FURTHER READING

Analin B, Mohanan A, Bakka K, Challabathula D (2020) Cytochrome oxidase and alternative oxidase pathways of mitochondrial electron transport chain are important for the photosynthetic performance of pea plants under salinity stress conditions. Plant Physiol Biochem. 154: 248-259.

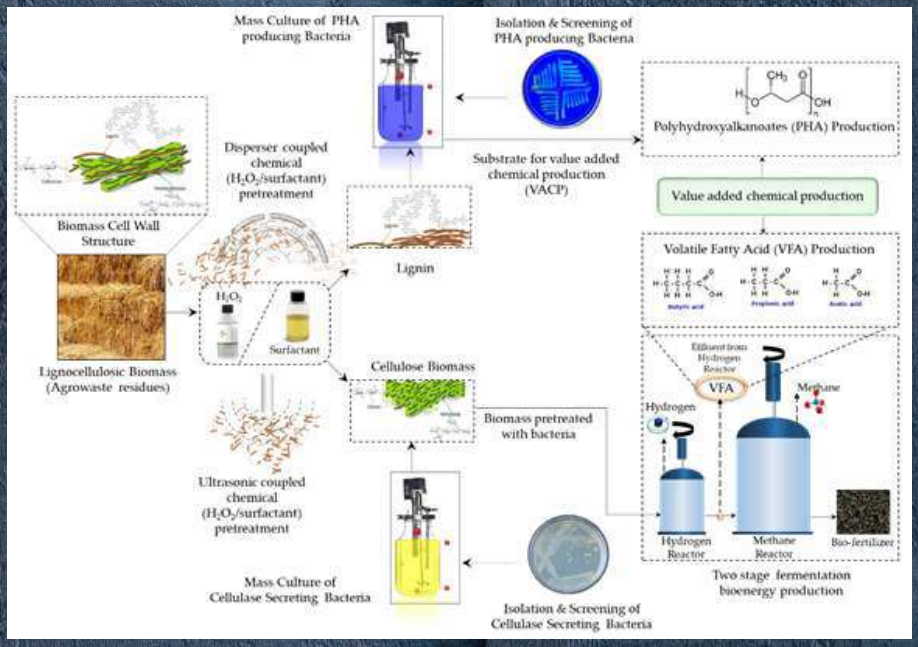
RESEARCH HIGHLIGHTS

Integrated Biorefinery on Lignocellulosic Waste for Value Added Products Recovery

J Rajesh Banu MSc MPhil PhD

Environmental Biotechnology Research Group, Department of Life Sciences, Central University of Tamil Nadu, Thiruvarur, India

Sustainability aspects have driven the industry to renewable sources for energy and chemical production. Fossil fuel has the sustainability issues of depletion, carbon emission and other environmental impacts. Recently bioenergy derived from lignocellulosic biomass such as agricultural residues has attracted more attention due to its higher production rate, easier availability and low cost. In addition, the ever increasing concern about the environmental issues related with the use of plastic bags is well-known for decades. In this aspect, bioplastic production (polyhydroxyalkanoate) could be used as an alternate due to their environmental friendly and easily degradable nature. A new concept portrayed in Figure 1 could contribute to social development of rural areas in India by utilizing agricultural waste as a raw material for the production of multiple products, to be able to reduce net greenhouse gas emissions by replacing fossil-based production.



It is expected that this knowledge would not only lead to an improvement of currently available energy sources, but could also enable the development of a sustainable process on a large-scale and reduces the net greenhouse gas emissions.

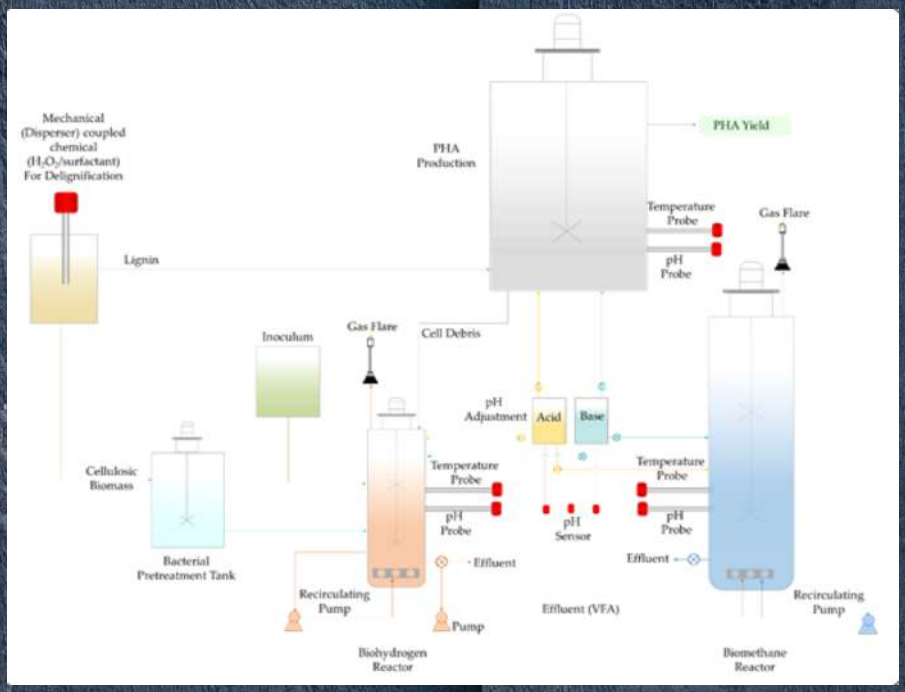


Figure 1: Proposed concept of integrated biorefinery

Objectives:

Utilization of lignin for value added chemical production

Isolation, screening and identification of PHA and cellulase secreting bacteria

Optimization of conditions for delignification and cellulosic hydrolysate separation

Arriving and optimizing design parameters for pilot scale experiment (Figure 2)

Cost benefit analysis and Life cycle assessment (LCA) of project outcome

FURTHER READING

Banu JR, Sugitha S, Kannah RY, Kavitha S, Yeom IT (2018) *Marsilea* Spp. - a novel source of lignocellulosic biomass : Effect of solubilized lignin on anaerobic biodegradability and cost of energy products. Bioresour Technol. 255: 220-228.

Banu JR, Kavitha S, Kannah RY, Poornima Devi T, Gunasekaran M, Kim SH, Kumar G (2019) A review on biopolymer production via lignin valorization. Bioresour Technol. 290: 121790.

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Bhatia SK, Jagtap SS, Bedekar AA, Bhatia RK, Patel AK, Pant D, Banu JR, Rao CV, Kim YG, Yang YH (2020) Recent advancements in lignocellulosic biomass pretreatment and detoxification : effect of key factors, technological improvements and challenges. Bioresour Technol. 300: 122724.

SCIENCE CAFE

The Oral Microbiome-Biofilm Axis: A Prelude to Onset of Oral Carcinogenesis?

Indranil Chattopadhyay MSc PhD

Cancer Biology Research Group, Department of Life Sciences,
Central University of Tamil Nadu, Thiruvarur, India

The author has expressed his ideas based on research findings from his lab that hints the association of formation of biofilm in the oral cavity with the development of inflammation and eventural onset of oral cancer. He proposes that harnessing beneficial bacteria in the oral compartment is key to a healthy life-style.

The Human Oral Microbiome Database (HOMD) reported that 687 species belong to 185 genera and 12 phyla of bacteria such as Bacteroidetes, Firmicutes, Actinobacteria, Proteobacteria, Fusobacteria, Spirochaetes, Synergistetes, Chlamydiae, Chloroflexi, SR1, Saccharibacteria (TM7) and Gracilibacteria (GN02). What is a biofilm? A typical biofilm is made up of mono or polymicrobial cells, polysaccharide, proteins, nucleic acids, and lipids. Oral biofilms are composed of 700 different microbial species, a strong extracellular matrix, which encompasses DNA, proteins, polysaccharides, and several other lipids and salivary glycoproteins, gigingival crevicular fluid, albumin, and host cell components. Bacterial colonization, interaction between bacterial cell surface adhesins and host receptors and extracellular matrix are the key factors for the development, and maturation of oral biofilms. Good oral hygiene is key to preventing access to pathogenic bacteria. Pathogens predominating in oral biofilms contribute to development of dental caries, periodontitis and oral cancer. Bacterial ability to bind to tissues in the oral cavity is the root cause of pathogenesis! Adherent bacteria are unable to attach for longer time-periods on shedding surfaces although teeth offers an excellent binding surface. Gram positive aerobe communities are localized in gingival margin whereas Gram negative anaerobes are found in supragingival biofilms.

Nutrients for bacteria? Saliva is the primary nutrient source for those residing in supragingival biofilms whereas gingival crevicular fluid offers nutrition for bacteria residing in subgingival biofilms. Formation of oral biofilms initially occurs at the supragingival region followed by the formation of subgingival biofilms. Subgingival biofilms are dominated by Gram negative obligate anaerobes. Gram positive aerobes such as *Actinomyces* spp. and oral streptococci are responsible for the initial colonization of dental surfaces. Streptococcal AGI/II proteins induces co-aggregation between *S. gordonii* and *A. oris*. Interaction between fimbriae of *Actinomyces naeslundii* with prolinerich salivary proteins that regulates interbacterial binding. The Gram negative anaerobic bacterium *Fusobacterium nucleatum* acts as a connecting link between early and late colonizers of the oral biofilms. Evidence also suggests that *F. nucleatum* supports the growth of *Porphyromonas gingivalis*. Metabolic products such as ammonia and organic acid of *F. nucleatum* favours growth of acid-sensitive bacterium *P. gingivalis* through enhancement of pH in biofilm. *P.gingivalis, Aggregatibacter actinomycetemcomitans, Prevotella intermedia, Eubacterium* spp., *Tannerella forsythia*, and a few others are considered as late colonizers of the oral biofilm. Gram positive oral bacteria such as streptococci use 17–21 amino acids long competence signalling peptides (CSP) and both Gram positive and Gram negative bacteria use autoinducer-2 (AI-2) to communicate each other in oral biofilms. Lactates of streptococci and lactobacilli has been utilized by *A. actinomycetemcomitans*. Glucosyltransferases (GTFB, GTFC, and GTFD) of cariogenic bacteria such as S. mutans are responsible for the synthesis of glucans. GbpA, GbpB, GbpC, and GbpD surface proteins of bacteria bind with the glucans. These enzymes and proteins are involved in sucrose-dependent pathway that induces plaque formation. Chemical communication among bacterial cells via expression of genes in response to high cell density is defined as quorum sensing.

Periodontal anaerobic pathogens such as *P. gingivalis*, *Prevotella intermedia* and *F. nucleatum* produce highest levels of AI-2. CSP is responsible for the formation of biofilm, bacteriocins, stress responses, acid tolerance and genetic conversion by *S. mutans*. Commensal and pathogenic bacterial species bypass host immune responses by forming biofilm. Streptococci produce oral adhesins, such as PaG, SspA, antigen I/II, amylase-binding proteins, and type 1 fimbriae-associated proteins. *S. gordonii* and *P. gingivalis* use AI-2 in oral biofilm. *S. gordonii* reduces dental plaque formation through production of hydrogen peroxide. Hydrogen peroxide of *S. gordonii* inhibits the growth of *Actinomyces naeslundii*. *F. nucleatum* can also associates with *S. cristatus*. Streptococci, actinomyces, and lactobacilli inhibit the growth of bacterial species by transforming the oral microenvironment acidic. Porphyromonas, campylobacters, *T. forsythia*, *Treponema denticola* and *A. actinomycetemcomitans* in the biofilm are responsible for augmenting inflammation in periodontitis. Evidence is also accumulating that chronic inflammation arising from biofilm formation in the oral cavity plays a solid role in increasing the risk of development of oral cancer.

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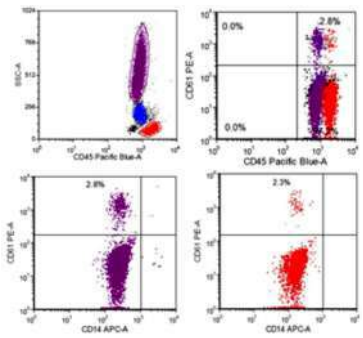
SCIENCE CAFE

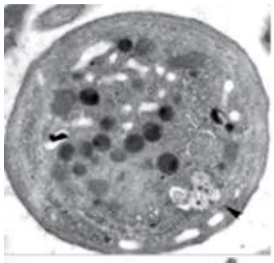
Mechanisms of Platelet Activation and Its Impact on Health and Disease

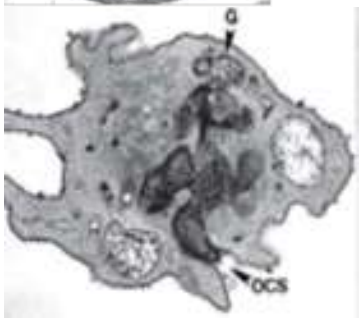
Meganathan Kannan MSc PhD

Blood and Vascular Biology Research Group, Department of Life Sciences, Central University of Tamil Nadu, Thiruvarur, India

The author is a platelet biologist, who describes herein, some of the key mechanisms implictaed with platelet activation and the impact it might have on health and disease manifestations.







Images panels: FACS analysis of activated platelets (top); Electron micrography of a normal (middle) and an activated (bottom) platelet.

Platelets become activated when they come in contact with exposed collagen in the areas of endothelial damage. In normal, resting platelets are in discoid shape, inactive and circulate in blood for about 7-10 days; upon activation the shape changes to a compact sphere with long dendritic extensions facilitating platelet deposition at the sites of injury. The morphological and physiological changes of the platelets and phosphatidylserine exposure help in stable platelet plug formation and thus participate in primary hemostasis. The platelet activation process involves various steps including adhesion, aggregation and secretion of intermediate molecules. The adhesion of circulating platelets to the vascular sub-endothelium is facilitated by von Willebrand Factor (VWF), which is a key for initiating platelet deposition and thrombus formation. A membrane glycoprotein, GPIbα, a constituent of GPIb-IX-V complex binds to VWF and mediates the process of platelet adhesion. The platelet GPIb comprises of two subunits, α and β, of which the α subunit exhibits binding site for VWF that facilitates the initial process of platelet adhesion on injured sub-endothelium. The immunoreceptor tyrosine-based activation motif (ITAM) containing proteins associated with GPIb-IX complex play an important role in signal transduction during the process of adhesion. Though GPIb-IX does not require ITAM for its primary signaling, this motif greatly helps the amplification of signaling initiated by GPIb-IX. Activated platelets (a) release granular contents of adenosine-di-phosphate that binds to P2Y1 and P2Y12 receptors, (b) synthesize thrombin that binds to protease-activated receptors and (c) produce thromboxane A2 which eventually, through G-protein coupled receptors (GPCRs), activate the GPIIb/IIIa (αIIbβ3) integrin receptors. Finally the activated integrin receptors bind to fibrinogen that results in aggregation of platelets. The mechanism of platelet activation is not only based on the receptor-mediated signaling, but also depends the bidirectional signaling of Inside-Out and Outside-In signaling across the plasma membrane. Upon platelet activation, the Inside-Out signaling, increases the affinity of integrin receptor towards its ligands such as fibrinogen. This is achieved with the help of a key protein, Talin, a cytoskeletal protein which induces integrin activation by binding to the cytoplasmic domain of β integrin receptor. The head domain of Talin binds to the integrin β cytoplasmic tail and thus mediates stable thrombus formation. Following inside-out signaling, a range of cellular events occur which mediate the augmentation of platelet activation and thrombus growth. Similar to Talin which plays a critical role in Inside-Out signaling, the process of Outside-In signaling utilizes multiple key adopter proteins such as Kindlin-3, Dab2, FAK, Syk, etc. Activated platelets aggregate and secrete biologically active intermediate substances that further potentiate platelet activation through autocrine as well as paracrine mechanisms. Defects in any of the functions result in platelet disorders such as Glanzmann thrombasthennia, Bernard Soulier syndrome, etc. Increased platelet activation has been associated with various pathological conditions such as acute coronary syndrome, stroke, peripheral vascular disease and other inflammatory diseases. In addition, the open canalicular system, a connector between the interor and surface of platelets, plays an importnt role in platelet activation as abnormalities of this system leads to the conditions called Budd-Chiari syndrome and May-Hegglin anomaly.

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Stefanini L, Bergmeier W (2018) Negative regulators of platelet activation and adhesion. J Thromb Haemost. 16(2): 220-30.

DLS Congratulates

Ms Arpita Shukla (R142001), Research Scholar working under the supervision of **Dr. Jayalakshmi Krishnan**, Department of Life Sciences defended her PhD research at a public viva voce held on 11th of June 2020. The title of her thesis was Seasonal fluctuations and insecticide resistance in *Aedes* (*Stegomyia*) *albopictus* (Skuse, 1895) in field populations in Thiruvarur District of Tamil Nadu, India. Her research work focused on mosquitoes, their seasonal changes and their resistance capacity at Thiruvarur district. The findings of her study are useful for public health vector control implementation by national programs. The faculty, staff and students of the Department of Life Sciences congratulate Ms Arpita and wishes her all the best in all her future endeavors and academic career.

PUBLICATIONS

Shukla A, Rajalakshmi A, Subash K, Jayakumar S, Arul N, Srivastava PK, Eapen A, Krishnan J (2019) Seasonal variations among dengue vector mosquitoes in rural settings of Thiruvarur District in Tamil Nadu, India. J Vector Borne Dis. (Accepted)

DLS Congratulates

Ms Madhusmita Panda (R142002), Research Scholar working under the supervision of **Dr. Indranil Chattopadhyay**, Department of Life Sciences, delivered the pre-thesis submission seminar on 10th of June 2020 via a Webex meeting at 11 AM at the Pro-VC Hall, Central University of Tamil Nadu, Thiruvarur. The title of her seminar was *Alterations in oral bacterial communities associated with oropharyngeal, hypopharyngeal and oral cancer in Assam (North-East India).*

PUBLICATIONS

Chattopadhyay I, Panda M (2019) Recent trends of saliva omics biomarkers for the diagnosis and treatment of oral cancer. J Oral Biosci. 61(2):84-94.

Chattopadhyay I, Verma M, Panda M (2019) Role of Oral Microbiome Signatures in Diagnosis and Prognosis of Oral Cancer. Technol Cancer Res Treat. 18:1533033819867354.

Panda M, Rai AK, Rahman T, Das A, Das R, Sarma A, Kataki AC, Chattopadhyay I (2020) Alterations of salivary microbial community associated with oropharyngeal and hypopharyngeal squamous cell carcinoma patients. Arch Microbiol. 202(4): 785-805.

DLS Congratulates

Ms N Sukritha (R142002), an iMSc alumnus of 2014-19, has been selected for a PhD programme in Chemical Ecology at the nGICE (Max Planck Next Generation Chemical Ecology) in Germany. She will be working on mosquito behaviour under the supervision of Prof. Rickard Ignell (SLU) in a collaborative project between the Swedish Agricultural University (Stockholm, Sweden), Max Planck Institute (Germany) and the Lund University (Scania, Sweden). The department congratulates Ms. Sukritha for bringing laurels to DLS and CUTN and wishes her with loads of good luck.

Journal Publications (January - April 2020)

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ACCESS BIOLOGY

The quarterly official newsletter of the Department of Life Sciences

APRIL 2020

The Professor & Head
Department of Life Sciences
School of Life Sciences
Central University of Tamil Nadu
Neelakudi Campus
Thiruvarur 610 005
Tamilnadu, India
eMail: hodlifesciences@cutn.ac.in.