Morfologia & Clinica

Studi in onore di Abdo Jurjus

Palermo University Prize
Dire delle qualità scientifiche ed umane proprie del prof. Abdo Jurjus (nato a Ras El Kharf in Libano il 5 Luglio del 1948, paese dove, nel 1972, consegna la laurea in Scienze Biologiche), risulta particolarmente stimolante. E ciò, proprio per quel dipingere il suo costante e proficuo interesse verso il vasto scenario delle scienze morfologiche, per le sue ricerche conformate sia nei laboratori della più antica Scuola di Medicina libanese, l’American University of Beirut (AUB), sia per le sue intense collaborazioni svolte in U.S.A., già con Robert Gallo, figura di vanglia negli studi sull’HIV, sviluppando importanti contributi sul cancro, sui processi infiammatori, sull’HIV e sui fattori di crescita.

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Describing the scientific and human qualities of Professor Abdo Jurjus is very inspirational. He was born on July 5th 1948 in Ras El Kharf, Lebanon, where he took his master degree in Biological Sciences in 1972. He has always been interested in studying the vast field of morphological sciences, has conducted his researches in the laboratories of the American University of Beirut (AUB), Lebanon’s oldest medical school. He has strictly cooperated with the American scientist Robert Gallo, a major figure in HIV studies, and has greatly contributed in studies on cancer, inflammatory processes, HIV and growth factors.
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Premessa
di Aldo Gerbino
Complesso Monumentale dello Steri
Sala Magna
21 | 06 | 2017
h 10,30 a.m.
Piazza Marina 61, Palermo

Palermo University Prize

Program

Greetings:
Fabrizio Micari, Palermo University Rector
Ada Maria Fiorema, Vice Rector to International Relations
Francesco Vigli, Dean of School of Medicine and Surgery

Speakers:
Giovanni Zuneta, Professor of Anatomy - Director of Biomedical and Neuroscience Department - Le Departamento Internazionale Ricerche di Biofaa, Italy
Aldo Gerina, Professor of Histology - Responsible of Tissue Banking
Angelo Leon, Professor of Histology and Embryology

Lectures:
Evolution of Anatomical Science Teaching
Prof. Abd Romanos Jurys
Professor of Morphological Sciences
American University of Beirut, Lebanon

Invited lectures:
Isabelle Miletich, DDS, BSc, MSc, PhD (Group leader)
King's College, London
Molecular Regulation of Salivary Gland Homoeostasis
Rosalyn Jurys, Professor of Anatomy
School of Medicine, George Washington University, Washington, USA
Anatomical Relation in Chewing Corns

Charles:
Francesco Cappello, Professor of Anatomy
Giovanni Tornadella, Professor of Surgery

Fabrizio Micari, Palermo University Rector
Palermo University Prize
Award Ceremony
to Prof. Abd Romanos Jurys

Abdo Romanos Jurys

Prof. JURUS, renowned Lebanese scientist and dean of his entire career to practice in the field of Morphology, he works at Bedossa's Institute of Health-NIH, as a well-dropping research on tumors.
For more info, he has been interested in seeking new, and knowledge in the field of classical and functional.

ATTENDANCE CERTIFICATE TO ALL PAR
Dysbiosis, Inflammatory Bowel Disease and colon cancer: there is a correlation?

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Key words: dysbiosis, Inflammatory Bowel Disease, IBD, colorectal cancer.

ABSTRACT
Dysbiosis has been linked to IBD. The intestinal microbiota plays a fundamental role in health and in the progression of diseases such as IBD and CRC. Many cancers arise from sites of infection and chronic inflammation. Patients with inflammatory bowel disease (IBD) have an increased risk of 10%-15% developing colorectal cancer. Therefore, a complete understanding of the composition and function of the gut microbiota is critical.

THE INTESTINAL MICROBIOTA: GENERAL ASPECTS
The microbiota is an ensemble of microorganisms that resides in the gastro-intestinal tract to form a “tissue into the organ”. Colonization occurs mainly in the colon that typically harbors more than 500 different species of bacteria.
In utero, the intestine of the mammalian fetus is sterile. At birth, the intestinal microbiota is acquired by ingesting maternal anal or vaginal organisms, ultimately developing into a stable community, with marked variations in microbial composition between individuals. The complex microbial population of the intestinal tract plays an important role in host nutrition and health, since different bacterial species establish a rich interaction network involving mutualism, symbiosis and pathogenicity. Colonization occurs mainly in the colon that typically harbors more than 500 different species of bacteria. It establishes a relation of mutual benefit with the gastro-intestinal tract through its metabolic activities.
The microbiota exerts various functions, such as the inhibition of pathogenic bacteria and the synthesis of short-chain fatty acids. It promotes the absorption of nutrients and minerals, acts as an immune system to the mucous membrane, and supports the synthesis of vitamins and amino acids and the metabolism of proteins.
Most of the bacterial population forming the microbiota is not pathogenic. However,
the balance between pathogenic and non-pathogenic bacteria promotes a positive influence in favor of the gastro-intestinal tract. Changes in the microbiota composition are mainly influenced by diet and age, as well as genetic factors.

Numerous studies performed in human and animal models have investigated changes in the composition of microbiota in several gastrointestinal inflammatory diseases, including IBD. The development of IBD is often associated with qualitative and quantitative disorders of the intestinal microbial flora (dysbiosis).

Resident bacteria play an important role in initiating and perpetuating intestinal inflammation in IBD. It is well known that the quality and quantity of intestinal microbiota vary with disease; some bacterial species may promote the development of a specific disease, while concurrently protecting the host from another disorder.

Dysmicrobism can cause alterations in the immune response and can, in particular, lead to gut associated lymphoid tissue (GALT) activation. Alteration of this equilibrium can produce dysbiosis and IBD. In particular, the change of a bacterial microflora into a saprophytic bacteria population may alter tight junctions, which are responsible for the correct functioning of the epithelial surface in the intestinal mucosa. Specifically, several authors have reported a reduction in the relative abundance of “beneficial bacteria”, such as Bifidobacteria and Lactobacilli, and an increase in potentially dangerous bacteria, such as E. coli, in gut inflammatory diseases, including IBD.

**DYSBIOSIS AND INFLAMMATORY BOWEL DISEASE**

Inflammatory bowel diseases (IBD) are chronic, recurrent and multifactorial conditions affecting the gastro-intestinal tract. IBD is diagnosed in 2.5–3 million people in Europe and more than 1 million people in the United States.

Although the aetiology of IBD is still not fully understood, it involves a complex interaction between genetic, luminal and environmental factors, including diet, cigarette smoke and drug exposure, infections, geography and stress, that trigger an inappropriate mucosal immune response. In IBD the suspected aetiological cause is a dysregulation of the intestinal immune system, in which it is possible to determine a cross-like immune reactivity against the resident microbiota. They are characterized by an aberrant inflammatory reaction towards the microbiota in the gastro-intestinal tract. Changes in the composition of intestinal microbiota and an abnormal immune response to gut microorganisms are likely to be the key factors in the onset and progression of IBD.

The pathogenetic mechanisms underlying the inflammatory response are still under investigation.

The ulcerative colitis (UC) and the Crohn's disease (CD) are the principal pathological entities of inflammatory bowel disease (IBD).
In UC, a large inflammatory infiltrate, mainly represented by neutrophils, is observed both in the mucous membrane and the underlocated membrane [3]. Most secreted cytokines are interleukin-4, interleukin-5, and interleukin-13. CD can involve any part of the intestinal mucosa, with a typically segmental lesion distribution, and presents unaffected areas of mucosa along the intestine. CD rarely affects the rectum, with a possible presence of fistulas, abscesses and/or anal stenosis. In CD, all the layers of the intestinal wall are involved, with a segmental distribution of the painful blisters, distinct from the inflammatory granulation tissue generated by the action of gamma interferon and TNF-ALPHA.

DYSBIOSIS, IBD AND COLORECTAL CANCER
The colorectal cancer (CRC) has been increasing in recent years and its mortality rates are very high. Patients with IBD have an increased risk of 10%-15% developing CRC that is a common disease of high economic costs in developed countries. Many cancers arise from sites of infection and chronic inflammation. The microbiota analysis on tissues and fecal materials has identified various microbial groups associated with CRC. Stool samples derived from CRC patients harbor a higher population of bacteria belonging to the group Bacteroides-Prevotella compared with normal controls.
It has been reported for several decades that carcinomas mostly develop from adenomas and chronic inflammations.
The big question is how could this chronic inflammation progress and express itself, and how could its complications lead to the formation of cancerous cells?
Increasing evidence indicates that dysbiosis favors the production of genotoxins and metabolites associated with carcinogenesis. It also involves another complex series interactions between immune cells (T cells B cells, NK cells, mast cells, macrophages), interleukins, bradykinins, complement system factors, prostaglandins, tumour necrosis factors, histamines, hormones and activated neutrophils products like myeloperoxidase, radicals and oxydants.

CONCLUSION
Although dysbiosis has been linked to IBD, it is still essential to clarify whether abnormal microbiota is the initiating factor that contributes to the development and persistence of IBD, or a secondary symptom of gut inflammation. The intestinal microbiota plays a fundamental role in health and in the progression of diseases such as IBD and CRC. Many cancers arise from sites of infection and chronic inflammation. Therefore, a complete understanding of the composition and function of the gut microbiota is critical.
REFERENCES


