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The expression of many bacterial genes adapts itself in an almost in stantaneous and reversible way to specific environmental changes. More specifically, the concentration of a number of

metabolites, a function of the amounts of enzymes involved in their synthesis or degradation, in turn retroacts on the rate of synthesis of these enzymes. The genetic bases for this regulation were established by JACOB and MONOD (1961). These authors also showed how the known elements of these regulatory mechanisms could be connected into a wide variety of circuits endowed with any desired degree of stability, in order to account for essentially irreversible processes like differentiation (MONOD and JACOB, 1961). The general principles used by JACOB and MONOD in their study of negative regulation were extended to positive regulation by ENGLESBERG et al. (1965). An independent approach permitted the discovery of positive controls in temperate bacteriophages (see below, III). Each control operation is mediated by a pair of complementary

genetic elements (hereafter called "control cell"): a control gene which produces a l control (or regulator) protein and a control site which is the target for the regulator protein. Negative control means that the control protein (repressor) prevents gene expression. One deals with positive control when the control protein (activator) is necessary for this expression. It has become apparent that, as initially postulated by JACOB and MONOD, control of gene expression operates, at least to a large extent, at the transcriptional level.

Bacterial plasmids are circular double-stranded DNA molecules that are physi cally separate from the bacterial chromosome. They are replicated and stably inherited in the extrachromosomal (autonomous) state. The plasmids of entero bacteria can be divided into two

distinct groups according to their size: (i) small plasmids with MW of less than 10 Mdal, and (ii) large plasmids with MW ranging from 50-100 Mdal. These two groups differ strikingly in their copy numbers per cell (multiplicity). Whereas most small plasmids are multicopy plasmids (20-100 copies per cell), large plasmids are normally present at a multiplicity similar to the number of chromosomal genome equivalents (oligo copy plasmids). Furthermore, large plasmids can promote the transfer of DNA by conjugation and are therefore classified as conjugative plasmids. Since this property depends on the presence of the tra operon, a 15-20 Mdal segment of DNA (Helmuth and Achtman, 1975), small plasmids are necessarily nonconju gative. Because of their inability to mediate DNA transfer, small plasmids have often been designated as "nontransmissible. " This is

clearly a misnomer since nonconjugative plasmids can in general be mobilized for conjugal transfer by a conjugative plasmid present in the same cell. Plasmids can further be classified with respect to their ability to continue replication in the absence of de novo protein synthesis (stable replication).

Measles, also called the greatest killer of children in history, still annually affects about 50 million individuals and causes close to a million deaths primarily in developing countries. Before the advent of measles vaccine some 30 years ago, these figures were roughly three times higher. Attenuated measles virus (MV) strains, all quite closely related to the original Edmonston isolate, have a very good record as a safe and highly efficacious vaccine and have brought down the measles toll in industrialized countries to

almost negligible levels. However, recent outbreaks in the USA and Europe have again brought the measles problem to public attention. Sadly enough, these outbreaks were more instrumental in inducing activities to drastically reduce and hopefully finally eradicate measles than were the ten thousand times higher number of victims in developing countries. To reach this goal, as detailed in this volume, apparently it is not enough to of the existing vaccine as was the rigorously enforce use case with smallpox eradication: the intricacies of measles disease phenomena, in particular the generalized immune suppression which favors secondary infections, require more basic knowledge of the virus-host interactions and probably the development of new vaccines for special applications such as first immunizations of very young infants in developing countries.

Our gut is colonized by numerous bacteria throughout our life, and the gut epithelium is constantly exposed to foreign microbes and dietary antigens. Thus, the gut epithelium acts as a barrier against microbial invaders and is equipped with various innate defense systems. Resident commensal and foreign invading bacteria interact intimately with the gut epithelium and can impact host cellular and innate immune responses. From the perspective of many pathogenic bacteria, the gut epithelium serves as an infectious foothold and port of entry for disseminate into deeper tissues. In some instances when the intestinal defense activity and host immune system become compromised, even commensal and opportunistic pathogenic bacteria can cross the barrier and initiate local and systematic infectious diseases. Conversely, some highly

pathogenic bacteria, such as those highlighted in this book, are able to colonize or invade the intestinal epithelium despite the gut barrier function is intact. Therefore, the relationship between the defensive activity of the intestinal epithelium against microbes and the pathogenesis of infective microbes becomes the basis for maintaining a healthy life. The authors offer an overview of the current topics related to major gastric and enteric pathogens, while highlighting their highly evolved host (human)-adapted infectious processes. Clearly, an in-depth study of bacterial infectious strategies, as well as the host cellular and immune responses, presented in each chapter of this book will provide further insight into the critical roles of the host innate and adaptive immune systems and their importance in determining the severity or completely preventing

infectious diseases. Furthermore, under the continuous threat of emerging and re-emerging infectious diseases, the topic of gut-bacteria molecular interactions will provide various clues and ideas for the development of new therapeutic strategies.

Binding of various ligands (hormones, neurotransmitters, immunological stimuli) to membrane receptors induces the following changes: 1. Receptor redistribution (clustering, "capping") 2. Conformational changes that can be detected by fluorescent probes 3. Alteration in membrane fluidity (spin label and fluorescence polarization probes) 4. Changes in fluxes of ions and metabolites 5. Increased phospholipid turnover (especially of phosphatidyl inositol) 6. Activation of membrane-bound enzymes (adenyl cyclase, ATPase, transmethylases). Some of the early

changes resulting from or associated with the binding (adsorption) of virions to the host cell membrane are of the same type. Adsorption of animal viruses to cells is the ftrst step in a chain of events resulting in the production of progeny virus on the one hand and in damage to cells and tissues on the other. In the classical studies of viral infection, cells are adsorbed with virus, usually for 60 min, and the changes induced by the virus in the host cell are recorded thereafter. In the past decade, more and more studies have been aimed at the events occurring in these ftrst 60 min of the so-called adsorption period. These studies deal with the nature of adsorption, e. g. , the ligand-receptor type of interaction between the virus and the cell membrane. Many receptors for viruses were identifted and so were the viral proteins which take part in adsorption.

One of the most promising new approaches for the prevention of HIV transmission, particularly for developing countries, involves topical, self-administered products known as microbicides. The development of microbicides is a long and complicated process, and this volume provides an overview of all the critical areas, from the selection of appropriate candidate molecules and their formulation, preclinical and clinical testing for safety and efficacy, strategies for product registration and finally, issues associated with product launch, distribution and access. The book will prove valuable to both those working in the field and all others who are interested in learning more about this product class, which has the potential to significantly impact the future of this devastating epidemic.

Continuous genetic variation and selection of virus subpopulations in the course of RNA virus replications are intimately related to viral disease mechanisms. The central topics of this volume are the origins of the quasispecies concept, and the implications of quasispecies dynamics for viral populations.

Scientific research on dengue has a long and rich history. The literature has been touched by famous names in medicine- Benjamin Rush, Walter Reed, and Albert Sabin, to name a very few- and has been fertile ground for medical historians . The advances made in those early investigations are all the more remarkable for the limited tools available at the time. The demonstration of a viral etiology for dengue fever, the recognition of

mosquitoes as the vector for transmission to humans, and the existence of multiple viral variants (serotypes) with only partial cross-protection were all accomplished prior to the ability to culture and characterize the etiologic agent. Research on dengue in this period was typically driven by circumstances. Epidemics of dengue created public health crises, although these were relatively short-lived in any one location, as the population of susceptible individuals quickly shrank. Military considerations became as a major driving force for research. With the introduction of large numbers of non-immune individuals into endemic areas, dengue could cripple military readiness, taking more soldiers out of action than hostile fire. Dengue and dengue hemorrhagic fever, which assumed pandemic proportions during the latter half of the last century, have shown no

indication of slowing their growth during this first decade of the twenty-first century. Challenges remain in understanding the basic mechanisms of viral replication and disease pathogenesis, in clinical management of patients, and in control of dengue viral transmission. Nevertheless, new tools and insights have led to major recent scientific advances. As the first candidate vaccines enter large-scale efficacy trials, there is reason to hope that we may soon "turn the corner" on this disease.