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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).

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.

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.

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.

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.

This book offers a comprehensive review of basic and clinical research on Varicella-zoster Virus, the only human herpesvirus for which vaccines to prevent both primary and recurrent infection are approved.