Chapter 8
CRISPR–Cas9 Technology for Medical Diagnostics

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ABSTRACT

CRISPR-Cas9 is a paradigm shift in the molecular diagnostics field that goes beyond its beginnings as a gene editing technology to become a radically new diagnostic platform. The review discusses the history and uses of CRISPR-based diagnostics in infectious diseases, oncology and genetic screening. CRISPR systems offer explicit, customizable, and molecular recognition features, which can be applied to create efficient, quick, and economical diagnostic systems. The combination of such technologies is one that will bring democratized, universally available molecular diagnostics, and change healthcare in resource-abundant and resource-constrained contexts, and eventually makes CRISPR a foundation of precision diagnostics.

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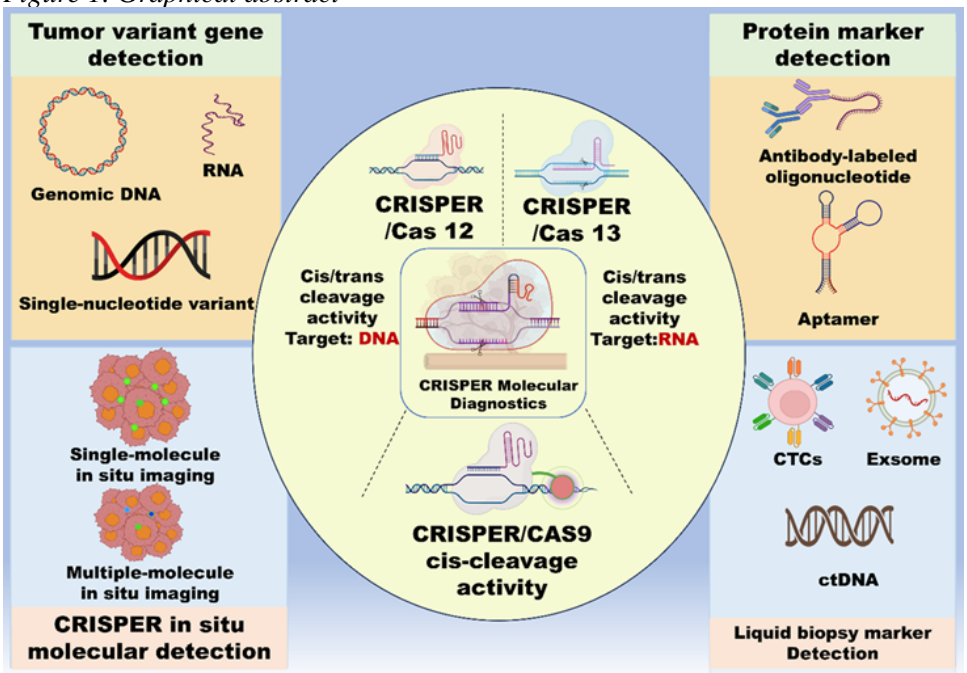
14.1 INTRODUCTION

Figure 1. Graphical abstract



Clustered regularly interspaced short palindromic repeats (CRISPR), initially identified in the Escherichia coli genome in 1987, constitute a component of the adaptive immune system found in prokaryotic organisms, including archaea and bacteria (Mohanraju et al., 2022).Further research into the structure and function of the CRISPR system has led to advancements in gene editing, gene therapy, and diagnostics, building on the original discovery of the system in bacteria as an adaptive immune system (Hossain, 2021). Adapted into an exact gene-editing program, the type II CRISPR system utilizing the Cas9 nuclease, in particular, this programmable system of RNA-guided defence became an appealing system to develop rapid, sensitive, and cost-effective diagnostics. genome modification, the specificity, programmability and molecular recognition properties of CRISPR-Cas9 immediately placed the program as an attractive platform to create rapid, sensitive, cost-effective diagnostics (Zhou & Simonian, 2024).

Diagnostic replacements gene editing takes advantage of the intrinsic capacity of the CRISPR-Cas proteins to find specific nucleic-acid sequences. CRISPR based diagnostics started with the realization that catalytically inactive forms like dCas9 still have a strong affinity to bind DNA but fail to cleave the target (Bhardwaj et al.,