Sumoylation and ubiquitination in infectious disease and cancer

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Sumoylation and ubiquitination are important posttranslational modifications for cellular protein functions as well as virus replication. While many viruses involve interactions with host proteins to interfere with or usurp cellular sumoylation for creating a suitable replication environment, little is known about sumoylation in antagonizing viral infection. Here, we show that sumoylation promotes EV71 3C protein ubiquitination for degradation, correlating with a decrease of EV71 in virus replication and cell apoptosis. Our results uncover a previously undescribed cellular regulatory event against EV71 virus replication and host cell apoptosis by sumoylation at 3C protease, serving as a template for sumoylation in antagonizing viral replication. In addition, acute promyelocytic leukemia (APL) is predominately caused by the chromosomal translocation between *RARA* and its counterpart such as *PML* and *PLZF*. While both retinoic acid and arsenic trioxide treatment targeting PML/RARA transcriptional repression and protein catabolism are successful to eradicate PML/RARA-elicited myeloid differentiation block, very little is known about effective treatment for PLZF/RARA-induced APL. We identified a deubiquitinating enzyme regulating PLZF/RARA stability and provided a potentially therapeutic intervention in PLZF/RARA-associated APL.