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Program AND Abstracts

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116. Inactivation of Respiratory Viruses Using Far-Infrared Radiant Heater

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Virus inactivation is important to control the viral diseases and spread. Not like vaccine or pharmacological drug, it can prevent various viruses from spreading between the environment, people and animals. As high temperature exposure could be a good option to inactivate viruses, we have tested the heat sensitivities of several viruses mostly respiratory, such as influenza viruses, human rhinoviruses, coronaviruses, and adenoviruses. Mineral heat ware (produced by Ecopartners Ltd, Seoul, Korea) was used as a heat source. Far-infrared radiant heater is a regenerative FIR heating system applied physical and bioware technologies. The wavelength of far infrared rays was 4 ~ 10 um. Test temperature was 170°C and 210°C. Most viruses were inactivated within 60 seconds at both temperatures but certain viruses showed incomplete inactivation. Understanding the relationship between heat sensitivities and physical characteristics of viruses may help the control of emerging viruses.

117. VIRIP – an Anti-HIV Host Peptide Output Hypothesis

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Alpha1-antitrypsin is known to be a precursor of bioactive peptides. Full-length protein proteolytic cleavage has been regarded as the only mechanism of these peptides production. VIRIP (virus-inhibitory peptide) is such a derivative, a potent anti-HIV host peptide (Münch J., 2007). It is also believed to be produced proteolytically, although its C-terminus proteolytic formation hasn't been demonstrated yet. Our previous data on *SERPINA1* gene coding region relative expression analysis implied the existence of a more fine mechanism – regulation at a transcriptional level. Northern hybridization with cRNA probe derived from exon 5 on different total RNA samples proved the *SERPINA1* gene short transcripts existance. Our hypothesis has been supported by an independent research (Matamala N., 2017). Here we hypothesize that VIRIP can be produced by immune cells from unique short transcript(s) containing coding region of exon4/exon5 and/or exon5, which are polyadenylated at a cleavage site (T) located 13 nt downstream of putative AACAAA polyadenylation signal, thus producing a stop-codon and a transcript lacking 3'-UTR and encoding the VIRIP sequence. It has been established that such 3'UTR-depleted transcripts are highly stable, that must be important in case of VIRIP. AAT full-length blood concentration in HIV-infected patients (around 30uM) tends not to exceed its normal levels (Bryan C.L., 2010), but VIRIP IC50 (4-20uM, depending on HIV subtype) is relatively high (Münch J., 2007). The existence of stable unique transcript(s) may increase local VIRIP level. Our hypothesis may help to solve such a discrepancy.

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