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Food for thought

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Corresponding Affiliation: Aff1

Aff1 Oregon Health Sciences University, OR, USA

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Context

Secretion of mucosal IgA constitutes an important component of the bronchial immune system. Indeed, the presence of antigen-specific IgA has been associated with protection against respiratory viruses such as respiratory syncytial virus and influenza, as well bacterial pathogens such as Chlamydia and meningococcus. Nonetheless, IgA knockout mice are able to mount a successful protective response to influenza virus following nasal challenge. The authors had previously noted that feeding mice with total parenteral nutrition (TPN) led to selective suppression of mucosal IgA that could be reversed with the administration of bombesin. The authors then sought to determine whether mice fed with TPN were less able to develop protective immunity to influenza virus following nasal challenge.

Significant findings

The authors challenged the mice with intranasal influenza virus at a dose expected to elicit a self-limiting upper respiratory tract infection, and that would be expected to confer protection (as defined by an absence of viral shedding) upon re-challenge. Following immunization, mice were fed with chow, TPN delivered intravenously, and TPN delivered via intragastric catheter. Protective immunity was observed upon rechallenge in both of the enterally fed groups, while the intravenous TPN group exhibited viral shedding at a level comparable to the non-immune control. To explain these findings, the authors compared the titers of serum anti-influenza IgG and nasal anti-influenza IgA as well as the number of antibody producing cells in the nasal passages and spleen. Feeding with intravenously delivered TPN resulted in a nearly sixfold reduction in nasotracheal influenza-specific IgA, with no diminution in IgG. A very modest decrease in the overall number of antibody forming cells was observed as well. From these data, the authors conclude that in the wild-type mouse intravenously delivered TPN nutrition leads to the selective loss of IgA, which in turn renders the mice susceptible to re-infection with influenza virus.

Comments

To the practitioner, the observation that intravenously delivered TPN can lead to diminished resistance against a viral pathogen is indeed food for thought. Whether this diminution is fully explained by the decreased level of IgA or whether other components of the immune response are also impaired remains to be more fully elucidated. Nonetheless, the study provides further justification for favoring enteral nutrition.

Methods

Viral challenge and shedding, antibody titers

Additional information

References

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