

CALL FOR PAPERS

In the beginning of the 20th century, Peyton Rous injected filtered extracts obtained from chicken sarcomas into healthy chickens to generate new sarcomas. After this finding, a growing number of pathogens have been found to be oncogenic. Microorganisms and their metabolites, as well as chronic inflammation, have also been considered to cause cancers. For example, the human papillomavirus (HPV) causes cervical squamous cell carcinoma and oropharyngeal cancer. Epstein-Barr virus (EBV) causes Burkitt lymphoma, nasopharyngeal cancer, and stomach cancer. Hepatitis B and C virus (HBV and HCV) cause hepatocellular carcinoma. Human T lymphotropic virus (HTLV), human herpes virus type 8 (HHV8), and *Helicobacter pylori* (*H. pylori*) are causative agents for adult T cell leukemia, Kaposi's sarcoma, and gastric cancer, respectively.

The mechanisms by which pathogenic microorganisms cause cancer vary. In the case of HPV, EBV, and HTLV, host cell proliferation is promoted by viral oncogenes suppressing the function of the *p53* gene and/or the *Retinoblastoma* (*RB*) gene. In the case of HBV and HCV, inflammation of hepatic cells increases the risk of cancer development. Not only does *H. pylori* cause gastritis, but its toxins can promote gastric cancer. It is also known that retrovirus infection causes tumor suppressor gene deficiency during the process of integration into the host gene. However, infection by these microorganisms is only the initial step of carcinogenesis, and cancer cannot occur by a single infection step alone. One underexplored way to study the relationship between infections and oncogenic events is to investigate the indirect role of infectious organisms that are not considered to be oncogenic. The oncogenic process may result from interactions between immune pathways involved in protection against infectious agents as well as cancer cells.

Almost 20% of all cancers worldwide are estimated to be associated with infections. However, cancers caused by infection include many preventable factors, such as lifestyle. We believe effective preventive measures exist for some of them, which include a potential target for novel cancer diagnostics, therapeutic approaches, and the possibility of prevention by vaccination.

This special issue welcomes papers concerning current knowledge of infection-associated cancers, spanning basic biology, and potential clinical applications.

Potential topics include but are not limited to the following:

- ▶ Molecular mechanisms to understand infection attributed cancers
- ▶ Tumor microenvironment including tumor immune response
- ▶ Development of novel biomarker for diagnosis and for predicting prognosis
- ▶ Animal models for studying infection-associated cancers
- ▶ New treatment or preventive strategy for infection-associated cancers

Authors can submit their manuscripts through the Manuscript Tracking System at <https://mts.hindawi.com/submit/journals/jo/inac/>.

Papers are published upon acceptance, regardless of the Special Issue publication date.

Lead Guest Editor

Hironori Yoshiyama, Shimane University, Shimane, Japan
yosiyama@med.shimane-u.ac.jp

Guest Editors

Keiji Ueda, Osaka University, Osaka, Japan
kueda@virus.med.osaka-u.ac.jp

Jun Komano, Nagoya Medical Center, Nagoya, Japan
jomano615@gmail.com

Hisashi Iizasa, Shimane University, Shimane, Japan
iizasah@med.shimane-u.ac.jp

Submission Deadline

Friday, 31 May 2019

Publication Date

October 2019