UNIVERSITI PUTRA MALAYSIA

TRANSCRIPTOMIC ANALYSIS OF EJ28 BLADDER CANCER CELLS PERSISTENTLY INFECTED WITH NEWCASTLE DISEASE VIRUS

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By

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Newcastle disease virus (NDV) is a highly contagious avian virus which leads to tremendous economy loss in poultry industry. Nevertheless, NDV shows good oncolytic activities on cancer cells by inducing apoptosis and therefore, it has been assessed in clinical trials for cancer treatment. Despite their promising potential, NDV was found to be able to persistently infect cancer cells *in vitro* without causing any cell death. The morphological characteristics of the cancer cells persistently infected by NDV has been described in the previous studies but the exact mechanism involved has yet to be elucidated. Hence, the objective of this study is to investigate the transcriptomic profiles of cancer cells persistently infected by NDV. By challenging with three rounds of infection with Malaysian velogenic strain AF2240, the NDV persistently infected bladder cancer cells EJ28 was established and was designated as EJ28P. The persistency of EJ28P was confirmed when they have grown to 4-fold of the original seeding number after 5 days post NDV infection; whilst its parental cells was nearly diminished in the same condition. Annexin V apoptosis assay revealed that approximately 60% of parental cells underwent apoptosis at 48 h post infection while the EJ28P was about 30%, which was insignificantly different from mock-infection. EJ28P reinfected with recombinant NDV AF2240 with green fluorescent protein (rAF-GFP) exhibited green fluorescent even after 25 passages. This further confirmed the persistency of NDV in EJ28P. Subsequently, total RNA of EJ28 and EJ28P were extracted and subjected to microarray analyses. A total of 355 genes, which were differentially expressed in EJ28P compared to EJ28, were identified using edgeR program, with their $\log_2$ fold change of 2 or more. Among them, 222 genes were up-regulated while 133 genes were down-regulated. Gene ontology (GO) of these genes were obtained using PANTHER Classification System and Database for annotation, visualisation and Integrated Discovery (DAVID). Genes associated to cytokine and apoptosis such as *IL6*, *IL8*, *GBP2*, *CXCL8*, *HER5* and *TNF* were down-regulated. Pathway analysis revealed that TGF-β signalling was downregulated in EJ28P, which could lead suppression of apoptotic pathway and promotion of cell proliferation. However, the anti-virus responses of EJ28P were not completely suppressed as interferon-induced transmembrane (*IFITM1*), an anti-viral

protein gene was up-regulated. On the other hand, genes associated to solute carrier families that are responsible for glucose and amino acids transportation across the membrane, were significantly up-regulated, suggesting that high demand of energy for virus activities or for the cancer cell's metabolism, or for both. In addition, genes related to keratin group were overexpressed possibly involved in maintaining the cell structural integrity. Since these expressed genes were not only beneficial to cell but also to virus, it may either reflect a competition on both cell and virus for each survival, or an interaction from both sides in order to maintain a co-existing form. Based on these analyses, it is postulated that impairment of apoptosis and defective in pro-inflammatory response, together with overexpression of solute carrier families and keratin groups, contribute to NDV persistent infection in bladder cancer cells. This study expands our understanding on persistent NDV infection in bladder cancer cells.

Abstrak tesis yang dikemukakan kepada Senat Universiti Putra Malaysia sebagai memenuhi keperluan untuk Ijazah Master Sains

ANALISIS TRANSKRIPTOM SEL KANSER PUNDI KENCING EJ28 YANG DIJANGKITI BERTERUSAN DENGAN *NEWCASTLE DISEASE VIRUS*

Oleh

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Newcastle disease virus (NDV) adalah virus burung yang berjangkit dan menyebabkan kerugian yang besar dalam industri penternakan ayam. Walau bagaimanapun, NDV berupaya untuk membunuh sel kanser dengan mencetuskan apoptosis, oleh itu, virus ini dikaji dalam fasa klinikal untuk rawatan kanser. Walaupun NDV berpotensi tinggi dalam rawatan kanser, NDV telah diketahui boleh menjangkiti sel kanser secara berterusan dalam keadaan *in vitro* tanpa menyebabkan kematian sel kanser. Ciri-ciri mofologi jangkitan berterusan NDV telah digambarkan dalam kajian sebelum ini tetapi mekanismenya masih belum diketahui. Oleh itu, objektif keseluruhan kajian ini adalah untuk menyiasat profil transkriptomik jangkitan berterusan NDV. Melalui pencabaran tiga kali dengan jangkitan NDV AF2240, kanser pundi kencing EJ28 yang dijangkiti secara berterusan dengan NDV telah diwujudkan dan dinamakan EJ28P. Selepas 5 hari dijangkiti dengan NDV, jangkitan beterusan EJ28P telah disahkan apabila bilangan sel ini bertambah 4 kali ganda berbanding dengan bilangan sel pada hari pertama. Bilangan sel induk EJ28 pula didapati berkurangan dalam tempoh ujian yang sama. Ujian apoptosis Annexin V menunjukkan bahawa lebih kurang 60% sel induk mengalami apoptosis selepas 48 jam dijangkiti oleh NDV. EJ28P pula, hanya mengalami lebih kurang 30% apoptosis, yang didapati lebih kurang sama dengan peratusan apoptosis sel yang tidak dijangkiti oleh NDV. EJ28P yang dijangkiti NDV berekombinan dengan protein fluorescen hijau masih dapat dikesan selepas dikultur 25 kali dalam flask. Ini membukti bahawa EJ28P yang dijangkiti oleh NDV adalah suatu jangkitan berterusan. Seterusnya, RNA EJ28P telah diekstrak dan diuji dengan analisis microarray. Sejumlah 355 gen telah dikenalpasti dengan program edgeR, dengan log2 perubahan lipat lebih daripada 2. Antara gen-gen ini, 222 gen megalami regulasi meningkat dan 133 gen mengalami regulasi menurun. Pencarian ontologi gen (GO) telah dijalankan melalui Sistem klasifikasi PANTHER dan Database for annotation, visualisation and Integrated Discovery (DAVID). Gen berkaitan dengan sitokin dan apoptosis seperti IL6, IL8, GBP2, CXCL8, HER5 and TNF mengalami regulasi menurun. Analisa lintasan mendedahkan pengisyaratan TGF-beta telah terbantut dalam EJ28P, menyebabkan lintasan apoptosis ditindas dan pada masa yang sama menggalakkan pertumbuhan sel. Namun itu, respon anti-virus EJ28P bukan ditindas sepenuhnya kerana interferon-induced transmembrane

(IFITM1), gen yang mengkod protein anti-virus mengalami regulasi meningkat. Sebaliknya, gen berkaitan dengan solute carrier families yang berfungsi dalam pengangkutan glukos dan asid amino melalui membran sel, mengalami regulasi meningkat, menunjukkan keperluan tenaga dalam aktiviti virus, metabolisma sel, ataupun kedua-duanya. Tambahan pula, gen yang berkaitan dengan kumpulan keratin mengalami ekspresi meningkat. Ini mungkin menunjukkan penglibatan pemeliharaan integriti struktur sel. Kajian ekspresi gen, menunjukkan bahawa gen-gen yang terekspres bukan sahaja membantu sel yang dijangkit untuk terus hidup, malah juga membantu rambatan virus Berdasarkan kepada analisis, faktor-faktor seperti kegagalan sel mencetus apoptosis dan inflamasi serta ekspresi solute carrier family dan keratin yang meningkat menyumbang kepada jangkitan berterusan NDV. Kesimpulannya, kajian ini mengembangkan pemahaman terhadap jangkitan berterusan NDV dan juga menyumbang kepada kajian berikutnya.

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This thesis was submitted to the Senate of Universiti Putra Malaysia and has been accepted as fulfilment of the requirement for the degree of Master of Science. The members of the Supervisory Committee were as follows:

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDICES

LIST OF ABBREVATIONS

BF	Biological Function
BSA	bovine serum albumin
CC	Cellular Component
°C	degree Celsius
cDNA	complementary deoxyribonucleic acid
CO_2	Carbon dioxide
Cy3	green cyanine fluorescent dye
Cy5	red cyanine fluorescent dye
DAPI	4',6-diamidino-2-phenylindole
DAVID	Database of Annotation, Visualisation and Integrated Discovery
DMEM	Dulbecco's Modified Eagle Medium
DNA	deoxyribonucleic acid
EDTA	ethylenediaminetetraacetic acid
FC	Fold change
GE	gene-end sequence
GFP	Green Fluorescent Protein
GM	Growth media
GO	Gene ontology
GS	gene start sequence
h	hour
HA	Haemagglutination
HEK	human embryonic kidney
HIV	human immunodeficiency virus
hpi	hour(s) post-infection
IFN	interferon
IPA	Ingenuity Pathway Analysis
IS	intergenic sequence
KEGG	Kyoto Encyclopedia of Genes and Genomes
L	Litre
µ	micro
M	Molarity
MF	Molecular Function
min	minute(s)
mL	Millilitre
mm	millimetre
MM	Maintenance media
MOI	multiplicity of infection
mRNA	messenger ribonucleic acid
NaCl	sodium chloride
NDV	Newcastle disease virus
nm	nanometer
PBS	Phosphate buffered saline
pH	Puissance of hydrogen
PI	Persistent infection
PPI	protein-protein interaction
rAF-GFP	recombinant NDV AF2240 harbouring GFP Protein gene
RBC	chicken red blood cells
RIN	RNA intergrity number

RNA	ribonucleic acid
Rpm	revolutions per minute
s	second(s)
TBFV	Tickborne Flavivirus
TNF	tumour necrosis factor
U	Unit
(v/v)	(volume/volume)
(w/v)	(weight/volume)

CHAPTER 1

INTRODUCTION

Newcastle disease virus (NDV) is an economically important avian paramyxovirus (Lancaster, 1976; Alexander, 1997). Its non-segmented single-stranded negative sensed RNA genome consists of six genes; which encode for nucleocapsid (NP), phosphoprotein (P), matrix (M), fusion (F), haemagglutinin-neuraminidase (HN) and large polymerase (L) proteins (Yusoff and Tan, 2001). The mesogenic and lentogenic NDV strain causes mild infection while the velogenic strains are highly contagious affecting both wild and domestic avian species alike (Songhua *et al*., 2003).

Despite their pathogenicity in avian species, NDV displays oncolytic properties enabling it to be utilised as a therapeutic agent for cancer treatment (Rusell, 2002). Early observation of its oncolytic potential was reported for leukaemia (Wheelock and Dingle, 1967), cervical cancer (Cassel and Garrett, 1965) and metastatic gastric cancer (Csatary, 1971). Since then, the virus has been studied extensively, both *in vivo* and *in vitro*, followed by clinical trials which showed promising results (Schirrmacher, 2016). Similar to other oncolytic viruses, NDV capitalises on the defective antiviral and apoptotic pathways in cancer cells to allow it to selectively replicate in tumour cells. The suppression of interferon type 1 antiviral pathways by tumour cells in order to evade immunosurveillance, have been reported to potentially permit oncolytic virus replication (Fiola *et al*., 2006; Krishnamuthy *et al*., 2006; Singh *et al*., 2012). Apart from that, overexpression of the anti-apoptotic proteins in tumour cells also promotes virus propagation (Mansour *et al*., 2011; Schirrmacher, 2017).

Interestingly, NDV and other RNA viruses such as measles virus, Sendai virus, and vesicular stomatitis virus were found to be able to cause persistent infections in cancer cell lines (Rima and Martin, 1976). The virus remains in these persistently infected cells without causing cellular apoptosis (Boldogh et al., 1996). This phenomenon could have evolved initially leading to viral tropism (Ahmed et al., 1981; Baric et al., 1999; Pinkert et al., 2011). Initial studies on persistent infection (PI) by NDV during 1960s and 1970s were more focused on the virus and its cellular infection properties (Rodriguez et al., 1967; Thacore and Youngner, 1969; Preble and Youngner, 1973a and 1973b). Since then, studies of NDV PI have been neglected until recently, Chia et al. (2014) reported persistent infection by NDV in the colorectal cancer cell line, SW480, which then produced PI virus with reduced plaques size. Rangaswamy et al. (2017) revealed that PI NDV consists of mutated HN and F genes after it persistently infected ovarian cancer cells. However, the role of the host cell in PI is still obscure.

To have a better understanding of the role of the host cells during persistent infection, microarray analysis will be used to study the transcriptomic profile of the cancer cells in response to persistent NDV infection. Microarray is a tool that can probe thousands of expressed genes and measures their expression level (Quankeenbush, 2006). It has been used to reveal PI mechanism of viruses including Hepatitis C virus (Woodhouse et al.,

2010), Yersinia virus (Avican et al., 2015) and Foot-and-Mouth disease virus (Eschbaumer et al., 2016). By comparing the gene expression profile of PI cells to their parental cells, a list of genes involved in PI can be determined and used for downstream studies.

Most of the previous report on NDV persistent infection focused on characteristics and morphological studies. Genetic information in NDV persistent infection has not been investigated. In view of this, a comprehensive study in genetic level is elementary in better understanding of NDV persistent infection. It is hypothesised that genes associated with cellular apoptosis and virus defence mechanism are supressed in persistent infected cancer cells.

The main objective of this study is to delineate the mechanism of persistent NDV infection in bladder cancer cells using microarray analysis. The specific aims are as follows:

1. To establish persistently NDV-infected bladder cancer cell line;
2. To characterise persistently NDV-infected bladder cancer cell line; and
3. To profile and identify the genes that are related to persistent NDV infection.

REFERENCES

Ahmad, U., Ahmed, I., Keong, Y. Y., Abd Manan, N. Abd. and Othman, F. (2015). Inhibitory and Apoptosis-Inducing Effects of Newcastle Disease Virus Strain AF2240 on Mammary Carcinoma Cell Line. *BioMed Research International* 2015.

Ahmed, R., Canning, W. M., Kauffman, R. S., Sharpe, A. H., Hallum, J. V. and Fields, B. N. (1981). Role of the host cell in persistent viral infection: Coevolution of L cells and retrovirus during persistent infection. *Cell* 25(2): 325-332.

Ahmed, S., Bradshaw, A., Gera, S., Dewan, M. Z. and Xu, R. (2017). The TGF-β/Smad4 Signaling Pathway in Pancreatic Carcinogenesis and Its Clinical Significance. *Journal of Clinical Medicine* 6(1): 5.

Alabsi, A., Bakar, S., Ali, R., Omar, A., Bejo, M., Ideris, A., and Ali, A. M. (2011). Effects of Newcastle Disease Virus Strains AF2240 and V4-UPM on Cytolysis and Apoptosis of Leukemia Cell Lines. *International Journal of Molecular Science* 12: 8645-8660.

Alexander, D. J. (1997). Newcastle disease and other avian Paramyxoviridae infections. In *Diseases of Poultry*,10th edn. Edited by Calnek, B. W. Ames, IA: Iowa State University Press; pp. 541-569.

Alves, N. L., Derks, I. A., Berk, E., Spijker, R., van Lier, R. A., Eldering, E. (2006). The Noxa/Mcl-1 axis regulates susceptibility to apoptosis under glucose limitation in dividing T cells. *Immunity* 24:703–716.

Avican, K., Fahlgren, A., Huss, M., Heroven, Ann. K., Beckstette, M., Dersch, P. and Fallman, M. (2015). Reprogramming of Yersinia from Virulent to Persistent Mode Revealed by Complex *In Vivo* RNA-seq Analysis. *PLoS Pathogens* 11(1): e1004600.

Baric, R. S., Sullivan, E., Hensley, L., Yount, B. and Chen, W. (1999). Persistent Infection Promotes Cross-Species Transmissibility of Mouse Hepatitis Virus. *Journal of Virology* 73(1): 638-649.

Batliwalla, F. M., Bateman, B. A., Serrano, D., Murray, D., Macphail, S., Maino, V. C., Ansel, J. C., Gregersen, P. K. and Armstrong, C. A. (1998). A 15-Year Follow-up of AJCC Stage III Malignant Melanoma Patients Treated Postsurgically with Newcastle Disease Virus (NDV) Oncolysate and Determination of Alterations in the CD8 T Cell Repertoire. *Molecular Medicine* 4: 783-794.

Bello, J. Omar. M., Nieva, L. O., Paredes, A. C., Gonzalez, A. M. F., Zavaleta, L. R. and Lizano, M. (2015). Regulation of the Wnt/β-Catenin Signaling Pathway by Human Papillomavirus E6 and E7 Oncoproteins. *Viruses* 7(8): 4734-4755.

Bergs, V. V., Henle, G., Deinhardt, F., Henle, W. (1958). Studies on persistent infections of tissue cultures. II. Nature of the resistance to vesicular stomatitis virus. *The Journal of Experimental Medicine* 108: 561-572.

Blaskovic, D. and Styk, B. (1967) Laboratory methods of virus transmission in multicellular organisms. In *Virology*. Edited by Maramorasch, K. and Koprocvski, H. vol. 1. New York: Academic Press; pp. 163-233.

Boldogh, I., Albrecht, T. and Porter, D. D. (1996). Chapter 46: Persistent Viral Infections. In *Medical Microbiology*. 4th edition. Edited by Baron, S. University of Texas Medical Branch at Galveston; pp 1-23.

Bragulla, H. H. and Homberger, D. G. (2009). Structure and functions of keratin proteins in simple, stratified, keratinized and cornified epithelia. *Journal of Anatomy* 214: 516–559.

Broer, A., Rahimi, F. and Broer, S. (2016). Deletion of Amino Acid Transporter ASCT2 (SLC1A5) Reveals an Essential Role for Transporters SNAT1 (SLC38A1) and SNAT2 (SLC38A2) to Sustain Glutaminolysis in Cancer Cells. *Journal of Biological Chemistry* 291(25): 13194-13205.

Burke, E., Dupuy, L., Wall, C. and Barik, S. (1998). Role of cellular actin in the gene expression and morphogenesis of human respiratory syncytial virus. *Virology* 252(1): 137-148.

Cassel, W. A., and Garrett, R. E. (1965). Newcastle disease virus as an antineoplastic agent. *Cancer* 18: 863–868.

Cassel, W. A., Murray, D. R. and Phillips, H. S. (1983). A phase II study on the postsurgical management of stage II malignant melanoma with a Newcastle disease virus oncolysate. *Cancer* 52: 856–860.

Cassel, W. A., Murray, D. R., Torbin, A. H., Olkowski, Z. L. and Moore, M. E. (1977). Viral oncolysate in the management of malignant melanoma. I. Preparation of the oncolysate and measurement of immunologic responses. *Cancer* 40(2): 672-679.

Cassel, W. and Murray, D. R. (1992). A ten-year follow-up on stage II Malignant Melanoma patients treated postsurgically with newcastle disease virus oncolysate. *Medical Oncology and Tumor Pharmacotherapy* 9(4): 169-171.

Caviness, K., Kuhn, J. H. and Palacios, G. (2017). Ebola virus persistence as a new focus in clinical research. *Current Opinion in Virology* 23: 43-48.

Chapman, N. M. and Kim, K. S. (2008). Persistent coxsackievirus infection: enterovirus persistence in chronic myocarditis and dilated cardiomyopathy. *Current Topics in Microbiology and Immunology* 323: 275-292.

Chia, S. L., Tan, W. S., Yusoff, K., and Shafee, N. (2012) Plaque formation by a velogenic Newcastle disease virus in human colorectal cancer cell lines. *Acta Virologica* 56: 345-347.

Chia, S., Yusoff, K. and Shafee, N. (2014). Viral persistence in colorectal cancer cells infected by Newcastle disease virus. *Virology Journal* 11:91.

Childs, K., Stock, N., Ross, C., Andrejeva, J., Hilton, L., Skinner, M., Randall, R., Goodbourn, S. (2007). mda-5, but not RIG-I, is a common target for paramyxovirus V proteins. *Virology* 359:190-200.

Corish, P. and Tyler-Smith, C. (1999). Attenuation of green fluorescent protein half-life in mammalian cells. *Protein Engineering* 12(12): 1035-1040.

Creixell, P., Reimand, J., Haider, S., Wu, G., Shibata, T., Vazquez, M., Mustonen, V., Gonzalez-Perez, A., Pearson, J., Sander, C., Raphael, B. J., Marks, D. S., Ouellette, B. F. F., Valencia, A., Bader, G. D., Boutros, P. C., Stuart, J. M., Linding, R., Lopez-Bigas, N., Stein, L. D. and Mutation Consequences and Pathway Analysis Working Group of the International Cancer Genome Consortium. (2015). Pathway and Network Analysis of Cancer Genomes. *Nature Methods* 12(7): 615-621.

Csatary, L. K. (1971). Viruses in the treatment of cancer. *The Lancet* 2(7728): 825.

Csatary, L. K., Gosztonyi, G., Szeberenyi, J., Fabian, Z., Liszka, V., Bodey, B. and Csatary, C. M. (2004). MTH-68/H oncolytic viral treatment in human high-grade gliomas. *Journal of Neuro-Oncology* 67: 83-93.

Csatary, L. K., Eckhardt, S., Bukosza, I., Czegledi, F., Fenyvesi, C., Gergely, P., Bodey, B. and Csatary, C. M. (1993). Attenuated Veterinary Virus Vaccine for the Treatment of Cancer. *Cancer Detection and Prevention* 17(6): 619-627.

Cummiskey, J. F., Hallum, J. V., Skinner, M. S., Leslie, G. A. (1973). Persistent Newcastle disease virus infection in embryonic chicken tracheal organ cultures. *Infection and Immunology* 8(4):657-64.

De, B. P., Lesoon, A. and Banerjee, A. K. (1991). Human parainfluenza virus type 3 transcription in vitro: role of cellular actin in mRNA synthesis. *Journal of Virology* 65(6): 3268-3275.

DiVito, K. A., Simbulan-Rosenthal, C. M., Chen, You-Shin., Trabosh, V. A. and Rosenthal, D. S. (2014). Id2, Id3 and Id4 overcome a Smad7-mediated block in tumorigenesis, generating TGF-β-independent melanoma. *Carcinogenesis* 35(4): 951-958.

Dohner, K. and Sodeik, B. (2004). The Role of the Cytoskeleton During Viral Infection. *Current Topics in Microbiology and Immunology* 285: 67-108.

Doi, T., Kwon, H., Honda, T., Sato, H., Yoneda, M. and Kai, C. (2016). Measles virus induces persistent infection by autoregulation of viral replication. *Scientific Reports* 6: 37163.

Dortmans, J. C. F. M., Koch, G., Rottier, P. J. M. and Peeters, B. P. H. (2011). Virulence of Newcastle disease virus: what is known so far? *Veterinary Research* 42: 122.

Duan, Z., Xu, H., Ji, X., and Zhao, J. (2015). Recombinant Newcastle disease virus-vectored vaccines against human and animal infectious diseases. *Future Microbiology* 10: 1307–1323.

Dufva, M. (2009). Introduction to Microarray Technology. In *DNA Microarrays for Biomedical Research: Methods and Protocols*, vol. 529. Edited by Dufva, M. Humana Press, a part of Springer Science Business Media; pp 1-22.

Eckert, B. S. (1986). Alteration of the distribution of intermediate filaments in PtK1 cells by acrylamide. II: Effect on the organization of cytoplasmic organelles. *Cell Motility and Cytoskeleton* 6(1):15-24.

Elankumaran, S., Rockeman, D. and Samal, S. K. (2006). Newcastle Disease Virus Exerts Oncolysis by both Intrinsic and Extrinsic Caspase-Dependent Pathways of Cell Death. *Journal of Virology* 80(15): 7522-7534.

El-Gebali, S., Bentz, S., Hediger, M. A. and Anderle, P. (2012). Solute carriers (SLCs) in cancer. *Molecular Aspects of Medicine* 34: 719–734.

Eschbaumer, M., Stenfeldt, C., Smoliga, G. R., Pacheco, J. M., Rodriguez, L. L., Li, R. W. and Zhu, J. (2016). Transcriptomic Analysis of Persistent Infection with Foot-and-Mouth Disease Virus in Cattle Suggests Impairment of Apoptosis and Cell-Mediated Immunity in the Nasopharynx. *PLoS One* 11(9): e0162750.

Espin, R., Roca, F. J., Cande, S., Sepulcre, M. P., Gonzalez-Rosa, J. M., Alcaraz-Perez, F., Meseguer, J., Cayuela, M. L., Mercader, N., Mulero, V. (2013). TNF receptors regulate vascular homeostasis in zebrafish through a caspase-8, caspase-2 and P53 apoptotic program that bypasses caspase-3. *Disease Models and Mechanisms* 6(2): 383-396.

Farrell, H. E., Vally, H., Lynch, D. M., Fleming, P., Shellam, G. R., Scalzo, A. A., Davis-Poynter, N. J. (1997). Inhibition of natural killer cells by a cytomegalovirus MHC class I homologue in vivo. *Nature* 386(6624): 510-514.

Fei, X., Qi, M., Wu, B., Song, Y., Wang, Y. and Li, T. (2012). MicroRNA-195-5p suppresses glucose uptake and proliferation of human bladder cancer T24 cells by regulating GLUT3 expression. *FEBS Letters* 586(4): 392-397.

Fiola, C., Peeters, B., Fournier, P., Arnold, A., Bucur, M. and Schirrmacher, V. (2006). Tumor selective replication of Newcastle disease virus: Association with defects of tumor cells in antiviral defence. *Tumor Immunology* 119(2): 328-338.

Fraser, G., Edwards, Helen. H., McNulty, M. S., and Ruben, J. M. S. (1976). Accidental persistent infection of cell lines by Newcastle disease virus, showing three unusual features — Defective neuraminidase, temperature sensitivity and intranuclear inclusions. *Archives of Virology* 50(1-2): 147-157.

Freeman, A. I., Zakay-Rones, Z., Gomori, J. M., Linetsky, E., Rasooly, L., Greenbaum, E., Rozenman-Yair, S., Panet, A., Libson, E., Irving, C. S., Galun, E. and Siegal, T. (2006). Phase I/II Trial of Intravenous NDV-HUJ Oncolytic Virus in Recurrent Glioblastoma Multiforme. *Molecular Therapy* 13(1): 221-228.

Garg, R. K. (2002). Subacute sclerosing panencephalitis. *Postgraduate Medical Journal* 78: 63-70.

Glickman, R. L., Syddall, R. J., Iorio, R. I., Sheehan, J. P. and Bratt, M. A. (1988). Quantitative basic residue requirements in the cleavage-activation site of the fusion glycoprotein as a determinant of virulence for Newcastle disease virus. *Journal of Virology* 62: 354-356.

Gotoh, B., Ohnishi, Y., Inocencio, N. M., Esaki, E., Nakayama, K., Barr, P. J., Thomas, G., and Nagai, Y. (1992). Mammalian subtilisin-related proteinases in cleavage activation of the paramyxovirus fusion glycoprotein: superiority of furin/PACE to PC2 or PC1/PC3. *Journal of Virology* 66: 6391-6397.

Gottlob, K., Majewski, N., Kennedy, S., Kandel, E., Robey, R. B., Hay, N. (2001). Inhibition of early apoptotic events by Akt/PKB is dependent on the first committed step of glycolysis and mitochondrial hexokinase. *Genes Development* 15:1406–1418.

Gu, L. H. and Coulombe, P. A. (2007). Keratin function in skin epithelia: a broadening palette with surprising shades. *Current Opinion in Cell Biology* 19: 13–23.

Gupta, P. S. P., Folger, J. K., Raiput, S. K., Lv, L., Yao, J., Ireland, J. J. and Smith, G. W. (2014). Regulation and Regulatory Role of WNT Signaling in Potentiating FSH Action during Bovine Dominant Follicle Selection. *PLoS One* 9(6): e100201.

Hanahan, D. and Weinberg, R. A. (2011). Hallmarks of Cancer: The Next Generation. *Cell* 144(5): 646-674.

Hatton, O. L., Arnold-Harris, A., Schaffert, S., Krams, S. M. and Martinez, O. M. (2014). The Interplay Between Epstein Barr Virus and B Lymphocytes: Implications for Infection, Immunity, and Disease. *Immunologic Research* 58(0): 268-276.

He, L., Vasiliou, K., and Nebert, D. W. (2009). Analysis and update of the human solute carrier (SLC) gene superfamily. *Human Genomics* 3:195–206.

Hecht, T. T. and Summers, D. F. (1974). Newcastle Disease Virus Infection of L Cells. *Journal of Virology* 14(1): 162-169.

Hediger, M. A., Romero, M. F., Peng, J.B., Rolfs, A., Takanaga, H., Bruford, E. A. (2004). The ABCs of solute carriers: physiological, pathological and therapeutic implications of human membrane transport proteins Introduction. *Pflügers Archiv: European Journal of Physiology* 447: 465–468.

Heiden, S., Grund, C., Roder, A., Granzow, H., Kuhnel, D., Mettenleiter, T. C. (2014). Different Regions of the Newcastle Disease Virus Fusion Protein Modulate Pathogenicity. *PLoS One* 9(12): e113344.

Henle, G. and Henle, W. (1966). Immunofluorescence in cells derived from Burkitt's lymphoma. *Journal of Bacteriology* 91(3):1248–1256.

Henle, G., Deinhardt, F., Bergs, V. V., Henle, W. (1958). Studies on persistent infections of tissue cultures. I. General aspects of the system. *The Journal of Experimental Medicine* 108(4):5 37–560.

Hill, V. M., Harmon, S. A. and Summers, D. F. (1986). Stimulation of vesicular stomatitis virus in vitro RNA synthesis by microtubule-associated proteins. *Proceedings of the National Academy of Sciences* 83: 5410-5413.

Ideris, A., Ibrahim, A. L., and Spradbrow, P. B. (1990). Vaccination of chickens against Newcastle disease with a food pellet vaccine. *Avian Pathology* 19: 371-384.

Im, J., Kim, B., Lee, J., Park, S., Ban, H. S., Jung, K. E. and Won, M. (2018). DDIAS suppresses TRAIL-mediated apoptosis by inhibiting DISC formation and destabilizing caspase-8 in cancer cells. *Oncogene* 37: 1251-1262.

Ito, Y., Nagai, Y. and Maeno, K. (1982). Interferon production in mouse spleen cells and mouse fibroblasts (L cells) stimulated by various strains of Newcastle disease virus. *Journal of General Virology* 62:349-352.

Kallewaard, N. L., Bowen, A. L., Crowe Jr, J. E. (2005). Cooperativity of actin and microtubule elements during replication of respiratory syncytial virus. *Virology* 331(1): 73-71.

Kane, M. and Golovkina, T. (2010). Common Threads in Persistent Viral Infections. *Journal of Virology* 84(9): 4116-4123.

Kim, S. H, Wanasen, N., Paldurai, A., Xiao, S., Collins, P. L. and Samal, S. K. (2013). Newcastle Disease Virus Fusion Protein Is the Major Contributor to Protective Immunity of Genotype-Matched Vaccine. *Plos One* 8(8): e74022.

Klatt, N. R., Chomont, N., Douek, D. C. and Deeks, S. G. (2013). Immune activation and HIV persistence: implications for curative approaches to HIV infection. *Immunological Reviews* 254(1): 326-342.

Krishnamurthy, S., Takimoto, T., Scroggs, R. A. and Portner, A. (2006). Differentially Regulated Interferon Response Determines the Outcome of Newcastle Disease Virus Infection in Normal and Tumor Cell Lines. *Journal of Virology* 80(11): 5145-5155.

Kuan, C. Y., Whitmarsh, A. J., Yang, D. D., Liao, G., Schloemer, A. J., Dong, C., Bao, J., Banasiak, K. J., Haddad, G. G., Flavell, R. A., Davis, R. J. and Rakic, P. (2003). Absence of excitotoxicity-induced apoptosis in the hippocampus of mice lacking the Jnk3 gene. *Proceedings of the National Academy of Sciences* 100:15184–15189.

Lamb, R. A. and Kolakofsky, D. (2007). Paramyxoviridae: the viruses and their replication. Edited by Knipe, D. M., Howley, P. M., and Griffin, E. D. Philadelphia, Lippincott-Raven Press; pp 1449-1496.

Lamb, R. A., and Kolakofsky, D. (1996). *Paramyxoviridae*: the viruses and their replication. In *Fields virology* 3rd edn, Vol. 1. Edited by Fields, B. N., Knipe, D. M. and Howley, P. M. Philadelphia, PA: Lippincott-Raven; pp 1177-1203.

Lamb, R. and Parks, G. (2007). Paramyxoviridae: The Viruses and Their Replication. Edited by Knipe, D. M., Howley, P. M., Griffin, D. E., Lamb, R. A., Martin, M. A, Roizman, B. and Straus, S. E. Lippincott Williams & Wilkins, Philadelphia; pp1449-1496.

Lancaster, J. E. (1976). A History of Newcastle Disease with Comments on its Economic Effects. *World's Poultry Science Journal* 32(2): 167-175.

Lasorella, A., Rothschild, G., Yokota, Y., Russell, R. G. and Iavarone, A. (2005). Id2 mediates tumor initiation, proliferation, and angiogenesis in Rb mutant mice. *Molecular Cell Biology* 25: 3563–3574.

Laurie, S. A., Bell, J. C., Atkins, H. L., Roach, J., Bamat, M. K., O'neil, J. D., Roberts, M. S., Groene, W. S. and Lorence, R. M. (2006). A Phase 1 Clinical Study of Intravenous Administration of PV701, an Oncolytic Virus, Using Two-Step Desensitization. *Clinical Cancer Research* 12(8): 2555-2562.

Lawton, P., Karimi, Z., Mancinelli, L. and Seto, J. T. (1986). Persistent infections with Sendai virus and Newcastle disease viruses. *Archives of Virology* 89:225-233.

Lorence, R. M., Scot Roberts, M. O'Neil, J. D., Groene, W. S., Miller, J. A., Mueller, S. N. and Bamat, M. K. (2007). Phase 1 Clinical Experience Using Intravenous Administration of PV701, an Oncolytic Newcastle Disease Virus. *Current Cancer Drug Targets* 7: 157-167.

Lu, J., Pan, Q., Rong, L., Liu, S. and Liang, C. (2011). The IFITM Proteins Inhibit HIV-1 Infection. *Journal of Virology* 85(5): 2126-2137.

Luby-Phelps, K. (2000). Cytoarchitecture and physical properties of cytoplasm: volume, viscosity, diffusion, intracellular surface area. *International Review of Cytology* 192:189 –221.

Ludlow, M., McQuaid, S., Cosby, S. L. Cattaneo, R., Rima, B. K., and Duprex, W. P. (2005). Measles virus superinfection immunity and receptor redistribution in persistently infected NT2 cells. *Journal of General Virology* 86: 2291–2303.

Maeno, K., Yoshii, S., Nagata, I. and Matsumoto, T. (1966). Growth of Newcastle Disease Virus in a HVJ Carrier Culture of HeLa Cells. *Virology* 29: 255-263.

Malapeira, J., Esselens, C., Serra, J. J. B., Canals, F. and Arribas, J. (2011). ADAM17 (TACE) regulates TGFβ signaling through the cleavage of vasorin. *Oncogene* 30: 1912-1922.

Mansour, M., Palese, P. and Zamarin, D. (2011). Oncolytic Specificity of Newcastle Disease Virus Is Mediated by Selectivity for Apoptosis-Resistant Cells. *Journal of Virology* 85(12): 6015-6023.

Matthews, J. D., Morgan, R., Sleigher, C. and Frey, T. K. (2013). DO viruses require the cytoskeleton? *Virology Journal* 10:121.

McChesney, M. B., Kerhl, J. H., Valsamakis, A., Fauci, A. S. and Oldstone, M B. (1987). Measles virus infection of B lymphocytes permits cellular activation but blocks progression through the cell cycle. *Journal of Virology* 61(11): 3441-3447.

McNulty, M. S., Gowans, E. J., Louza, A. C. and Fraser, G. (1977). An electron microscopic study of MDBK cells persistently infected with Newcastle disease virus. *Archives of Virology* 53(3): 185-195.

Millar, N. S., Chambers, P. and Emmerson, P. T. (1988). Nucleotide sequence of the fusion and haemagglutination-neuraminidase glycoprotein gens of Newcastle disease virus, strain Ulster: molecular basis for variations in pathogenicity between strains. *The Journal of General Virology* 69 (Pt 3): 613-620.

Miller, M. B. and Tang, Y-W. (2009). Basic Concepts of Microarrays and Potential Applications in Clinical Microbiology. *Clinical Microbiology* 22(4): 611-633.

Mlera, L., Lam, J., Offerdahl, D. K., Martens, C., Sturdevant, D., Turner, C. V., Porcella, S. F. and Bloom, M. E. (2016). Transcriptome Analysis Reveals a Signature Profile for Tick-Borne Flavivirus Persistence in HEK 293T Cells. *mBio* 7(3): e00314-e00316.

Mudhasani, R., Tran, J. P., Retterer, C., Radoshitzky, S. R., Kota, K. P., Altamuraa, L. A., Smith, J. M., Packard, B. Z., Kuhn, J. H., Costantino, J., Garrison, A. R., Schmaljohn, C. S., Huang, I-C., Farzan, M. and Bavari, S. (2013). IFITM-2 and IFITM-3 but Not IFITM-1 Restrict Rift Valley Fever Virus. *Journal of Virology* 87(15): 8451-8464.

Murray, D. R., Cassel, W. A., Torbin, Arlene, H., Olkowski, Z. L., Moore, M. E. (1977). *Cancer* 40(2): 680-686.

Nagai, Y., Hamaguchi, Y. M. and Toyoda, T. (1989). Molecular biology of Newcastle disease virus. *Progress in Veterinary Microbiology and Immunology* 5: 16-64.

Naniche, D., Wild, T. F., Rabourdin-Combe, C. and Gerlier, D. (1993). Measles virus haemagglutinin induces down-regulation of gp57/67, a molecule involved in virus binding. *Journal of General Virology* 74(6): 1073–1079.

Nascimento, R., Costa, H. and Parkhouse, R. M. (2012). Virus manipulation of cell cycle. *Protoplasma* 249: 519–528.

Nash, K. L. and Lever, A. M. L. (2004). Green fluorescent protein: green cells do not always indicate gene expression. *Gene Therapy* 11: 882-883.

Ogasawara, T., Gotoh, B., Suzuki, H., Asaka, J., Shimokata, Kaoru, Rott, R., and Nagai, Y. (1992). Expression of factor x and its significance for the determination of paramyxovirus ropism in the chick embryo. *The European Molecular Biology Organization Journal* 11: 467-472.

Park, M-S., Garcia-Sastre, A., Cros, J. F., Basler, C. F. and Palese, P. (2003). Newcastle Disease Virus V Protein Is a Determinant of Host Range Restriction. *Journal of Virology* 77(17): 9522-9532.

Pecora, A. L., Rizvi, N., Cohen, G. I., Meropol, N. J., Sterman, D., Marshall, J. L., Goldberg, S., Gross, P., O'Neil, J. D., Groene, W. S., Roberts, M. S., Rabin, H., Bamat, M. K., and Lorence, R. M. (2002). Phase I Trial of Intravenous Administration of PV701, an Oncolytic Virus, in Patients with Advanced Solid Cancers. *Journal of Clinical Oncology* 20: 2251-2266.

Perrot, C. Y., Javelaud, D. and Mauviel, A. (2013). Insights into the Transforming Growth Factor-β Signaling Pathway in Cutaneous Melanoma. *Annals of Dermotology* 25(2): 135-144.

Pinkert, S., Klingel, K., Lindig, V., Dorner, A., Zeichhardt, H., Spiller, O. B. and Fechner, H. (2011). Virus-Host Coevolution in a Persistently Coxsackievirus B3-Infected Cardiomyocyte Cell Line. *Journal of Virology* 85(24): 13409-13419.

Preble, O. T. and Youngner, J. S. (1973a). Temperature-sensitive defect of mutants isolated from L cells persistently infected with Newcastle disease virus. *Journal of Virology* 12(3): 472–480.

Preble, O. T. and Youngner, J. S. (1973b). Selection of Temperature-Sensitive Mutants During Persistent Infection: Role in Maintenance of Persistent Newcastle Disease Virus Infections of L Cells. *Journal of Virology* 12(3): 481-491.

Puhlmann, J., Puehler, F., Mumberg, D., Boukamp, P., and Beier, R. (2010). Rac1 is required for oncolytic NDV replication in human cancer cells and establishes a link between tumorigenesis and sensitivity to oncolytic virus. *Oncogene* 29(15): 2205–2216

Quackenbush, John. (2006). Microarray Analysis and Tumor Classification. *The new England Journal of Medicine* 354: 2463-2472.

Rabbani, M. A. G., Ribaudo, M., Guo, J-T. and Sailen, B. (2016). Identification of Interferon-Stimulated Gene Proteins That Inhibit Human Parainfluenza Virus Type 3. *Journal of Virology* 90(24): 11145-11146.

Radtke, K., Dohner, K. and Sodeik, B. (2006). Viral interactions with the cytoskeleton: a hitchhiker's guide to the cell. *Cell Microbiology* 8(3): 387-400.

Randall, R. E. and Griffin, D. E. (2017). Within host RNA virus persistence: mechanisms and consequences. *Current Opinion in Virology* 23: 35-42.

Rangaswamy, U. S., Wang, W., Cheng, X., McTammey, P., Carroll, D., Jin, H. (2017). Newcastle disease virus establishes persistent infection in tumor cells in vitro: contribution of the cleavage site of fusion protein and second sialic acid binding site of hemagglutinin-neuraminidase. *Journal of Virology* 91(16): e00770-17.

Raposo, R. A. S., Rougvie, M. D. M., Paquin-Proulx, D., Brailey, P. M., Cabino, V. D., Zdinak, P. M., Thomas, A. S., Huang, Szu-han, Beckerle, G. A., Jones, R. B. and Nixon, D. F. (2017). IFITM1 targets HIV-1 latently infected cells for antibody-dependent cytolysis. *The Journal of Clinical Investigation* 2(1): e85811.

Rathmell, J. C., Fox, C. J., Plas, D. R., Hammerman, P., Cinalli, R. M., Thompson, C. B. (2003). Akt-directed glucose metabolism can prevent Bax conformation change and promote growth factor-independent survival. *Molecular Cell Biology* 23:7315–7328.

Rawadi, G. and Dussurget, O. (1995). Advances in PCR based Detection of Mycoplasmas Contaminating Cell Cultures. *PCR Methods and Applications* 4: 199-208.

Reichard, K. W., Lorence, R. M., Cascino, C. J., Peeples, M. E., Walter, R. J., Fernando, M. B., Reyes, H. M. and Greager, J. A. (1992) Newcastle disease virus selectively kills human tumor cells. *Journal of Surgical Research* 52: 448–453.

Rima, B. K. and Martin, S. J. (1976). Persistent Infection of Tissue Culture Cells by RNA. *Medical Microbiology and Immunology* 162: 89-118.

Rima, B. K., and Duprex, W. P. (2005) Molecular mechanisms of measles virus persistence. *Virus Research* 111(2): 132-147.

Rodriguez, J. E. and Henle, W. (1964). Studies on Persistent Infections of Tissue Cultures V. The Initial Stages of Infection of L(Mcn) Cells by Newcastle Disease Virus. *The Journal of Experimental Medicine* 19(6): 895–922.

Rodriguez, J. E., Meulen, V. T. and Henle, W. (1967). Studies on Persistent Infections of Tissue Culture VI. Reversible Changes in Newcastle Disease Virus Populations as a Result of Passage in L Cells or Chick Embryos. *Journal of Virology*: 1-9.

Rout, S. N., and Samal, S. K. (2008). The Large Polymerase Protein Is Associated with the Virulence of Newcastle Disease Virus. *Journal of Virology* 82: 7828-7836.

Rusell, S. J. (2002). RNA viruses as virotherapy agents. *Cancer Gene Therapy* 9: 961-966.

Sakaguchi, T., Matsuda, Y., Kiyokage, R., Kawahara, N., Kiyotani, K, Katunuma, N., Nagai, Y. and Yoshida, T. (1991). Identification of endoprotease activity in the trans Golgi membranes of rat liver cells that specifically processes in vitro the fusion glycoprotein precursor of virulent Newcastle disease virus. *Virology* 184: 504-512.

Schirrmacher, V., Haas, C., Bonifer, R., Ahlert, T., Gerhards, R. and Ertel, C. (1999). Human tumor cell modification by virus infection: An efficient and safe way to produce cancer vaccine with pleiotropic immune stimulatory properties when using Newcastle disease virus. *Gene Therapy* 6: 63–73

Schirrmacher, V. (2016). Fifty Years of Clinical Application of Newcastle Disease Virus: Time to Celebrate! *Biomedicines* 4(3): 16.

Schirrmacher, V. (2017). Immunobiology of Newcastle Disease Virus and Its Use for Prophylactic Vaccination in Poultry and as Adjuvant for Therapeutic Vaccination in Cancer Patients. *International Journal of Molecular Sciences* 18: 1103-1123.

Schoggins, J. W. and Rice, C. M. (2011). Interferon-stimulated genes and their antiviral effector functions. *Current Opinion in Virology* 1(6): 519-525.

Selvaraj, S. and Natarajan, J. (2011). Microarray Data Analysis and Mining Tools. *Bioinformation* 6(3): 95-99.

Sen, G. C and Peters, G. A. (2007). Viral stress-inducible genes. *Advances in Virus Research* 70: 233–263.

Shangguan, L., Ti, X., Krause, U., Hai, B., Zhao, Y., Yang, Z. Liu, F. (2012). Inhibition of TGF-b/Smad Signaling by BAMBI Blocks Differentiation of Human

Mesenchymal Stem Cells to Carcinoma-Associated Fibroblasts and Abolishes Their Protumor Effects. *Stem Cells* 30(12): 2810–2819.

Singh, P. K., Doley, J., Kumar, G. R., Sahoo, A. P. and Tiwari, A. K. (2012). Oncolytic viruses & their specific targeting to tumour cells. *Indian Journal of Medical Research* 136(4): 571-584.

Snow, A. L., Chen, L. J., Nepomuceno, R. R., Krams, S. M., Esquivel, C. O. and Martinez, O. M. (2001). Resistance to Fas-Mediated Apoptosis in EBV-Infected B Cell Lymphomas Is Due to Defects in the Proximal Fas Signaling Pathway. *The Journal of Immunology* 167(9): 5404-5411.

Songhua, S., Chaogang, S., Caozhe, X., Jian, Z., Yongqiang, H., Jianhua, W. and Zuxun, G. (2003). Differentiation of velogenic, mesogenic and lentogenic strains of Newcastle disease virus by multiplex RT-PCR. *Annals of Applied Biology* 142: 49-54.

Steward, M., Vipond, I. B., Millar, N. S. and Emmerson, P. T. (1993). "RNA editing in Newcastle disease virus." *The Journal of General Virology* 74(12): 2539-2574.

Stojdl, D. F., Lichty, B., Knowles, S., Marius, R., Atkins, H., Sonenberg, N. and Bell, J. C. (2000). Exploiting tumor-specific defects in the interferon pathway with a previously unknown oncolytic virus. *Nature* Medicine 6: 821-825.

Takimoto, T. and Portner, A. (2004). Molecular mechanism of paramyxovirus budding. *Virus Research* 106(2): 133-145.

Tarca, A. L., Romero, R. and Draghici, S. (2006). Analysis of microarray experiments of gene expression profiling. *American Journal of Obstetrics & Gynecology* 195(2): 373-388.

Tayeb, S., Zakay-Rones, Z. and Panet, A. (2014). Therapeutic potential of oncolytic Newcastle disease virus a critical review. *Oncolytic Virotherapy* 4: 49-62.

Thacore, H. and Youngner, J. S. (1969). Cells Persistently Infected with Newcastle Disease Virus I. Properties of Mutants Isolated from Persistently Infected L Cells. *Journal of Virology* 4(3): 244-251.

Toivola, D. M., Strnad, P., Habtezion, A. and Omary, M. B. (2010). Intermediate filaments take the heat as stress proteins. *Trends in Cell Biology* 20: 79–91.

Tsai, P., Lin, C-C., Sun, H-Y., Lee, J-C., Chang, T-T. and Young, K-C. (2017). Viral dynamics of persistent hepatitis C virus infection in high-sensitive reporter cells resemble patient's viremia. *Journal of Microbiology, Immunology and Infection* 2017.

Uchiumi, A., Yamashita, M. and Katagata, Y. (2012). Downregulation of keratins 8, 18 and 19 influences invasiveness of human cultured squamous cell carcinoma and adenocarcinoma cells. *Experimental and Therapeutic Medicine* 3: 443-448.

Vale, R. D. (2003). The molecular motor toolbox for intracellular transport. *Cell* 112: 467–480.

Vidal, S. and Kolakofsky, D. (1989). Modified model for the switch from Sendai virus transcription to replication. *Journal of Virology* 63: 1951–1958.

Warren, C. J., Griffin, L. M., Little, A. S., Huang, I-C., Farzan, M. and Pyeon, D. (2014). The Antiviral Restriction Factors IFITM1, 2 and 3 Do Not Inhibit Infection of Human Papillomavirus, Cytomegalovirus and Adenovirus. *PLoS One* 9(5): e96579.

Werner, T. (2007). Bioinformatics applications for pathway analysis of microarray data. *Current Opinion in Biotechnology* 19(1): 50-54.

Wheelock, E. F., and Dingle, J. H. (1964). Observations on the repeated administration of viruses to a patient with acute leukemia. *The New England Journal of Medicine* 271: 645–651.

Xu, J., Sun, Y., Li, Y., Ruthel, G., Weiss, S. R., Raj, A., Beiting, D. and Lopez, C. B. (2017). Replication defective viral genomes exploit a cellular pro-survival mechanism to establish paramyxovirus persistence. *Nature Communications* 8: 799.

Yan, X., Lin, Z, Chen, F., Zhao, X., Chen, H., Ning, Y. and Chen, Y-G. (2009). Human BAMBI cooperates with Smad7 to inhibit TGF-β Signaling. *Journal of Biological Chemistry* 284(44): 30097–30104.

Yu, Xiao-hui., Cheng, Jin-long., Xue, Jia., Jin, Ji-hui., Song, Yang., Zhao, Jing., and Zhang, Guo-zhong. (2017). Roles of Polymerase-associated Protein Genes in Newcastle Disease Virus Virulence. *Frontiers in Microbiology* 8: 161.

Yusoff, K., and Tan, W. S. (2001) Newcastle disease virus: macromolecules and opportunities. *Avian Pathology* 30: 439–455.

Zhao, Y., Altman, B. J., Coloff, J. L., Herman, C. E., Jacobs, S. R., Wieman, H. L., Wofford, J. A., Dimascio, L. N., Ilkayeva, O., Kelekar, A., Reya, T., Rathmell, J. C. (2007). Glycogen synthase kinase 3alpha and 3beta mediate a glucosesensitive antiapoptotic signaling pathway to stabilize Mcl-1. *Molecular Cell Biology* 27: 4328–4339.

Zhao, Y., Wieman, H. L., Jacobs, S. R. and Rathmell, J. C. (2008). Mechanisms and Methods in Glucose Metabolism and Cell Death. *Methods Enzymology* 442: 439-457.

Zuylen, W. J., Rawlinson, W. D. and Ford, C. E. (2016). The Wnt pathway: a key network in cell signalling dysregulated by viruses. *Reviews in Medical Virology* 26(5): 340-55.

Zwezdaryk, K. J., Combs, J. A., Morris, C. A. and Sillivan, D. E. (2016). Regulation of Wnt/β-catenin signaling by herpesviruses. *World Journal of Virology* 5(4): 144-154.