About OMICS Group

OMICS Group is an amalgamation of Open Access publications and worldwide international science conferences and events. Established in the year 2007 with the sole aim of making the information on Sciences and technology 'Open Access', OMICS Group publishes 500 online open access scholarly journals in all aspects of Science, Engineering, Management and Technology journals. OMICS Group has been instrumental in taking the knowledge on Science & technology to the doorsteps of ordinary men and women. Research Scholars, Students, Libraries, Educational Institutions, Research centers and the industry are main stakeholders that benefitted greatly from this knowledge dissemination. OMICS Group also organizes 500 International conferences annually across the globe, where knowledge transfer takes place through debates, round table discussions, poster presentations, workshops, symposia and exhibitions.

About OMICS International Conferences

OMICS International is a pioneer and leading science event organizer, which publishes around 500 open access journals and conducts over 500 Medical, Clinical, Engineering, Life Sciences, Pharma scientific conferences all over the globe annually with the support of more than 1000 scientific associations and 30,000 editorial board members and 3.5 million followers to its credit.

OMICS Group has organized 500 conferences, workshops and national symposiums across the major cities including San Francisco, Las Vegas, San Antonio, Omaha, Orlando, Raleigh, Santa Clara, Chicago, Philadelphia, Baltimore, United Kingdom, Valencia, Dubai, Beijing, Hyderabad, Bengaluru and Mumbai.

.

Genome-Wide Transcriptomes Integrate Animal Intra- and Inter-Organism Processes and Evoke the Ecoimmunological Concept in Animal Health

Yongming Sang, D.Sc., Ph.D.

College of Veterinary Medicine

Kansas State University, Manhattan, KS 66506, USA



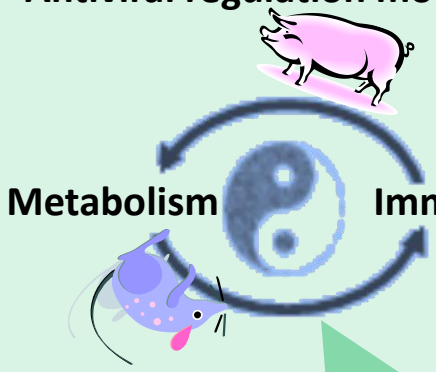
(07/28/2015, Transcriptome-2015)

III. Integrative Strategy in Animal Health and Nutrition:

Current Projects

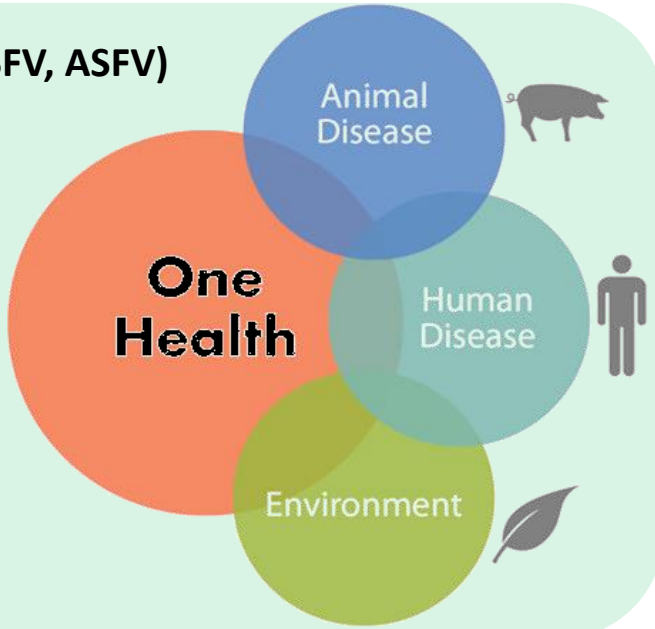
USDA: (1) Immunogenetics and immunoreagent development **($48,000, 2014-2018) (2)** Macrophage-based antiviral regulation **($499,436, 2013-2017)**; **(3)** Molecular and functional diversity of interferons (USDA pending)

Antiviral regulation model (PRRSV, PEDV, SIV, DCoV, CSFV, ASFV)



Metabolism **Immunity** = **Two Facets of One task of animal health & nutrition**

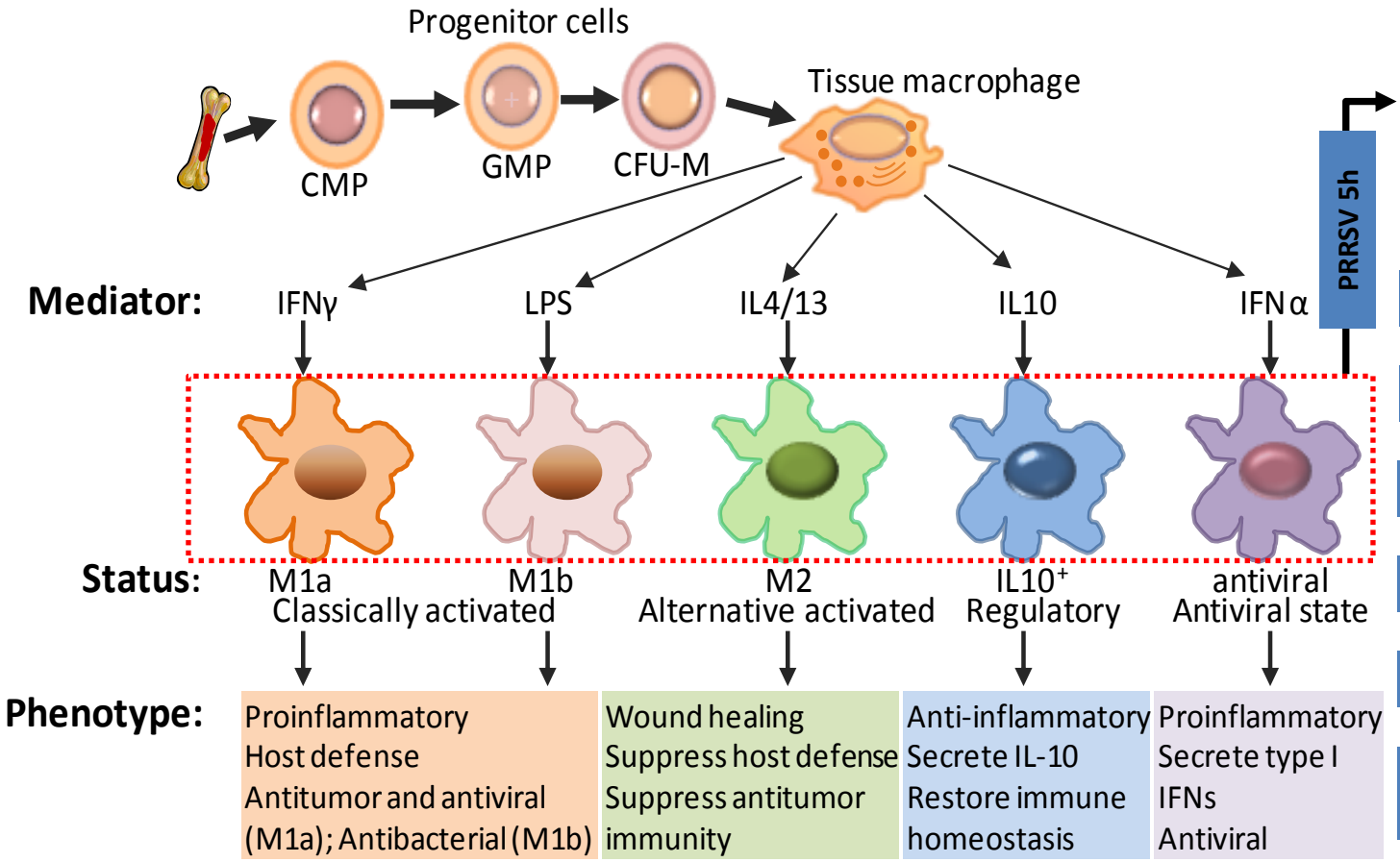
Obesity model (Had-36)

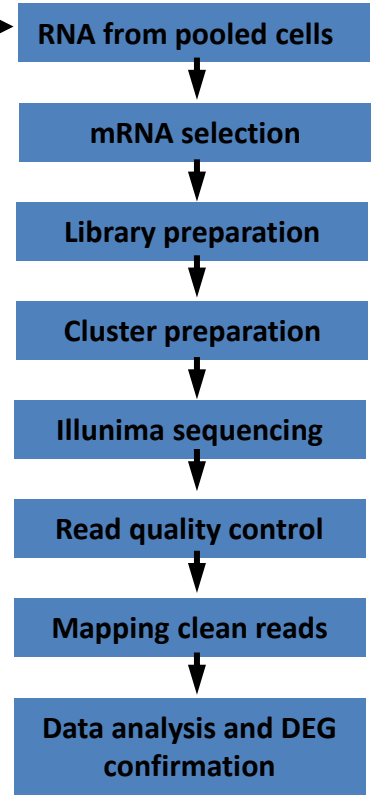


NIH: Rat obesity induced by high-fat-diet plus Had-36 infection (NIH pilot: **$50,000**; and R15 pending)

Google images

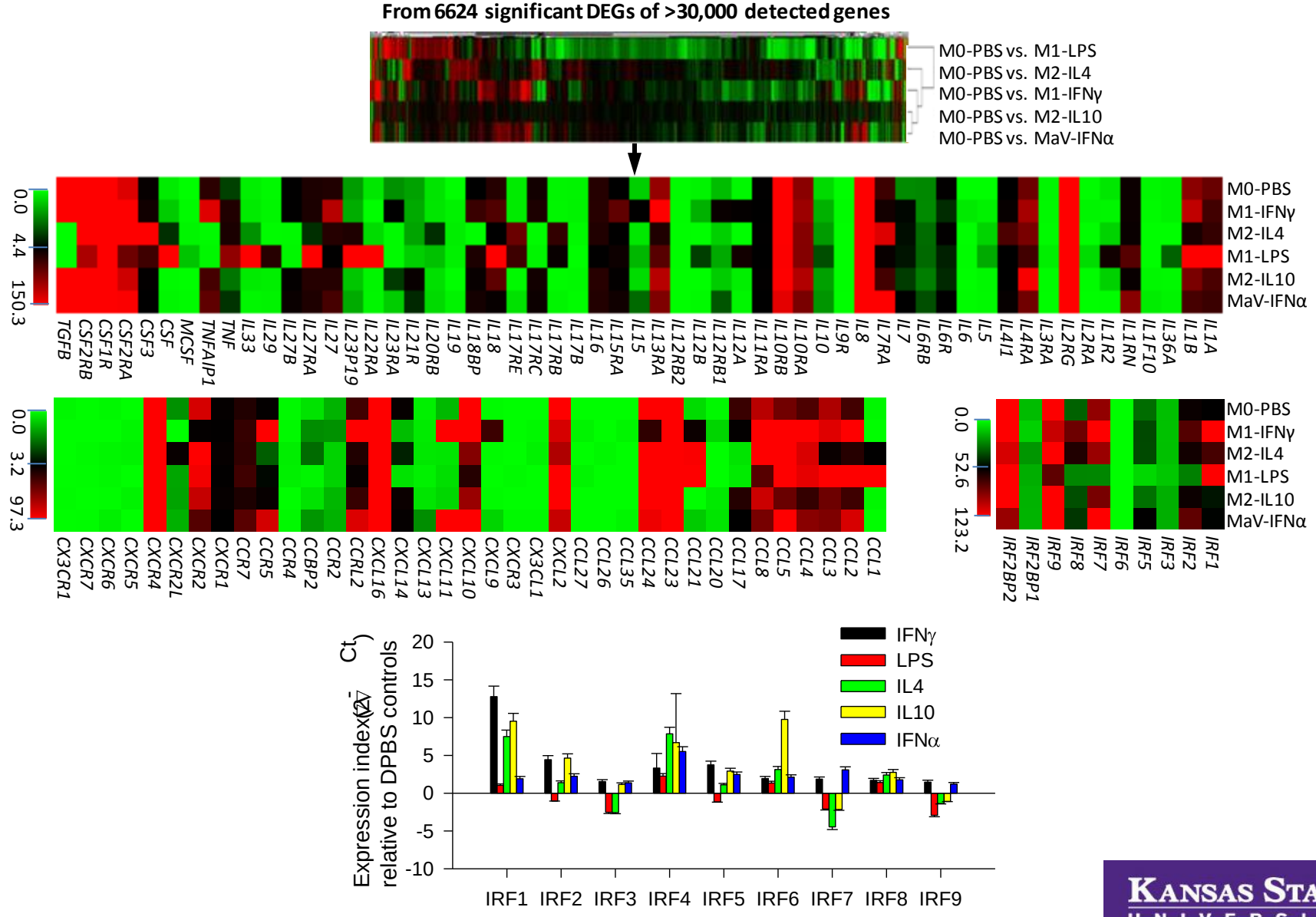
Our Macrophage Polarization and RNA-seq Procedure





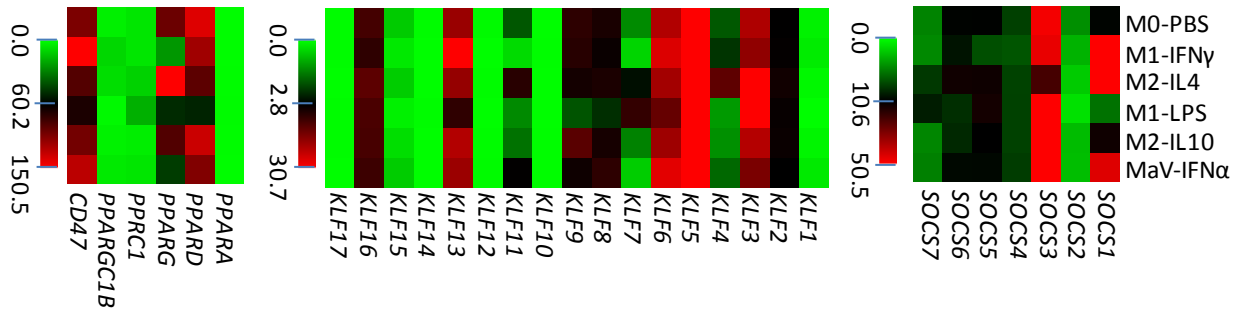
KANSAS STATE UNIVERSITY

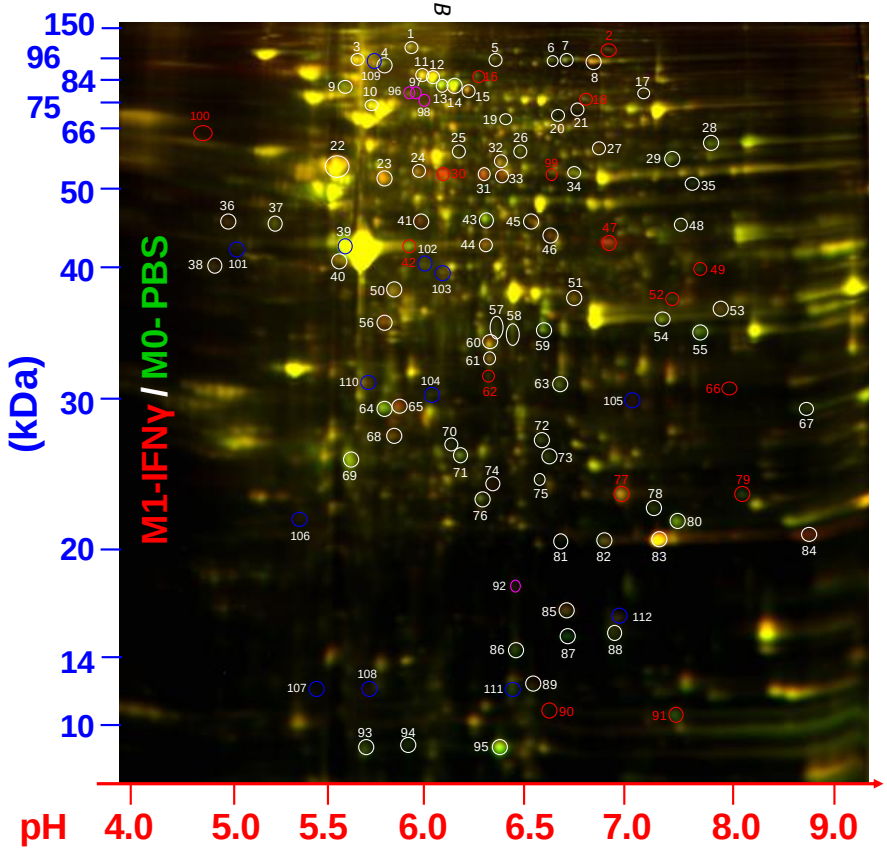
DEGs Related to Cytokines, Chemokines and IRFs, and RT-PCR Confirmation

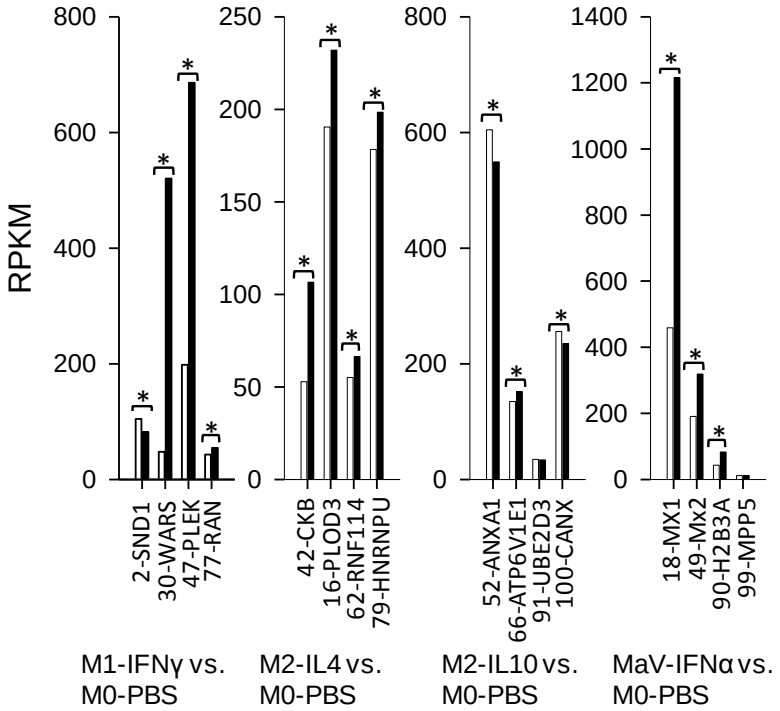


KANSAS STATE UNIVERSITY

DEGs Related to Transcription Factors that are Important for Macrophage Activation, and Proteomic Confirmation



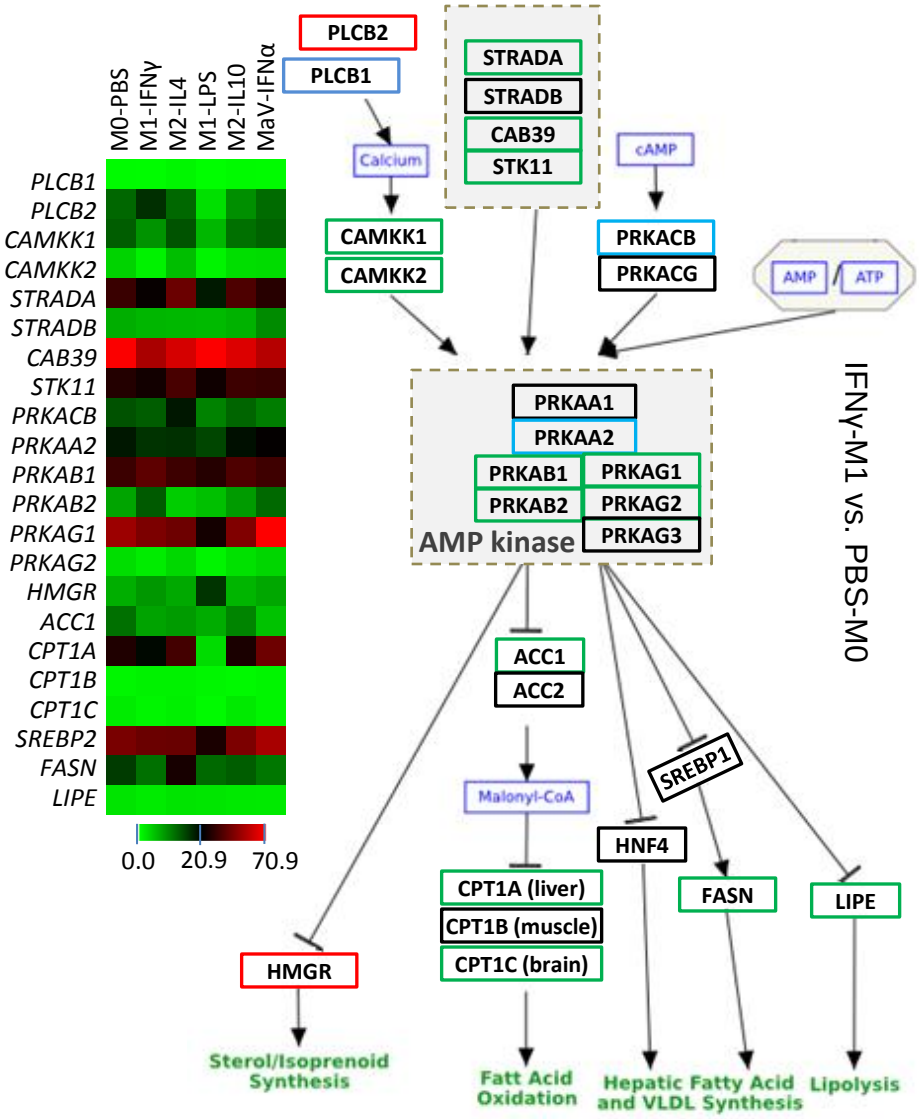


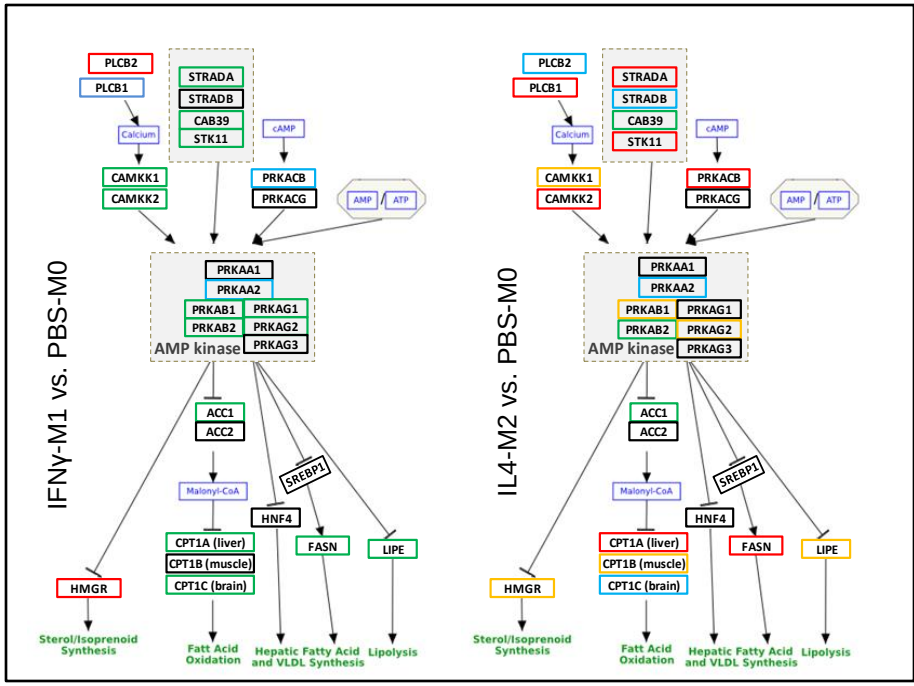


Spence S, et al., Immunity. 2013 Jan 24;38(1):66-78. Alder JK, et al. J Immunol. 2008 Apr 15;180(8):5645-52.

KANSAS STATE UNIVERSITY

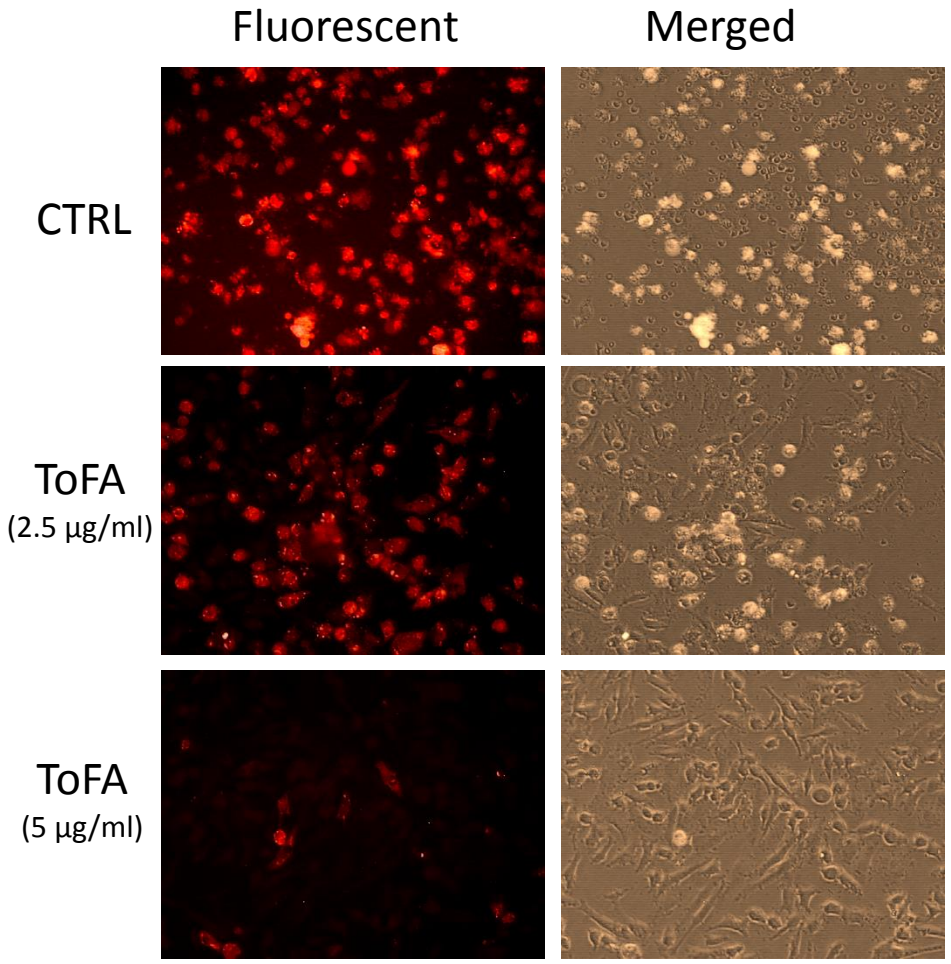
DEGs in AMPK Pathway in Mediation of Lipid Metabolism



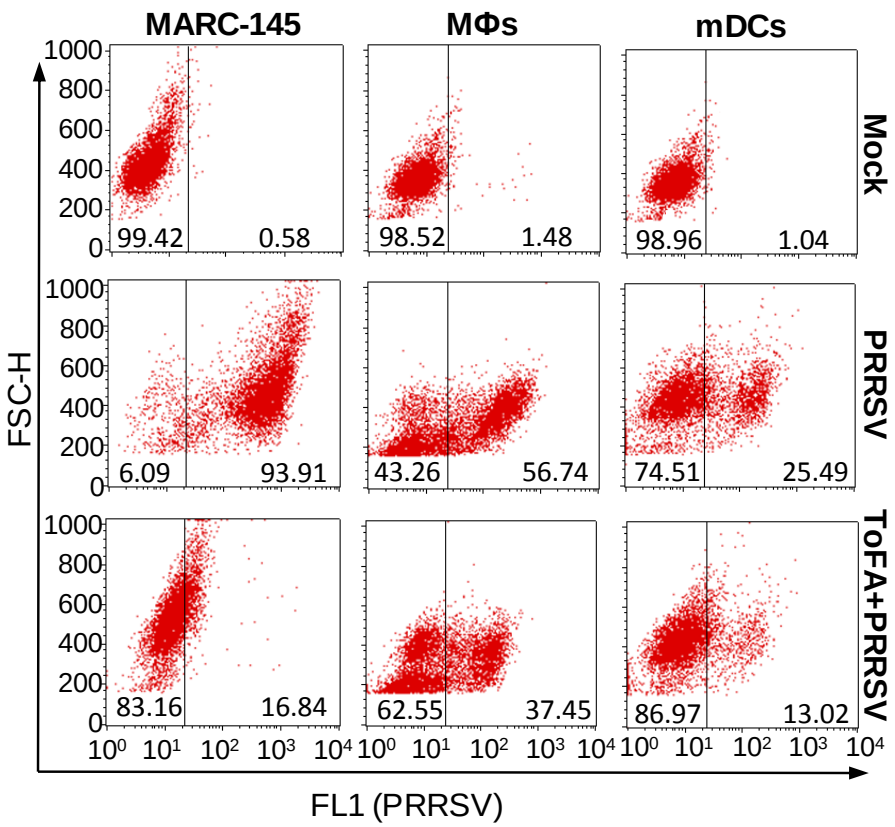


KANSAS STATE UNIVERSITY

Regulation of PRRSV infection *via* AMPK Pathway and Lipid Metabolism



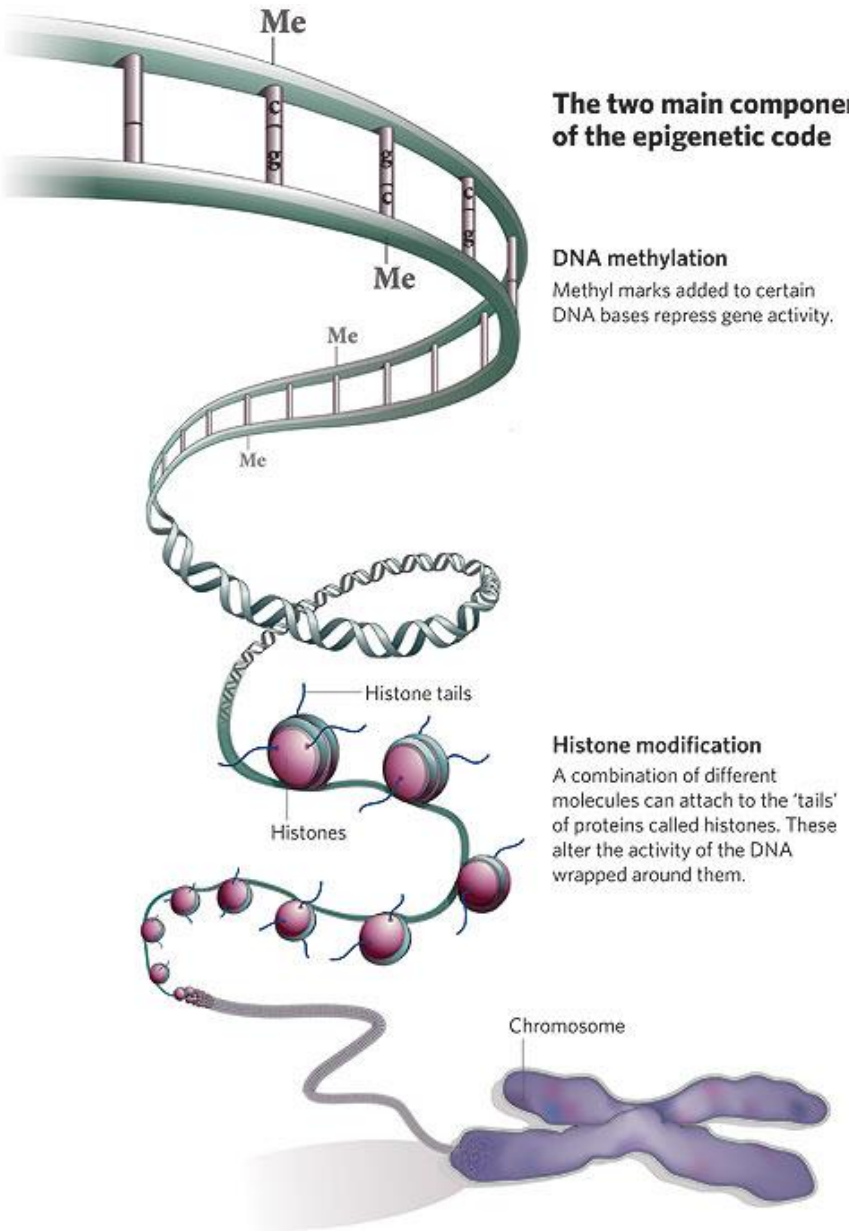
mDCs infected with DsRed-PRRSV for 48 h

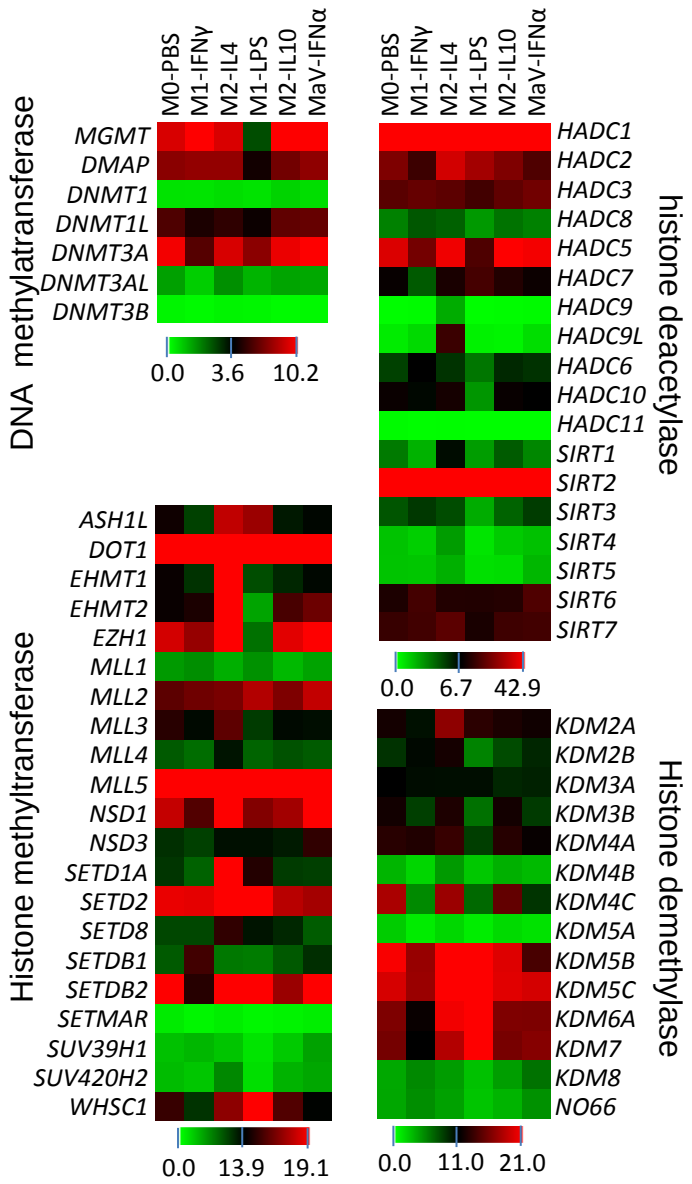


ToFA (5-(Tetradecyloxy)-2-furoic acid), a competitive inhibitor of acetyl-CoA carboxylase (ACC)

KANSAS STATE UNIVERSITY

DEGs Critical for Epigenetic Regulation

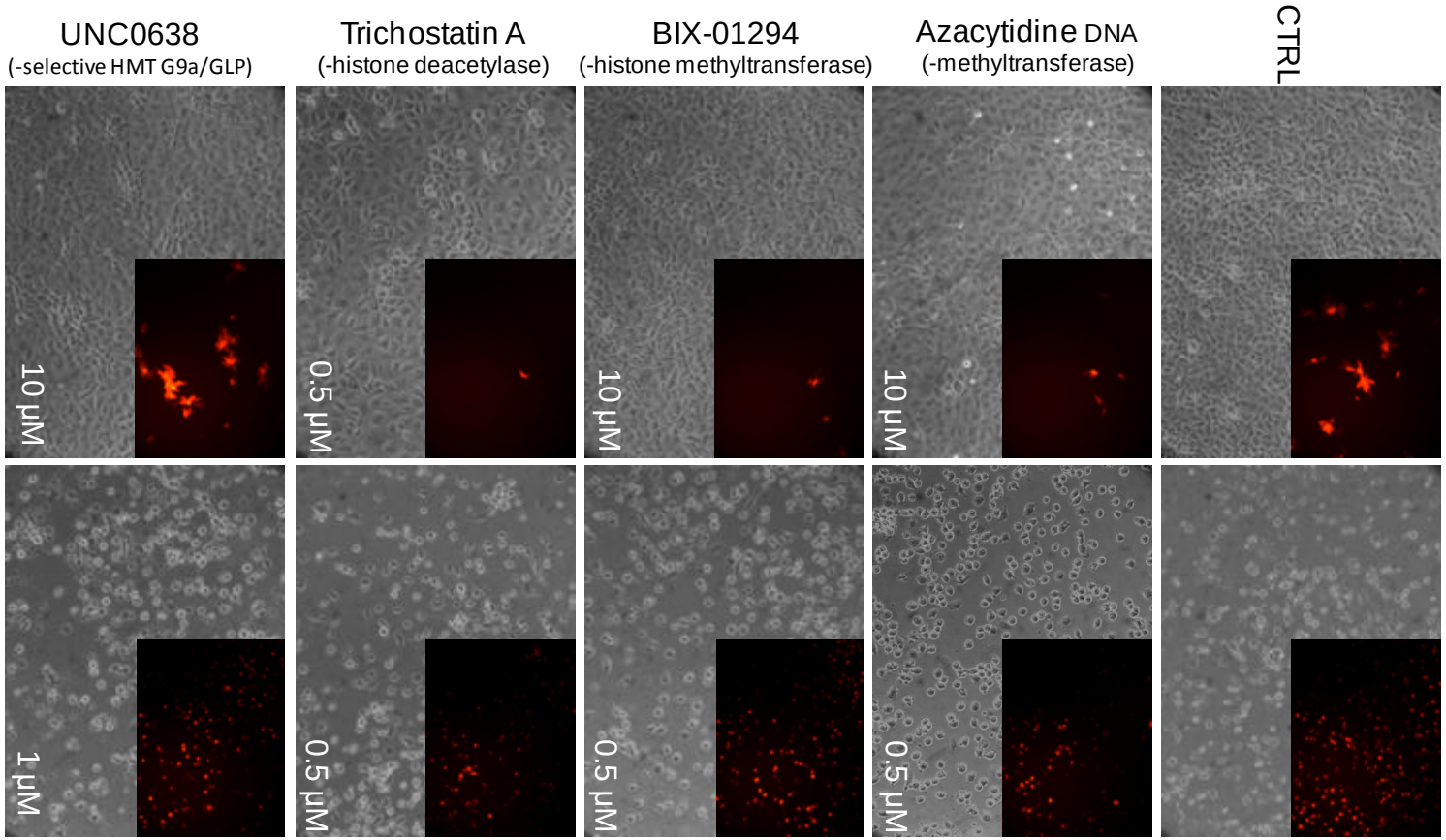




http://www.eusem.com/main/CH/epi

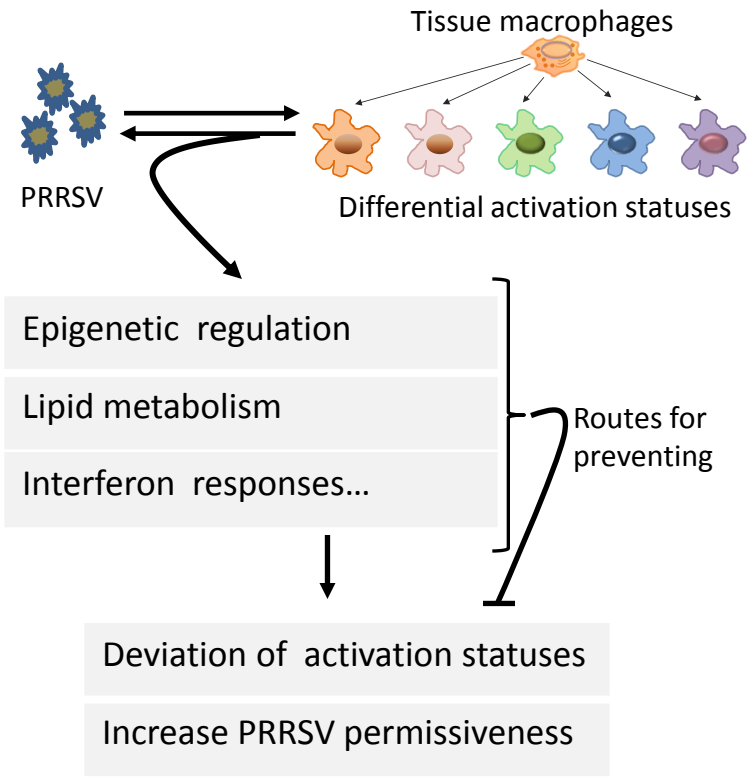
KANSAS STATE UNIVERSITY

Regulation of PRRSV infection *via* Epigenetic Regulation



KANSAS STATE UNIVERSITY

Summary



1. **Porcine monocytic cells at different activation statuses, interact differentially with PRRSV**

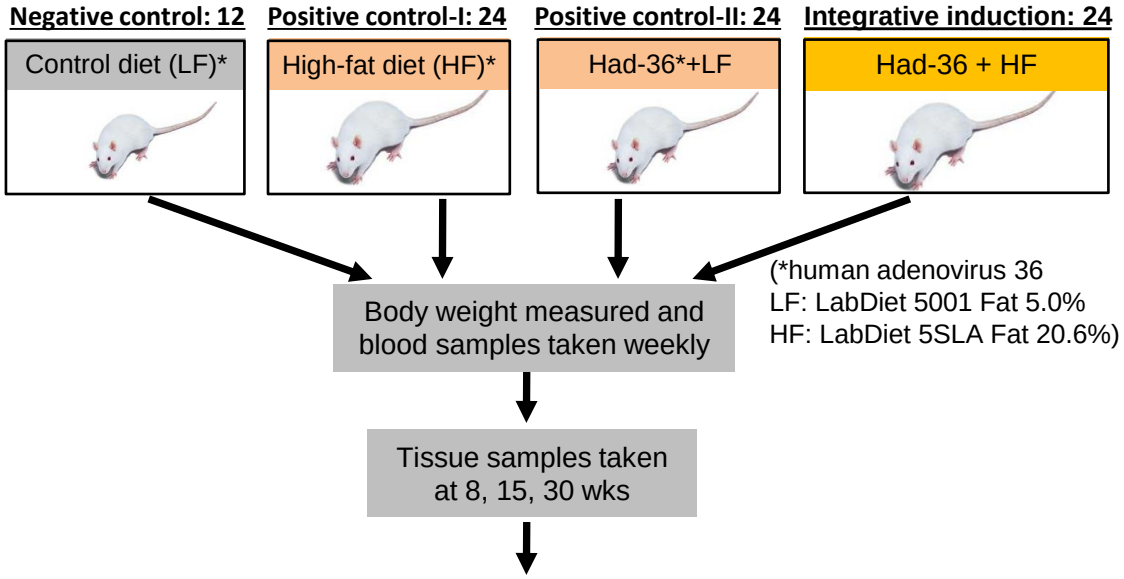
2. **Key genes in the pathways such as lipid metabolism, epigenetics and interferon signaling are revealed among significant DEGs.**

3. **Signature genes revealed in PRRSV—infected macrophages at each activation status infer the mechanism of PRRSV-cell interaction.**

4. **Understanding and modulating cell activation status of monocytic innate immune cells provides a framework for optimizing antiviral immunity**

Objective and Approach

Male Wistar Rats: 6-wk old [Serum negative to Ad., Body Weight (BW): 125±5g]



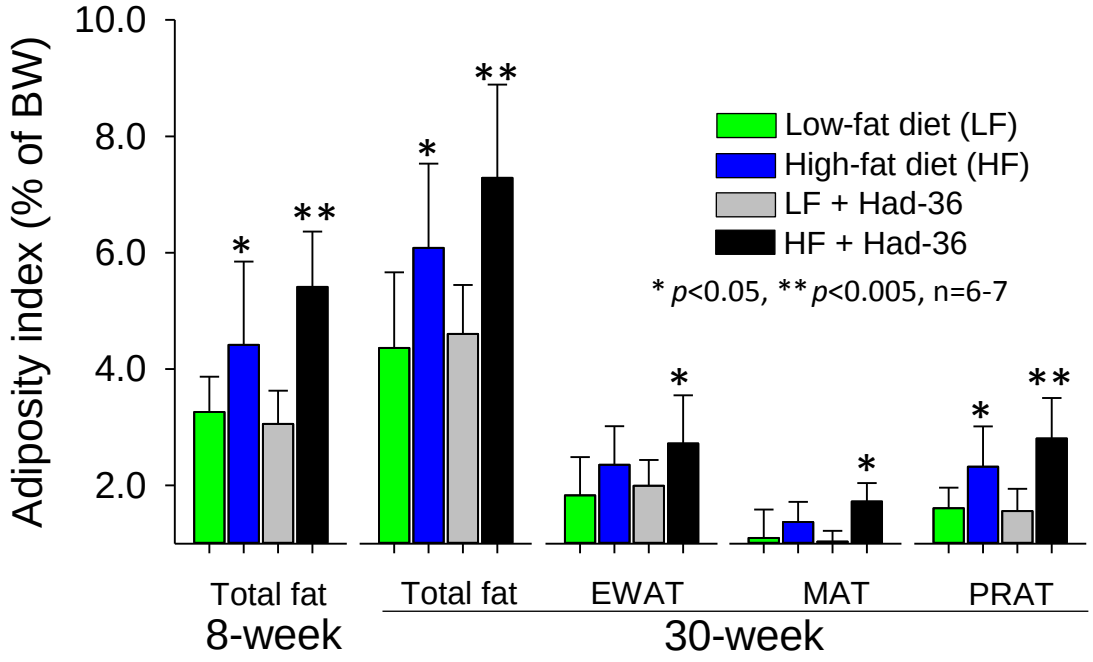
Aims:

1. Develop rat obesity model integratively induced by a high-fat diet and Had-36 infection
2. Validate obesity phenotypes including body weight, fat index, plasma leptin level, and parameters associated with gut/adipose inflammation
3. Extract ileal RNA from LF lean, Had-36+LF, HF and Had-36+HF obese groups
4. Perform transcriptomic analyses using 8-wk ileal and hypothalamus samples
5. Analyze gene ontology and pathway integration, and determine obesity-linked innate immune genes

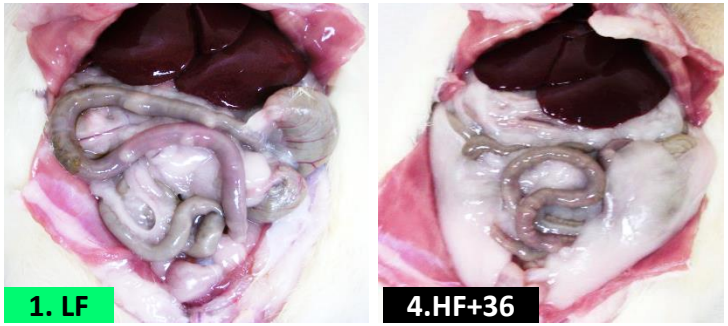
de La Serre CB, et al., Am J Physiol Gastrointest Liver Physiol. 2010 Aug;299(2):G440-8.

Results and Discussion

HF + Had-36 infection synergistically increased adiposity indexes in total fat or visceral fats in three adipose depots

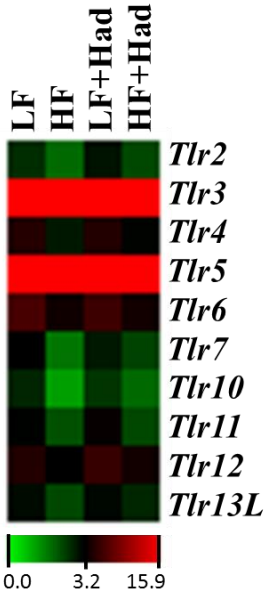


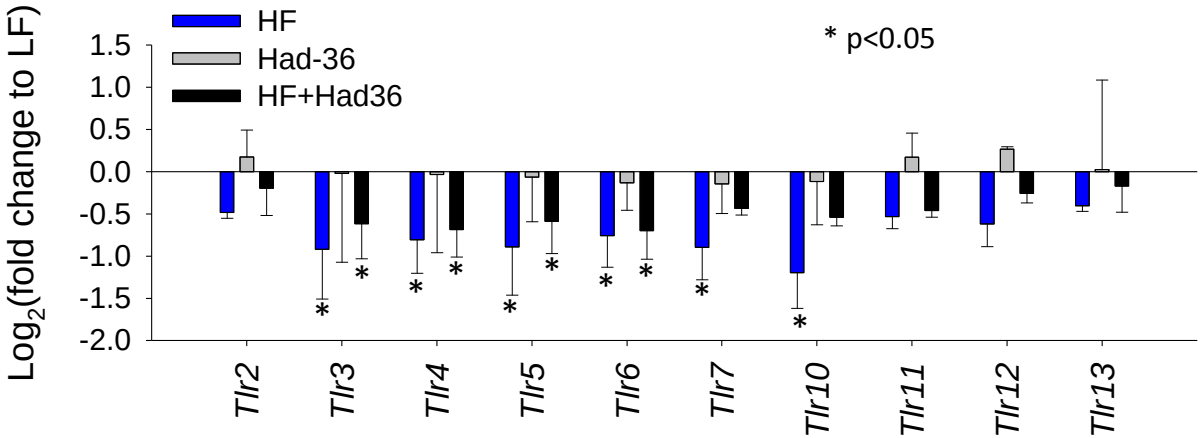
EWAT, MAT and PRAT: epididymal , mesenteric, and perirenal white adipose tissue

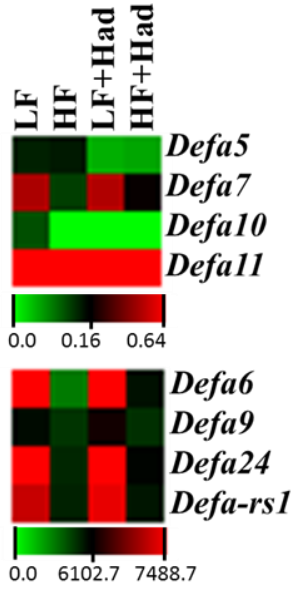


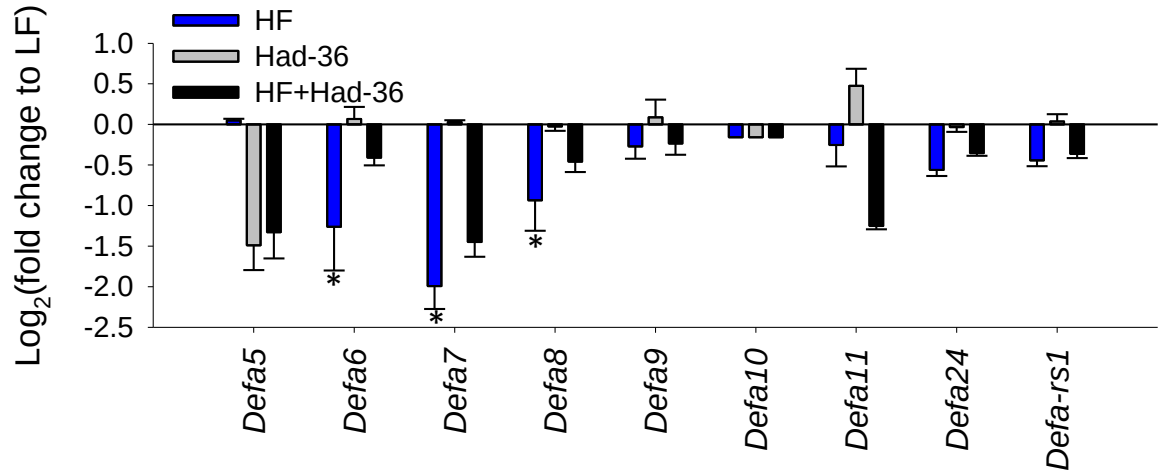
Results and Discussion

Profiling of Significant Differential Expression Genes



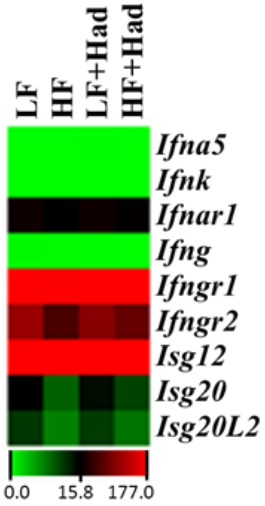


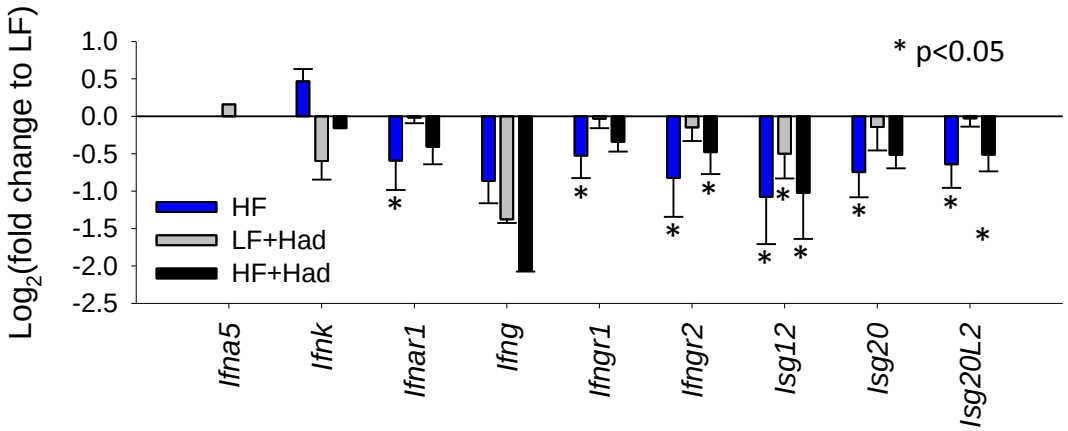


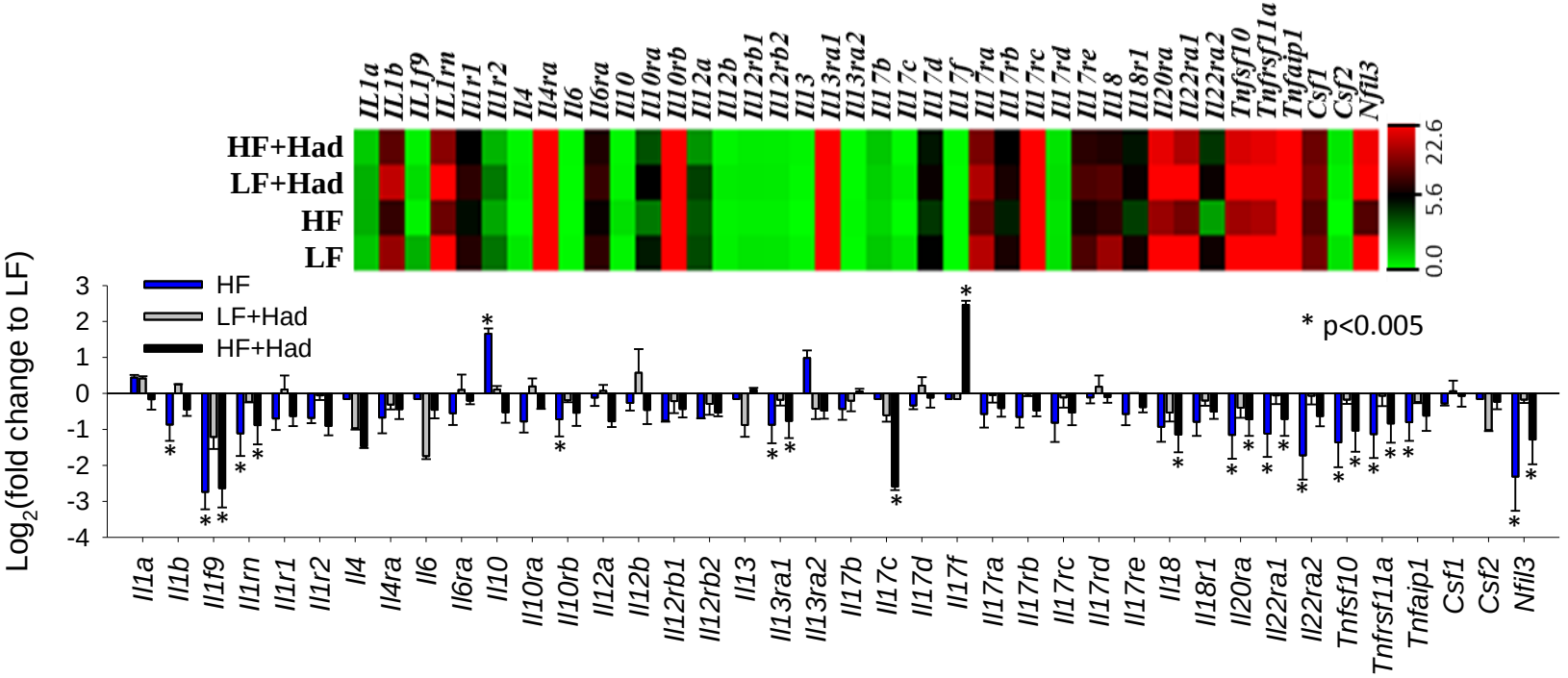


Results and Discussion

Profiling of Significant Differential Expression Genes

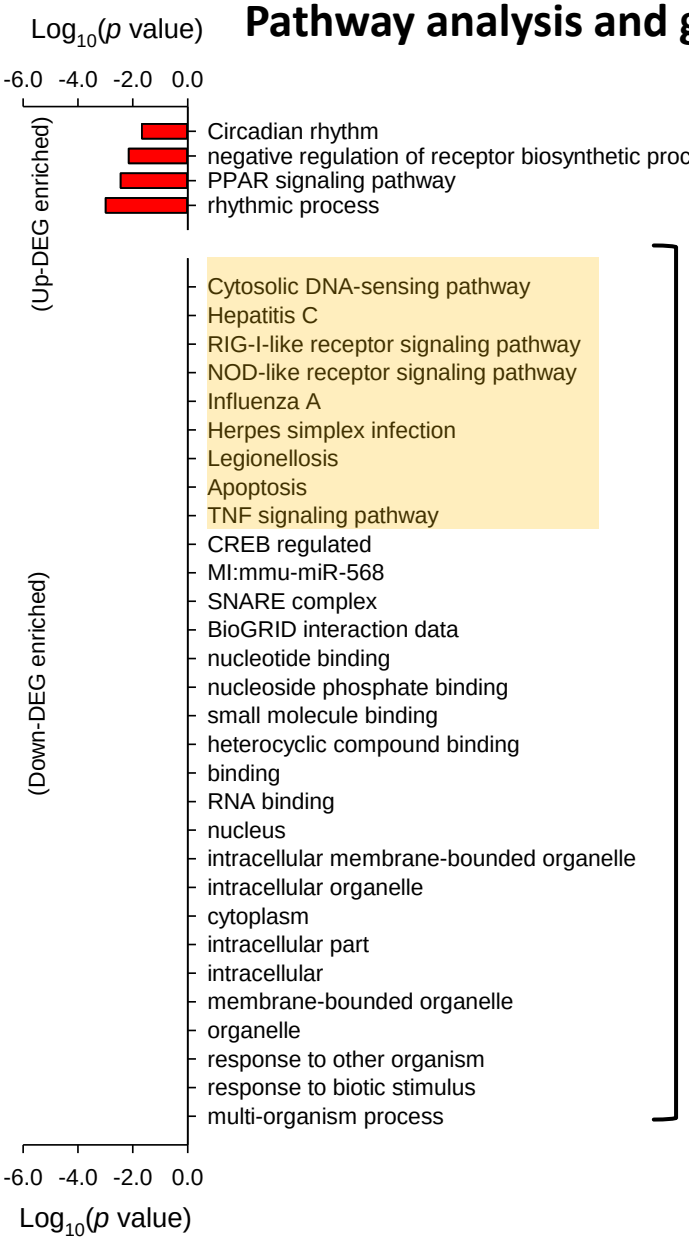






Results and Discussion

Pathway analysis and gene ontology (GO) analysis



HF + Had-36 infected group compared with control group

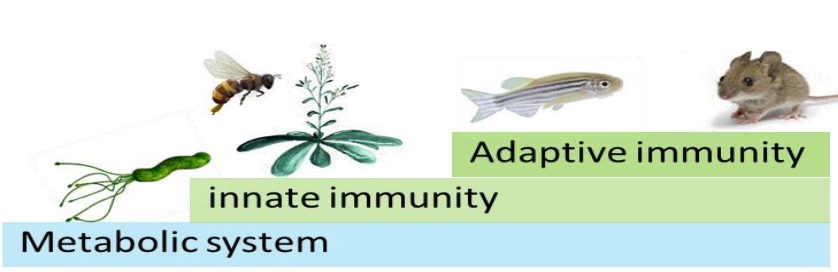
All 30, including 10 disease/immunity related (highlighted), pathways show little direct relation with lipid metabolic pathways but are highly relevant to intracellular and inter-organism communication ***(p<0.05)***

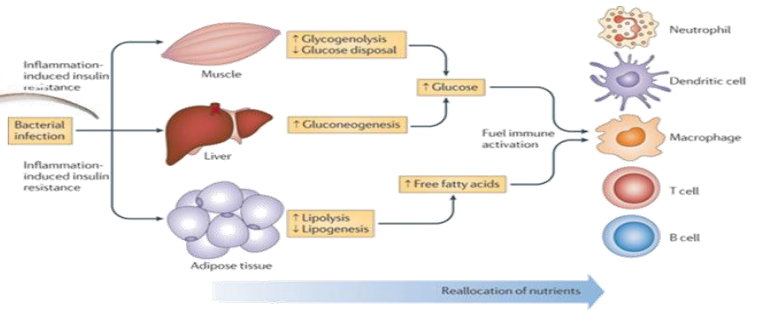
Summary

1. Infection of Had-36 synergizes with the induction of obesity by high-fat diet in rats; however, Had-36 infection has little effect in obesity induction in rats fed the control diet.

2. Either high-fat diet or Had-36 infection increases gut permeability and inflammation but has no synergistic effect (actually having antagonistic effect)

3. At 8-wk post treatment, rat ileal transcriptome shows significant down-regulation of multiple families of immune and metabolic genes in the HF and HF+Had-36 groups, as well as reprograming of circadian rhythm pathway.





Chawla A, et al. Nat Rev Immunol. 2011, 11:738-49.

Acknowledgements

• Laboratory colleagues:



•Collaborators:

Dr. Frank Blecha, KSU
Dr. Raymond R. R. Rowland, KSU
Dr. Joan Lunney, USDA-ARS
Dr. Jishu Shi, KSU
Dr. Wenjun Ma, KSU
Dr. Laura Millar, USDA-ARS
Dr. Zhihua Jiang, WSU
Dr. Christopher Tuggle, ISU
Dr. Chris Netherton (Pirbright, UK)
Dr. Bryan Charleston (Pirbright, UK)
Dr. Linda Dixon (Pirbright, UK)
Dr. Ruth Welti (Kansas Lipdomics)
Dr. Robert Goodband (KSU, Animal Science) and Mr. Frank Jennings Jr (KSU, Animal farms)

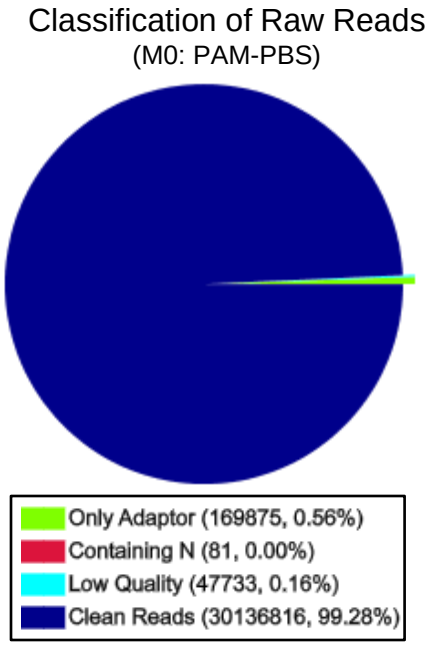
•Funding resources:

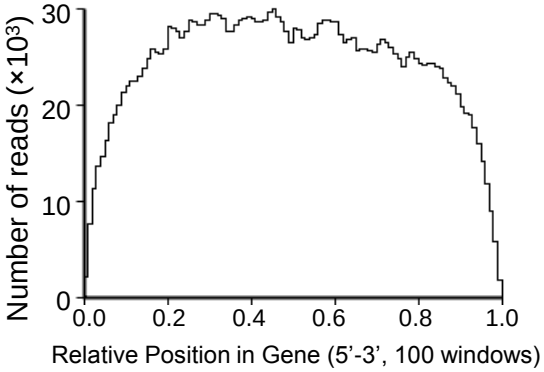
• The studies are supported in part by (1) USDA-NIFA-COOP-2010-002475 (Co-PI: Sang, KSU); USDA NIFA AFRI 2015-67015-23216 (Co-PI: Sang, KSU), and in particular (3) USDA-NIFA-2013-67015-21236 (PI: Sang, KSU)
•NIH 1R15HD066377-01 (Co-PI: Sang KSU), Pilot project (PI. Sang, KSU) of NIH NCRR-COBRE-P20-RR017686 (PD: Daniel Marcus, KSU)

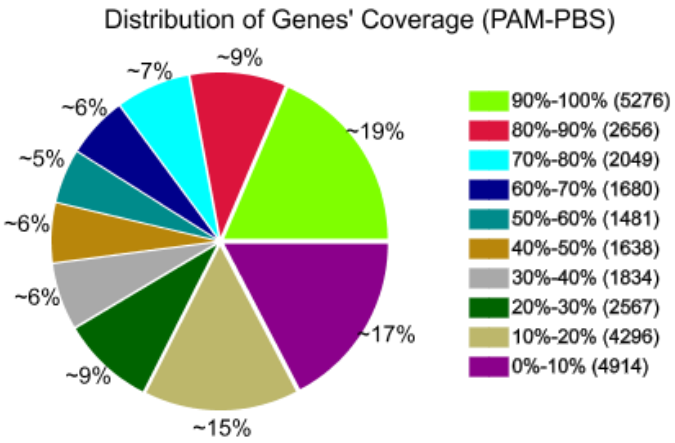
KANSAS STATE UNIVERSITY

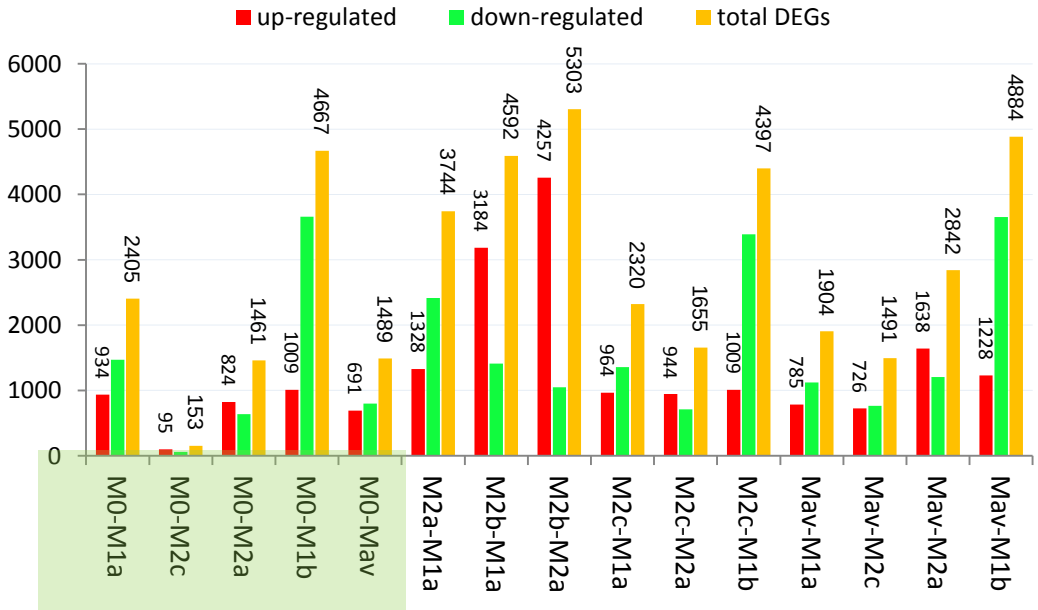
Thank You!

RNA-seq Data Quality Control and DEG Analysis







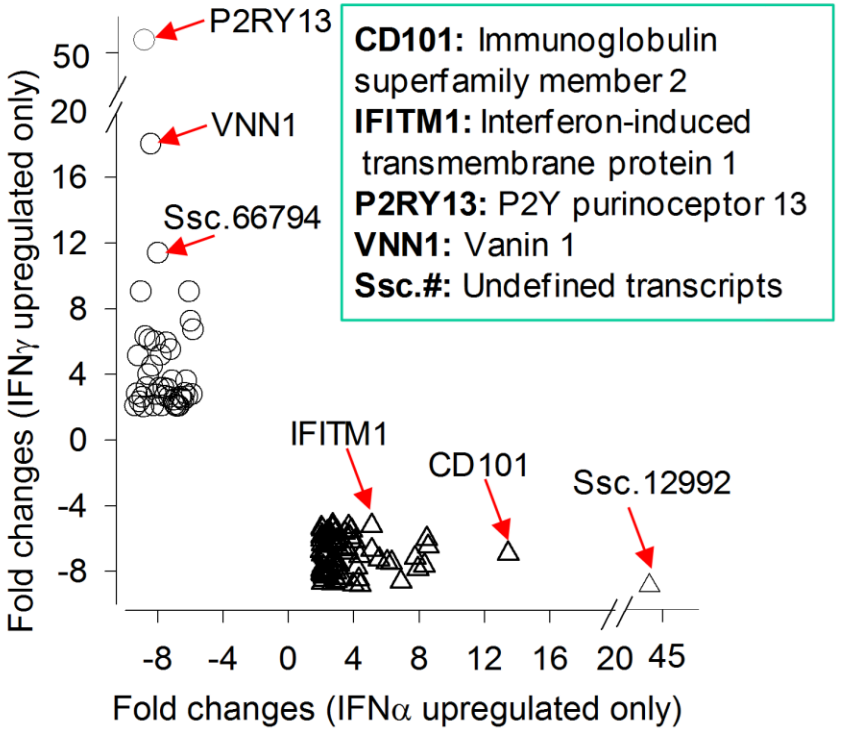


KANSAS STATE UNIVERSITY

Profiling Signature Genes of Each Activation Status

Table 1: Numbers of potential gene makers identified in each activation status.

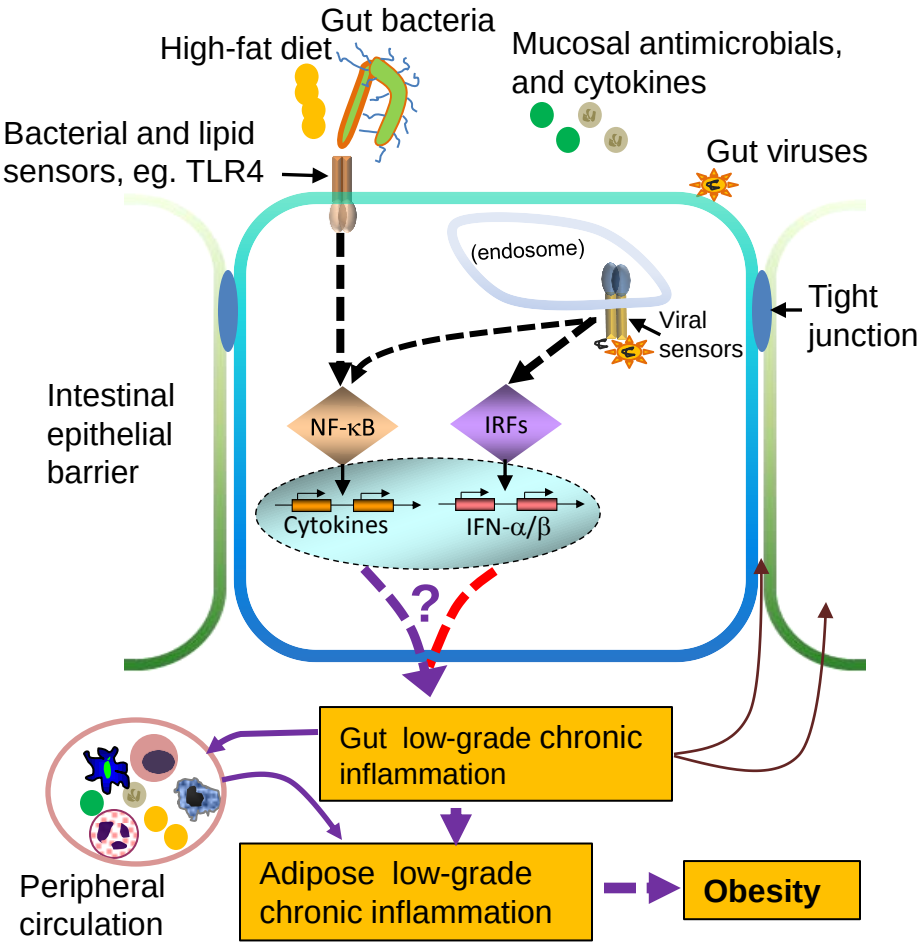
Status	DEG number
(1) IFNγ-M1	44
(2) IFNα-MaV	72
(3) Co-IFNγ & IFNα	88
(4) IL4-M2	234
(5) LPS-M1	72
(6) IL10-M2	9
Total	519



KANSAS STATE UNIVERSITY

Background and Hypothesis

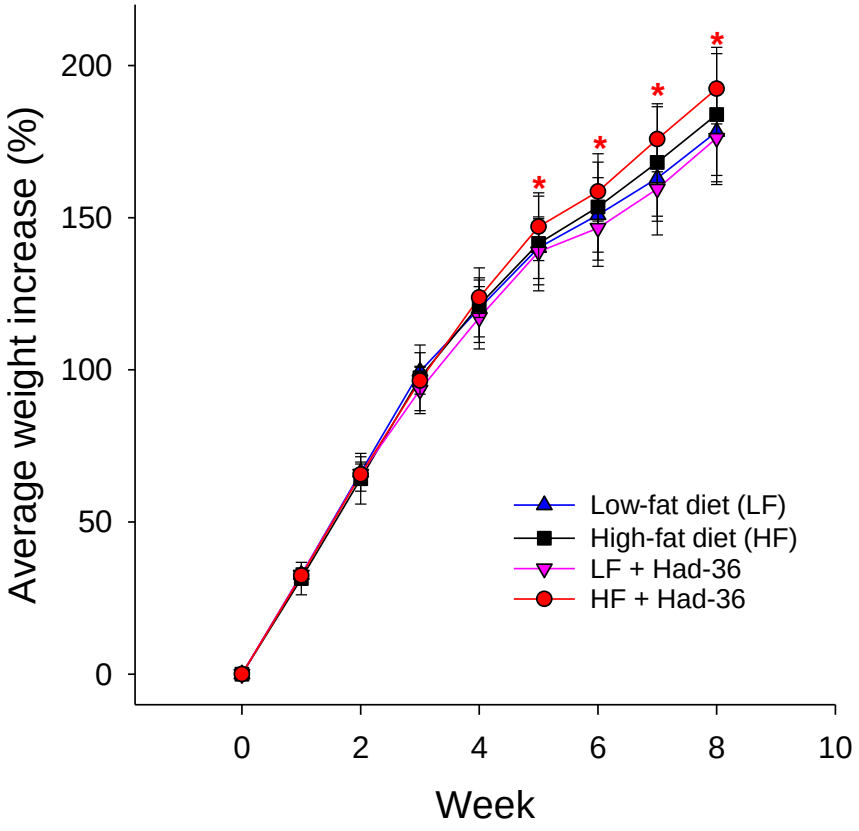
Hypothesis: Viral infection and high-fat diet can induce obesity independently, and in particular may contribute to prevalence of obesity synergistically.

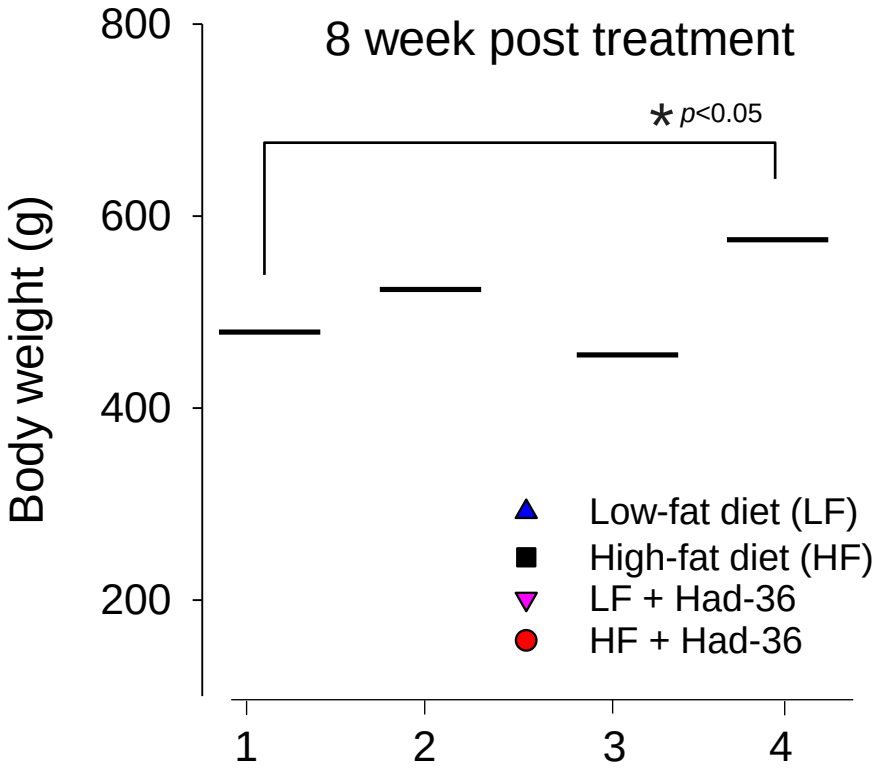


Mitra AK, Clarke K. Viral obesity: fact or fiction? Obes Rev. 2010 Apr;11(4):289-96.
Dhurandhar NV. Insulin sparing action of adenovirus 36 and its E4orf1 protein. J Diabetes Complications. 2013 Mar-Apr;27(2):191-9.

Results and Discussion

HF + Had-36 infection synergistically increased BW after 5 wk
Had-36 infection without HF had little effect

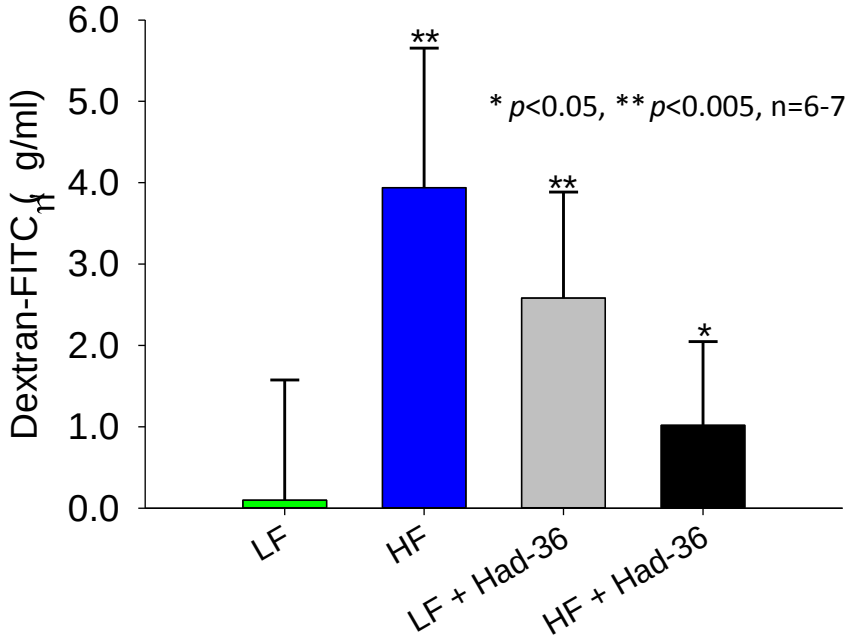


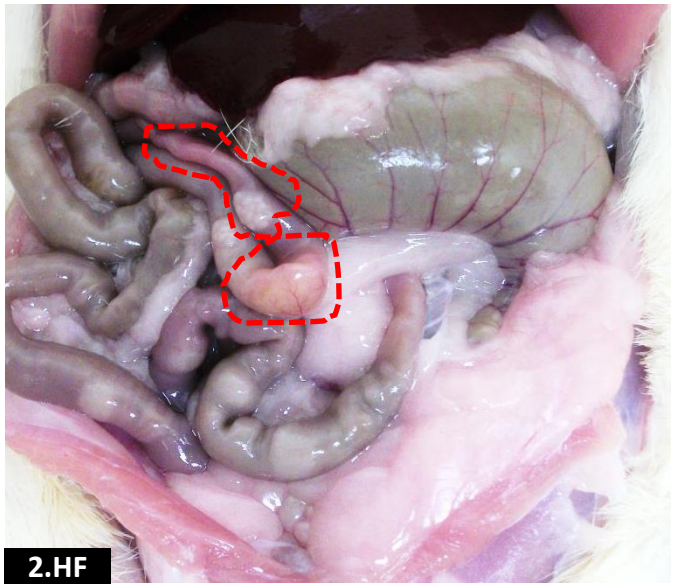


Ave. BW diff: 20-30 g between HF groups with LF groups

Vander Wal JS, Huelsing J, Dubuisson O, Dhurandhar NV. An observational study of the association between adenovirus 36 antibody status and weight loss among youth. Obes Facts. 2013;6(3):269-78.

Results and Discussion

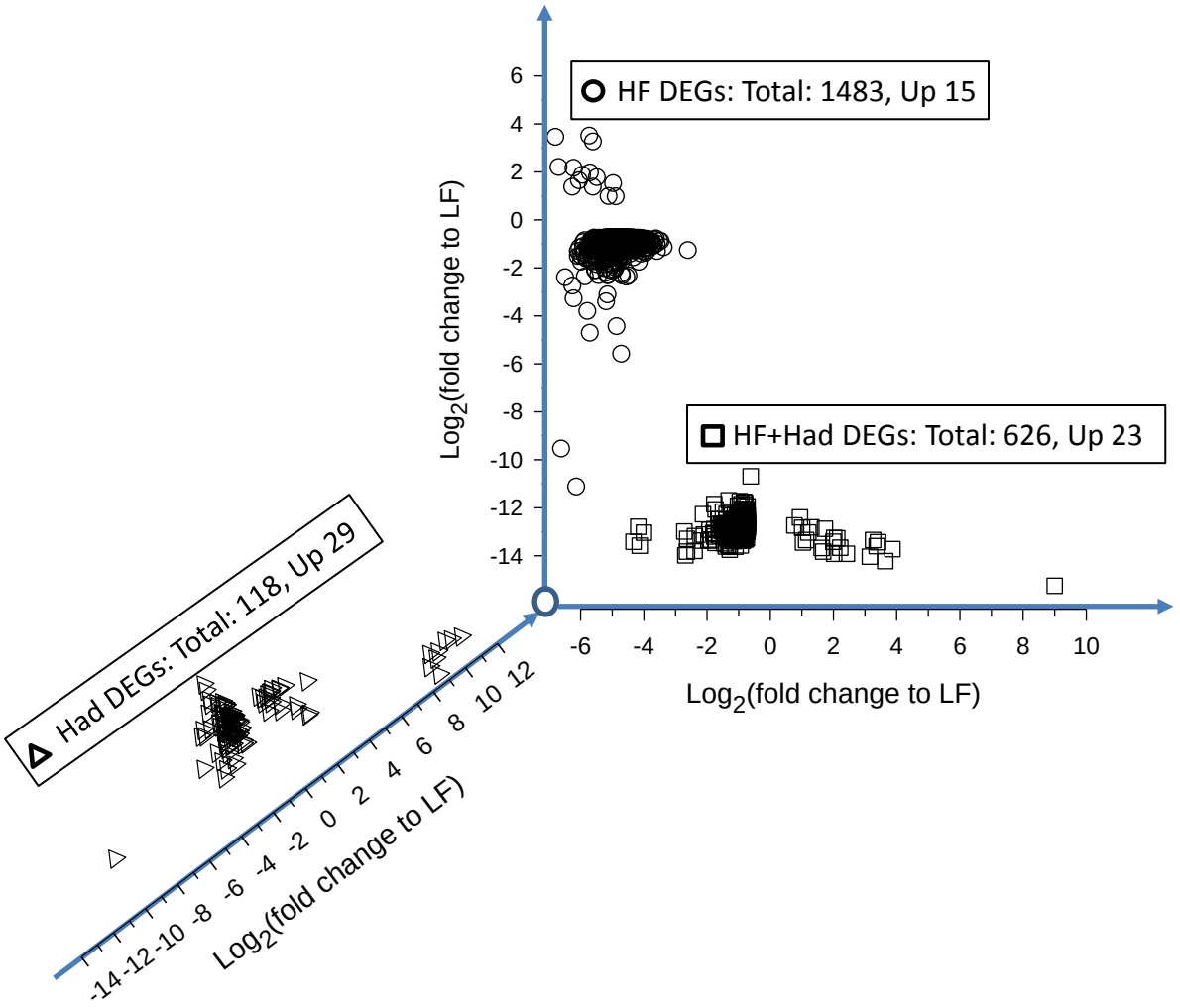




Either high-fat diet or Had-36 infection increases gut permeability but has no synergistic effect (actually having antagonistic effect) . Thus, the development of obesity is associated but may not proportionally correlated with an increase in gut permeability.

Results and Discussion

Profiling of Significant Differential Expression Genes



Let Us Meet Again

We welcome you all to our future conferences of OMICS International

Please Visit:

http://transcriptomics.conferenceseries.com/

http://conferenceseries.com/

http://www.conferenceseries.com/genetics-and-molecular-biology-conferences.php