# Curriculum Vitae

**Name:** Haijiang Lin, MD, PhD, M.Sc

## A. Education/ Training

| INSTITUTION AND LOCATION | DEGREE (if applicable) | END DATE | FIELD OF STUDY |
|---|---|---|---|
| Weifang Medical University, Weifang | MD | 07/1985 | Medicine |
| McGill University, Montreal, Canada | PhD | 02/2000 | Molecular Biology & Biochemistry |
| Dana-Farber Cancer Institute, Harvard Medical School, Boston, Massachusetts | Postdoctoral Fellow | 04/2002 | Cell Biology, Kinetochore-Microtubule attachment |
| Massachusetts Institute of Technology, Cambridge, Massachusetts | Postdoctoral Fellow | 10/2006 | Epigenetic, DNA methylation and Cancer |
| University of South Carolina, Greenville, South Carolina | Resident | 06/2009 | General Surgery |
| University of Texas Medical Branch at Galveston, Galveston, Texas | Resident | 06/2012 | Ophthalmology |
| Mass. Eye and Ear Infirmary, Harvard Medical School, Boston, Massachusetts | Fellow | 06/2015 | Medical Retina |

## B. Positions and Honors

### Positions and Employment

2006 - 2008 Assistant Professor, University of Texas Medical Branch at Galveston , Galveston, TX

2013 - Instructor, Harvard Medical School, Boston, MA

2016 - Assistant Professor, University of Massachusetts Medical School, Worcester, MA

### Other Experience and Professional Memberships

2000 - Reviewer, Biomedical Journal

2000 - 2007 Reviewer, Molecular and Cellular Biology

2002 - Reviewer, Cancer Research

2002 - 2006 Reviewer, Cell

2002 - 2006 Reviewer, Science

2002 - 2006 Reviewer, Journal of Cellular Biology

2007 - Reviewer, PLoS One

2009 - Member, Association for Research in Vision and Ophthalmology (ARVO)

| | |
|---|---|
| 2009 - | Member, American Academy of Ophthalmology (AAO) |
| 2010 - | Reviewer, Molecular Vision |
| 2011 - | Reviewer, Experimental Eye Research |
| 2013 - | Reviewer, European Journal of Medicine |

**Honors**

| | |
|---|---|
| 1995 - 1998 | Scholarship, McGill University, Canada |
| 1997 - 2000 | Pauline Blidner-Krupp Student Award, Royal Victoria Hospital Institute, Canada |
| 2000 - 2002 | Abraham Postdoctoral Fellowship, Dana-Farber Cancer Institute, Harvard Medical School, U.S. |
| 2001 | Experimental Mouse Genetic Award , Jackson Laboratory, U.S. |
| 2001 - 2004 | Fellowship (top sixth ranked in 268 applicants), Canadian Institute of Health Research, Canada |
| 2004 - 2005 | Pilot Project Award , Cancer Center, Massachusetts Institute of Technology, U.S. |
| 2012 | Research Award, Department of Ophthalmology, University of Texas Medical Branch at Galveston, U.S. |

## C. Contribution to Science

1. My contribution to ubiquitin-26S proteasome system. I identified and characterized two novel genes that are involved in ubiquitin conjugation and deubiquitination. A. I identified the mammalian ubiquitine conjugating enzyme gene, UBC7 and found a loop of this enzyme determines the affinity to the ubiquitin activating enzyme (E1) and conjugation of ubiquitin to selected target proteins. UBC7 with this loop was unable to accept Ubiquitin from ubiquitin-activating enzyme (E1) but was able to accept NEDD8 from E1. This works contributes to understanding the mechanism lead ubiquitin conjugating enzyme to conjugate ubiquitin to specific target protein and distinguish Ubiquitin and NEED8. B. I identified a new gene, which has two splicing isoforms coding for two deubiquitinating enzymes, UBP- t1 and UBP-t2 with identical core regions (UBPt-core) but distinct N termini, which determine the sub- cellular distribution and modulate substrate specificity. These two-part works have significant impact in ubiquitin field.

    a. Lin H, Yin L, Reid J, Wilkinson KD, Wing SS. Divergent N-terminal sequences of a deubiquitinating enzyme modulate substrate specificity. J Biol Chem. 2001 Jun 8;276(23):20357-63. PubMed PMID: 11278432.

    b. Lin H, Keriel A, Morales CR, Bedard N, Zhao Q, Hingamp P, Lefrançois S, Combaret L, Wing SS. Divergent N-terminal sequences target an inducible testis deubiquitinating enzyme to distinct subcellular structures. Mol Cell Biol. 2000 Sep;20(17):6568-78. PubMed PMID: 10938131; PubMed Central PMCID: PMC86134.

c. Lin H, Wing SS. Identification of rabbit reticulocyte E217K as a UBC7 homologue and functional characterization of its core domain loop. J Biol Chem. 1999 May 21;274(21):14685-91. PubMed PMID: 10329663.

2. My contribution to cancer epigenetic research. I identified the function of DNA methyl-transferase, Dnmt1, Dnmt3a and Dnmt3b in the development of cancer. A. I created all splicing isoforms of de novo DNA methyltransferase, Dnmt3a and Dnmt3b induction transgenic mice. I found that Dnmt3b1 enhanced the number and average size of colon tumors in Apc Min/+ mice. The overexpression of Dnmt3b1 caused loss of imprinting and increased expression of Igf2 as well as methylation and transcriptional silencing of the tumor suppressor genes Sfrp2, Sfrp4, and Sfrp5. Importantly, we found that Dnmt3b1 but not Dnmt3a1 efficiently methylates the same set of genes in tumors and in non- tumor tissues, demonstrating that de novo methyltransferases can initiate methylation and silencing of specific genes in phenotypically normal cells. B. I created Dnmt3b conditional knock out mice. Using the conditional inactivation of Dnmt3b in Apc (Min/+) mice, I demonstrate that the loss of Dnmt3b has no impact on microadenoma formation, but a significant decrease in the formation of macroscopic colonic adenomas. For the first time, we demonstrated that Dnmt3b plays a critical role in the transition of microadenoma to macroscopic adenomas. C. We detected that DNA hypomethylation promotes early lesions in the colon and liver, but suppresses later stages of intestinal tumorgenesis through a loss of Heterozygosity (LOH) mechanism using Dnmt1 hypomorphic alleles to reduce genomic methylation. This is the first time to report that a dual role of genomic DNA hypomethylation in the development of cancer.

a. Subramanyam D, Belair CD, Barry-Holson KQ, Lin H, Kogan SC, Passegué E, Blelloch R. PML- RAR{alpha} and Dnmt3a1 cooperate in vivo to promote acute promyelocytic leukemia. Cancer Res. 2010 Nov 1;70(21):8792-801. PubMed PMID: 20861188; PubMed Central PMCID: PMC3021794.

b. Linhart HG, Lin H, Yamada Y, Moran E, Steine EJ, Gokhale S, Lo G, Cantu E, Ehrich M, He T, Meissner A, Jaenisch R. Dnmt3b promotes tumorigenesis in vivo by gene-specific de novo methylation and transcriptional silencing. Genes Dev. 2007 Dec 1;21(23):3110-22. PubMed PMID: 18056424; PubMed Central PMCID: PMC2081977.

c. Lin H, Yamada Y, Nguyen S, Linhart H, Jackson-Grusby L, Meissner A, Meletis K, Lo G, Jaenisch R. Suppression of intestinal neoplasia by deletion of Dnmt3b. Mol Cell Biol. 2006 Apr;26(8):2976- 83. PubMed PMID: 16581773; PubMed Central PMCID: PMC1446955.

d. Yamada Y, Jackson-Grusby L, Linhart H, Meissner A, Eden A, Lin H, Jaenisch R. Opposing effects of DNA hypomethylation on intestinal and liver carcinogenesis. Proc Natl Acad Sci U S

A. 2005 Sep 20;102(38):13580-5. PubMed PMID: 16174748; PubMed Central PMCID: PMC1224663.

3. My contribution to studying the molecular pathology of AMD. A. I found that mitochondrial DNA (mtDNA) damage increased and repair capacity decreased with aging, especially in RPE cells from the macular region. Most importantly, the mtDNA damage was positively, while repair capacity negatively correlated with the severity of AMD. In addition, more mitochondrial heteroplasmic mutations were detected in AMD patient macular RPE cells. B. We found that cytosolic accumulation of damaged mtDNA fragments induces RPE cells to secrete the inflammatory cytokines IL-6 and IL-8, and prime the NLRP3 inflammasome in a manner that depends on the size of mtDNA fragment, but not on its specific sequence. This induction was mediated by STING (Stimulator of Interferon Genes) and NF-κB pathway. C. I found that miR-23a reduce RPE cell death induced by H2O2 through regulation of Fas but miR-23a expression is significantly down regulated in AMD patient macular RPE cells and may lead RPE cells to loss this protection. D. We found that miR-17-3p increased the cell death under oxidative stress by down regulating the expression of the antioxidant enzymes manganese superoxide dismutase (MnSOD) and thioredoxin reductase-2 (TrxR2). We found miR-17- 3p to be elevated in AMD patient macular RPE cells and possible contribute to RPE cell death under- oxidative stress in AMD patient.

    a. Tian B, Maidana DE, Dib B, Miller JB, Bouzika P, Miller JW, Vavvas DG, Lin H. miR-17-3p Exacerbates Oxidative Damage in Human Retinal Pigment Epithelial Cells. PLoS One. 2016;11(8): e0160887. PubMed PMID: 27505139; PubMed Central PMCID: PMC4978424.

    b. Dib B, Lin H, Maidana DE, Tian B, Miller JB, Bouzika P, Miller JW, Vavvas DG. Mitochondrial DNA has a pro-inflammatory role in AMD. Biochim Biophys Acta. 2015 Nov;1853(11 Pt A):2897-906. PubMed PMID: 26305120; PubMed Central PMCID: PMC5330253.

    c. Lin H, Qian J, Castillo AC, Long B, Keyes KT, Chen G, Ye Y. Effect of miR-23 on oxidant-induced injury in human retinal pigment epithelial cells. Invest Ophthalmol Vis Sci. 2011 Aug 9;52(9):6308- 14. PubMed PMID: 21693609.

    d. Lin H, Xu H, Liang FQ, Liang H, Gupta P, Havey AN, Boulton ME, Godley BF. Mitochondrial DNA damage and repair in RPE associated with aging and age-related macular degeneration. Invest Ophthalmol Vis Sci. 2011 Jun 1;52(6):3521-9. PubMed PMID: 21273542; PubMed Central PMCID: PMC3109040.

4. My contribution to exploring the novel therapeutic strategy for AMD and studying the regulation of gene expression associated with AMD. A. We found that high dose atorvastatin, HMG CoA (3-hydroxy-3- methylglutaryl-coenzyme reductase inhibitors, increased the phagocytic function of RPE cells as well as rescued the cells from the phagocytic dysfunction induced by pathologic stimuli relevant to AMD, such as cholesterol crystals and oxidized low-density lipoproteins (ox-LDL), potentially by increasing the cellular membrane fluidity. Furthermore, atorvastatin was able to block the induction of interleukins IL-6 and IL-8 triggered by cholesterol crystals and ox-LDL. This study demonstrated that statin preserve the phagocytic function of the RPE cells while also exhibiting anti-inflammatory properties. Patients with intensive statin treatment showed regression of some high-risk features of AMD. B. I found that the drug delivery nanoparticle, PEG-PLGA, had the lowest cytotoxicity to RPE cells and retina vascular endothelial cells. In addition, PEG-PLGA NPs penetrated and distributed throughout the cell, C. The gene for the high temperature requirement A1 (HTRA1) is thought to be important in many diseases, including AMD and rheumatoid arthritis (RA). We demonstrated that LPS significantly increased HTRA-1 expression in the joint tissue in mice with arthritis. This induction is mediated by NF-κB. Meanwhile, IFN-γ negatively controls HTRA1 expression through activation of the p38 MAPK/STAT1 pathway. Our study offers new insights into the molecular regulation of HTRA1 expression and its role in RA pathogenesis, which may have significant impact on clinical therapy for RA and possibly other HTRA1-related diseases, such as AMD.but preferentially in mitochondria, a property that may prove benefit in the treatment of diseases with mitochondrial dysfunction, such as AMD.

    a. Tian B, Al-Moujahed A, Bouzika P, Hu Y, Notomi S, Tsoka P, Miller JW, Lin H, Vavvas DG. Atorvastatin Promotes Phagocytosis and Attenuates Pro-Inflammatory Response in Human Retinal Pigment Epithelial Cells. Sci Rep. 2017 May 24;7(1):2329. PubMed PMID: 28539592; PubMed Central PMCID: PMC5443823.

    b. Lin H, Yue Y, Maidana DE, Bouzika P, Atik A, Matsumoto H, Miller JW, Vavvas DG. Drug Delivery Nanoparticles: Toxicity Comparison in Retinal Pigment Epithelium and Retinal Vascular Endothelial Cells. Semin Ophthalmol. 2016;31(1-2):1-9. PubMed PMID: 26959123; PubMed Central PMCID: PMC5405708.

    c. Hou Y, Lin H, Zhu L, Liu Z, Hu F, Shi J, Yang T, Shi X, Guo H, Tan X, Zhang L, Wang Q, Li Z, Zhao Y. The inhibitory effect of IFN-γ on protease HTRA1 expression in rheumatoid arthritis. J Immunol. 2014 Jul 1;193(1):130-8. PubMed PMID: 24907345.

    d. Hou Y,Lin H, Zhu L, Liu Z, Hu F, Shi J, Yang T, Shi X, Zhu M, Godley BF, Wang Q, Li Z, Zhao Y. Lipopolysaccharide increases the incidence of collagen-induced arthritis in mice through induction of protease HTRA-1 expression. Arthritis Rheum. 2013

Nov;65(11):2835-46. PubMed PMID: 23982886.

5. My contribution in identifying proteins involved in microtubule-kinetochore attachment. I demonstrated that the budding yeast CLIP-170 orthologue Bik1 is a component of the kinetochore-microtubule binding interface, which is essential in polyploidy cells. The polyploidy-specific requirement for BIK1 enabled us to characterize BIK1 without eliminating nonhomologous genes, providing a new approach to circumventing the overlapping function that is a common feature of the cytoskeleton. In polyploid cells, Bik1 is required before anaphase to maintain kinetochore separation and therefore contributes to the force that opposes the elastic recoil of attached sister chromatids. This protein is required for the viability of polyploids and has potential implications for cancer therapeutics.

    a. Lin H, de Carvalho P, Kho D, Tai CY, Pierre P, Fink GR, Pellman D. Polyploids require Bik1 for kinetochore-microtubule attachment. J Cell Biol. 2001 Dec 24;155(7):1173-84. PubMed PMID: 11756471; PubMed Central PMCID: PMC2199317.

## D. Peer reviewed publications

1. **Lin, H.,** Keriel, A., Morales, C.R., Bedard, N., Zhao, Q., Hingamp, P., Lefrancois, S., Combaret, L. and Wing, S.S.. 'Divergent N-terminal sequences target an inducible testis deubiquitinating enzyme to distinct subcellular structures'. ***Mol Cell Biol***, 2000. 20 (17):6568-6578.

2. **Lin, H**. and Wing, S.S.. 'Identification of rabbit reticulocyte E217K as a UBC7 homologue and functional characterization of its core domain loop'. ***J Biol Chem***, 1999. 274 (21):14685-14691

3. **Lin, H.,** Yin, L., Reid, J., Wilkinson, K.D. and Wing, S.S.. 'Divergent N-terminal sequences of a deubiquitinating enzyme modulate substrate specificity'. ***J Biol Chem***, 2001. 276 (23):20357-20363.

4. **Lin, H.,** de Carvalho, P., Kho, D., Tai, C.Y., Pierre, P., Fink, G.R. and Pellman, D.. 'Polyploids require Bik1 for kinetochore-microtubule attachment'. ***J Cell Biol,*** 2001. 155 (7): 1173-1184.

5. Yamada, Y., Jackson-Grusby, L., Linhart, H., Meissner, A., Eden, A**., Lin, H**. and Jaenisch, R., (2005). 'Opposing effects of DNA hypomethylation on intestinal and liver carcinogenesis'. ***Proc Natl Acad Sci U S A*****,** 2005. 102 (38):13580-13585.

6. **Lin, H.,** Yamada, Y., Nguyen, S., Linhart, H., Jackson-Grusby, L., Meissner, A., Meletis, K., Lo, G. and Jaenisch, R.. 'Suppression of intestinal neoplasia by deletion of Dnmt3b'. ***Mol Cell Biol***, 2006. 26 (8):2976-2983.

7. Linhart, H.G**., Lin, H**., Yamada, Y., Moran, E., Steine, E.J., Gokhale, S., Lo, G., Cantu, E., Ehrich, M., He, T., Meissner, A. and Jaenisch, R.. 'Dnmt3b promotes tumorigenesis in vivo by gene-specific de novo methylation and transcriptional silencing'. ***Genes and Development,*** 2007. 21 (23):3110-3122.

8. Jarrett, S.G., **Lin, H**., Godley, B.F. and Boulton, M.E.. 'Mitochondrial DNA damage and its potential role in retinal degeneration'. ***Prog Retin Eye Res***, 2008. 27 (6):596-607.

9. Subramanyam, D., Belair, C.D., Barry-Holson, K.Q., **Lin, H**., Kogan, S.C., Passegue, E. and Blelloch, R., 'PML-RAR{alpha} and Dnmt3a1 cooperate in vivo to promote acute promyelocytic leukemia'. ***Cancer Res*****,** 2010. 70 (21):8792-8801

10. **Lin, H**., Xu, H., Liang, F.Q., Liang, H., Gupta, P., Havey, A.N., Boulton, M.E. and Godley, B.F., 'Mitochondrial DNA damage and repair in RPE associated with aging and age-related macular degeneration'. ***Invest Ophthalmol Vis Sci*****.** 2011. 52(6):3521-9.

11. **Lin H.,** Qian JQ., Castillo A., Long B., Keyes KT., Chen G., and Ye Y.. Effect of miR-23 on oxidant-induced injury in human retinal pigment epithelium cells. ***Invest Ophthalmol Vis Sci*****.** 2011. 52(9):6308-14

12. Hou Y, **Lin H (co-equal first author),** Zhu L, Liu Z, Hu F, Shi J, Yang T, Shi X, Zhu M, Godley BF, Wang Q, Li Z, Zhao Y. Lipopolysaccharide increases the incidence of collagen-induced arthritis in mice through induction of protease HTRA-1 expression. ***Arthritis Rheum***. 2013 Nov;65(11):2835-46.

13. Weis B., Schmidt J., Maamar J., Raj A., **Lin H**, Tóth C., Kristina Riedmann K., Seitz K.,Ho a., Jaenisch R. and LinhartH. Inhibition of Intestinal Tumor Formation by Deletion of the DNA-Methyltransferase-3a in the $APC^{Min}$ Mouse Model. ***Oncogen (Nature series)*** 2014 May 19;0. doi: 10.1038/onc.2014.114.

14. Hou Y, **Lin H (co- first author)**, Zhu L, Liu Z, Hu F, Shi J, Yang T, Shi X, Guo H, Tan X, Zhang L, Wang Q, Li Z, Zhao Y. The Inhibitory Effect of IFN-γ on Protease HTRA1 Expression in Rheumatoid Arthritis. ***J Immunol***. 2014 Jun 6. pii: 1302700.

15. Matsumoto H., Murakami Y., Kataoka K., **Lin H**.,, Connor KM., Miller JW., Zhou D., Avruch J., Vavvas DG.. Mammalian STE20-like kinase 2, not kinase 1, mediates photoreceptor cell death during retinal detachment. ***Cell Death Dis.*** 2014 May 29;5:e1269. doi: 10.1038/cddis.2014.218.

16. Hu Y., **Lin H**. (**co- first author)**, Dib B., Atik A., Bouzika P., Lin C., Yan Y., Tang S., Miller JW and Vavvas DG. Cholesterol crystals induce inflammatory cytokines expression in ARPE-19 cells by activating the NF-κB pathway. ***Discov Med***. 2014 Jul-Aug;18(97):7-14.

17. Dib B, **Lin H** (**co- first author)**, Maidana DE, Tian B, Miller JB, Bouzika P, Miller JW, Vavvas DG. Mitochondrial DNA has a pro-inflammatory role in AMD**. *BBA Molecular Cell*** 2015 Nov;1853(11 Pt A):2897-906. doi: 10.1016/j.bbamcr.2015.08.012

18. Maidana DE., Tsoka P., Tian B., Dib D., Matsumoto H., Kataoka, K., **Lin H.,** Miller JW., and Vavvas DG.. A Novel ImageJ Macro for Automated Cell Death Quantitation in the Retina ***Invest Ophthalmol Vis Sci.*** 2015;56::10.1167/15-17599

19. **Lin H,** Yan Y, Maidana DE, Bouzika P, Atik A, Matsumoto H, Miller JW,. Vavvas DG.. Drug Delivery Nanoparticles: Toxicity Comparison in Retinal Pigment Epithelium and Retinal Vascular Endothelial Cells. ***Semin Ophthalmol***. 2016;31(1-2):1-9.

20. Tian B, Maidana DE, Dib B, Miller JB, Bouzika P, Miller JW, Vavvas DG, **Lin H.** miR-17-3p Exacerbates Oxidative Damage in Human Retinal Pigment Epithelial Cells. **PLoS One.** 2016 Aug 9;11(8):

21. **Lin H,** Ro[h] M, Matsumoto H, Atik A, Bouzika P, Alhatem A, Miller JW and.Vavvas DG. Blocking the Necroptosis Pathway Decreases RPE and Photoreceptor Damage Induced by $NaIO_3$ In revision ***Invest Ophthalmol Vis Sci***

22. Miller JB. **Lin H** (**co- first author)**, Atik A., Bouzika P., Yan Y., Gragoudas ES., Miller JW and Vavvas DG.. Iron Rescue of Deferoxamine Toxicity in human retinal pigment epithelium cells. In revision ***Invest Ophthalmol Vis Sci***

23. Tian B., Al-Moujahed A., Bouzika P., Hu Y., Notomi S, Tsoka P., Miller JW., **Lin H**. **(Co-correspond author)** and Demetrios G. Vavvas. Atorvastatin Promotes Phagocytosis and Attenuates Pro-Inflammatory Response in Human Retinal Pigment Epithelial Cells (submitted)

In preparation

24. Maidana DE, Dib B, Tian B, Miller JB, Miller JW, Vavvas DG and **Lin H (corresponding author).** MicroRNA-192 Mediates Glucose Induced Epithelial to Mesenchymal Transition in Retinal Pigment Epithelium.

25. Bouzik P, **Lin H (co- first author),** Atik A, Yan Y, Hu Y, Miller JW, Gradoudas GS and Vavvas DG. Verteporfin Without Photoactivation: Effect of the Photosensitizer on Ocular Cell Lines

26. Bouzik P, **Lin H (co- first author),** Atik A, Yan Y, Hu Y, Miller JW, Gradoudas GS and Vavvas DG. Effect of Verteporfin on different Cancer Cell Lines

27. Atik A, **Lin H (co- first author),** Bouzika P, Yan Y, Hu Y, Miller JW and Vavvas DG Sodium thiosulfate protects against cisplatin toxicity in ARPE-19 cells

**Invited Speaker in last 3 years**

| | | |
|---|---|---|
| 4/29/2018 | ARVO Honolulu, Hawaii | Cytosolic Accumulation of nDNA Fragments in RPE Cells and the Development of AMD |
| 2/23/2018 | University of Massachusetts Medical School | Cytosolic Accumulation of nDNA Fragments in RPE Cells and the Development of AMD |
| 4/28/2017 | Tianjin Medical University | Damaged Genomic DNA Accumulation in RPE cells and Development of AMD |
| 4/10/2017 | Changchun Tongren Eye Hospital | Damaged Genomic DNA Accumulation in RPE cells and Development of AMD |
| 4/19/2016 | Kellogg Eye Center, University of Michigan | Mitochondrial DNA damage and pro-inflammatory function in AMD |
| 12/17/2015 | Scheie Eye Institute University Of Pennsylvania | Mitochondrial DNA damage and pro-inflammatory function in AMD |
| 9/20/2019 | Shanzhen Eye Hospital | **Damaged nuclear DNA in RPE cells triggers inflammation, Senescence and VEGFreleasing in the development of AMD.** |
| 9/19/2019 | Shenzhan University | **Damaged nuclear DNA accumulates in retinal pigment epithelium cells of age-related** |

| | | |
|---|---|---|
| | | **macular degeneration patients.** |
| 9/18/2018, | Zhonshan Ophthalmic Center, Sun Yat-sen University | **Damaged nuclear DNA accumulates in retinal pigment epithelium cells of age-related macular degeneration patients.** |
| 9/22/2018 | Tianjin Eye Hospital, Tianjin Medical University, International Retina Day | **Damaged nuclear DNA in RPE cells triggers inflammation, Senescence and VEGFreleasing in the development of AMD.** |
| 9/13/2018 | Institute of Zoology, Chinese national academic of science | Damaged nuclear DNA accumulates in retinal pigment epithelium cells of age-related macular degeneration patients. |