BIOGRAPHICAL SKETCH

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NAME: Edward A. Fisher, MD, PhD, MPH

POSITION TITLE: Leon H. Charney Professor of Cardiovascular Medicine

eRA COMMONS USER NAME (credential, e.g., agency login): eafisher

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Harpur College, SUNY, Binghamton NY	BA	1971	Mathematics
New York Univ. Sch. of Med., NY, NY	MD	1975	Medicine
Univ. of North Carolina, Chapel Hill, NC	MPH	1978	Epidemiology
MIT, Cambridge, MA	PhD	1982	Biochem./Nutrition
University of Oxford	MA	2010	-

A. Personal Statement

The Fisher lab has long been interested in lipid/lipoprotein metabolism as well as in atherosclerosis and its regression and imaging. The laboratory uses a wide variety of biochemical, cell biology, genetic, and molecular biology techniques to better understand these processes. Among other accomplishments, we have discovered major degradation pathways that regulate VLDL production by the liver, have developed novel models of atherosclerosis regression and experimental techniques and systems biological approaches to analyze molecular changes in plaques, and have adapted HDL and other nanoparticles to deliver imaging and therapeutic agents to plaques.

B. Positions and Honors

Postgraduate training

1975-1977 Pediatric Resident, Duke Hospital, Durham, NC
1978-1981 Fellowship, Division GI/Nutrition, Children's Hospital, Boston, MA
1978-1981 Fellowship, Clinical Research Center, MIT, Cambridge, MA
1981-1984 Medical Staff Fellow (Lab. of Molecular Biol.), NIH, Bethesda, MD

Faculty appointments

1984-1987 Assistant Professor of Pediatrics, University of Pennsylvania, Philadelphia, PA
1987-1995 Assistant to Associate Professor of Biochemistry, Medicine, and Pediatrics, Medical College of Pennsylvania, Philadelphia, PA
1995-1999 Director of Lipoprotein Research, Cardiovascular Institute and Associate Professor of Medicine and Cell Biology, Mount Sinai School of Medicine, and Guest Investigator, Rockefeller University, NY, NY
1999- 2003 Same as above, but Professor of Medicine, Pediatrics, and Biochemistry
2003- Leon H. Charney Professor of Cardiovascular Medicine and Professor of Cell Biology, New York University School of Medicine; Adjunct faculty, Rockefeller University, NY, NY

Hospital and administrative appointments

2003-Dir., Vascular Biology and Disease Research Program; Dir., NYU Center for the Prevention of Cardiovascular Disease; both at NYU School of Medicine, NY, NY

Awards, honors, memberships

1968 Chemical Rubber Company Award in Chemistry
1969 Distinguished Independent Work in Mathematics
1996 President, NY Lipid Club
1998-2007 Editorial Board, Journal of Lipid Research
2002-2007 Editorial Board, Journal of Biological Chemistry

2003	Leon H. Charney Endowed Professorship in Cardiovascular Medicine, NYU School of Medicine
2005	Faculty inductee, Alpha Omega Alpha honor society, NYU School of Medicine chapter
2005	Elected member, Interurban Clinical Club
2005-	Editorial Board, Journal of Clinical Investigation
2005	Ruth Gray Memorial Lecture, Northwestern University/Evanston Hospital
2007	Solomon A. Berson Medical Alumni Achievement Award in Basic Science, NYU School of Medicine
2007-	Editor, ATVB; Editor-in-Chief, 2011-2012
2007	ACC/Pfizer Visiting Professor in Preventive Cardiology (served at University of Virginia)
2008	Special Recognition Award in the Field of Arteriosclerosis, ATVB Council/American Heart Association
2009	Elected to the American Association of Physicians
2010-2011	George Eastman Visiting Professor, Oxford University
2011	Chair, Gordon Research Conference on Atherosclerosis
2012	Elected to the Association of University Cardiologists
2012	Chair, Keystone Symposium on Molecular Basis of Vascular Inflammation and Atherosclerosis
2013	George Lyman Duff Lecture award, American Heart Association
2016	National Lipid Association Award for "Remarkable accomplishments in lipidology"

C. Contributions to Science

Complete list of published work in My Bibliography (~250 papers):
http://www.ncbi.nlm.nih.gov/sites/myncbi/edward.fisher.1/bibliograpahy/40847556/public/?sort=date&direction=descending

1) Regulation of VLDL and apolipoprotein B100 (apoB) production: Unlike other secretory proteins, apoB abundance is regulated not by its level of synthesis, but by its degradation. I and my collaborators have been the first to discover 3 main pathways that accomplish apoB degradation. The earliest begins at the cell surface (a re-uptake process) and is regulated by LDL receptor activity (Williams K et al., JBC 1997). The nascent lipoprotein particles are then directed to the lysosome for degradation:

Identification of the next degradative process came from studies in which lipid transfer in the ER to the translocating apoB polypeptide is insufficient. ApoB begins to misfold and becomes targeted to the ER-associated degradation pathway, and ultimately, to the cytosolic ubiquitin-proteasome machinery. Our report was the first example of a wild-type mammalian protein subject to this type of quality control, and the paper has had a significant impact on both the lipoprotein and the cell biology communities:

I. **Fisher, E.A.**, Zhou, M., Mitchell, D.M., Wu, X., Omura, S., Wang, H., Goldberg A.L., and Ginsberg, H.N. (1997). The degradation of apolipoprotein B100 is mediated by the ubiquitin-proteasome pathway and involves heat shock protein (HSP) 70. J. Biol. Chem. 272:20427-20434.

We went on to explore in more mechanistic detail the roles of HSP70, as well as other chaperone molecules, in apoB degradation by ERAD, and some of the seminal results are found in this review:
II. Brodsky, JL, and **Fisher, EA**. (2008). The many intersecting pathways underlying apolipoprotein B secretion and degradation. Trends Endocrinol Metab. Sep;19(7):254-9. PMCID: PMC3216472

In addition to apoB-ERAD during lipid insuffiency, we also were studying the mechanistic basis of the reduction in plasma levels of VLDL in mammals (including man) consuming diets rich in fish oil fatty acids EPA and DHA. We discovered an apoB degradative process regulated by lipid peroxides generated from EPA and DHA. This work showed that not all oxidant stress was necessarily harmful in the liver: essentially, by modulating the "oxidant tone", the degradation of apoB and its availability for VLDL assembly/section could be adjusted:
III. Pan, M, Cederbaum, AI, Zhang, YL, Ginsberg, HN, Williams, KJ, and **Fisher, EA**. (2004). Lipid peroxidation and oxidant stress regulate hepatic apolipoprotein B degradation and VLDL production. J. Clin. Invest. 113:1277-87. (with commentary) PMCID: PMC398425

We subsequently found that autophagy was the responsible degradative process:
IV. Meihui Pan, Vatsala Maitin, Sajesh Parathath, Ursula Andreo, Sharron X. Lin, Carly St. Germain, Zemin Yao, Frederick R. Maxfield, Kevin Jon Williams, and Edward A. Fisher. (2008). Pre-secretory oxidation, aggregation, and autophagosomal destruction of apolipoprotein-B: a pathway for late-stage quality control. Proc. Natl. Acad. Sci. USA 105:5862-7. PMCID: PMC2311371

Thus, in addition to the quality control in the ER, there is also a surveillance system in the Golgi, and perhaps other post-ER compartments, that serves to prevent the forward movement in the secretory pathway of products that not correctly processed. There is a growing list of other clinically relevant metabolic/genetic factors (including insulin and sortilin) in which autophagy is a regulator of VLDL/apoB secretion.

2) The regression of atherosclerosis and its molecular analyses: Progression of this disease has been the major focus of most labs. Regression was underserved not only scientifically, but also strategically, given that the majority of adults coming for risk reduction therapies already have significant plaque burdens. I set out to develop mouse models that would reproducibly and potently manifest plaque regression. In 2001 we introduced an aortic transplantation model, which we quickly applied to study whether a selective increase in HDL could regress plaques. Indeed it did, providing strong and direct evidence cited in the current debates about the importance of HDL as an atheroprotective agent:
I. James X. Rong, Jie Li, Ernane D. Reis, Robin P Choudhury, Hayes M. Dansky, Valerie Elmalem, John T. Fallon, Jan L. Breslow, **Edward A. Fisher**. (2001). Remodeling of advanced atherosclerotic lesions in apolipoprotein E-deficient mice by elevation of HDL cholesterol levels. Circulation 104:2447-2452. (with commentary)

We have since used the transplant model to be the first to show that macrophages could emigrate from plaques, that both forms of LXRs are required for regression, and that during regression, the inflammatory state of plaque macrophages is highly responsive to changes in the microenvironment, as revealed by the enrichment in macrophages with features of alternatively-active M2 cells (publications in PNAS, JCI, Circulation, et al.). Examples of many of these features are found in another study of HDL:
II. Feig JE, Rong JX, Shamir R, Sanson M, Vengrenyuk Y, Liu J, Rayner K, Moore K, Garabedian M, **Fisher EA**. (2011). HDL promotes rapid atherosclerosis regression in mice and alters inflammatory properties of plaque monocyte-derived cells. Proc. Natl. Acad. Sci. U S A. 108(17):7166-71. PMCID: PMC3084076.

In this and other studies, analytical power was significantly increased by molecular assays made possible by our introducing into the atherosclerosis field the use of laser capture microdissection (LCM) to isolate plaque cells of any type or location:
III. E. Trogan, R. Choudhury, H. Dansky, J.L. Breslow, and **E.A. Fisher**. (2002). Laser capture microdissection analysis of gene expression in macrophage cells from atherosclerotic lesions of apolipoprotein E-deficient mice. Proc. Natl. Acad. Sci. USA 99:2234-2239. PMCID: PMC122348

We also showed that state-of-the art transcriptome profiling/systems biological analyses to discover regression-related factors could be accomplished with RNA isolated by LCM:
IV. Ramsey SA, Vengrenyuk Y, Menon P, Podolsky I, Feig JE, Aderem A, ***Fisher EA**, *Gold ES (co-corresponding authors). (2014). Epigenome-guided analysis of the transcriptome of plaque macrophages during atherosclerosis regression reveals activation of the Wnt signaling pathway. PLoS Genet. 10(12):e1004828. PMCID: PMC4256277

We also developed other, non-surgical, models of plaque regression in collaboration with a number of colleagues/collaborators, including Steve Young (UCLA; the Reversa model; Circulation 2011), With Kathryn Moore (NYU; anti-miR33 treatment; JCI 2010), Jonathan Smith and Stan Hazen (Cleveland Clinic; HDL infusion; ATVB 2014). We have applied these and other models to clinically relevant topics in cardiovascular disease; e.g., with Ira Goldberg (NYU) we showed that, as in patients, diabetes attenuates the benefits of aggressive lipid lowering (Diabetes 2011; Cell Metab. 2013), and in recent studies with Kathryn Moore, that this can be overcome with anti-miR33 therapy (Circ. Res. 2011).

3) Imaging and therapy of atherosclerosis by HDL carriers of imaging and therapeutic agents: In 1998 I collaborated with Zahi Fayad (Mount Sinai) and published the first MRI images of mouse atherosclerosis

(Circulation 1998). I then developed an approach in which MRI imaging enhancing agents could be loaded on to HDL particles. These particles readily entered plaques, where they dramatically improved the signal to noise ratio to reveal even structural features:
I. Frias JC, Ma Y, Williams KJ, *Fayad ZA, ***Fisher EA** (co-corresponding authors). (2006). Properties of a versatile nanoparticle platform contrast agent to image and characterize atherosclerotic plaques by magnetic resonance imaging. Nano Lett. 6(10):2220-4. (cover article)

These efforts have expanded to show that HDL cannot only carry imaging agents for MR, but also for CT and PET. Some of the advances are summarized in review articles, one of which is cited below:
II. Skajaa T, Cormode DP, Falk E, Mulder WJ, **Fisher EA**, Fayad ZA. (2010). High-Density Lipoprotein–Based Contrast Agents for Multimodal Imaging of Atherosclerosis. Arterioscler Thromb Vasc Biol. 30(2):169-76. PMCID: PMC2826843

By incorporating "targeting" molecules on the HDL, we can further resolve plaque compositional changes, as demonstrated in our Reversa model:
III. Chen W, Cormode DP, Vengrenyuk Y, Herranz B, Feig JE, Klink A, Mulder WJ, ***Fisher EA**, *Fayad ZA (co-corresponding authors). (2013). Collagen-specific peptide conjugated HDL nanoparticles as MRI contrast agent to evaluate compositional changes in atherosclerotic plaque regression. JACC Cardiovasc Imaging. 6(3):373-84. PMCID: PMC3653172

Current efforts are also directed at delivering therapeutic agents to plaques:
IV. Duivenvoorden R, Tang J, Cormode DP, Mieszawska AJ, Izquierdo-Garcia D, Ozcan C, Otten MJ, Zaidi N, Lobatto ME, van Rijs SM, Priem B, Kuan EL, Martel C, Hewing B, Sager H, Nahrendorf M, Randolph GJ, Stroes ES, Fuster V, **Fisher EA**, Fayad ZA, Mulder WJ. (2014). A statin-loaded reconstituted high-density lipoprotein nanoparticle inhibits atherosclerotic plaque inflammation. Nat Commun. 5:3065. PMCID: PMC4001802

D. Research Support

Ongoing Research Support:

NHLBI R01HL127930-01 PI: E. Fisher 12/1/2015 – 11/30/2019
Molecular Regulation of Apoprotein B Degradation
This project focuses on the cell biology of VLDL assembly and secretion as regulated by the degradation of apolipoprotein B.

NIDDK R01DK095684 E. Fisher, I. Goldberg, PIs 4/01/2012 – 3/31/2017
Diabetes-mediated effects on myeloid precursors and vascular complications
The focus of this project is to identify the mechanisms by which diabetes impairs atherosclerosis regression.

NHLBI R01HL117226 E. Fisher, M. Garabedian, PIs 12/23/2013 – 11/30/2017
Regulation of LXR alpha by glucose and cholesterol in diabetes and atherosclerosis
This project supports studies on the effects of post-translational modifications of LXR alpha on the regulation of gene expression occurring in macrophages under conditions of hyperlipidemia and hyperglycemia.

NIBIB R01EB009638-09A1 PI: Z. Fayad; E. Fisher, W. Mulder, co-Is 8/1/2013 – 7/31/2017
Theranostic HDL nanoparticles for inflammatory macrophages in atherosclerosis
The overall goal is to use nanoparticles to image mouse atherosclerotic plaques. Dr. Fisher's aims are to exploit the properties of HDL to deliver imaging agents to plaques and develop HDL as a molecular imaging tool.

NHLBI R01HL112793 J. Miano, E. Fisher, PIs 8/1/2013 – 3/31/2017
Regulation and function of AKAP12A in the vessel wall
The aim of this grant is to investigate the role of AKAP12A in vascular smooth muscle cell differentiation.

NHLBI P01HL092969 PI: K. Bornfeldt 4/01/2015 – 3/31/2020
Cardiovascular Disease and Diabetes Program Project
Project 2: Dyslipidemia and Atherosclerosis Regression (Ira Goldberg, E. Fisher, PIs)
The effects of elevated plasma triglycerides on the regression of atherosclerosis when LDL cholesterol is lowered will be tested using a variety of mouse models.

NHLBI R01HL130630 E. Fisher, A. Rodriguez, S. Wassmer, PIs 9/15/2015 – 5/31/2019
Beta-catenin signaling in endothelial cells during cerebral malaria
The goal of this grant is to study the Wnt signaling pathway in endothelial cells and how this relates to cerebral malaria.

NHLBI R01HL084312 E. Fisher, P. Loke, K. Moore, PIs 4/1/2010 – 03/31/2020
Molecular regulation of atherosclerosis regression
The project supports studies on the molecular events occurring in foam cells in plaques undergoing regression in novel mouse models we and our collaborators have developed.

NHLBI R01HL129433 E. Fisher, K. Moore, PIs 4/1/2016-3/31/2020
HDL and atherosclerosis regression
This project supports studies on the functional characteristics of HDL needed to regress atherosclerosis and the underlying molecular mechanisms.

Completed Research Support

NHLBI P01HL098055-05 S. Hazen, PI 9/8/2010 – 5/31/2016
HDL AND ATHEROSCLEROSIS REGRESSION (Project 2; E. Fisher, PI)
Subcontract CLEVELAND CLINIC LERNER
Investigation into the molecular pathways by which HDL promotes the regression of atherosclerosis in mouse models.

NHLBI P01HL098055 S. Hazen, PI 9/8/2010 – 3/31/2016
REGRESSION OF ATHEROSCLEROSIS CORE (Core C; E. Fisher, PI)
Subcontract CLEVELAND CLINIC LERNER COM-CWRU
Investigation into the molecular pathways by which HDL promotes the regression of atherosclerosis in mouse models.

NHLBI R24OD018339 01 D. Levy, PI, E. Fisher, Co-I 2/1/2013 – 2/28/2016
Restoring Animal Research Resources Lost Due to Super Storm Sandy
This is a Hurricane Sandy supplement that covers losses related to R01EB009638 and HHSN268201000045 in nanoparticle research that were not covered by the Sandy supplements to the parent grants.

NHLBI P01HL098055-04S1 S. Hazen, PI 12/1/2013 – 11/30/2015
HDL AND ATHEROSCLEROSIS REGRESSION – Hurricane Sandy Supplement (Project 2 – Fisher)
Subcontract CLEVELAND CLINIC LERNER
This Hurricane Sandy supplement funded studies on the molecular pathways by which HDL promotes the regression of atherosclerosis in mouse models.

NIH HHSN2682010000045C Z. Fayad (PI); E. Fisher, W. Mulder (Co-Is) 8/13/10 – 7/31/15
Program of Excellence in Nanotechnology: Translational Nanomedical Therapies for Cardiac and Vascular Diseases
This project supports efforts in Dr. Fisher's lab to test the potential for nanoparticles to deliver LXR agonists specifically to atherosclerotic plaques in order to avoid systemic exposure.

NHLBI R01 HL58541 E. Fisher, PI 7/15/97 – 11/30/14
Molecular regulation of apoprotein B degradation
This project focuses on the regulation of the regulation of apoB lipoprotein assembly and secretion by the ubiquitin-proteasome pathway and other degradative mechanisms.

PUBLICATIONS

A. Peer-reviewed original investigations:

1. Smith, A.B. and Fisher, E. Statistical assessment of chemical effects on human reproduction. New Zealand Statistician 15: 12-16, 1980.

2. Fisher, E. and Gahl, W. Cysteamine in treatment of type III hyperlipidemia. Lancet II 8308: 1131-1132, 1982.

3. Fisher, E., Blum, C., Zannis, V. and Breslow, J. Independent effects of dietary saturated fat and cholesterol on plasma lipids, lipoproteins, and apolipoprotein E. J. Lipid Res. 24: 1039-1048, 1983.

4. Gahl, W.A., Gregg, R.E., Hoeg, J.M. and Fisher, E. In vivo alteration of a mutant human protein using the free thiol cysteamine. Am. J. Med. Genet. 20: 409-417, 1985.

5. Fisher, E. and Felsenfeld, G. Comparison of the folding of ß-globin and ovalbumin gene containing chromatin isolated from chicken oviduct and erythrocytes. Biochemistry 25: 8010-8016, 1986.

6. Fisher, E., Carroll, R., Cortner, J., and Surrey, S. Transcriptional activity of the genes for apoproteins A-I and E in the neonatal rat liver. Atherosclerosis 68: 249-253, 1987.

7. Fisher, E., Jung, A., and Cortner, J. Artifacts in RFLP analyses of human DNA samples co-precipitated with t-RNA. Clinical Chemistry 33: 2304-2305, 1987.

8. Fisher, E., Anbari, A., Klurfeld, D., and Kritchevsky, D. Independent Effects of Diet and Nutritional Status on Apoprotein B Gene Expression in Rabbit. Arteriosclerosis 8, 797-803, 1988.

9. Kritchevsky, D., Tepper, S.A., Davidson, L.M., Fisher, E., and Klurfeld, D.M. Experimental atherosclerosis: interaction of animal and vegetable protein with saturated or unsaturated fat. Atherosclerosis 75: 123-127, 1989.

10. Fisher, E. Change in chromatin organization of the 3'-flanking region of the rat apoprotein E gene in neonatal rats after an increase in transcriptional activity. Atherosclerosis 76: 29-33, 1989.

12. Wong, S.H., Fisher, E.A., and Marsh, J.B. Comparative effects of eicosapentaenoic and docosahexaenoic acids on apoB messenger RNA and secretion of newly synthesized VLDL in HepG2 cells. Arteriosclerosis 9: 836-841, 1989.

13. Zolfaghari, R., Harrison, E.H., Ross, A.C., and Fisher, E.A. Expression in Xenopus oocytes of rat liver mRNA coding for bile salt-dependent cholesteryl ester hydrolase. Proc. Natl. Acad. Sci. USA 86: 6913-6916, 1989.

14. Williams, K.J., Brocia, R.W., and Fisher, E.A. The unstirred water layer as a site of control of apolipoprotein B secretion. J. Biol. Chem. 265: 16741-16744, 1990.

15. Bass, J., Fisher, E.A., Prack, M.M., Williams, D.L., and Marsh, J.B. Macrophages from nephrotic rats regulate apolipoprotein E biosynthesis and cholesterol content independently. J. Clin. Invest. 87: 470-475, 1991.

16. Sparks, J.D., Zolfaghari, R., Sparks, C.E., Smith, H., and Fisher, E.A. Impaired hepatic apolipoprotein B and E translation in streptozotocin diabetic rats. J. Clin. Invest. 89: 1418-1430, 1992.

17. Zolfaghari, R., Harrison, E.H., Han, J.H., Rutter, W.J., and Fisher, E.A. Tissue and species differences in bile salt-dependent cholesteryl esterase activity and gene expression. Arteriosclerosis and Thrombosis 12: 295-301, 1992.

18. Aalto-Setala, K., Fisher, E.A., Chen, X., Chajek-Shaul, T., Hayek, T., Zechner, R., Walsh, A., Ramakrishnan, R., Ginsberg, H.N., and Breslow, J.L. Mechanism of hypertriglyceridemia in human apolipoprotein(apo)CIII transgenic mice: Diminished VLDL fractional catabolic rate associated with increased apo CIII and reduced apo E on the particles. J. Clin. Invest. 90: 11889-11900, 1992.

19. Furth, E., Sprecher, H., Fisher, E.A., Fleishman, H.D., and Laposata, M. An in vitro model for essential fatty acid deficiency: HepG2 cells permanently maintained in lipid-free medium. J. Lipid Res. 33: 1719-1726, 1992.

20. Wang, H., Chen, X., and Fisher, E.A. N-3 fatty acids stimulate intracellular degradation of apoprotein B in rat hepatocytes. J. Clin. Invest. 91: 1380-1389, 1993.

21. Zolfaghari, R., Glick, J.M., and Fisher, E.A. The effects of varying the expression of a neutral cholesteryl ester hydrolase on the turnover of cholesteryl ester in rat hepatoma cells. J. Biol. Chem. 268: 13532-13538, 1993.

22. Zolfaghari, R., Chen, X., and Fisher, E.A. Simple method for extracting RNA from cultured cells and tissue with guanidine salts. Clin. Chem. 39: 1408-1411, 1993.

23. Chen, X., Sparks, J.D., Yao, Z., and Fisher, E.A. Hepatic polysomes that contain apoprotein B mRNA have unusual physical properties. J. Biol. Chem. 268: 21007-21013, 1993.

24. Reisher, S., Fisher, E.A., and Feinstein, S.I. Rabbit and rat liver nuclei both contain proteins which bind to the regions controlling apolipoprotein A-I gene expression. Eur. J. Biochem. 216: 247-253, 1993.

25. Wang, H., Yao, Z., and Fisher, E.A. The effects of n-3 fatty acids on the secretion of carboxy-truncated forms of human apoprotein B. J. Biol. Chem. 269: 18514-18520, 1994.

26. Heinemann, T., Metzger, S., Fisher, E.A., Breslow, J.L., and Huang, L. Alternative polyadenylation of apolipoprotein B RNA is a major cause of B-48 protein formation in rat hepatoma cell lines transfected with human apoB-100 minigenes. J. Lipid Res. 35:2200-2211, 1994.

27. Morlock-Fitzpatrick, K.R., and Fisher, E.A. The effects of O- and N-linked glycosylation on the secretion and bile salt-stimulation of pancreatic carboxy ester lipase activity. Proc. Soc. Exp. Biol. Med. (P.S.E.M.) 208:186-190, 1995.

28. Shamir, R., Johnson, W.J., Zolfaghari, R., Lee, H.S., and Fisher, E.A. Role of bile salt-dependent cholesteryl ester hydrolase in the uptake of micellar cholesterol by intestinal cells. Biochemistry 34:6351-6358,1995.

29. Burkey, B.F., France, D., Wang, H., Ma, X., Nottage, B., Kowal, C., Diffenderfer, M., Marsh, J.B., Paterniti, J.R., and Fisher, E.A. Overexpression of human apolipoprotein A-I in transgenic rats and the hyperlipoproteinemia associated with experimental nephrosis. J. Lipid Res.36:1463-1473, 1995.

30. Azrolan, N., Odaka, H., Breslow, J.L., and Fisher, E.A. Dietary fat elevates hepatic apoA-I production by increasing the fraction of apolipoprotein A-1 mRNA in the translating pool. J. Biol. Chem. 270:19833-19838, 1995.

31. Shamir, R., Johnson, W.J., Morlock-Fitzpatrick, K., Zolfaghari, R., Li, L., Mas, E., Lombardo, D., Morel, D.W., and Fisher, E.A. Pancreatic carboxyl ester lipase: A circulating enzyme that modifies normal and oxidized lipoproteins in vitro. J.Clin.Invest. 97:1696-1704, 1996.

32. Marsh, J.B., Diffenderfer, M.R., Fisher, E.A., Sowden, M., Dong, M., Paterniti, J.R., and Burkey, B. F. Effect of experimental nephrosis on hepatic lipoprotein secretion and urinary lipoprotein excretion in rats expressing the human apolipoprotein A-I gene. J. Lipid Res. 37:1113-1124, 1996.

33. Werth, V.P., Williams, K.J., Fisher, E.A., Bashir, M., Rosenbloom, J., and Shi, X. UVB irradiation alters cellular responses to cytokines: role in extracellular matrix gene expression. J. Invest. Derm. 108:290-294, 1997.

34. Chen, X., Harrison, E.H., and Fisher, E.A., Molecular cloning of the cDNA for rat hepatic, bile salt-dependent cholesteryl ester hydrolase demonstrates identity with pancreatic carboxylester lipase. Proc. Soc. Exp. Biol. Med. (P.S.E.M.) 215:186-191, 1997.

35. Fisher, E.A., Zhou, M., Mitchell, D.M., Wu, X., Omura, S., Wang, H., Goldberg A.L., and Ginsberg, H.N. The degradation of apolipoprotein B100 is mediated by the ubiquitin-proteasome pathway and involves heat shock protein 70. J. Biol. Chem. 272:20427-20434, 1997.

36. Zolfaghari, R., Shamir, R., Fisher, E.A. Properties of pancreatic carboxyl ester lipase in mRNA-injected Xenopus oocytes and transfected mammalian hepatic and intestinal cells. Methods in Enzymology 286:80-101, 1997.

37. Fuki, I., Kuhn, K.M., Lomazov, I.R., Rothman, V.L., Tuszynski, G.P., Iozzo, R.V., Swenson, T.L., Fisher, E.A., and Williams, K.J The Syndecan Family of Proteoglycans: Novel Receptors Mediating Internalization of Atherogenic Lipoproteins In Vitro. J. Clin. Invest. 100:1611-1622, 1997.

38. Bruneau, N., Nganga, A., Fisher, E.A., and Lombardo, D. *O*-glycosylation of C-terminal tandem-repeated sequences regulates the secretion of rat pancreatic bile-salt dependent lipase. J. Biol. Chem. 272:27353-27361, 1997.

39. Zaiou, M., Azrolan, N., Hayek, T., Wang, H., Wu, L., Haghpassand, M., Cizman, B., Madaio, M.P., Milbrandt, J., Marsh, J., Breslow, J.L., and Fisher, E.A. The full induction of human apoprotein A-I gene expression by the experimental nephrotic syndrome in transgenic mice depends on cis-acting elements in the proximal 256 base-pair promoter region and the trans-acting factor early growth response factor 1. J. Clin. Invest. 101:1699-1707, 1998.

40. Fayad, Z.A., Fallon, J.T., Wehrli, S., Dansky, H., Poon, M., Badimon, J., Charlton, S., Fisher, E.A., Breslow, J.L., and Fuster, V. Noninvasive in vivo

high-resolution imaging of atherosclerotic lesions in genetically engineered mice. Circulation 98:1541-1547, 1998.

41. Zhou, M., Fisher E.A., and Ginsberg, H.N. Regulated co-translational ubiquitination of apolipoprotein B100. J. Biol. Chem. 273:24649-24653, 1998.

42. Mitchell, D.M., Zhou, M., Pariyarath, R., Wang, H., Aitchison, J., Ginsberg, H.N., and Fisher, E.A. Apoprotein B100 has a prolonged interaction with the translocon during which its lipidation and translocation change from dependence on the microsomal triglyceride transfer protein to independence. Proc. Natl. Acad. Sci. USA 95:14733-14738, 1998.

43. Weng, W., Li, L., van Bennekum, A.M., Potter, S.H., Harrison, E.H., Blaner, W.S., Breslow, J.L., and Fisher, E.A. Intestinal absorption of dietary cholesteryl ester is decreased but retinyl ester absorption is normal in carboxyl ester lipase knockout mice. Biochemistry 38:4143-4149,1999.

44. Van Bennekum, A.M., Li, L., Piantedosi, R., Shamir, R., Vogel, S., Fisher, E.A., Blaner, W.S., and Harrison, E.H. Carboxyl ester lipase overexpression in rat hepatoma cells and CEL deficiency in mice have no impact on hepatic uptake or metabolism of chylomicron-retinyl ester. Biochemistry 38:4150-4156, 1999.

45. Van Bennekum, A.M., Fisher, E.A., Blaner, W.S., and Harrison, E.H. Hydrolysis of retinyl esters by pancreatic triglyceride lipase. Biochemistry 39:4900-4906, 2000.

46. Pan, M., Liang, J., Fisher, E.A., and Ginsberg, H.N. Inhibition of translocation of nascent apolipoprotein B across the endoplasmic reticulum membrane is associated with selective inhibition of the synthesis of apolipoprotein B. J. Biol. Chem. 275:27399-27405, 2000.

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