# Curriculum Vitae

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| **Name** | Professor Asif Ahmed | |
| **Office Address** | School of Life & Health Sciences<br>Aston University<br>Aston Triangle<br>Birmingham<br>B4 7ET, UK<br><br>Tel: +44 121 204 4967<br>E-mail: asif.ahmed@aston.ac.uk | |
| **Present Position** | Pro-Vice Chancellor (Health)<br>Aston University | 1st Oct 2012 – |
| | Chair of Vascular Biology<br>Aston University (HEFCE) | 1st Oct 2012 – |
| **Previous Position** | Assistant Principal International<br>Post-Doctoral Training, University of Edinburgh | 2010 – 2012 |
| | Gustav Born Chair of Vascular Biology<br>University of Edinburgh | 2010 – 2012 |
| | Professor of Reproductive Physiology<br>University of Birmingham (HEFCE) | 1998 – 2010 |
| | Senior Lecturer (UoB) | 1996 – 1998 |
| | Lecturer (UoB) | 1993 – 1996 |
| **Post-doctoral Training** | University of Cambridge | 1989 – 1993 |
| **Postgraduate** | University College, University of London<br>PhD Thesis: *Blood Platelets and Antithrombin III Studies during Cardiopulmonary Bypass* | 1986 – 1989 |
| **Undergraduate** | King's College, University of London<br>Degree: Pharmacology, BSc (Hons) | 1980 – 1983 |
| **Schools** | Southgate College, London N14<br>Aylward School, London N18 | 1978 – 1980<br>1971 – 1977 |
| **Fellowship** | Fellow of the Molecular Medicine Society<br>Founder Member of EVBO | 1998 –<br>2006 – |
| **Editorial Board** | Laboratory Investigation<br>Vascular Cell<br>ISRN Vascular Medicine | 1998 – 2007<br>2010 –<br>2011 – |
| **Visiting Professor** | Stanford University School of Medicine | 2009 – 2011 |
| **External Committees** | Member of MRC PSMB Board (2008 – 2010)<br>Italian Cancer Research (AIRC) Preferred Reviewer<br>Member of R & D Committee, Women's NHS Trust | |
| **External Assessor** | For Reader and Chair promotion at University of Bristol, University of Cambridge, University of Leicester, Imperial College London, Harvard Medical School, Hull York Medical School, Stanford University | |

**External Examiner** University of Cambridge, University of London, University of Nottingham, University of Sheffield, University of Ulster, Free University, University of Amsterdam, University of Singapore, Open University, University of Milan and other international universities

**Patents**

(i) International Application Number: PCT/GB95/02801
International Publication Number: WO 96/1662

(ii) International Application Number: PCT/GB95/01301
International Publication Number: WO 95/33454
United States Patent Number: 5,981,470

**Impact** Function and regulation of growth factors receptors and second messengers in angiogenesis and endothelial dysfunction. My laboratory has been at the forefront of heme oxygenase (Hmox) in Pregnancy (Acevedo and Ahmed, 1998; Ahmed, 2011). In 2000, our discovery that placental Hmox activity is protective against oxidant injury and inflammatory responses led to major research focus on endogenous protective enzyme in preeclampsia. In 2004, we identified soluble Flt-1 as the single most important molecule responsible for angiogenic imbalance in preeclampsia by demonstrating that its removal restored angiogenic balance in preeclamptic samples (Ahmad and Ahmed, 2004). My laboratory subsequently went on to identify Hmox1 and its gaseous by-product, carbon monoxide (CO), as endogenous inhibitors of soluble Flt-1 and soluble endoglin, the natural anti-angiogenic factors that are responsible for the major clinical signs of preeclampsia. Thus, increasing HO activity should provide protection against preeclampsia. As statins up-regulate Hmox1, this is the scientific rationale for undertaking the World's first RCT on pravastain in pregnancy, StAmP Trial (Statins to Ameliorate early onset Pre-eclampsia). Positive outcome of this study will lead to complete change in the clinical management of this condition and save many hundreds of thousands of lives (see Review Ahmed A, Thromb Res. 2011 Feb;127S3:S72-S75).

**Major Grants as Principal Investigator**

MRC (Programme Grant) The Role of Endogenous Vascular Protection Factors in Preeclampsia. Total FEC: £1,619,738.

MRC (Strategic grant) Angiogenic Biomarkers as Predictive Tests for Early Onset Preeclampsia: A Population-Based Study. Total FEC: £2,008,404.

MRC (Strategic Grant). A proof of principle randomised placebo-controlled trial to ameliorate early onset preeclampsia. (Shortlisted under the IES Platform Call.) Total FEC: £499,753.

BHF (Programme Grant). Molecular Mechanisms of Maternal Vascular Homeostasis. £481,638.

**Symposium Organiser**

28th May – 1st June 2012 Chair and Organiser of the 7th International Congress on Heme oxygenase, John McIntyre Conference Centre, Edinburgh.

30th Sept – 4th Oct 2009 Chair of Heme oxygenases: Role in Physiological Processes Beyond Heme Degradation and a member of the scientific committee at 6th International Conference Heme Oxygenase, Miami, Florida.

21st – 25th Sept 2008 Co-chair of Predictors of Preeclampsia & Angiogenesis at 16th World Congress ISSHP

**Conference Invited Speaker**

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| 8th International Workshop on Reproductive Immunology | 2012 |
| 36th Eastern Canadian Perinatal Investigators Meeting | 2012 |
| 18th ISSHP World Meeting, Geneva | 2012 |
| First European Conference on the Biology of Hydrogen Sulfide | 2012 |
| 6th European Meeting for Vascular Biology & Medicine | 2011 |
| Gordon Research Conference on Vascular Biology | 2010 |
| 6th International Conference Heme Oxygenase, Miami, Florida | 2009 |
| Biochemical Society - Molecular and Cellular Mechanisms | 2009 |
| 16th ISSHIP World Congress, Washington | 2008 |
| Trans-Institute Angiogenesis Research Program, NIH, Washington DC | 2007 |
| 5th International Conference Heme Oxygenase, Krakow, Poland | 2007 |
| Tox Talx 8, Ralston Retreat in Mill Valley, California | 2007 |
| AngioNET, Belfast | 2007 |
| The Macdonald UK Obstetric Medicine Society | 2007 |
| Mayo Clinic Angiogenesis Symposium | 2006 |
| 15th ISSHIP World Congress, Lisbon | 2006 |
| The Physiology Society, University College London | 2006 |

**International Collaborators**

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| 1. | **Institution** | University of Florida, Gainesville |
| | **Collaborator** | Professor Mike Boulton (Joint Wellcome Trust Grant & Publications) |
| 2. | **Institution** | Duke University Medical Center, Durham |
| | **Collaborator** | Dr Christopher D Kontos (Joint Grants & Publications) |
| 3. | **Institution** | UT Houston Medical School, Houston |
| | **Collaborator** | Dr Yang Xia (Joint Publications) |
| 4. | **Institution** | The GBF, Braunschweig |
| | **Collaborator** | Dr Herbert A Weich (Joint Publications) |
| 5. | **Institution** | Yale School of Medicine, New Haven |
| | **Collaborator** | Dr Irina A Buhimschi (Joint Grant & Publications) |
| 6. | **Institution** | Mayo Clinic College of Medicine, Rochester |
| | **Collaborator** | Professor Debabrata Mukhopadhyay (Joint Publications) |
| 7. | **Institution** | Max Delbrueck Center for Molecular Medicine, Berlin |
| | **Collaborator** | Dr Ferdinand le Noble (Joint Publications) |
| 8. | **Institution** | Cardiovasculaire Inserm Lariboisiere, Inserm U689, Paris |
| | **Collaborator** | Dr Stephanie Lehoux (Joint grant – www.EVGN.org) |
| 9. | **Institution** | University of Barcelona, Maternal-Fetal Medicine, Barcelona |
| | **Collaborator** | Professor Eduard Gratacós (M Curie Programme & Publications) |
| 10. | **Institution** | VIB, Institute for Biotechnology, Leuven |
| | **Collaborator** | Professor Peter Carmeliet (joint publication) |
| 11. | **Institution** | Beth Israel Deaconess Medical Center, Boston |
| | **Collaborator** | Dr Leo Otterbein (joint publication) |
| 12. | **Institution** | 1st Faculty of Medicine, Charles University, Prague |
| | **Collaborator** | Professor Libor Vítek |

**Contribution to the field of Reproductive and Vascular Biology**

1. My early research identified heparin as the culprit for post-operative bleeding following bypass surgery and provided a solution by demonstrating that slow infusion of protamine sulphate eliminated endothelial-bound heparin as it returned into the circulation which reduced post-operative blood loss (*J Cardiovasc Surg* 27:600-3).

2. I showed that phospholipase D could be activated in the absence of phospholipase C activity. This was the first study to show that a growth factor (basic FGF) could activate phospholipase D in endothelial cells and this was independent of inositol-lipid hydrolysis (*Am J Physiol* 266: C206-C212, 1994). Subsequently, we showed that VEGF also stimulated phospholipase D but this was dependent upon phospholipase C activity (*Lab Invest*. 75:427-37, 1996).

3. My group was the first to publish the expression and regulation of angiotensin II receptor subtype in human endometrium and the identification of a novel high-affinity angiotensin II binding site, which is now referred to as an atypical receptor (*J Clin Invest* 96: 848-857, 1995).

4. The first evidence of an autocrine loop for VEGF and its receptor. We showed that VEGF and its receptor were expressed on trophoblast and VEGF stimulated parathyroid hormone-related protein (PTHrP) release from trophoblast (*Growth Factors* 12:235-43, 1995).

5. The first publication to demonstrate that VEGF Receptor-1 negatively regulated VEGFR-2 mediated cell proliferation from my group (*Lab Invest* 76:779-791, 1997).

6. We were the first to propose that placental pathology may arise due to an alteration in the homeostasis of local oxygen-sensitive growth factors such as PlGF and VEGF and that increased levels of endogenous soluble VEGFR-1 antagonize the beneficial effects of VEGF in preeclampsia (*Trophoblast Res*. 10:215-258, 1997).

7. Uterine quiescence is a prerequisite for a successful pregnancy, but what keeps it in this state was not known. Dr Carmen Acevedo and I showed that heme oxygenase-1 and not nitric oxide synthase keeps the human uterus in a relaxed state during pregnancy (*J Clin Invest* 101:949-955, 1998).

8. We were the first to show that angiotensin-II could stimulate the release of vasorelaxants (NO and PTHrP). We showed that $AT_1$ receptor is expressed on trophoblast and that its expression is reduced in intrauterine growth restricted placenta. We proposed this could compromise nutritional transfer from mother to fetus. A number of groups are pursuing this line of research initiated by my laboratory (*J Clin Invest* 101:442-454, 1998).

9. We identified site of expression of PAF receptors in human endometrium and its intracellular signalling and functional role in the endometrium (*FASEB J* 12:831-843, 1998).

10. We showed that there was increased expression of PlGF in proliferative diabetic retinopathy (*Lab Invest* 78:109-116, 1998). Additional studies support the idea that anti-PlGF therapy is a viable treatment for PDR and cancer.

11. Morphological studies show poor placental vascular development in intrauterine growth restriction (IUGR). We have provided a molecular explanation for the observed poor angiogenesis in the pathogenesis of IUGR and showed that its placenta growth factor (PlGF) upregulation may lead to abnormal placental angiogenesis (*Lab Invest* 79:151-170, 1999).

12. The first evidence for the role of heme oxygenase-1 as an endogenous placental factor involved in cytoprotection and placental blood vessel relaxation was recently described. This work provides potentially a new approach to study the disease of preeclampsia (*Mol Med* 6:391-409, 2000).

13. Recently my group showed that the angiopoietins and their cognate receptor, Tie2, which are required for angiogenesis, are expressed and functionally active in non-endothelial cells (trophoblast), and that their effects appear to be distinct from those observed in endothelial cells. Furthermore, this ligand/receptor system appears to be dysregulated in IUGR (*Am J Pathol* 156:2185-2199, 2000).

14. In tumour epithelial cells, we showed that VEGFR-2-mediated mitogenesis is negatively regulated by VEGFR-1 via nitric oxide (*Am J Pathol* 158:265-273, 2001).

15. Our work on endothelial biology has led to three key findings: (i) Nitric Oxide (NO) as a molecular switch for endothelial cell differentiation in response to VEGF-induced capillary network formation. (ii) VEGFR-1 (flt-1) is a signalling receptor whose function is to promote branching angiogenesis mediated by NO. (iii) The function of VEGFR-2 is to promote proliferation and migration in an unidirectional fashion (non-branching angiogenesis that is negatively regulated by VEGFR-1 and NO.) (*Am J Pathol* 159:993-1008, 2001).

16. We demonstrated that excessive menstrual bleeding (menorrhagia) is due not to a primary defect in angiogenesis, but due to down-regulation of angiopoietin-1 that is involved in blood vessel maturation. In other words, menorrhagia is a result of failure of blood vessel maturation (*Am J Pathol* 160:773-80, 2002).

17. PlGF-deficient mice display abnormalities in VEGF-dependent retinal angiogenesis and myocardial angiogenesis in response to ischemia. We showed that PlGF contributes to pathological angiogenesis by prolonging endothelial cell survival via the PI3K pathway. PlGF displays a capacity to retain capillary networks and to promote survival of endothelial cells in long-term serum-deprived cultures by up-regulating survival signals (*Diabetes* 52:2959-68, 2003).

18. We showed that VEGF induced prolonged HO-1 expression and activity in endothelial cells and that HO-1 inhibition abrogated VEGF-driven angiogenesis. We think that during chronic inflammation HO-1 displays two roles. First an anti-inflammatory action inhibiting leukocyte infiltration and second, the promotion of VEGF-driven non-inflammatory angiogenesis which facilitates tissue repair (*Blood* 103:761-6, 2004).

19. Cervical ripening is critical for a successful delivery. VEGF is a potent angiogenic polypeptide that promotes vascular permeability. We have provided the first evidence for a key role of VEGF in the process of cervical ripening and proof-of-principle for novel drug targets to either promote or inhibit cervical ripening in the absence of adverse uterotonic effects in vivo. (WO 96/1662).

20. We were the first to propose that elevation in soluble Flt-1 may promote preeclampsia (Ahmed, 1997; *Trophoblast Res* 10:215-258). We showed that removal of sFlt-1 restored angiogenesis in preeclampsia. These findings identified sFlt-1 as the factor responsible for angiogenic imbalance in preeclampsia (*Cir Res* 95:884-891, 2004).

21. We provided direct evidence for endothelial Flt-1 function in angiogenesis and identified Tyr794 of VEGFR-1 as a key residue in NO-driven *in vitro* angiogenesis (*Circ Res* 99:715-22, 2006).

22. Identified heme-oxygenase-1 as an inhibitor of sFlt-1 and sEndoglin and statin as a potential therapeutic agent against preeclampsia (*Circulation* 115:1789-97, 2007).

23. I provided a new role of Angiopoietin-2 (Ang2) by showing that it could act as an atheroprotective factor in atherosclerosis-prone ApoE null mice (*Circ Res* 104: 1333-1336, 2009).

24. Using mouse and human studies, we showed that an increase in the maternal sFlt-1 if kept below a critical threshold level during pregnancy is not always associated with adverse outcomes; it depends on dose and duration of exposure (*Circulation* 124:2543-53, 2011 and *J Cell Mol Med* 14:1857-67, 2010).

25. The most commonly accepted hypothesis is that f*etal metabolic programming* leads secondarily to diseases associated with cardiovascular disease, such as obesity, diabetes mellitus, and hypertension. We provide an alternative hypothesis that fetal growth restriction induces primary cardiac changes that persist into childhood (*Circulation* 121:2427-36, 2010 and *PLoS One* 4:e5155, 2009).

26. Dissected the contribution of systemic inflammation and anti-angiogenic factors in preeclampsia and showed that excessive systemic inflammation is unlikely to be the main contributor to severe preeclampsia (*Angiogenesis* 2012).

27. My group was amongst the first laboratories to signal the importance of vascular growth factors in pregnancy and pinioned the concept of angiogenic imbalance theory in preeclampsia in the mid-90s. I am the Principal Investigator for the World's first randomized controlled clinical trial on statins in pregnancy StAmP Trial (*Thromb Res* 127S3:S72-S75, 2011).

**Publications from Ahmed's Laboratory**

Cudmore MJ, Hewett PW, Ahmad S, Wang KQ, Cai M, Al-Ani B, Fujisawa T, Sissaoui S, Ramma W, Miller M, Newby DE, Gu Y, Barleon B, Weich H and **Ahmed A**. (2012) Identification and functional characterisation of VEGF-A receptor heterodimerisation. ***Nat Comms*** 3:972. doi: 10.1038/ncomms1977.

Ramma W, Buhimschi IA, Zhao G, Dulay AT, Nayeri UA, Buhimschi CS, **Ahmed A**. (2012) The elevation in circulating anti-angiogenic factors is independent of markers of neutrophil activation and systemic inflammation in preeclampsia. ***Angiogenesis*.** 15(3):341-8.

Rosenberg VA, Buhimschi IA, Lockwood CJ, Paidas MJ, Dulay AT, Ramma W, Abdel-Razeq SS, Zhao G, Ahmad S, **Ahmed A**, Buhimschi CS. (2011). Heparin elevates circulating soluble Fms-Like Tyrosine immunoreactivity in pregnant women receiving anticoagulation therapy. ***Circulation*** 124(23):2543-53.

Cudmore MJ, Ahmad S, Sissaoui S, Ramma W, Ma B, Fujisawa T, Al-Ani B, Wang K, Cai M, Crispi F, Hewett PW, Gratacós E, Egginton S and **Ahmed A**. (2012) Loss of Akt activity increases circulating soluble endoglin release in preeclampsia: identification of inter-dependency between Akt-1 and heme oxygenase-1. ***Eur Heart J*** 33:1150-8. Epub 2011 Mar 16.

Ahmad S, Hewett PW, Al-Ani B, Sissaoui S, Fujisawa T, Cudmore MJ, **Ahmed A**. (2011) Autocrine activity of soluble Flt-1 controls endothelial cell function and angiogenesis. ***Vascular Cell*.** 3:15.

Cudmore MJ, Ramma W, Cai M, Fujisawa T, Ahmad S, Al-Ani B, **Ahmed A.** (2012) Resveratrol inhibits the release of soluble fms-like tyrosine kinase (sFlt-1) from human placenta. ***Am J Obstet Gynecol.*** 2012 Mar;206(3):253.e10-5. Epub 2011 Nov 25.

Krueger J, Liu D, Scholz K, Zimmer A, Shi Y, Klein C, Siekmann A, Schulte-Merker S, Cudmore M, **Ahmed A**, le Noble F. (2011) Flt1 acts as a negative regulator of tip cell formation and branching morphogenesis in the zebrafish embryo. ***Development.*** 138:2111-20.

Cai J, Wu L, Qi X, Shaw L, Li Calzi S, Caballero S, Jiang WG, Vinores SA, Antonetti D, **Ahmed A**, Grant MB, Boulton ME. (2011) Placenta growth factor-1 exerts time-dependent stabilization of adherens junctions following VEGF-induced vascular permeability. ***PLoS One.*** 6(3):e18076.

Ahmad S, Cudmore MJ, Wang K, Hewett P, Potluri R, Fujisawa T, **Ahmed A**. (2010) Angiopoietin-1 induces migration of monocytes in a tie-2 and integrin-independent manner. ***Hypertension.*** 56:477-83.

Gould P, Gu M, Liao J, Ahmed S, Cudmore MJ, **Ahmed A** and Vatish M. (2010) Urotensin-II stimulates the release of placental sflt-1 under hypoxic conditions and UT-II receptor is upregulated in preeclampsia. ***Hypertension*** 56:172-8.

Crispi F, Bijnens B, Figueras F, Bartrons J, Eixarch E, Le Noble F, **Ahmed A**, Gratacós E. (2010) Fetal growth restriction results in remodeled and less efficient hearts in children. ***Circulation*** 121(22):2427-36.

Wegiel B, Gallo D, Raman K, Karlsson J, Ozanich B, Chin BY, Tzeng E, Ahmad S, **Ahmed** A, Baty C, Otterbein L. (2010) Nitric Oxide-Dependent Endothelial Progenitor Cell Mobilization by Carbon Monoxide Enhances Endothelial Repair Following Vascular Injury. ***Circulation*** 121:537-48.

Al-Ani B, Hewett P, Cudmore M, Fujisawa T, Saifeddine M, Williams H, Ramma W, Sissaoui S, Padma-Sheela Jayaraman PS, Ohba M, Ahmad S, Hollenberg MD and **Ahmed A**. (2010) Activation of PAR2 Induces sVEGFR-1 Release via PKC and EGF Receptor Transactivation in Endothelial Cells. ***Hypertension*** 55:689-97.

Mee CJ, Farquhar M, Harris HJ, Ramma W, **Ahmed A**, Maurel P, Bicknell R, Balfe P, McKeating JA. (2010) Hepatitis C virus infection reduces hepatocellular polarity in a vascular endothelial growth factor dependent manner. ***Gastroenterology*** 138:1134-1142.

Bergmann A, Ahmad S, Cudmore MJ, Gruber AD, Wittschen P, Lindenmaier W, Christofori G, Gross V, M.D; da Costa Gonzalves AC, Gröne HJ, **Ahmed A**, Weich HA. (2010) Reduction of circulating soluble Flt-1 alleviates preeclampsia-like symptoms in a mouse model. ***J Cell Mol Med*** 14:1857-67.

**Ahmed A**, Fujisawa T, Niu XL, Ahmad S, Al-Ani B, Chudasama K, Abbas A, Potluri R, Bhandari V, Findley CM, Lam G, Huang JD, Hewett PW, Cudmore MJ, and Kontos CD (2009). Angiopoietin-2 (Ang2) confers atheroprotection in atherosclerosis-prone ApoE null mice. ***Circ Res*** 104: 1333-1336.

Tintu A, Rouwet E, Verlohren S, Brinkmann J, Ahmad S, Crispi F, van Bilsen M, Carmeliet P, Staff AC, Tjwa M, Cetin I, Gratacos E, Hernandez-Andrade E, Hofstra L, Jacobs M, Lamers WH, Morano I, Safak E, **Ahmed A**, le Noble F. (2009) Hypoxia induces dilated cardiomyopathy in the chick embryo: mechanism, intervention, and long-term consequences. ***PloS One*** 4:e5155.

Crispi F, Hernandez-Andrade E, Pelsers MM, Plasencia W, Benavides-Serralde JA, Eixarch E, Le Noble F, **Ahmed A**, Glatz JF, Nicolaides KH, Gratacos E. (2008) Cardiac dysfunction and cell damage across clinical stages of severity in growth restricted fetuses. ***Am J Obstet Gynecol*** 199:254.e1-8.

Kanasaki K, Palmsten K, Sugimoto H, Ahmad S, Hamano Y, Xie L, Parry S, Augustin HG, Gattone Jr VH, Folkman J, Strauss III JF, and Kalluri R (2008) Deficiency in Catechol-O-Methyltransferase and 2-Methoxyestradiol is Associated with Preeclampsia. ***Nature*** 453:1117-21.

Zhou CC, Ahmad S, Xia L, Mi, T, Abbasi S, Xia L, Day MC, Ramin SM, **Ahmed A**, Kellems RE and Xia Y. (2008) Autoantibody from women with preeclampsia induces sFlt-1 production via AT1 receptor and calcineurin/NFAT signalling. ***Hypertension*** 51:1010-9.

******Cudmore M, Ahmad S, Al-Ani B, Coxal H, Devey L, Wigmore S, Hewett P and **Ahmed A** (2007). Negative regulation of soluble VEGFR-1 release by a heme-oxygenase-1-dependent pathway. ***Circulation*** 115:1789-97.

Roberts JR, Perkins GD, Fujisawa T, Pettigrew KA, Gao F, **Ahmed A**, Thickett DR. (2007) Vascular endothelial growth factor promotes physical wound repair and is anti-apoptotic in primary distal lung epithelial and A549 cells. ***Crit Care Med*** 35:2164-70.

Zhou CC, Ahmad S, Xia L, Hewett, P, Abbasi S, **Ahmed A**, Kellems RE and Xia Y. (2007) Angiotensin II-induced soluble fms-Like tyrosine kinase 1 (sFlt-1) synthesis and secretion via calcineurin signalling. ***Circ Res*** 100:88-95.

Findley CM, Cudmore M, **Ahmed A**, Kontos CD. (2007) VEGF Induces Soluble $Tie_2$ Shedding via a Phosphoinositide 3-Kinase/Akt-dependent Pathway to Modulate $Tie_2$ Signaling. ***Arterioscler Thromb Vasc Biol*** 27:2619-26.

Ahmad S, Hewett PW, Wang P, Al-Ani B Cudmore M, Fujisawa T, Wang P, Haigh JJ, Le Noble F, Mukhopadhyay D and **Ahmed A**. (2006) Direct evidence for endothelial Flt-1 function in nitric oxide-Mediated Angiogenesis. ***Circ Res*** 99:715-22.

Al-Ani B, Hewett P, Ahmed S, Ahmad S and **Ahmed** A. (2006) The Release of Nitric Oxide from S-nitrosothiols promotes angiogenesis. ***PLoS One*** 1:e25.

Hewett PW, Daft EL, Laughton CA, Ahmad S, **Ahmed A**, Murray JC. (2006) Selective inhibition of the human tie-1 promoter with triplex-forming oligonucleotides targeted to Ets binding sites. ***Mol Med*** 20: 8-16.

Cudmore M, Ahmad S, Al-Ani B, Hewett PW, Ahmed S, and **Ahmed A**. (2006) VEGF-E induces angiogenesis via a nitric oxide mediated cGMP independent mechanism. ***Biochem Biophys Res Commun*** 345:1275-82.

Cai J, Jiang WG, **Ahmed A**, Boulton M. (2006) Vascular endothelial growth factor-induced endothelial cell proliferation is regulated by interaction between VEGFR-2, SH-PTP1 and eNOS. ***Microvasc Res*** 71:20-31.

Ahmad S and **Ahmed A** (2005) Anti-angiogenic effect of soluble VEGFR-1 in placental angiogenesis. ***Endothelium*** 12:89-95.

Ahmad S and **Ahmed A** (2004) Elevated Placental Soluble Flt-1 inhibits angiogenesis in Preeclampsia. ***Cir Res*** 95:884-891.

Buhimschi IA, Dussably L, Buhimschi CS, **Ahmed A**, Weiner CP. (2004). Physical and biomechanical characteristics of rat cervical ripening are not consistent with increased collaganase activity. ***Am J Obstet Gynecol*** 191: 1695-704.

Belgore F, Blann A, Neil D, **Ahmed A**, Lip GY. (2004) Localisation of members of the vascular endothelial growth factor (VEGF) family and their receptors in human atherosclerotic arteries***. J Clin Pathol*** 57:266-72.

Bussolati B, **Ahmed A**, Pemberton H, Landis RC, Di Carlo F, Haskard DO and Mason JC. (2004) Bifunctional role for VEGF-induced Heme Oxygenase-1 in vivo: induction of angiogenesis and inhibition of leukocytic infiltration. ***Blood*** 103:761-6.

Cai J, Ahmad S, Jiang WG, Huang J, Kontos CD, Boulton M. and **Ahmed A**. (2003). Activation of vascular endothelial growth factor receptor-1 sustains angiogenesis and Bcl-2 expression via the phosphatidylinositol 3-kinase pathway in endothelial cells. ***Diabetes*** 52:2959-68.

Perkins J, St John J, and **Ahmed A**. (2002) Modulation of trophoblast cell death by oxygen and EGF. ***Mol Med*** 8:846-857.

Hewett P, Nijjar S, Shams M, Gupta J. and **Ahmed A**. (2002) Down-regulation of angiopoietin-1 expression in menorrhagia. ***Am J Pathol*** 160: 773-80.

Bussolati B, Dunk CE, Grohmann M, Kontos CD, Mason J. and **Ahmed A** (2001) Vascular endothelial growth factor receptor-1 (VEGFR-1) receptor suppresses VEGFR-2-mediated mitogenesis and promotes endothelial cell organisation via nitric oxide. ***Am J Pathol*** 159: 993-1008.

Ramsden JD, Helen C Cocks HC, Shams M, Nijjar S, Watkinson JC, Sheppard MC, **Ahmed A**, Eggo MC (2001) Tie-2 is expressed on thyroid follicular cells, is increased in goiter and regulated by TSH through cyclic AMP. ***J Clin Endocriol Metab*** 86(6):2709-16.

Cassoni P, Sapino A, Munaron L, Deaglio S, Chini B, Graziani A, **Ahmed A**, Bussolati B. (2001) Activation of functional oxytocin receptors stimulates cell proliferation in human trophoblast and choriocarcinoma cell lines. ***Endocrinology*** 142:1130-1136.

Dunk C, **Ahmed A**. (2001) Vascular endothelial growth factor receptor-2-mediated mitogenesis is negatively regulated by vascular endothelial growth factor receptor-1 in tumor epithelial cells. ***Am J Pathol*** 158: 265-273.

Dunk C, **Ahmed A**. (2001) Expression of VEGF-C and activation of its receptors VEGFR-2 and VEGFR-3 in placenta ***Histol Histopath*** 16: 359-75.

***Ahmed A**, Rahman M, Zhang X, Acevedo CA, Nijjar S, Rushton I, Bussolati B, St John J (2000) Induction of placental heme oxygenase-1 is protective against TNFa-induced cytotoxicity and promotes vessel relaxation. ***Mol Med*** 6: 391-409.

Hornig C, Barleon B, Ahmad S, Vuorela P, **Ahmed A**, Weich HA (2000) Release and complex formation of soluble VEGFR-1 from endothelial cells and biological fluids. ***Lab Invest*** 80:443-54.

Dunk C, Shams M, Nijjar S, Rhaman M, Qiu Y, Bussolati B, **Ahmed A**. (2000) Angiopoietin-1 and angiopoietin-2 activate trophoblast Tie-2 to promote growth and migration during placental development. ***Am J Pathol*** 156:2185-2199.

**Ahmed A**, Dunk C, Ahmad S, Khaliq A. (2000) Regulation of placental VEGF and PlGF and soluble Flt-1 by oxygen. Placenta 21: Trophoblast Res Suppl 14: S1-S9.

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Brockelsby J, Hayman R, **Ahmed A**, Warren A, Johnson I, Baker P. (1999) VEGF via VEGFR-1 (Flt-1) mimics preeclamptic plasma in inhibiting uterine blood vessel relaxation in pregnancy: Implications in the pathogenesis of preeclampsia. ***Lab Invest*** 79: 1101-1111.

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Khaliq A, Dunk CE, Shams M, Li X-F, Whittle MJ, Weich H, and **Ahmed A**. (1999) Hypoxia down-regulates placenta growth factor whereas fetal growth restriction up-regulates placenta growth factor expression: Molecular evidence for "placental hyperoxia" in intrauterine growth restriction. ***Lab Invest*** 79:151-170.

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*****= Translated into the Clinical trial StAmP.