CURRICULUM VITAE
SALOMON AMAR D.D.S., Ph.D.

PERSONAL DATA

Business Address: Boston University Medical Center,
Department of Periodontology and Oral Biology,
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EDUCATION

[----]	Aquiba School, Strasbourg, France	**B.S.:** Mathematics and Physics
[----]	Univ. Louis Pasteur, Strasbourg, France	**D.D.S.:**
[----]	Univ. Louis Pasteur, Strasbourg, France	**Certificate:** Histology Cytology
[----]	Univ. Louis Pasteur, Strasbourg, France	**M.S.:** Skeletal Tissues and Apatites
[----]	Univ. Louis Pasteur, Strasbourg, France	**Certificate:** Periodontology
[----]	Univ. Louis Pasteur, Strasbourg, France	**Ph.D.:** Developmental Biology
[----]	Northwestern University, Chicago, USA	**Postdoctoral Fellow:** Biochemistry-Molecular Biology
[----]	Eastman Dental Center, Rochester, USA	**Certificate:** Periodontology
[----]	Boston University, USA	**D.M.D.**

EXPERIENCE IN HIGHER EDUCATION

2006- Present: Boston University	Associate Dean for Research
2001-Present: Boston University	Professor of Periodontology and Oral Biology
1997-Present: Boston University	Research Associate Professor of Biochemistry
2004-present: FDA	Panel Member: Dental Product Panel, CDRH, FDA
1996-2004: FDA	Consultant: Dental Product Panel, CDRH, FDA.
1995-2001: Boston University	Associate Professor of Periodontology and Oral Biology
1992- 1995: Eastman Dental Center	Assistant Professor of Periodontology
1994- 1997: University of Rochester	Adjunct Assistant Professor of Pathology
1994- 1997: University of Rochester	Adjunct Assistant Professor of Hematology
1990-1992: Northwestern University	Assistant Professor,

		Oral Biology Division and Department of Periodontics.
1990-1992	Northwestern University	Coordinator of Continuing Education
[----------]	Northwestern University	Post doctoral Fellow, Department of Oral Biology
[----------]	Medical School Strasbourg, France	Research Assistant
1985-1986	Dental School Strasbourg, France	Instructor

HONORS AND AWARDS:

- Research First Prize of Alpha Omega Fraternity International Division, [----].
- Young Investigator Award, [----], IADR-AADR.
- Diplomate of the American Board of Periodontology
- Lady Davis Fellowship, [----].
- NIH-NHLBI RFA Reviewer: 2002 and 2003.
- NIH-NIDCR RFA Center For Discoveries Reviewer 2004.
  NIH CSR Ad-hoc reviewer.

EXPERIENCE OTHER THAN HIGHER EDUCATION

Private Clinical Practice, Epinal, France, 1986 (Part-time).
Private Clinical Practice limited to Periodontics, Epinal France, 1987-1989; (Part-time).
Private Clinical Practice limited to Periodontics, Rochester, NY, 1994-1995, (Part-time).
Private Clinical Practice limited to Periodontics, Boston, MA, 1995-present, (Part-time).

Professional Licensure:

Illinois Temporary Dental Teaching License, Illinois, [----------]
Illinois Dental License: Active
Illinois Specialty License: Periodontology, Active
New York State Dental License: Active
Massachusetts Dental License: Active
Diplomate of the American Board of Periodontology 2000

Hospital Affiliation:

Attending Dentist: Franciscan Children Hospital
Attending Dentist: Boston Medical Center

EDITORIAL BOARDS

Associate Editor:
Oral Diseases
Editorial Board
Journal of Dental Research
Grand Rounds in Oral-Systemic Medicine

Research Interests and Activities

1. Research

1.1. Inflammation and Host response:

1.1.1. Cytokines:

The host response to infection plays a major role in the resolution inflammatory diseases and in particular periodontal diseases. However the overproduction of proinflammatory mediators during this inflammatory phase is deleterious to the host. Among the proinflammatory mediators secreted during the inflammatory process the most prominent are IL-1 and TNF-alpha. Using a TNF and/or IL-1 -deficient mice, we quantified the relative contribution of each of these mediators in *Porphyromonas gingivalis*-induced bone resorption. IL-1 was found to be the most potent while a synergistic effect was observed with TNF-alpha. These findings led us to propose the hypothesis that if IL-1 and TNF expression can be suppressed but not eliminated, outcome variables of the periodontitis could be largely improved. This hypothesis was tested using an experimental periodontitis monkey model. Local application of blockers to IL-1 and TNF in the gingiva can prevent bone loss and the inflammatory reaction, naturally occurring in the animals receiving the placebo control. These data highlight the important role of IL-1 and TNF in mediating periodontal inflammation and bone loss and demonstrate for the first time that bone loss and inflammation in periodontitis can be controlled by local application of blockers to IL-1 and TNF.

Given that IL-1 and TNF could not account for all the occurring bone loss, we investigated other mediators. Under the same condition, the contribution of Prostaglandin's and IL-11 was determined. IL-11 or PG were found to be the major mediators equally capable of mediating bone resorption at low doses of *P.g.* LPS while at high doses of *P.g.* LPS IL-1 and TNF were the major mediators. These data provide for the first time the relative contribution of each mediator in *P.g.* LPS-induced bone loss and propose that chronic bone loss (low LPS doses) can be prevented by indomethacin or anti-IL-11 while acute bone loss (high LPS doses) can be prevented by blockers to IL-1 and TNF. Should chronic bone diseases such as Rhumathoid arthritis or Periodontitis progress continuously a therapeutic approach involving anti-PG's or anti-IL-11 would be better suited whereas if these diseases progress by burst of acute bone loss, a therapeutic approach involving blockers to IL-1 and TNF would be more appropriate.

1.1.2. Transcriptional Regulation:

The overexpression of cytokines (IL-1; TNF) in inflammatory processes is extremely detrimental for the host. Our approach to reduce deleterious effects associated with the overexpression of these cytokines consisted in identifying molecular factors controlling cytokine gene expression in inflammatory processes and particularly in gingivitis and in periodontitis.

Tumour necrosis factor alpha (TNF-α) is a potent inflammatory mediator and has been implicated in the pathophysiology of a variety of diseases including Crohn's disease, inflammatory bowel disease or IBS, multiple sclerosis and rheumatoid arthritis. We recently cloned a novel transcription factor that we can show binds to the TNF-α promoter and regulates transcription in lipopolysaccharide-treated macrophages by repressing significantly TNF gene expression. Binding of this protein, which we have named LPS-induced TNF-α factor (LITAF), to its recognition element in the TNF-α promoter leads to the up-regulation of TNF-α expression. Thus, pharmacological inhibition of LITAF appears to be an attractive therapeutic strategy for the treatment of the diseases mentioned above, as being upstream of TNF-α it would block the production of this inflammatory cytokine. The inhibition of this factor has resulted in a substantial reduction of TNF-alpha and could be used in therapeutic approaches aimed at dampening down excess of TNF-alpha (i.e. Rheumatoid Arthritis; Crohn's Disease; Periodontal Disease). Given the role of NFkB in cytokine gene regulation, the discovery of this novel transcription factor named LITAF controlling TNF gene expression is viewed as a milestone in the transcriptional regulation

of cytokine genes. We have continued with this project by expressing high levels of recombinant LITAF that we have purified for structural analysis. We are currently carrying out structure-function experiments using X-ray diffraction; a high-resolution structure will enable the design of efficacious functional inhibitors of LITAF that could represent lead compounds for use in drug development programs aimed at treating inflammatory diseases.

1.1.3. Atherosclerosis:

Finally, we have conclusively demonstrated for the first time in a mouse model that *Porphyromonas gingivalis,* the principal microorganism in periodontal diseases, can aggravate and even trigger the development of atherosclerotic lesions. These results provide for the first time a causal relation between Porphyromonas gingivalis and atherosclerosis and pave the way for the complete elucidation of bacterial induced atherosclerosis.

1.2. Wound healing:

In our wound healing studies we identified several candidate genes responsible for periodontal regeneration that are now evaluated in various animal models. These findings establish for the first time the existence of an adult stem cell in the periodontal compartment (with a reduced apoptotic rate compared to other cells) and provide new understanding of periodontal homeostasis and regeneration. Furthermore, effort was made to identify critical factors involved in driving periodontal wounds into the regeneration of periodontal structures after periodontal diseases. Recently using cDNA array technologies we isolated and cloned a new factor involved in tissue regeneration. The overexpression of this factor hold promise in regenerating tissues lost from disease processes.

1.3. Human Clinical Approach:

At a clinical level, I have contributed to studies aimed a determining whether chronic inflammatory diseases such as periodontitis can affect the endothelial function and lead to atherosclerosis.

I am also serving as a consultant for Health Care Industry and biotechnology companies.

2. Teaching

* Periodontics, Instructor, 1985-1988, Dental School, Strasbourg (France), Undergraduate.
* Periodontics, Instructor, 1990- 1992, Northwestern University Dental School, Undergraduate.
* Periodontics II, Lecturer, 1990-1992, Northwestern University Dental School, Undergraduate.
* Periodontics III, Lecturer, 1990-1992, Northwestern University Dental School, Undergraduate.
* Advanced Periodontics, Lecturer, 1990-1992, Northwestern University Dental School, Undergraduate.
* Pathology Review, Course Director, 1990-1992, Northwestern University Dental School, Graduate Students.
* Advanced Anatomy and Histology, Course Director, 1991-1992, Northwestern University Dental School, Graduate Students.

Oral Anatomy and Histology, Lecturer, 1991-1992, Northwestern University Dental School, Undergraduate Students
* Current Literature Review Seminar, Course Director, 1991-1992, Northwestern University Dental School, Graduate Students.
* Periodontics I, II, III, Lecturer, 1990-1992, Northwestern University Dental School, Continuing Education.
* Research Laboratory teaching, 1989-1992, Oral Biology Division, Northwestern University, Graduate Students.
* Research Laboratory teaching, 1992-1995, Department of Periodontology, Eastman Dental Center, Graduate Students.
* Biology of the Periodontium: 1993-present, Eastman Dental Center, Graduate Students
*Literature Review Seminar, Instructor, 1994-1995, Eastman Dental Center, Graduate Students.
* Clinical Periodontics: Instructor in the Graduate Periodontal Clinic, 1994-1995, Eastman Dental Center, Graduate Students.
* Oral Biology 1: Course Director, 1996-present, Boston University, Dental Students
* Literature Review Seminar, Course Director, 1995-2004, Boston University, Graduate Students.
* Periodontology III: Course Director, 1996-2005, Boston University, Graduate Students.

PROFESSIONAL ACTIVITIES

Memberships

American Dental Association
International Association for Dental Research
American Association for Dental Research
American Association of Oral Biologists
American Academy of Periodontology
International Academy of Periodontology
The Wound Healing Society
International Endotoxin Society

Meetings and Conferences

Boston University Chairman and PI of the Organizing Committee for the NIH-Sponsor Teleconference: "Behavioral Aspect of Dentistry", October 16, 1996 Boston, MA.

Scientific Advisory Committee to the NIH-sponsored Meeting: Molecular Mechanisms of Host Cell Interactions in Periodontal Diseases; March 14-17, 1997, St. Petersburg, Florida.

Guest Editor with Drs. C. Genco and T. E. Van Dyke of Journal of Clinical Infectious Diseases Special Issue on "Molecular Mechanisms of Host Cell Interactions in Periodontal Diseases"

IADR/AADR Symposium Organizer: Molecular and Cellular aspect of Periodontal Wound Healing: March 2000, Washington DC.

Guest Editor for two special issues in wound healing: Journal of Parodontologie and Oral Implantologie. Vol 22, 4, November 2003 and Vol 23, 1 March 2004.

Committee Participation

Committee on Academic Freedom: Faculty Council Boston University: Member
Committee on Financial Affairs: Faculty Council Boston university: Member
AADR Constitution Committee: Member 1998-2001
AADR Award and Fellowship: Member 2001-2005
AADR Hatton Award Committee: Member 2005-present
Curriculum Committee: Boston University School of Dental Medicine: Member
Predoctoral Course Director Committee: 2004-present

GRANTS AND CONTRACTS:

Active

NIDCR/NIH R01 DE 014079
Role of LITAF in Inflammatory Processes
1/01/02-12/30/06 direct : $900,000
P.I.: Salomon Amar

NIDCR/NIH R01 DE 015345
Systemic Endothelial Consequences of Periodontal Disease
5/01/04-4/30/09 direct $2,594,488
P.I.: Salomon Amar

NIDCR/NIH R01 DE 015989
Functional Genomics in Periodontal Host-Parasite interactions
3/01/04-11/30/07 direct $1,615,000
P.I.: Salomon Amar

NHLBI/NIH R01 HL076801
Infection, Inflammation and Atherosclerosis
1/01/05-12/31/09 direct $1,615,000
P.I.: Salomon Amar

PATENTS

Transcription factor regulating TNF-alpha
Patent: US 6566501-A 2
20-MAY-2003;

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PUBLICATIONS:

1. Textbooks:

- Robinson P. J and **Amar S**.: Influence of Pregnancy in the Oral Cavity. Clinical Obstetrics. Vol. 2: Chap. 15, 1-6, 1992.

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2. Refereed Journals

- **Amar S**., Karcher-Djuricic V., Meyer J.M. and Ruch J.V.: Lingual (Root-Analog) and Labial (Crown- Analog) Mouse incisor dentin promotes ameloblast differentiation. Arch. Anat. Microsc. Morphol. Exp. 75: 229-239, 1986.

- **Amar S**., Karcher-Djuricic V., Meyer J.M., and Ruch J.V.: Root-analog and crown-analog mouse incisor dentin promotes ameloblast differentiation: An evidence of absence of heterotypic cell contacts. Proc. Finn. Dent. Soc. 83:225-236, 1987.

- **Amar S** and Ruch J.V.: Mouse incisor lingual inner dental epithelium does not contain potential ameloblasts. Med. Sci. Res. 15:949-950, 1987.

- Tziafas D., **Amar S**., Staubli A., Meyer J.M., and Ruch J.V.: Effects of glycosaminoglycans oni i*n vitro* growing mouse dental cell. Arch. Oral. Biol. 33:735-740, 1988.

- Tenenbaum H., **Amar S**., and Klewansky P.: Orificial Plasmocytosis: A periodontal localization. Ann. Derm. Ven. 115:479-482, 1988.

- **Amar S**., Tenenbaum H., and Cuisinier F.J.G.: Characteristics of early onset periodontitis: A report of two cases. J. Parodont. 8:53-59, 1989.

- **Amar S**., Luo W., Snead M.L., and Ruch J.V.: Amelogenin gene expression in mouse incisor heterotopic recombinations. Differentiation. 41:56-61, 1989.

- Cuisinier F.J.G., Tenenbaum H., and **Amar S**.: Comparative study of different biological membranes in sem. J. Parodont. 8:271-278, 1989.

- Veis A., Sires B., Clohisy J., Sabsay B., and **Amar S**.: Rat incisor dentin contains a factor which alters the phenotypic expression and stimulates chondrogenesis in fibroblast-like cells i*n vitro*. Biomaterials 11:35-37, 1990.

- **Amar S**., Sires B. and Veis A.: A rat incisor dentin matrix protein can induce neonatal rat muscle fibroblasts, in culture, to express phenotypic products of chondroblastic cells. J. Biol. Bucc. 25:55-60, 1991.

- **Amar S.**, Sires B., Sabsay B., Clohisy J. and Veis A.: The isolation and partial characterization of a rat incisor dentin matrix polypeptide with i*n vitro* chondrogenic activity. J. Biol. Chem. 266:8609-8818, 1991.

- **Amar S**., Fabre M., and Ruch J.V.: Effects of ascorbate-deficiency on collagen secretion and resorption on cultured mouse incisor germs. Connect. Tissue Res. 28:125-142, 1992.

- Yamada J., **Amar S.**, and Petrungaro P.: Psoriasis-associated periodontitis: A case report. J. Periodontol. 63:854-857, 1992.

- Takashiba S., Shapira L., **Amar S.**, and Van Dyke T. E.: Cloning and characterization of human TNF-α promoter region. Gene 131:307-308, 1993.

- Massarat T., **Amar S**., Veis A. and Osetek E. : Immunolocalization of phenotypic cartilage macromolecules within fibroblastic cellular nodules induced by chondrogenic inducing agents *in vitro*. Northwest Dent Res 5:25-29, 1994.

- **Amar S**. and Chung K.M.: Cellular biology advances in periodontal regeneration: Clinical Implications. Curr. Opin. Periodont. 2: 128-140, 1994.

- Shapira L, Takashiba S, **Amar** S and Van Dyke T.E.: *Porphyromonas gingivalis* lipopolysaccharide stimulation of human monocytes: Dependence on serum and CD14 receptor. Oral Microbiol Immunol. 9:112-117, 1994.

- Shapira L., Takashiba S., Champagne C., **Amar** S. and Van Dyke T.E.: Induction of TNF-α and IL-1β secretion by lipopolysaccharide in human adherent monocytes requires the generation of two distinct intracellular signals: Involvement of protein kinase C and tyrosine kinase. J. Immunol. 153:1818-1824, 1994.

- **Amar S** and Chung K.M.: Influence of hormonal variation on the periodontium in women. Periodontology 2000 6: 79-87, 1994.

- Shapira L., Champagne C., Gordon B., **Amar S**. and Van Dyke T.E.: Lipopolysaccharide (LPS) priming of superoxide release by human neutrophils: Role of membranal Cd14 and serum LPS binding protein. Inflammation 19:289-295, 1995.

- **Amar S** and Chung K.M.: Molecular and cellular biology research in periodontal regeneration and its clinical implication. Ann. Acad. Med. Singapore, 24:58-67, 1995.

- Takashiba S., Van Dyke T.E., Shapira L. and **Amar S**.: Lipopolysaccharide-inducible and salicylate-sensitive nuclear factor(s) on human tumor necrosis factor-alpha promoter. Infect. Immun. 63:1529-1534, 1995.

- **Amar S**., Petrungaro P., Amar A. and Van Dyke T.E: Immunolocalization of bone matrix macromolecules in human periodontal regeneration tissues. Arch. Oral. Biol., 40:653-661, 1995.

- **Amar S.**: Implications of cellular and molecular biology advances in periodontal regeneration. Anat. Rec. 245:361-373, 1996.

- Landi L., Pretel R., Ross K. And **Amar S.**: Factors Responsible For Failures Associated With Guided Tissue Regeneration Procedures. J. Parodont. Impl. Oral. 15: 129-151, 1996

- Takashiba S., Van Dyke T.E. and . **Amar S**.: Inhibition of nuclear factor kappa B subunit P65 mRNA accumulation in LPS-stimulated human monocytic cells treated with sodium salicylate. Oral. Microbiol. Immunol. 11: 420-424, 1996.

- **Amar S.**, Van Dyke T.E., Eugster H.P., Schultze N., Koebel P. and Blüthmann H.: Tumor necrosis factor (TNF)-induced cutaneous necrosis is mediated by TNF receptor 1. J. Inflam. 47:180-189, 1997.

- **Amar S.**, Chung K.M., Nam S.H., Karatzas S. , Myokai F. and Van Dyke T.E.: Markers of bone and cementum formation accumulate in tissues regenerated in periodontal defects treated with expanded polytetrafluoroethylene membranes. J. Perio. Research. 32:148-158, 1997.

- Landi L., **Amar S.**, Pollins S. and Van Dyke T.E.: Host mechanisms in the pathogenesis of periodontal diseases. Curr. Opin. Periodontol., 4:3-10,1997.

- Assuma R., Oates T., Cochran D., **Amar S**. and Graves D.T.: IL-1 And TNF antagonists inhibit the inflammatory response and bone loss in experimental periodontitis. J. Immunol. 160:403-409, 1998.

- Shapira L, Champagne C., Van Dyke T. E. and **Amar S**: Strain-dependent activation of monocytes and inflammatory macrophages by lipopolysaccharide of *Porphyromonas gingivalis*. Infect. Immun. 66:2736-42, 1998.

- Genco A.C., Van Dyke T.E. and **Amar S.**: Animal models for P*orphyromonas gingivalis*-mediated periodontal diseases. Trends Microbiol. 11:444-449, 1998.

- Graves D.T., Delima A., Assuma R. , **Amar S**, Oates T., and Cochran D. : IL-1 and TNF antagonists inhibit the progression of inflammatory cell infiltration toward alveolar bone in experimental periodontitis. J Periodont. 69:1419-1424, 1998.

- Myokai F., Takashiba S., Lebo R. and **Amar S.**: A novel LPS-induced transcription factor regulating TNF-α gene expression: Molecular cloning, sequencing, characterization, and chromosomal assignment. Proc. Natl. Acad. Sci. 96: 4518-4523, 1999.

- Karatzas S., Zavras A., Greenspan D. and **Amar S**. : Histologic observations of Periodontal wound healing after treatment with perioglas™ in non-human primates. Int. J. Perio. Rest. Dent. 19: 489-499, 1999.

- Chiang C. Y., Kyritsis G, Graves D.T., and **Amar S**: IL-1 and TNF activity partially account for calvarial bone resorption induced by local injection of LPS. Infect. Immun. 67: 4231-4236, 1999.

- Tohme Z. N., **Amar S.** and Van Dyke T. E.: Moesin functions as an LPS receptor on human monocyte. Infect. Immun. 67: 3215-3220, 1999.

- Takashiba S., Van Dyke T.E., **Amar S**, Murayama Y., Soskolne A. W. and Shapira L: differentiation of monocytes into macrophages primes the cells for rapid and enhanced TNF-α production: Role of nuclear factor Kb. Infect. Immun. 67: 5573-5578, 1999.

- Soolari A.S., Champagne C., Punzi J.S., **Amar S.**, Van Dyke T.E.: Serum modulation of neutrophil response to *Porphyromonas gingivalis* LPS in periodontal disease. J. Int. Acad. Periodontol. 4: 101-109:1999.

- Delima A. J, Oates T., Assuma R., Schwartz Z., Cochran D, **Amar S**. and Graves D.T: Soluble antagonists to interleukin-1 (IL-1) and tumor necrosis factor (TNF) inhibits loss of tissue attachment in experimental periodontitis. J Clin Periodontol. 28:233-240, 2001.

- **Amar S.**, Oyaisu K., Li L. and Van Dyke T.E.: Moesin: A potential LPS receptor on human monocytes. J. Endotox. Res. 7:281-286, 2001.

- Graves D.T., Nooh N., Gillen T., Davey M., Patel S., Cottrell D. and **Amar S**: Interleukin-1 plays a critical role in oral but not dermal wound healing. J. Immunol. 167:5316-20, 2001.

- Li L., Messas E., Batista E. L. Levine R.A. and **Amar S**.: *Porphyromonas gingivalis* infection accelerates the progression of atherosclerosis in an Apoe (+/-) murine model. Circulation 105: 861-867, 2002.

- Han X.Z. Bolcato A.L. and **Amar S**.: Identification of genes differentially expressed in cultured human osteoblasts versus human fibroblasts by DNA microarray analysis. Connec. Tissue Res. 2002; 43:63-75.

- Han X.Z. and **Amar S**.: Identification of genes differentially expressed in cultured human periodontal ligament fibroblasts versus human gingival fibroblasts by DNA microarray analysis. J. Dent. Res. 6:399-405, 2002.

- Li L., Khansari A., Graves D.T., and **Amar S**.: Contribution of interleukin-11 and prostaglandin(s) in lipopolysaccharide-induced bone resorption i*n vivo.* Infect. Immun. 70:3915-3922, 2002.

- **Amar S.** and Han X.: Regulation of tumour necrosis factor-α gene expression: Applied Genomics and Proteomics 1: 31-45, 2002.

- Delima A.J., Karatzas S., **Amar S.** and Graves D.T.: Inflammation and tissue loss caused by periodontal pathogens is reduced by interleukin-1 antagonists. : J. Infect. Dis. 186:511-516, 2002.

-Tang X. Fenton M. J. and **Amar S.**: Identification and functional characterization of a novel binding site on TNF-α promoter. Proc. Natl. Acad. Sci. U S A.; 100 :4096-4101. 2003.

- Han X. Z. and **Amar S**.: IGF-1 signaling enhances cell survival in periodontal ligament fibroblasts versus gingival fibroblasts. J. Dent. Res. 82:454-459, 2003.

- Santana R. B., Xu L., Chase B. H., **Amar S.**, Graves D. T., and Trackman P. C.: A role for advanced glycation end products in diminished bone healing in type I diabetes. Diabetes. 52:1502-10. 2003.

- **Amar S.**, Gokce N., Morgan S., Loukideli M., Van Dyke T.E. and Vita J.A.: Periodontal disease is associated with brachial artery endothelial dysfunction and systemic inflammation. Arterioscler. Thromb. Vasc. Biol. 23:1245-1249, 2003.

- Han X. Z. and **Amar S**.: IGF-1 signaling preferentially enhances cell survival in cultured periodontal ligament fibroblasts versus gingival fibroblasts. J. Perio. 74:1176-1182, 2003.

- **Amar S.** and Han X.: The impact of periodontal infection on systemic diseases. Med Sci Monit. 9: 291-299; 2003.

- Han X. Z. and **Amar S.**: Secreted frizzled-related protein 1 (Sfrp1) protects fibroblast from ceramide-induced apoptosis. J. Biol. Chem. 279 : 2832-2840, 2004

- Bolcato-Bellemin A. L., Mattei M.G., Lebo R. and **Amar S**.: Molecular cloning and characterization of the mouse LITAF cDNA implications in the regulation of the tumor necrosis factor-α (TNF-α) gene expression. J. Endotox. Res. 10:15-23, 2004.

- Iontcheva I., **Amar S.**, Zawawi K.H., Kantarci A. and Van Dyke T.E.: A role for moesin in lipopolysaccharide stimulated signal transduction. Infect. Immun. 72: 2312-2320, 2004.

- Zhang X., Kohli M., Zhou Q., Graves D.T, and **Amar S.**: Short- and long-term effects of IL-1 and TNF antagonists on periodontal wound healing. J. Immunol. 173:3514-3523, 2004

- Chi H., Messas E., Levine R.A., Graves D.T., and **Amar S.**: Interleukin-1 receptor signaling mediates atherosclerosis associated with bacterial exposure and/or high-fat diet in a murine ApoE heterozygote model: pharmacotherapeutic implications. Circulation 110:1678-1685, 2004.

- Lu H., Raptis M., Black E., Stan M., **Amar S.**, and Graves D.T.: Influence of diabetes on the exacerbation of an inflammatory response in cardiovascular tissue**.** Endocrinology. 145: 4934-4939, 2004.

- Zhou Q., Desta T., Graves D.T. and **Amar S.**: Cytokine profiling of macrophages exposed to *Porphyromonas gingivalis,* its LPS or its FimA. Infect Immun. 73:935-43, 2005.

- Tang X. Levy-Marciano D. Susan E. Leeman and **Amar S.**: LPS induces the interaction of a transcription factor, LPS-induced TNF-α factor, and STAT6(B) with effects on multiple cytokines. Proc. Natl. Acad. Sci. U S A.; 102 :5132-5137, 2005.

- Graves D.T., Ghada N. Huafei L., Desta T. and **Amar S.**: *Porphyromonas gingivalis* fimbriae are pro-inflammatory but do not play a prominent role in the innate immune response to *P.*

gingivalis. J. Endotox. Res. 11: 13-18, 2005.

- Petrungaro P.S. and **Amar S**.: Localized Ridge Augmentation with Allogenic Block Grafts Prior to Implant Placement: Case Reports and Histologic Evaluations. Implant Dent. 14:139-148, 2005.

- Zhou Q. and **Amar S**.: Identification of Proteins Differentially Expressed in Human Monocytes Exposed to *Porphyromonas gingivalis* and its Purified Components by High Throughput Immunoblotting. Infect. Immun. 74:1204-1214, 2006

- Leone C. W., Bokhadhoor H., Kuo D., Desta T., Yang J., Siqueira M.F., **Amar S.** and Graves D.T.: Immunization Enhances Inflammation and Tissue Destruction in Response to *Porphyromonas gingivalis.* Infect. Immun. 74: 2286–2292, 2006.

- Li C.H. and **Amar S.**: Role of secreted frizzled related protein 1 (SFRP1) in wound healing. J. Dent. Res. 85: 374-378, 2006.

- [----------] In press.

- [----------] In press.

- [----------] In press.

3. Non-refereed publications

- **Amar S**.: Contribution to the study of juvenile periodontitis: A bacteriological and immunological study. Odontology Thesis, Strasbourg 1986.

- **Amar S**.: Contribution to the study of epigenetic control mechanisms in dental cell terminal differentiation. Ph.D. Thesis, Strasbourg 1989