

# Ira S. Richards, Ph.D., FACFE

## Occupational and Clinical Toxicology

Associate Professor: Toxicology and Internal Medicine, Colleges of Public Health and Medicine at the University of South Florida. Graduate instruction in environmental and occupational toxicology, industrial toxicology, pathobiology, industrial hygiene, leading to master of science and doctor of philosophy degrees.



Experience with petrochemical and agrochemical industries/manufacturing/general industrial/environmental/workplace covering a wide range of classes of chemicals including pesticides, solvents, heavy metals, irritants, particulates, carcinogens, pharmaceuticals, illicit substances.

Well-published. Professionally-affiliated with Society of Toxicology, American Academy of Clinical Toxicology, American Chemical Society.

Recognitions include: Sigma Xi, Delta Omega. Board Certified Diplomate and Fellow of American College of Forensic Examiners, listings in Who's Who Science and Engineering.

RECENTLY PUBLISHED: Principles and Practice of Toxicology in Public Health, Jones and Bartlett Publisher (2007).

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# *Ira S. Richards, Ph.D., F.A.C.F.E.*

**Toxicologist and Associate Professor  
Colleges of Public Health and Medicine  
University of South Florida**

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## **Education**

Ph.D. (1976). New York University

M.S. (1972). University of Massachusetts

B.S. (1970). Long Island University

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## **Recognitions and Memberships**

- Society of Toxicology
- International Society on Toxinology
- American Academy of Clinical Toxicology
- American Chemical Society
- New York Academy of Sciences
- Board Certified Forensic Examiner (American Board of Forensic Examiners)
- Diplomate and Fellow American Board of Forensic Examiners
- Sigma Xi
- Delta Omega
- Who's Who in Science and Engineering (1994-1995; 1996-1997; 1999-2000)
- Who's Who in the World (1995-1996)
- International Society of Police Surgeons

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## Present Academic Position

Associate Professor, Department of Environmental and Occupational Health, School of Public Health, University of South Florida, Tampa, Florida; Associate Professor, Department of Internal Medicine, College of Medicine, University of South Florida.

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### Curriculum Responsibilities\*

- Pathobiology of Human Disease
- Environmental and Occupational Toxicology
- Industrial Toxicology
- Xenobiotic Metabolism
- Industrial Hygiene
- Environmental and Occupational Health
- Directed Research
- Special Project
- Independent Study
- Supervised Field Experience
- Thesis
- Dissertation

\*All courses are graduate level as part of the M.S., Ph.D., or M.P.H. degrees

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## University Service Activities (Selected)

- University of South Florida Committee on Campus Issues Related to Security Following 9/11/01
  - University of South Florida Chemical Safety Committee (Chair) 1994-
  - University of South Florida Faculty Senate ( 1991-1994 )
  - University of South Florida Committee on Committees (1991-1994)
  - University of South Florida Health Science Center Institutional Review Board (1991- 1993).
  - University of South Florida Laboratory Animal Medicine Ethics Committee (1993-1995).
  - Human Resources (Chair) 1986-1989
  - Computer (1987-1989)
  - Graduate Programs and Curriculum (1989-1990; 1998, 2002 )
- 

## Research Interests

Pulmonary Toxicology  
Cardiac Toxicology  
Biochemical Toxicology

## Special Research Skills

Cellular Electrophysiology; in vitro  
Toxicology; Cell Culture; Organ  
Perfusion

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## Professional Service Activities (Selected)

- Board of Director, American Heart Association, Florida Affiliate (1995-1996).
- Consultant to the Tampa Bay Poison Information Center, The Tampa General Hospital (1987-).

- Member of the State of Florida Toxic Substances Advisory Council (1992- 1996).
- Training of HazMat Specialists- City of Tampa Fire Department.
- Consultant to industry, e.g., Rohm and Haas Chemical, Honeywell .
- Consultant to the University of South Florida ( University -wide Chemical Hazards Evaluation and Development of Chemical Hygiene Plan ).
- Consultant and Expert Witness on Occupational Chemical Exposures.
- Contributor to Continuing Education Program of the American College of Chest Physicians .
- Contributor to Educational Programs of the American Lung Association, American Heart Association.
- Reviewer for Professional Publications, e.g., European Journal of Pharmacology; International Journal of Environmental Health Research., American Industrial Hygiene Association Journal, Life Sciences.
- 1995 Chairman for Cardiovascular Toxicology Program at annual meeting of the Society of Toxicology.
- Consultant to Southwest Florida Water Management District on pesticide related health effects.
- Hazardous Substances Consultant and Emergency Responder Manatee County Sheriff's Office
- Hazardous Substances Consultant and Emergency Responder City of Tampa Fire and Rescue
- Hazardous Substances Advisor and Emergency Responder to Florida Department of Environmental Protection, Division of Law Enforcement
- Hazardous Substances Consultant and Emergency Responder to City of Fort Meyers Fire Department

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## **Scholarly Activities**

### **Selected Chapters and Reviews**

- [Richards, I.S.](#), and Brooks, S.M. 1995. Respiratory Toxicology. Chapter 14, 166-180. In: Environmental Medicine: Concepts and Practice. S.M. Brooks, Ed. Mosby and Company .

- Fernandez-Caldas, E., Fox, R.W., [Richards, I.S.](#), Varney, T.C., and Brooks, S.M. 1995. Indoor Air Pollution. Chapter 37, 419-437. In: Environmental Medicine: Concepts and Practice. S.M. Brooks, Ed. Mosby and Company .
- [Richards, I.S.](#) 1991. Health effects of illicit cocaine use. Pulmonary and Critical Care Update. American College of Chest Physicians 7(2), 1-7.

### **Selected Abstracts**

- Moulvi, F.I. and [Richards, I.S.](#) 1998. Effects of eastern diamondback rattlesnake (*Crotalus adamanteus*) venom on electromechanical aspects of isolated perfused rat heart. *The Toxicologist* 42, 286.
- Sutton, E.T., Norman, J.G., Rao, P.S., Graham, L., Newton, C.A., and [Richards, I.S.](#) 1998. Pulmonary endothelium and epithelium are maintained and reduced neutrophil infiltration is found post-endotoxin in Type 1 interleukin-1 receptor knockout mice. *FASEB J.* 12: A992.
- Sutton, E.T., Price, J.M., [Richards, I.S.](#), Graham, L.B. and Rao, P.S. 1997. Serum nitric oxide (NO) levels during septic shock depends on the mode of endotoxin (ENDT) administration. *FASEB J.* 11: A80
- [Richards, I.S.](#) 1996. Ethanol potentiates the depressant effects of cocaine in human fetal myocardium in vitro. *The Toxicologist* 30, 336.
- Sutton, E.T., Norman, J.G., Glaccum, M., and [Richards, I.S.](#) 1996. Endothelial structural integrity is maintained during endotoxic shock in a Type-1 interleukin-1 receptor knockout mouse. *The Toxicologist* 30, 336.
- [Richards, I.S.](#), and Leikauf, G. 1995. Formalin induces spontaneous oscillations of membrane potential in human airway smooth muscle to Substance P. *The Toxicologist* 15 (1), 96.
- Sutton, E.T., Norman, J.G., Baker, C.H., and [Richards, I.S.](#) 1995. Structural integrity of tracheal epithelial cells in septic shock and ARDS can be maintained by blockade of the interleukin-1 receptor. *The Toxicologist* 15 (1), 97.
- [Richards, I.S.](#), Baker, N., and Leikauf, G. 1994. Formalin-induced human airway smooth muscle contractions are electrically coupled to membrane depolarizations. *The Toxicologist*, 14 (1), 253.
- Sutton, E.T., Baker, C.H., and [Richards, I.S.](#) 1994. Structural damage to epithelial cells in septic shock and ARDS can be caused by interaction of neutrophils and endotoxin. *The Toxicologist*. 14 (1), 81.

- Rosbolt, J., and Richards, I.S. 1993. Formalin produces alterations in human smooth muscle contractility. *The Toxicologist* 13, 261.
- Richards, I.S. 1991. Sodium metabisulfite produces depolarizations and contractions in human airways smooth muscle via a histaminergically-mediated mechanism. *American Review of Respiratory Disease*. 143 (4) A 755.
- Nanasi, P.P., Knilans, T.K., Varro, A., Richards, I.S., and Lathrop, D.A. 1990. Biphasic effect of tetraethylammonium on canine purkinje fiber action potential configuration. *Journal of Molecular and Cellular Cardiology* 22, 53.
- Richards, I.S., Kulkarni, A.P., and Pierce, R. 1990. Florida-Red Tide toxins-induce airway smooth muscle depolarization. *The Toxicologist* 10, 175.
- Richards, I.S. 1989. Ethanol alters membrane potentials of airway smooth muscle and suppresses tetraethylammonium-induced bronchospasm. *American Review of Respiratory Disease* 139, 613 .
- Richards, I.S., and Kulkarni, A.P. 1989. Cocaine-induced alterations of electromechanical activity in human fetal heart in vitro. *The Toxicologist* 9, 147.

### **Selected Manuscripts**

- Sutton, E.T., Norman, J., Rao, P.S., Graham, L.B., and Richards, I.S. 2000. Pulmonary endothelial and epithelial integrity and neutrophil infiltration following endotoxin in interleukin-1 receptor knockout mice. *Shock* 13, 117-125.
- Brooks, S.M., Fox, R., Lockey, R., Hammad, Y., Richards, I.S., Giovinco-Barbas and Jenkins, K. 1998. The spectrum of irritant-induced asthma: Sudden and not-so-sudden onset and the role of allergy. *Chest* 113, 42-49.
- Richards, I.S. 1997. Ethanol potentiates the depressant effect of cocaine in human fetal myocardium in vitro. *Journal of Toxicology-Clinical Toxicology* 35, 365-371.
- Sutton, E. Truitt, Norman, J.G., Newton, A., Hellerman, G.R. and Richards, I.S. 1997. Endothelial structural integrity is maintained during endotoxic shock in a transgenic mouse model with type I IL-1 receptor "knockout". *Shock* 7 (2) 105-110.

- Dietrich, C., [Richards, I.S.](#), Bernard, T., and Y. Hammad. 1996. Human stress protein response to formaldehyde exposure. *Exp. Toxic Pathol.* 48, 518.
- [Richards, I.S.](#), and Brooks, S.M. 1995. Respiratory Toxicology. Chapter 14, 166-180. In: *Environmental Medicine: Concepts and Practice*. S.M. Brooks, Ed. Mosby and Company
- Fernandez-Caldas, E., Fox, R.W., [Richards, I.S.](#), Varney, T.C., and Brooks, S.M. 1995. Indoor Air Pollution. Chapter 37, 419-437. In: *Environmental Medicine: Concepts and Practice*. S.M. Brooks, Ed. Mosby and Company .
- Leikauf, G.D., Swiecicowski, A.L., Rosbolt, J.P., and [Richards, I.S.](#) 1993. Aldehyde-Induced airway hyperreactivity. *Proc. Hung. Biochem. Soc.* 3, 235-240.
- Kulkarni, A.P., Cai, Y., and [Richards, I.S.](#) 1992. Rat pulmonary lipooxygenase: dioxygenase activity and role in xenobiotic metabolism. *International Journal of Biochemistry* 24, 255- 261.
- Mitra, A., [Richards, I.S.](#), Kitchin, K., Conolly, R., and Kulkarni, A.P. 1992. Mirex induces ornithine decarboxylase activity in female rat liver. *Journal of Biochemical Toxicology* 5, 1- 6.
- Mitra, A., [Richards, I.S.](#), and Kulkarni, A.P. 1992. Dose, time and route dependency of the induction of rat hepatic ornithine decarboxylase by 12-O-tetradecanoylphorbol 13-acetate. *Drug and Chemical Toxicology.* 15, 67-79.
- Kulkarni, A.P., Edwards, J.H., and [Richards, I.S.](#) 1992. Metabolism of 1,2-dibromoethane in the human fetal liver. *General Pharmacology* 23, 1-5.
- Nanasi, P.P., Knilans, T.K., [Richards, I.S.](#), Varro, A., and Lathrop, D.A. 1992. Biphasic effect of tetraethylammonium on canine purkinje action potential configuration. *General Pharmacology* 23, 733-738.
- [Richards, I.S.](#), Kulkarni, A., and Brooks, S.M. 1991. Human fetal tracheal smooth muscle produces spontaneous electromechanical oscillations that are Ca<sup>2+</sup>-dependent and cholinergically-potentiated. *Developmental Pharmacology and Therapeutics* 16, 22-28.
- [Richards, I.S.](#), Kulkarni, and Bremner, W.F. 1990. Cocaine induced arrhythmias in human fetal myocardium in vitro: Potential mechanism for fetal death in utero. *Pharmacology and Toxicology* 66, 150-154.

- [Richards, I.S.](#), Kulkarni, A., Brooks, S., and Pierce, R. 1990. Florida red-tide toxin (brevetoxins) produce depolarization of airway smooth muscle. *Toxicon* 28, 1105-1111.
- [Richards, I.S.](#), Miller, L., Solomon, D., Kulkarni, A., Brooks, S., and Sperelakis, N. 1990. Azelastine inhibits acetylcholine-induced contraction and depolarization in human airway smooth muscle. *European Journal of Pharmacology* 186, 331-334.
- Kulkarni, A.P., Mitra, A., Chaudhuri, J., Byczkowski, J.Z. and [Richards, I.S.](#) 1990. Hydrogen peroxide: A potent activator of dioxygenase activity of soybean lipoxygenase. *Biochemical and Biophysical Research Communications* 166, 417-423.
- Kulkarni, A.P., Chaudhuri, J., Mitra, A., and [Richards, I.S.](#) 1989. Dioxygenase and peroxidase activities of soybean lipoxygenase: Synergistic interaction between linoleic acid and hydrogen peroxide. *Research Communications in Chemical Pathology and Pharmacology* 66, 287-295.
- [Richards, I.S.](#), Kulkarni, A., and Brooks, S.M. 1989. Ethanol-induced bronchodilatation in TEA-treated canine tracheal smooth muscle is mediated by a  $\beta$ -adrenoceptor-dependent mechanism. *European Journal of Pharmacology* 167, 155-160.
- [Richards, I.S.](#), Kulkarni, A., Brooks, S.M., Lathrop, D.A., Bremner, W.F., and Sperelakis, 1989. A moderate concentration of ethanol alters cellular membrane potentials and decreases contractile force of human fetal heart. *Developmental Pharmacology and Therapeutics* 13, 51-56.

### **Presentations at Professional Meetings (Selected)**

- Pulmonary endothelium and epithelium are maintained and reduced neutrophil infiltration is found post-endotoxin in Type 1 interleukin-1 receptor knockout mice. Society for Experimental Biology. 1998.
- "Ethanol Potentiates the Depressant Effects of Cocaine in Human Fetal Myocardium in vitro". Society of Toxicology. 3/14/96. Anaheim, California.
- "Endothelial Structural Integrity is Maintained During Endotoxic Shock in a Type-1 Interleukin-1 Receptor Knockout Mouse". Society of Toxicology. 3/14/96. Anaheim, California.

- "Cholinergically Mediated Contractile Response of in vitro Porcine Airway Smooth Muscle to Biocides". Society of Toxicology. 7/5/95 Seattle, Washington.
- "Formalin Induces Spontaneous Oscillations of Membrane Potential in Human Airway Smooth Muscle to Substance P". Society of Toxicology. 3/6/95 Baltimore, Maryland.
- "Structural Integrity of Tracheal Epithelial Cells in Septic Shock and ARDS Can Be Maintained By blockade of the Interleukin- 1 Receptor". 5th International Symposium on the Correlation Between in vitro and in vivo Investigations in Inhalation Toxicology. Hanover, Germany. 2/22/95.
- "Human Stress Protein Response to Formaldehyde Exposure". American Industrial Hygiene Conference. 5/24/94. Anaheim, California.
- "Formalin Produces Alterations in Human Airway Smooth Muscle Contractility". Society of Toxicology. 3/17/94. Dallas, Texas.
- "Safety Evaluation of Carpet Emissions and Other Indoor Pollutants: Formalin -Induced Human Airway Smooth Muscle Contractions Are Electrically Coupled to Membrane Depolarizations". Society of Toxicology. 3/16/94. Dallas, Texas.
- Effect of Formaldehyde Vapors on Stress Response in Human Leukocytes". Society of Toxicology. 3/17/93. New Orleans, Louisiana.
- "Cholinergic Potentiation of Spontaneous Electromechanical Activity in Human Fetal Airway Smooth Muscle". Society of Toxicology. 2/14/90, Miami, Florida.
- "Florida Red-Tide Toxins Induce Airway Smooth Muscle Depolarization". 1990. Society of Toxicology.
- Biphasic Effect of Tetraethylammonium on Canine Purkinje Fiber Action Potential Configuration. American Society of Cardiology / International Society for Heart Research. April 10, 1990. Chicago, Illinois.
- "Structural Damage To Epithelial Cells In Septic Shock And ARDS Can Be Caused By Interaction Of Neutrophils (PMN) and Endotoxin (ENDT)". Society of Toxicology, 3/1/89, Atlanta, Georgia.
- "Cocaine-induced alterations of electromechanical activity in human fetal heart". 1989 Florida Conference on Poison Information: Biological and Behavioral Aspects of Toxic Exposures. 5/13/89. Howard Johnson Plaza-Hotel, Tampa, Florida.



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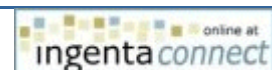
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**1:** [Toxicol Ind Health](#). 2006 Mar;22(2):59-63.



Lin

**Formalin produces depolarizations in human airway smooth muscle in vitro.**

**Richards IS, DeHate RB.**

Department of Environmental and Occupational Health, College of Public Health, University of South Florida, Tampa, FL, USA.  
 irichard@hsc.usf.edu

Respiratory irritants may result in airway smooth muscle (ASM) depolarization and bronchoconstriction. We examined the effect of formalin on membrane potentials in human ASM in two types of in vitro preparations: strip preparations, which contain functional sensory and motor nerve endings and cultured cells, which lack these nerve endings due to the tissue dissociation process. Depolarizations occurred in atropine-treated strip preparations in response to formalin exposures, but not in similarly-treated cultured cells, suggesting a role for non-cholinergic mediators in formalin-induced depolarization. It is suggested that formalin may act as an irritant to produce bronchoconstriction that is mediated by the release of endogenous substance P (SP) from peripheral sensory nerve endings. This is supported by our observation that exogenous SP produced depolarizations of a magnitude similar to those produced by formalin in both strip preparations and cultured cells. In addition, capsaicin, which releases endogenous SP from nerve endings, produced depolarizations of a magnitude similar to formalin in strip preparations, but was without effect in cultured cells.

PMID: 16716036 [PubMed - indexed for MEDLINE]

## Related Links

Florida red-tide toxins (brevetoxins) produce depolarization of airway smooth muscle. [Toxicol. 1990]

Potassium channel modulation: a new drug principle for regulation of smooth muscle contractility. Studies on isolated airways and arteries. [Dan Med Bull. 1996]

Capsaicin-sensitive stretch responses in ferret trachealis muscle. [J Physiol. 1994]

Effects of tachykinins and capsaicin on the mechanical and electrical activity of the guinea-pig isolated trachea. [Br J Pharmacol. 1997]

Evidence for non-adrenergic non-cholinergic contractile responses in bovine and porcine trachea. [Am Rev Respir Dis. 1997]

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**2:** [Shock](#). 2000 Feb;13(2):117-25.

Lin

**Pulmonary endothelial and epithelial integrity and neutrophil infiltration after endotoxin in interleukin-1 receptor knockout mice.**

## Related Links

Endothelial structural integrity is maintained during endotoxic shock in an interleukin-1 type 1 receptor knockout mouse. [Am J Physiol. 2000]

[Sutton ET](#), [Norman J](#), [Rao PS](#), [Graham LB](#), [Newton CA](#), [Richards IS](#).

Department of Physiology and Biophysics, College of Medicine, University of South Florida, Tampa 33612, USA.

Previously we found the structural integrity of the aortic endothelium was maintained after the administration of endotoxin in type 1 interleukin-1 (IL-1) receptor knockout mice. In this study, we investigated further the integrity of pulmonary vascular endothelium, airway epithelial, pulmonary microvasculature, and neutrophil infiltration into the microvasculature and respiratory air spaces. Adult male C57BL/129J wild-type mice and C57BL/129J knockout mice possessing a homozygous deletion of the type 1 IL-1 receptor received the following intraperitoneal injections; 1) *Escherichia coli* endotoxin (ENDT) (10 mg/kg), 2) ENDT (2 mg/kg given for 4 days), or (3) saline vehicle. Wild-type and knockout control animals receiving saline vehicle showed normal endothelial and epithelial ultrastructure with intact membranes. Pulmonary endothelial cell damage was found only in wild-type mice given a single 10 mg/kg endotoxin dose. Airway epithelial damage was found only in wild-type mice given a repetitive dose of endotoxin (2 mg/kg for 4 days). Neutrophil infiltration increased only in mice given a single dose of endotoxin (10 mg/kg) with the wild-type increasing by 32% and the knockouts by 6% compared with the saline control for that group respectively. Serum IL-6 and nitric oxide (indicators of septic shock severity and lethality) significantly increased only in the mice given 10 mg/kg of endotoxin. The maintenance of pulmonary endothelial and epithelial cell integrity and the decrease of neutrophil infiltration in the IL-1 knockout mice suggest that IL-1 contributes significantly to the severity of endotoxin-induced sepsis.

PMID: 10670841 [PubMed - indexed for MEDLINE]

knockout mouse. [Shock. 1997

Early response cytokines and innate immunity: essential roles for TNF receptor 1 and type I IL-1 receptor during *Escherichia coli* pneumonia in mice. [J Immunol. 2001

Inducible nitric oxide synthase is not required in the development of endotoxin tolerance in [Shock. 2002

Endogenous IL-17 as a mediator of neutrophil recruitment caused by endotoxin exposure in mouse airways. [J Immunol. 2003

Attenuation of hypoxic pulmonary vasoconstriction by endotoxemia requires 5-lipoxygenase. [Am J Physiol. 2001

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**3:** [Shock](#). 1997 Feb;7(2):105-10.

Lin

**Endothelial structural integrity is maintained during endotoxic shock in an interleukin-1 type 1 receptor knockout mouse.**

[Sutton ET](#), [Norman JG](#), [Newton CA](#), [Hellermann GR](#), [Richards IS](#).

Department of Physiology and Biophysics, College of Medicine, University of South Florida, Tampa 33612, USA.

The derangement of arterial endothelial cell morphology is a good indicator of a severe shock state. Because interleukin (IL)-1 has been implicated in this process, we examined the structural integrity of aortic endothelial cells in conjunction with serum IL-6 concentrations and nitric oxide levels, which are known to increase during endotoxemia in animals genetically devoid of the type 1 IL-1 receptor. Endotoxin (10 mg/kg *Escherichia coli*, injected intraperitoneally) (LD100) or saline vehicle was administered to adult male C57BL/129J wild-type control mice and C57BL/129J knockout mice possessing a

### Related Links

Pulmonary endothelial and epithelial integrity and neutrophil infiltration after endotoxin in interleukin-1 receptor knockout mice. [Shock. 2000

Maintenance of dynamic microvascular function and structure in a rat model of endotoxic shock by blockade of th interleukin-1 receptor. [Shock. 1995

Endothelial nitric oxide synthase-deficient mice exhibit increased susceptibility to endotoxin-induced acute renal failure. [Am J Physiol. 2004

Resistance to endotoxin shock in transgenic mice overexpressing endothelial nitric oxide synthase. [Am J Physiol. 2000

Endotoxemic renal failure in mice: Role of tumor necrosis factor

homozygous deletion of the type 1 IL-1 receptor. The integrity of the aortic endothelium was determined by comparisons of ultrastructure. Mice injected with sterile vehicle showed normal endothelial ultrastructure with intact membranes. Wild-type and knockout control animals receiving saline vehicle showed a complete aortic endothelium (29.11 +/- .27 and 30.85 +/- .21 intact endothelial cells per millimeter of internal elastic lamina (IEL), respectively,  $p = \text{N.S.}$ ). Endotoxin-treated wild-type animals showed extensive endothelial damage with most sections showing only denuded IEL on the luminal surface (1.83 +/- .38 cells/mm IEL,  $p < .001$  vs. control). Knockout animals treated with endotoxin showed complete maintenance of endothelial structural integrity (34.08 +/- .57 cells/mm IEL,  $p < .001$  vs. endotoxin-treated wild type) with ultrastructural morphology appearing identical to those given saline vehicle. Also, no apparent correlation was observed between serum IL-6 concentrations or serum nitric oxide levels and aortic endothelial damage. The maintenance of endothelial integrity in animals devoid of the IL-1 receptor confirms earlier observations of endothelial cell protection with IL-1 receptor antagonism and suggests that IL-1 contributes significantly to sepsis-induced endothelial damage.

PMID: 9035286 [PubMed - indexed for MEDLINE]

independent of inducible nitric oxide synthase. [Kidney Int. 2001]

[See all Related Articles...](#)

4: [J Toxicol Clin Toxicol.](#) 1997;35(4):365-9.

Lin

#### Ethanol potentiates the depressant effects of cocaine in human fetal myocardium in vitro.

**Richards IS.**

Department of Environmental and Occupational Health, College of Public Health, University of South Florida, Tampa 33612-3805, USA. irichard@com1.med.usf.edu

**BACKGROUND:** Cocaine and ethanol use is widespread in our society and is not uncommon in pregnant women. Previous work has demonstrated that acute exposure to cocaine produced significant effects on the configuration of human fetal myocardial action potentials and contractility in vitro. It has been hypothesized that these target-specific effects of cocaine may provide a plausible mechanism to account for fetal arrhythmia or sudden fetal death in utero. **OBJECTIVE:** This study was conducted to determine if treatment with a low concentration of ethanol (200 mg/L) would predispose human fetal myocardium to cocaine-induced toxicity in vitro. **METHODS:** Fetal hearts (12-14 weeks) were obtained at the time of elective abortion and transported to the laboratory in cold physiological salt solution. The force of muscle contractions and transmembrane potentials of ventricular walls were studied during external electrical stimulation in a specially constructed superfusion chamber. **RESULTS:** When a concentration of cocaine (200 micrograms/L physiological salt solution) that singly produced only modest myocardial depression was used in combination with a concentration of ethanol which alone produced nonsignificant changes,

#### Related Links

Cocaine-induced arrhythmia in human foetal myocardium in vitro possible mechanism for foetal death in utero. [Pharmacol Toxicol. 1990]

A moderate concentration of ethanol alters cellular membrane potentials and decreases contractile force of human fetal heart. [Dev Pharmacol Ther. 1989]

Individual and combined effects of ethanol and cocaine on intracellular signals and gene expression. [Neurosci Biomed Psychol. 1996]

Cocaine and alcohol synergism in taste perception learning. [Pharmacol Biochem Behav. 1998]

Prenatal cocaine, alcohol, and undernutrition differentially alter mineral and protein content in fetal rats. [Pharmacol Biochem Behav. 1998]

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marked depression and block of action potentials and contractility resulted. CONCLUSION: The combined use of ethanol and cocaine produces fetal myocardial depression which is greater than that predicted from the effects of these chemicals individually and may have significant in utero implications.

PMID: 9204096 [PubMed - indexed for MEDLINE]

5: [Exp Toxicol Pathol.](#) 1996 Nov;48(6):518-9.

Lin

**Human stress protein response to formaldehyde exposure.**

[Dietrich CJ](#), [Richards IS](#), [Bernard TE](#), [Hammad YY](#).

College of Public Health, University of South Florida, Tampa, USA.

PMID: 8954337 [PubMed - indexed for MEDLINE]

**Related Links**

Biochemical analysis of the stress protein response in human oesophageal epithelium. [Gut. 1997]

Expression of cytoprotective proteins, heat shock protein 70 and metallothioneins, in tissues of *Ostrea edulis* exposed to heat and heavy metals. Stress Chaperones. 2004

Heat shock proteins hsp32 and hsp70 as biomarkers of an early response? In vitro induction of heat shock proteins after exposure of cell culture to carcinogenic compounds and their real mixtures. [Mutat Res. 2003]

Heat shock protein 70 as an indicator of early lung injury caused by exposure to arsenite. [Mol Cell Biochem. 2005]

Glutamine protects articular chondrocytes from heat stress and NO-induced apoptosis with HSP70 expression. Osteoarthritis Cartilage. 2006

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6: [Gen Pharmacol.](#) 1992 Jul;23(4):733-8.

Lin

**Biphasic effect of tetraethylammonium on canine purkinje fibre action potential configuration.**

[Nanasi PP](#), [Knilians TK](#), [Richards IS](#), [Varro A](#), [Lathrop DA](#).

Department of Paediatrics, University of Cincinnati College of Medicine, Ohio.

1. Using conventional microelectrode techniques a biphasic effect of tetraethylammonium (5 mmol/l) on the configuration of action potentials recorded from isolated canine Purkinje fibres: action potentials were first shortened (early effect) and then lengthened (late effect) by tetraethylammonium. 2. The early effect of tetraethylammonium also included lengthening of phase 1 duration and elevation of the plateau amplitude. These early effects reached steady-state within the first 3 min of superfusion and were readily reversed within 3 min of initiating washout of the drug. 3. The late effect (gradual

**Related Links**

Electrophysiological effects of cesium and tetraethylammonium in canine Purkinje fibres. [J Cardiovasc Pharmacol. 1991]

Electrophysiological effects of EGIS-7229, a new antiarrhythmic agent, in isolated mammalian and human cardiac Purkinje fibres. [J Pharmacol Ther. 1997]

Different mechanisms underlying the repolarization of narrow and wide action potentials in pyramidal cells and interneurons of cat motor cortex. [Neuroscience. 1996]

Differences in the electrophysiologic response of four canine ventricular cell types to alpha 1-adrenergic agonists. [Circulation. 1999]

Age-related changes in Purkinje fiber action potentials of adult

lengthening of repolarisation during phase 3) failed to reach steady-state within the initial 60 min of superfusion and was not reversible. 4. The early effects of tetraethylammonium were more marked at slow driving rates and were not affected by blockade of alpha- and beta-adrenoceptors using 1 mumol/l phentolamine and 1 mumol/l propranolol. 5. The early effects of tetraethylammonium were mimicked by 4-aminopyridine (0.5 mmol/l), and in the presence of 4-aminopyridine tetraethylammonium failed to induce further changes in action potential morphology. 6. The early effects of tetraethylammonium may be due to inhibition of the transient outward current. 7. The rapid onset and reversibility of these early effects suggest that tetraethylammonium may act from outside the cell membrane.

PMID: 1356876 [PubMed - indexed for MEDLINE]

dogs.

[Circ Res. 1978

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**7:** [Int J Biochem.](#) 1992 Feb;24(2):255-61.

Lin

**Rat pulmonary lipoxygenase: dioxygenase activity and role in xenobiotic metabolism.**

[Kulkarni AP](#), [Cai Y](#), [Richards IS](#).

Department of Environmental and Occupational Health, College of Public Health, University of South Florida, Tampa 33612.

1. Dioxygenase activity and the ability of pregnant rat lung lipoxygenase to oxidize xenobiotics were examined in vitro under a variety of experimental conditions. 2. More than 90% of the dioxygenase activity towards linoleic acid in the lung homogenate was found to be associated with the cytosolic fraction. The cytosolic enzyme exhibited pH optima at 6.5 and 9.5, the activity being two-fold greater at pH 9.5. To observe maximal dioxygenase activity (about 0.7 mumol of 13-hydroperoxylinoleic acid formed/min per mg protein) at pH 9.5, the presence of 6.0 mM linoleic acid was required. 3. Benzidine oxidation occurred at maximal rate of pH 6.5 when the reaction medium contained 1.0 mM benzidine and 13.5 mM linoleic acid. All eight xenobiotics tested were oxidized at significant rates by the lung cytosolic lipoxygenase. 4. Both dioxygenase activity and benzidine oxidation were inhibited by the inhibitors of lipoxygenase, viz. nordihydroguaiaretic acid, BHT, caffeic acid, esculetin, and gossypol, in a concentration-dependent manner. 5. The results suggest that oxidation of xenobiotics by lipoxygenase may be an important pathway of metabolism in the mammalian lung.

PMID: 1733791 [PubMed - indexed for MEDLINE]

**Related Links**

Lipoxygenase activity in rat embryos and its potential for xenobiotic oxidation. [Int J Neonate.](#) 1993

Dioxygenase and co-oxidase activities of rat hepatic cytosolic lipoxygenase. [J Biochem Toxicol.](#) 1994

Dioxygenase and hydroperoxidase activities of rat brain cytosolic lipoxygenase. [Pathol Pharmacol.](#) 1992

Hydroperoxidase activity of lipoxygenase: a potential pathway for xenobiotic metabolism in the presence of linoleic acid. [Pathol Pharmacol.](#) 1988

Isolation and some properties of dioxygenase and co-oxidase activities of adult human liver cytosolic lipoxygenase. [Toxicol.](#) 1996

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**8:** [Gen Pharmacol.](#) 1992 Jan;23(1):1-5.

Lin

**Metabolism of 1,2-dibromoethane in the human fetal liver.**

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A novel model to assess

**Kulkarni AP, Edwards J, Richards IS.**

Florida Toxicology Research Center, College of Public Health, University of South Florida, Tampa 33612.

1. Toxicity of 1,2-dibromoethane requires bioactivation via glutathione S-transferase. Since this enzyme is undetectable in the fetus of several laboratory animal species during early gestation, in vitro studies were carried out with human fetal liver to assess potential fetotoxicity. 2. Glutathione S-transferase occurs abundantly in the human fetal liver cytosol and its titer is equal to or exceeds that found in adult human liver when estimated using 1-chloro-2,4-nitrobenzene as the second substrate. 3. Human fetal liver cytosolic glutathione S-transferase metabolized 1,2-dibromoethane with a high efficiency (mean +/- SD specific activity of 3.10 +/- 0.83 nmol/min/mg protein). This reaction was enzymatic in nature and the rate of conjugation was proportional to the concentration of reduced glutathione, 1,2-dibromoethane and the enzyme present in the reaction medium. 4. A significant bioactivation with a possibility of only limited detoxication via cytochrome P-450-dependent oxidation suggests that human fetus may be at greater risk from 1,2-dibromoethane toxicity than adult.

PMID: 1592216 [PubMed - indexed for MEDLINE]

developmental toxicity of dihaloalkanes in humans: bioactivation of 1,2-dibromoethane by the isozymes of human fetal liver glutathione S-transferase. *Environ Health Perspect.* 1992

Rat hepatic glutathione S-transferase-mediated embryotoxic bioactivation of ethylene dibromide. *Teratology.* 1992

The in vitro metabolism and bioactivation of 1,2-dibromoethane (ethylene dibromide) by human liver. *Biochem Biophys Res Commun.* 1986

Glutathione-mediated binding of dibromoalkanes to DNA: specificity of rat glutathione-S-transferase and dibromoalkane substrates. *Carcinogenesis.* 1984

[35S]-labeling of the Salmonella typhimurium glutathione pool to assess glutathione-mediated DNA binding by 1,2-dibromoethane. *J Biol Chem.* 2003

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**9:** [Dev Pharmacol Ther.](#) 1991;16(1):22-8.

Lin

**Human fetal tracheal smooth muscle produces spontaneous electromechanical oscillations that are Ca<sup>2+</sup> dependent and cholinergically potentiated.**

**Richards IS, Kulkarni A, Brooks SM.**

College of Public Health, University of South Florida, Tampa.

Although the electromechanical properties of, and the cholinergic innervation to adult airway smooth muscle has been extensively studied, the little information is available on developing human airway smooth muscle, and the role of cholinergic mechanisms in regulating bronchomotor tone. A total of 7 tracheae obtained at the time of elective abortion and between 12-16 weeks of gestational development were used in this study. For each trachea, muscle tension and transmembrane potentials were measured simultaneously using an isometric force transducer and a standard 3-M KCl-filled glass microelectrode. All preparations showed spontaneous electrical oscillations approximately 8 mV in amplitude, which could be increased using electrical field stimulation, or exogenously applied acetylcholine. This was accompanied by a corresponding increase in muscle tension. Atropine (0.1 microM) abolished this potentiation, but had no apparent effect on the oscillations. Slow-wave activity was completely suppressed in the absence of extracellular Ca<sup>2+</sup>, or in the presence of verapamil (1 microM) or quinidine (1 microM). It appears that these oscillations of membrane potential may be potentiated by cholinergic mechanisms which regulate cell membrane ion

**Related Links**

Phorbol ester effects on coupling mechanisms during cholinergic contraction of swine tracheal smooth muscle. *J Physiol.* 1989

Electromechanical effects of leukotriene D4 on ferret tracheal muscle and its muscarinic responsiveness. *Lung.* 1989

Excitation and contraction in bovine tracheal smooth muscle. *J Physiol.* 1975

Postjunctional alpha 1- and beta-adrenoceptor effects of noradrenaline on electrical slow waves and phasic contractions of cat colon circular muscle. *Br J Pharmacol.* 1995

Azelastine and desmethylazelastine suppress acetylcholine-induced contraction and depolarization in human airway smooth muscle. *Am J Respir Crit Care Med.* 1990

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channels, thus serving to change excitability in a rhythmic manner.

PMID: 1879248 [PubMed - indexed for MEDLINE]

10: [Eur J Pharmacol.](#) 1990 Sep 21;186(2-3):331-4.

Lin

**Azelastine and desmethylazelastine suppress acetylcholine-induced contraction and depolarization in human airway smooth muscle.**

**Richards IS, Miller L, Solomon D, Kulkarni A, Brooks S, Sperelakis N.**

Department of Environmental and Occupational Health, College of Public Health, University of South Florida, Tampa 33612.

We examined the effects of a new anti-asthmatic drug, azelastine, and its principal metabolite, desmethylazelastine, on the in vitro electromechanical response of human airway smooth muscle during cholinergic stimulation. Membrane potential and isometric force were simultaneously measured using an intracellular microelectrode and a microforce transducer.

Desmethylazelastine significantly suppressed acetylcholine-induced depolarization and contraction at  $10^{-6}$  M, whereas azelastine produced similar results at  $10^{-4}$  M, suggesting that the metabolite may be the principal compound acting upon the airway smooth muscle cell.

PMID: 1981189 [PubMed - indexed for MEDLINE]

**Related Links**

Azelastine inhibits agonist-induced electromechanical activity in canine tracheal muscle. [Chest.](#) 1989

Effects of azelastine on contraction of guinea pig tracheal smooth muscle. [\[Eur J Pharmacol.](#) 1990

Effects of azelastine on vagal neuroeffector transmission in canine and human airway smooth muscle. [\[J Allergy Clin Immunol.](#) 1990

Influence of epithelial removal on the antihistaminic activity of azelastine in the guinea-pig airway smooth muscle. [\[Agents Actions.](#) 1993

Effects of azelastine on membrane currents in tracheal smooth muscle cells isolated from the guinea-pig. [\[Eur J Pharmacol.](#) 1994

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11: [Pharmacol Toxicol.](#) 1990 Feb;66(2):150-4.

Lin

**Cocaine-induced arrhythmia in human foetal myocardium in vitro: possible mechanism for foetal death in utero.**

**Richards IS, Kulkarni AP, Bremner WF.**

College of Public Health, Tampa, Florida 33612-3899.

We examined the acute in vitro effects of cocaine on cell membrane potentials and contractility of 12-16 week old human foetal heart, to better assess the potential for the induction of serious arrhythmia, in utero, by this abused substance. Ventricular preparations were maintained in a tissue bath, and continuously provided with oxygen and glucose during the measurement of membrane potentials with microelectrodes, and developed force of contractions with microforce transducers. Cocaine (600 ng/ml) had a significant effect on the ability of the heart to produce action potentials of normal rising velocity, amplitude, and duration. Within 90 min., all electromechanical activity had ceased. Under the conditions of our study, the effects of cocaine were reversible, however, reversibility in vitro may have no counterpart in utero, and irreversible loss of cardiac function may result.

PMID: 2315267 [PubMed - indexed for MEDLINE]

**Related Links**

A moderate concentration of ethanol alters cellular membrane potentials and decreases contractile force of human fetal heart. [\[Dev Pharmacol Ther.](#) 1989

Ethanol potentiates the depressant effects of cocaine in human fetal myocardium in vitro. [\[Toxicol Clin Toxicol.](#) 1997

Cocaine suppression of triggered activity. A possible mechanism of antiarrhythmic action. [\[Circulation.](#) 1994

Effects of gallium chloride on changes in action potentials and contraction in guinea pig ventricular muscle. [\[Cancer Res.](#) 1996

Actions of adenosine and isoproterenol on isolated mammalian ventricular myocytes. [\[Am J Physiol.](#) 1985

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12: [Toxicol.](#) 1990;28(9):1105-11.

Lin

**Florida red-tide toxins (brevetoxins) produce depolarization of airway smooth muscle.**

**Richards IS, Kulkarni AP, Brooks SM, Pierce R.**

College of Public Health, University of South Florida, Tampa 33612.

Crude preparations of brevetoxin (PBTX) produce airway contraction; however, it is not known if this toxin-induced mechanical response is coupled to changes in airway smooth muscle membrane potential. Membrane potentials and contractility of in vitro canine trachealis smooth muscle preparations were simultaneously measured with a microelectrode and microforce transducer before and during exposure to either the crude toxin (0.01-1.2 micrograms/ml), or the purified fractions PBTX-2 or PBTX-3 (0.01-0.07 micrograms/ml). Membrane potentials in cultured airway smooth muscle-reaggregate preparations were similarly studied. Toxins produced concentration-dependent depolarizations and contractions in in vitro preparations. These responses were not obtained in the presence of either the muscarinic blocking agent atropine, the sodium channel blocker tetrodotoxin (TTX), 0 mM extracellular Ca<sup>2+</sup>, or the Ca<sup>2+</sup> channel blocker verapamil. The toxins were without effect in cultured cells, whereas acetylcholine produced depolarizations which were blocked in the presence of atropine, but not TTX. This suggested the presence of functional cholinergic receptors in cultured cells, and the PBTX-induced release of endogenous acetylcholine from peripheral nerve endings in the in vitro airway smooth muscle response.

PMID: 2260108 [PubMed - indexed for MEDLINE]

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In vitro red tide toxin effects on human bronchial smooth muscles

Lower airway smooth muscle contraction induced by *Ptychodiscus brevis* (red tide) toxin. [J Allergy Clin Immunol. 1987

The site of action of *Ptychodiscus brevis* toxin within the parasympathetic axonal sodium channel h gate in airway smooth muscle. [J Allergy Clin Immunol. 1984

The mechanism of *Ptychodiscus brevis* toxin-induced rat vas deferens contraction

Effects of toxin of red tide, *Ptychodiscus brevis*, on canine tracheal smooth muscle: a possible new asthma-triggering mechanism

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13: [Res Commun Chem Pathol Pharmacol.](#) 1989 Nov;66(2):287-96.

Lin

**Dioxygenase and peroxidase activities of soybean lipoygenase: synergistic interaction between linoleic acid and hydrogen peroxide.**

**Kulkarni AP, Chaudhuri J, Mitra A, Richards IS.**

Florida Toxicology Research Center, College of Public Health, University of South Florida, Tampa 33612.

The interaction of H<sub>2</sub>O<sub>2</sub> with soyabean lipoygenase was investigated in the presence of linoleic acid. Dioxygenase activity was significantly higher at pH 9.0 than at pH 6.0. H<sub>2</sub>O<sub>2</sub> at concentrations less than 1.0 nM stimulated linoleic acid oxidation synergistically and the magnitude of synergism was higher at pH 9.0. Linoleic acid dependent peroxidase activity towards benzidine, guaiacol, tetramethylbenzidine (TMBD) and tetramethylphenylenediamine (TMPD) was higher at pH 9.0, whereas pyrogallol and ABTS oxidation rates were higher at pH 6.0. H<sub>2</sub>O<sub>2</sub> supported oxidation of benzidine, guaiacol, pyrogallol and ABTS was higher at pH 6.0,

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The effect of linoleic acid on pH inside sodium bis(2-ethylhexyl) sulfosuccinate reverse micelles in isoctane and on the enzymic activity of soybean lipoygenase

Aldrin epoxidation. Catalytic potential of lipoygenase coupled with linoleic acid oxidation

whereas TMPD, TMBD exhibited higher oxidation rates at pH 9.0. H<sub>2</sub>O<sub>2</sub> in the presence of linoleic acid produced synergism in xenobiotic metabolism and depending upon the substrate in question upto 11-fold increase in oxidation rate was noted.

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**14:** [Eur J Pharmacol.](#) 1989 Aug 11;167(1):155-60.

**ELSEVIER** Lin  
FULL-TEXT ARTICLE

**Ethanol-induced bronchodilatation in TEA-treated canine tracheal smooth muscle is mediated by a beta-adrenoceptor-dependent mechanism.**

**Richards IS, Kulkarni AP, Brooks SM.**

Department of Environmental and Occupational Health, University of South Florida, College of Public Health, Tampa 33612-3899.

The effects of moderate concentrations of ethanol (8-34 mM) on the electromechanical activity of airway smooth muscle cells of canine trachealis, stimulated by the spasmogen tetraethylammonium (TEA), are described for in vitro and cultured reaggregate preparations. Ethanol produced a concentration-dependent hyperpolarization, and suppression of action potentials in smooth muscle preparations, in vitro, whereas it was without effect in cultured airway smooth muscle cells. In the presence of the beta-adrenoceptor antagonist propranolol (1 microM), ethanol had no effect on in vitro preparations. Isoproterenol (0.1 microM) produced hyperpolarization and suppression of action potentials in airway smooth muscle of both preparations. These effects were not observed when propranolol was additionally present. This suggests that both in vitro, and cultured airway smooth muscle preparations maintained their beta-receptors, and that ethanol caused the release of endogenous catecholamine from adrenergic nerve endings which apparently remained intact in in vitro, but not in cultured airway smooth muscle preparations.

PMID: 2550255 [PubMed - indexed for MEDLINE]

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The relaxant action of nicorandil in guinea-pig isolated trachealis. [\[Br J Pharmacol. 1986\]](#)

Electrophysiological and other aspects of the relaxant action of isoprenaline in guinea-pig isolated trachealis. [\[Br J Pharmacol. 1985\]](#)

cAMP suppresses CA<sup>2+</sup>-dependent electrical activity of airway smooth muscle induced by ATPA. [\[Am J Physiol. 1987\]](#)

Relaxant actions of isoprenaline on guinea-pig isolated tracheal smooth muscle. [\[Br J Pharmacol. 1995\]](#)

8-Bromo-cyclic GMP abolishes TEA-induced slow action potential in canine trachealis muscle. [\[Br J Pharmacol. 1986\]](#)

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**15:** [Dev Pharmacol Ther.](#) 1989;13(1):51-6.

Lin

**A moderate concentration of ethanol alters cellular membrane potentials and decreases contractile force of human fetal heart.**

**Richards IS, Kulkarni A, Brooks SM, Lathrop DA, Bremner WF, Sperelakis N.**

College of Public Health, University of South Florida, Tampa.

The effects of ethanol, although well studied in the adult myocardium, have been little studied in fetal tissue. Experiments in pregnant animals suggest that ethanol compromises fetal myocardial performance, in utero;

#### Related Links

Cocaine-induced arrhythmia in human foetal myocardium in vitro possible mechanism for foetal death in utero. [\[Pharmacol Toxicol. 1990\]](#)

Ethanol potentiates the depressant effects of cocaine in human fetal myocardium. [\[Toxicol Clin Toxicol. 1997\]](#)

Effects of alcohol on intracellular pH regulators and electromechanical parameters in human myocardium. [\[Am J Physiol Exp Res. 2005\]](#)

however, the physiological mechanism(s) remains obscure. The present report examines, *in vitro*, the effects of a moderate concentration of ethanol (20 mM) directly on cell membrane potentials and contractility of human fetal left ventricle as determined using intracellular microelectrodes and microforce transducers. We observed significant decreases in action potential amplitude, upstroke velocity, duration of repolarization, and the force of contractions. These effects were reversible. As ethanol crosses the placenta, our findings suggest that moderate concentrations of ethanol, as occur during 'social drinking', may temporarily compromise fetal myocardial performance *in utero*.

PMID: 2776585 [PubMed - indexed for MEDLINE]

Effect of ethanol on guinea pig ventricular action potentials. *Electrocardiol.* 1981

The action of ethanol upon the action potential and contraction of ventricular muscle. *Receptiv Struct Metab.* 1975

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**16:** [J Appl Physiol.](#) 1987 Jan;62(1):175-9. Lin



**cAMP suppresses CA<sup>2+</sup>-dependent electrical activity of airway smooth muscle induced by TEA.**

**Richards IS, Ousterhout J, Sperelakis N, Murlas CG.**

Using intracellular microelectrodes, we investigated whether exogenous dibutyryl adenosine 3',5'-cyclic monophosphate (DBcAMP) or forskolin influenced the electrical effects of tetraethylammonium (TEA) on canine tracheal smooth muscle. We found that 20 mM TEA depolarized airway smooth muscle cells from a resting membrane potential ( $E_m$ ) of  $-59 \pm 4$  mV (mean  $\pm$  SD) to  $-45 \pm 2$  mV and caused spontaneous action potentials (AP's) to develop, which were  $33 \pm 2$  mV in amplitude. These were totally abolished in 0 Ca<sup>2+</sup> solution. DBcAMP (1 mM) suppressed the development of this TEA-induced electrical activity and the phasic contractions electrically coupled to it. DBcAMP had no significant effect on  $E_m$  in the absence of TEA however. Forskolin (1  $\mu$ M) produced similar effects. Our findings suggest that Ca<sup>2+</sup> is the principal ion responsible for the inward current associated with the TEA-induced AP's in airway smooth muscle, and that adenosine 3',5'-cyclic monophosphate may suppress the electrogenesis of this current.

PMID: 3031001 [PubMed - indexed for MEDLINE]

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8-Bromo-cyclic GMP abolishes TEA-induced slow action potential in canine trachealis muscle. *Electrocardiol.* 1986

cAMP accelerates the decay of stretch-activated inward currents in guinea-pig urinary bladder myocytes. *[J Physiol.* 1995

Excitation and contraction in bovine tracheal smooth muscle. *[J Physiol.* 1975

Effect of tetraethylammonium on tonic airway smooth muscle: initiation of phasic electrical activity. *[Am J Physiol.* 1975

The effect of tetraethylammonium chloride on potassium permeability in the smooth muscle cell membrane of canine trachealis. *[Electrocardiol.* 1981

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**17:** [Eur J Pharmacol.](#) 1986 Sep 9;128(3):299-302. Lin

**8-Bromo-cyclic GMP abolishes TEA-induced slow action potentials in canine trachealis muscle.**

**Richards IS, Murlas C, Ousterhout JM, Sperelakis N.**

Using intracellular microelectrodes, we investigated whether 8-bromo-guanosine 3':5'-cyclic monophosphate (cGMP) influenced the electromechanical effects of tetraethylammonium (TEA) on canine tracheal smooth

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cAMP suppresses CA<sup>2+</sup>-dependent electrical activity of airway smooth muscle induced by TEA. *J Appl Physiol.* 1987

Effects of nitric oxide (NO) and NO donors on the membrane conductance of circular smooth muscle cells of the guinea-pig proximal colon. *[Br J Pharmacol.* 1996

muscle. We found that 20 mM TEA depolarized airway smooth muscle cells from  $-58 \pm 3$  mV (means  $\pm$  S.D.) to  $-44 \pm 2$  mV and caused spontaneous action potentials (APs) to develop which were  $31 \pm 2$  mV in amplitude. These APs, and the phasic contractions electrically coupled to them, were totally abolished in buffer containing 0.1 mM cGMP. Our findings suggest that cGMP markedly affects the channels mediating TEA-induced APs in airway smooth muscle.

PMID: 3024999 [PubMed - indexed for MEDLINE]

Ethanol-induced bronchodilatation in TEA-treated canine tracheal smooth muscle is mediated by a beta-adrenoceptor-dependent mechanism. [Eur J Pharmacol. 1989]

Cyclic nucleotides augment the phasic action of tetraethylammonium on guinea-pig trachealis. [Eur J Pharmacol. 1989]

Effects of 4-aminopyridine and tetraethylammonium chloride on the electrical activity and cable properties of canine tracheal smooth muscle. [Pharmacol Exp Ther. 1983]

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**18: Urology.** 1981 Jul;18(1):1-6.

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**Current status of endocrinologic effects of vasectomy.**

**Richards IS, Davis JE, Lubell I.**

PMID: 6789528 [PubMed - indexed for MEDLINE]

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The pituitary-testicular axis in the streptozotocin diabetic male rat: evidence for gonadotroph, Sertoli cell and Leydig cell dysfunction. [Endocrinology. 1981]

Pituitary and gonadal function in prepubertal and pubertal boys with hypogonadism. [Acta Endocrinol (Copenh). 1981]

Pituitary and gonadal function in chromatin positive XXY Klinefelter's syndrome. [Exp Med. 1973]

Chromatin-negative Klinefelter's syndrome with normal testes and serum gonadotropins and testosterone. [Fertil Steril. 1971]

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Principles and Practice of

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