Curriculum Vitae

Alan Kim Johnson
May 2017

Identification Data

Born August 15, 1942
Appointed to present rank of Professor on July 1, 1982
First appointed to University of Iowa as Assistant Professor on August 1, 1973

Education

Pennsylvania State University	1960-1964	Chemistry & Life Sciences	B.S., 1964
Temple University	1964-1966	Psychology	M.A., 1966
University of Pittsburgh	1966-1970	Psychobiology	Ph.D., 1970

Academic and Professional Experience

January 1965 - August 1965: Research Assistant, Dr. Irwin M. Spigel, Temple University; "Contribution of activity and visual deprivation and social stimulation on runway performance of the painted turtle (Chrysemys); the effect of stimulant drugs on active avoidance in the painted turtle."

April 1966 - August 1966: Graduate Research Assistant, Dr. Robert A. Patton, University of Pittsburgh; "The effect of alcohol on non-discriminated avoidance in the rat."

September 1966 - August 1969: National Science Foundation Trainee; sponsor, Dr. Alan E. Fisher, University of Pittsburgh; "Effects of central chemical stimulation upon self-stimulation; effects of central chemical stimulation upon electrically elicited behaviors; taste preferences under cholinergic-induced thirst."

September 1969 - November 1970: Graduate Research Assistant, Dr. Alan E. Fisher, University of Pittsburgh.

1968 - 1970: Psychobiology Curriculum Committee, University of Pittsburgh.

Spring Term 1970, Summer Term 1970: University of Pittsburgh Teaching Fellow; taught lecture and laboratory courses in Experimental Psychology, Learning, and Motivation.

December 1970 - August 1973: NIH Postdoctoral Fellow, Institute of Neurological Sciences, University of Pennsylvania; Sponsor, Dr. Alan N. Epstein; "Neural and biochemical mechanisms of thirst."

August 1973 - June 1978: Assistant Professor, Department of Psychology, University of Iowa.

January 1 - December 31, 1977: Visiting Professor, Department of Pharmacology, University of Heidelberg, G.F.R.

July 1978 - June 1982: Associate Professor, Department of Psychology, University of Iowa.

June 1978 - May 1979: Visiting Scientist, Regional Primate Research Center, University of Washington.

May 1982 - August 1982: Visiting Associate Professor, Department of Pharmacology, University of Texas Health Sciences Center at San Antonio.

May 1982 - August 1982: Associate Research Scientist, Southwest Foundation for Research and Education, San Antonio.

July 1982 - present: Professor, Department of Psychological and Brain Sciences, University of Iowa.

Academic and Professional Experience (continued)

November 1983 - present: Professor, Department of Pharmacology, University of Iowa.
July, 2000: F. Wendell Miller Distinguished Professor, College of Liberal Arts and Sciences, University of Iowa
August, 2000 -present: Professor, Department of Health and Human Physiology, University of Iowa

Professional Organizations

The American Physiological Society
Society for Neuroscience
International Behavioral Neuroscience Society
American Heart Association
Inter-American Society of Hypertension
Society for the Study of Ingestive Behavior
Serotonin Club

Awards, Honors, Special Recognition

NIH Postdoctoral Fellowship, 1971-1973
NIMH Research Career Development Award, 1975-1980; 1980-1985
American Men and Women of Science, since 1975
Research Fellow of the Alexander von Humboldt Foundation, G.F.R., 1977-1978
The Council for High Blood Pressure Research of the American Heart Association, Elected Fellow 1978
Brain and Behavioral Sciences Associate, 1979-present
High Blood Pressure Research Medical Advisory Board, American Heart Association
Awardee of National Academy of Sciences--National Research Council Travel Grant to participate in XXVIII International Congress of Physiological Sciences, Budapest, Hungary, July 1980
National Heart, Lung, & Blood Institute, The Behavioral Medicine Branch--Task Group Member--Working Conference on Circulation, Neurobiology & Behavior, 1981
National Heart, Lung, & Blood Institute, The Behavioral Medicine Branch--Task Group Member--Working Conference on Cardiovascular Instrumentation: Applicability of New Technology to Biobehavioral Research 1982
National Heart, Lung, & Blood Institute, The Behavioral Medicine Branch of Heart & Vascular Diseases--Consultant, 1981-present
Fellow, The Cardiovascular Section of the American Physiological Society, 1983-present
Fellow, The Society for Behavioral Medicine, 1986-present
The Council on Circulation of the American Heart Association, Elected Fellow 1987
Fellow, The Academy of Behavioral Medicine Research, 1987-present
Phi Beta Delta International Honor Society, 1988-present
Visiting Scientist Award--Medical Research Council of Canada (to support travel and living expenses for research in electrophysiology with Dr. L.P. Renaud, McGill University, June 1, 1988 to September 30, 1988).
Visiting Professor and Lectureship - State of Sao Paulo, Brazil, August 1995.
Visiting Lectureship - Heritage Foundation, Alberta, Canada, March 1996.
Rudy Clarenburg Lectureship - Kansas State University, Manhattan, KS, November 6-11, 1997.

Awards, Honors, Special Recognition (continued)

Ernest H. Starling Distinguished Lecturer of the American Physiological Society Water and Electrolyte Homeostasis Section, April 1999.
Howard Hughes Visiting Professor, University of Texas Health Science Center, San Antonio, TX, June 1999.
International Union of Physiological Sciences Commission on the Physiology of Food and Fluid Intake. Term of Office 1998-2003; Chair 2001-2003.
F. Wendell Miller Distinguished Professor, July, 2000-2010
Honorary Member of Brazilian Physiological Society (SBFis), August, 2007
Allan and Maria Myers International Visiting Fellow Award, 2012-2013, University of Melbourne, Melbourne, Australia

Scientific Advisory Boards

University of Pennsylvania, NIMH Program Project Grant on Sodium Appetite, 1990-92.
International Scientific Advisory Board for the Max-Planck-Institut für physiologische und klinische Forschung, W.G. Kerckhoff-Institute, in Bad Nauheim, Germany, 1998-present
Scientific Advisory Board of the Dalton Cardiovascular Research Center, University of Missouri, Columbia, Missouri, 1999-2004
External Advisor to the University of Wyoming, NIH Center for Biomedical Research Excellence (COBRE) Program, 2000-2005
Editorial Consultant, 2004 Edition of the *World Book Encyclopedia*
External Advisor to the Louisiana State University Health Sciences Center, NIH Center for Biomedical Research Excellence (COBRE) Program, 2007-present
University of Alabama at Birmingham Comprehensive Neuroscience Center External Advisory Committee, 2008-present; Site Visit October, 2009
External Advisor to the Department of Physiology, Paulista State University, Araraquara, Brazil, 2008-present

Professional Service

Associate Editor: *American Journal of Physiology: Regulatory, Integrative, and Comparative Physiology*, January, 1996-2001; 2007-2013
Editorial Boards: *American Journal of Physiology: Regulatory, Integrative, and Comparative Physiology*, 1984-present
Experimental Neurology, Neuroendocrine and Homeostatic Mechanisms section, 2001-present

North American Representative - International Commission on the Physiology of Food and Fluid Intake (ICPFFI) - The International Union of Physiological Sciences (IUPS) 1988-2003, ICPFFI President 2001-03.
Public Affairs Committee: American Physiological Society, 2002-04.
Nominating Committee: Society for the Study of Ingestive Behavior; Member, 1999-2001; Chair 2000-01
Board of Directors, Society for the Study of Ingestive Behavior, 2009-12

Journal Reviews: *Alcohol*
American Journal of Physiology
Appetite
Behavioral Neuroscience
Biological Psychiatry

Brain Research
Brain Research Bulletin
Brazilian Journal of Medical and Biological Research
Canadian Journal of Physiology and Pharmacology
Circulation Research
Clinical and Experimental Hypertension
Developmental Psychobiology
Endocrinology
Experimental Neurology
Experimental Physiology
Federation Proceedings
Hypertension
Journal of the Autonomic Nervous System
Journal of Pharmacology and Experimental Therapeutics
Lipids
Neuroendocrinology
Neuroscience Letters
Obesity
Peptides
Pflugers Archiv: European Journal of Physiology
Pharmacology Biochemistry & Behavior
Physiological Psychology
Physiology and Behavior
Proceeding of the National Academy of Sciences USA
Psychological Reviews
Science
Stress
Trends in Endocrinology and Metabolism Cell Press

Granting Agency Reviews

Consultant: Behavioral Medicine Branch, Division of Heart and Vascular Disease, National Heart, Lung and Blood Institute

Site Visit Team Member:

1. Program Project Review of A. Cowley for NHLBI Review Branch - May 1982
2. Program Project Review of D. Reis for NHLBI Review Branch - April 1983
3. Program Project Review of A. Cowley for NHLBI Review Branch - November 1984
4. Program Project Review of M. Prinz for NHLBI Review Branch - May 1985
5. Review of Medical College of Wisconsin Application for NIH Medical Scientist Training Program - May 1987
6. Program Project Review of J. Douglas for NHLBI Review Branch - February 1989

Granting Agency Reviews (continued)

7. Program Project Review of B. Zimmerman for NHLBI Review Branch - February 1990
8. Program Project Review of N. Rowland for NIA - January 1993
9. Program Project Review of C. Ferrario for NHLBI - September 1994
10. Program Project Review of V. Bishop for NHLBI - February 1996

Ad Hoc Reviewer:

1. National Science Foundation - Neurobiology
2. The Veterans Administration
3. National Institutes of Health
4. National Institutes of Health, ADAMH
5. Medical Research Council of Canada
6. International Human Frontier Science Program
7. Alberta Heritage Foundation for Medical Research
8. Louisiana Medical Foundation
9. Oak Ridge Associated Universities

Study Section Membership:

1. Cardiovascular and Renal Ad Hoc Study Section DRG NIH 1979-1981
2. Experimental Cardiovascular Sciences Study Section DRG NIH 1981-1983
3. Bio-Psychology Study Section DRG NIH 1986-1990.
4. Cellular and Molecular Basis of Disease Review Committee, June 1987.
5. National Space Biomedical Research Institute Peer Review Panel on Neurovestibular Adaptations, August 2000.
6. Neuroendocrinology, Neuroimmunology and Behavior Study Section (NNB), October 2000.
7. NIH Behavioral Neuroscience Fellowship Study Section, February 2007.
8. NIH Neuroendocrinology, Neuroimmunology, Rhythms, and Sleep Study Section, November 2013; February, 2014, October 2014
9. NIH Integrative and Clinical Endocrinology and Reproduction, February 2016

Research

Publications

1. Antelman, S. M., Johnson, A. K., & Fisher, A. E.: Survival of cholinergic drinking following nephrectomy. Physiology and Behavior, 1972, 8, 1169-1170.
2. Johnson, A. K., & Fisher, A. E.: Taste preferences for sucrose solutions and water under cholinergic and deprivation thirst. Physiology and Behavior, 1973, 10, 607-612.
3. Johnson, A. K., & Fisher, A. E.: Tolerance for quinine under cholinergic versus deprivation induced thirst. Physiology and Behavior, 1973, 10, 613-616.

4. Johnson, A. K.: The role of the cerebral ventricular system in angiotensin-induced thirst. In: G. Peters, J. T. Fitzsimons, & L. Peters-Haefeli (Eds.), Control Mechanisms of Drinking. Berlin-Heidelberg-New York: Springer-Verlag, 1975, pp. 117-122.
5. Johnson, A. K., & Epstein, A. N.: The cerebral ventricles as the avenue for the dipsogenic action of intracranial angiotensin. Brain Research, 1975, 86, 399-418.
6. Buggy, J., Fisher, A. E., Hoffman, W. E., Johnson, A. K., & Phillips, M. I.: Ventricular obstruction: Effect on drinking induced by intracranial injection of angiotensin. Science, 1975, 190, 72-74.
7. Johnson, A. K., & Schwob, J. E.: Cephalic angiotensin receptors mediating drinking to systemic angiotensin II. Pharmacology Biochemistry & Behavior, 1975, 3, 1077-1084.
8. Shrager, E. E., Osborne, M. J., Johnson, A. K., & Epstein, A. N.: Entry of angiotensin into cerebral ventricles and circumventricular structures. In: D. S. Davies, & J. L. Reid (Eds.), Central Action of Drugs in Blood Pressure Regulation. Baltimore, MD: University Park Press, 1975, pp. 65-67.
9. Johnson, A. K., & Buggy, J.: A critical analysis of the site of action for the dipsogenic effect of angiotensin II. In: J. P. Buckley, C. M. Ferrario, & M. F. Lokhandwala (Eds.), Central Actions of Angiotensin and Related Hormones. Elmsford, NY: Pergamon Press, 1977, pp. 357-386.
10. Fitzsimons, J. T., Epstein, A. N., & Johnson, A. K.: The peptide specificity of receptors for angiotensin-induced thirst. In: J. P. Buckley, C. M. Ferrario, & M. F. Lokhandwala (Eds.), Central Actions of Angiotensin and Related Hormones. Elmsford, NY: Pergamon Press, 1977, pp. 405-415.
11. Schwob, J. E., & Johnson, A. K.: Angiotensin-induced dipsogenesis in domestic fowl (Gallus gallus). Journal of Comparative and Physiological Psychology, 1977, 91, 182-188.
12. Johnson, A. K., Phillips, M. I., Mohring, J., & Ganten, D.: Angiotensin- induced drinking in rats with hereditary hypothalamic diabetes insipidus. Neuroscience Letters, 1977, 4, 327-330.
13. Buggy, J., Fink, G. D., Johnson, A. K., & Brody, M. J.: Prevention of the development of renal hypertension by anteroventral third ventricular tissue lesions. Circulation Research, 1977, 40(Suppl. I), I110-I117.
14. Buggy, J., & Johnson, A. K.: Preoptic-hypothalamic periventricular lesions: Thirst deficits and hypernatremia. American Journal of Physiology, 1977, 233, R44-R52.
15. Buggy, J., & Johnson, A. K.: Anteroventral third ventricle periventricular ablation: Temporary adipsia and persisting thirst deficits. Neuroscience Letters, 1977, 5, 177-182.
16. Johnson, A. K., & Buggy, J.: Periventricular preoptic-hypothalamus is vital for thirst and normal water economy. American Journal of Physiology, 1978, 234, R122-R129.
17. Rusterholz, D. B., Long, J. P., Flynn, J. R., Glyn, J. R., Barfnecht, C. F., Lind, R. W., & Johnson, A. K.: Inhibition of apomorphine-induced behaviors by derivatives of 2-amino-1,2,3,4-tetrahydronaphthalene. Archives Internationale de Pharmacodynamie et de Therapie, 1978, 232, 246-260.
18. Buggy, J., Fisher, A. E., Hoffman, W. E., Johnson, A. K., & Phillips, M. I.: Subfornical organ: A dipsogenic site of action of angiotensin II. Science, 1978, 201, 379-381.

Publications (continued)

19. Johnson, A. K., Simon, W., Schaz, K., Ganten, U., Ganten, D., & Mann, J. F. E.: Increased blood pressure responses to central angiotensin II in spontaneously hypertensive rats. Klinische Wochenschrift, 1978, 56, 47-49.
20. Ganten, D., Johnson, A. K., Mann, J. F. E., Rettig, R., Schelling, P., & Wiedemann, G.: Chronic beta-blockade: Reduction of blood pressure and drinking responses to centrally administered angiotensin. In: W. Maurer, A. Schomig, R. Dietz, & P. R. Lichtlen (Eds.), Beta-Blockade 1977. Stuttgart: Georg Thieme Verlag, 1978, pp. 108-112.
21. Buggy, J., & Johnson, A. K.: Angiotensin-induced thirst: Effects of third ventricular obstruction and periventricular ablation. Brain Research, 1978, 149, 117-128.
22. Brandes, J. S., & Johnson, A. K.: Recovery of feeding in rats following frontal neocortical ablations. Physiology and Behavior, 1978, 20, 763-770.

23. Fink, G. D., Buggy, J., Haywood, J. R., Johnson, A. K., & Brody, M. J.: Hemodynamic effects of electrical stimulation of forebrain angiotensin and osmosensitive sites. American Journal of Physiology, 1978, 235, H445-H451.
24. Brody, M. J., Fink, G. D., Buggy, J., Haywood, J. R., Gordon, F. J., & Johnson, A. K.: The role of the anteroventral third ventricle (AV3V) region in experimental hypertension. Circulation Research, 1978, 43, I2-I13.
25. Fitzsimons, J. T., Epstein, A. N., & Johnson, A. K.: Peptide antagonists of the renin-angiotensin system in the characterization of receptors for angiotensin-induced drinking. Brain Research, 1978, 153, 319-331.
26. Buggy, J., Fink, G. D., Haywood, J. R., Johnson, A. K., & Brody, M. J.: Interruption of the maintenance phase of established hypertension by ablation of the anteroventral third ventricle (AV3V) in rats. Clinical and Experimental Hypertension, 1978, 1, 337-353.
27. Johnson, A. K., Hoffman, W. E., & Buggy, J.: Attenuated pressor responses to intracranially injected stimuli and altered antidiuretic activity following preoptic-hypothalamic periventricular ablation. Brain Research, 1978, 157, 161-166.
28. Brody, M. J., Fink, G. D., Buggy, J., Haywood, J. R., Gordon, F. J., Knuepfer, M., Mow, M., Mahoney, L., & Johnson, A. K.: Critical role of the anteroventral third ventricle (AV3V) region in development and maintenance of experimental hypertension. In: P. Meyer, & H. Schmitt (Eds.), Nervous System and Hypertension. New York: John Wiley & Sons, 1979, pp. 76-84.
29. Buggy, J., Hoffman, W. E., Phillips, M. I., Fisher, A. E., & Johnson, A. K.: Osmosensitivity of rat third ventricle and interactions with angiotensin. American Journal of Physiology, 1979, 236, R75-R82.
30. Gordon, F. J., Haywood, J. R., Brody, M. J., Mann, J. F. E., Ganten, D., & Johnson, A. K.: Effect of anteroventral third ventricle (AV3V) lesions on the development of hypertension in spontaneously hypertensive rat. Japanese Heart Journal, 1979, 20(Suppl. I), 116-118.
31. Johnson, A. K.: Role of the periventricular tissue of the anteroventral third ventricle in body fluid homeostasis. In: P. Meyer, & H. Schmitt (Eds.), Nervous System and Hypertension. New York: John Wiley & Sons, 1979, pp. 106-114.
32. Bealer, S. L., Phillips, M. I., Johnson, A. K., & Schmid, P. G.: Anteroventral third ventricle lesions reduce antidiuretic response to angiotensin II. American Journal of Physiology, 1979, 236, E610-E615.
33. Bealer, S. L., & Johnson, A. K.: Sodium consumption following lesions surrounding the anteroventral third ventricle. Brain Research Bulletin, 1979, 4, 287-290.
34. Bealer, S. L., & Johnson, A. K.: Preoptic-hypothalamic periventricular lesions: Impairment of thirst-motivated behavior. Physiology and Behavior, 1979, 22, 841-846.

Publications (continued)

35. Gordon, F. J., Brody, M. J., Fink, G. D., Buggy, J., & Johnson, A. K.: Role of central catecholamines in the control of blood pressure and drinking behavior. Brain Research, 1979, 178, 161-173.
36. Johnson, A. K.: The analysis of drinking behavior: The need for defining physiological parameters and not for proliferating constructs. The Behavioral and Brain Sciences, 1979, 2, 107-108.
37. Brody, M. J., & Johnson, A. K.: Role of the anteroventral third ventricle region in fluid and electrolyte balance, arterial pressure regulation, and hypertension. In: L. Martini, & W. F. Ganong (Eds.), Frontiers in Neuroendocrinology (Vol. 6). New York: Raven Press, 1980, pp. 249-292.
38. Mann, J. F. E., Johnson, A. K., & Ganten, D.: Plasma angiotensin II: Dipsogenic levels and angiotensin-generating capacity of renin. American Journal of Physiology, 1980, 238, R372-R377.
39. Shrager, E. E., & Johnson, A. K.: Anteroventral third ventricle (AV3V) region ablation: Chronic elevations of plasma renin concentration. Brain Research, 1980, 190, 554-558.

40. Bealer, S. L., & Johnson, A. K.: Preoptic-hypothalamic periventricular lesions alter food-associated drinking and circadian rhythms. Journal of Comparative and Physiological Psychology, 1980, 94, 547-555.
41. Carithers, J., Bealer, S. L., Brody, M. J., & Johnson, A. K.: Fine structural evidence of degeneration in supraoptic nucleus and subfornical organ of rats with lesions in the anteroventral third ventricle. Brain Research, 1980, 201, 1-12.
42. Mahoney, L. T., Haywood, J. R., Corry, R., Patel, P. N., Johnson, A. K., & Brody, M. J.: The role of the renal afferent nerves in the pathogenesis of experimental renal and mineralocorticoid hypertension. In: H. Villarreal (Ed.), Hypertension. New York: John Wiley & Sons, 1981, pp. 195-198.
43. Gordon, F. J., & Johnson, A. K.: Electrical stimulation of the septal area in the rat: Prolonged suppression of water intake and correlation with self-stimulation. Brain Research, 1981, 206, 421-430.
44. Johnson, A. K., Buggy, J., Fink, G. D., & Brody, M. J.: Prevention of renal hypertension and of the central pressor effect of angiotensin by ventromedial hypothalamic ablation. Brain Research, 1981, 205, 255-264.
45. Johnson, A. K., Mann, J. F. E., Rascher, W., Johnson, J. K., & Ganten, D.: Plasma angiotensin II concentrations and experimentally induced thirst. American Journal of Physiology, 1981, 240, R229-R234.
46. Carithers, J., Dellmann, H. D., Bealer, S. L., Brody, M. J., & Johnson, A. K.: Ultrastructural effects of anteroventral third ventricle lesions on supraoptic nuclei and neural lobes of rats. Brain Research, 1981, 220, 13-29.
47. Schelling, P., Meyer, D., Loos, H.-E., Speck, G., Johnson, A. K., Phillips, M. I., & Ganten, D.: Renin activity in different brain regions of spontaneously hypertensive rats. In: J.P. Buckley, & C.M. Ferrario (Eds.), Central Nervous System Mechanisms in Hypertension. New York: Raven Press, 1981, pp. 397-406.
48. Rettig, R., Ganten, D., & Johnson, A. K.: Isoproterenol-induced thirst: Renal and extrarenal mechanisms. American Journal of Physiology, 1981, 241, R152-R157.
49. Lind, R. W., & Johnson, A. K.: Periventricular preoptic-hypothalamic lesions: Effects on isoproterenol induced thirst. Pharmacology, Biochemistry & Behavior, 1981, 15, 563-565.
50. Mann, J. F. E., Johnson, A. K., Rascher, W., Genest, J., & Ganten, D.: Thirst in the rat after ligation of the inferior vena cava: Role of angiotensin II. Pharmacology Biochemistry & Behavior, 1981, 15, 337-341.

Publications (continued)

51. Brody, M. J., & Johnson, A. K.: Role of forebrain structures in models of experimental hypertension. In: Disturbances in Neurogenic Control of the Circulation, Clinical Physiology Series, American Physiological Society. Baltimore: Williams & Wilkins Co., 1981, pp. 105-117.
52. Simon, W., Schaz, K., Mann, J. F. E., Ganten, U., Johnson, A. K., Unger, Th., Rascher, W., & Ganten, D.: The effects of beta-adrenoreceptor blockers on blood pressure responses to central angiotensin II. Neuropharmacology, 1981, 20, 719-726.
53. Tucker, D. C., & Johnson, A. K.: Behavioral correlates of spontaneous hypertension. Neuroscience & Biobehavioral Reviews, 1981, 5, 463-471.
54. Lind, R. W., & Johnson, A. K. Central and peripheral mechanisms mediating angiotensin-induced thirst. In: D. Ganten, M. Printz, M. I. Phillips, B. A. Scholkens (Eds.), The Renin Angiotensin System in the Brain. Berlin-Heidelberg-New York: Springer-Verlag, 1982, pp. 353-364.

55. Tucker, D. C., & Johnson, A. K.: Developmental heart rate differences between spontaneously hypertensive and Wistar-Kyoto rat pups. In: W. Rascher, D. Clough, & D. Ganten (Eds.), Hypertension Mechanisms: The Spontaneously Hypertensive Rat As a Model to Study Human Hypertension. Stuttgart-New York: Schattauer, 1982, pp. 383-386.
56. Galeno, T. M., Johnson, A. K., & Brody, M. J.: Hemodynamic response to stimulation of the amygdala in spontaneously hypertensive rats. In: W. Rascher, D. Clough, & D. Ganten (Eds.), Hypertension Mechanisms: The Spontaneously Hypertensive Rat As a Model to Study Human Hypertension. Stuttgart-New York: Schattauer, 1982, pp. 375-379.
57. Gordon, F. J., Haywood, J. R., Brody, M. J., & Johnson, A. K.: Effect of lesions of the anteroventral third ventricle (AV3V) on the development of hypertension in spontaneously hypertensive rats. Hypertension, 1982, 4, 387-393.
58. Matsuguchi, H., Sharabi, F. M., Gordon, F. J., Johnson, A. K., & Schmid, P. G.: Blood pressure and heart rate responses to microinjection of vasopressin into the nucleus tractus solitarius region of the rat. Neuropharmacology, 1982, 21, 687-693.
59. Galeno, T. M., Van Hoesen, G. W., Maixner, W., Johnson, A. K., & Brody, M. J.: Contribution of the amygdala to the development of spontaneous hypertension. Brain Research, 1982, 246, 1-6.
60. Lind, R. W., Van Hoesen, G. W., & Johnson, A. K.: An HRP study of the connections of the subfornical organ of the rat. Journal of Comparative Neurology, 1982, 210, 265-277.
61. Lind, R. W., & Johnson, A. K.: Subfornical organ-median preoptic connections and drinking and pressor responses to angiotensin II. Journal of Neuroscience, 1982, 2, 1043-1051.
62. Mitchell, L. D., Barron, K., Brody, M. J., & Johnson, A. K.: Two possible actions for circulating angiotensin II in the control of vasopressin release. Peptides, 1982, 3, 503-507.
63. Lind, R. W., & Johnson, A. K.: On the separation of functions mediated by the AV3V region. Peptides, 1982, 3, 495-499.
64. Johnson, A. K.: Neurobiology of the periventricular tissue surrounding the anteroventral third ventricle (AV3V) and its role in behavior, fluid balance, and cardiovascular control. In: O. A. Smith, R. A. Galosy, & S. M. Weiss (Eds.), Circulation, Neurobiology and Behavior. New York: Elsevier, 1982, pp. 277-295.
65. Hartle, D. K., Lind, R. W., Johnson, A. K., & Brody, M. J.: Localization of the anterior hypothalamic angiotensin II pressor system. Hypertension, 1982, 4(Suppl. II), II-159-II-165.
66. Schelling, P., Meyer, D., Loos, H. E., Speck, G., Phillips, M. I., Johnson, A. K., & Ganten, D.: A micromethod for the measurement of renin in brain nuclei: Its application in spontaneously hypertensive rats. Neuropharmacology, 1982, 21, 455-463.

Publications (continued)

67. Bealer, S. L., Haywood, J. R., Gruber, K. A., Buckalew, V. M., Fink, G. D., Brody, M. J., & Johnson, A. K.: Preoptic-hypothalamic periventricular lesions reduce natriuresis to volume expansion. American Journal of Physiology, 1983, 244, R51-57.
68. Lind, R. W., & Johnson, A. K.: A further characterization of the effects of AV3V lesions on ingestive behavior. American Journal of Physiology, 1983, 245, R83-R90.
69. Sladek, C. D., & Johnson, A. K.: Effect of anteroventral third ventricle lesions on vasopressin release by organ-cultured hypothalamo-neurohypophyseal explants. Neuroendocrinology, 1983, 37, 78-84.
70. Haywood, J. R., Fink, G. D., Buggy, J., Boutelle, S., Johnson, A. K., & Brody, M. J.: Prevention of two-kidney one-clip renal hypertension in rat by ablation of (AV3V) tissue. American Journal of Physiology, 1983, 245, H683-H689.
71. Lind, R. W., Ohman, L. E., Lansing, M. B., & Johnson, A. K.: Transection of subfornical organ neural connections diminishes the pressor response to intravenously infused angiotensin II. Brain Research, 1983, 275, 361-364.

72. Brody, M. J., Hartle, D. K., Lind, R. W., & Johnson, A. K.: Evidence for the participation of specific hypothalamic pathways in the pathogenesis of hypertension. In: H. Villarreal, & M. P. Sambhi (Eds.), Topics in Pathophysiology of Hypertension. Martinus Nijhoff Publishers, 1984, pp. 275-281.
73. Knuepfer, M. M., Johnson, A. K., & Brody, M. J.: Vasomotor projections from the anteroventral third ventricle (AV3V) region. American Journal of Physiology, 1984, 247, H139-H145.
74. Knuepfer, M. M., Johnson, A. K., & Brody, M. J.: Identification of brainstem projections mediating hemodynamic responses to stimulation of the anteroventral third ventricle (AV3V) region. Brain Research, 1984, 294, 305-314.
75. Lind, R. W., Thunhorst, R. L., & Johnson, A. K.: The subfornical organ and the integration of multiple factors in thirst. Physiology and Behavior, 1984, 32, 69-74.
76. Tucker, D. C., & Johnson, A. K.: Development of autonomic control of heart rate in genetically hypertensive and normotensive rats. American Journal of Physiology, 1984, 246, R570-R577.
77. Tucker, D. C., Bhatnagar, R. K., & Johnson, A. K.: Genetic and environmental influences on developing autonomic control of heart rate. American Journal of Physiology, 1984, 246, R578-R586.
78. Knuepfer, M. M., Johnson, A. K., & Brody, M. J.: Effect of subfornical organ ablation on the development of renal hypertension. Clinical and Experimental Hypertension, 1984, A6, 1027-1034.
79. Bealer, S. L., Carithers, J., & Johnson, A. K.: Fluid regulation, body weight and drinking responses following hypothalamic knife cuts. Brain Research, 1984, 305, 239-245.
80. Carithers, J., Bealer, S. L., & Johnson, A. K.: The effects of transverse cuts caudal to the preoptic recess on the hypothalamo-neurohypophyseal neurosecretory system. Brain Research, 1984, 305, 247-257.
81. Mahoney, L. T., Haywood, J. R., Correy, R., Patel, N. P., Johnson, A. K., & Brody, M. J.: Possible mechanism of protection from renal hypertension by anteroventral third ventricle (AV3V) lesions: Role of renal afferent nerves. In: Sambhi, M. P. (Ed.), Fundamental Fault in Hypertension. The Hague, Netherlands: Martinus Nijhoff Publishers, 1984, pp. 207-212.

Publications (continued)

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Description of Research Program

Overview

The major research direction of the Johnson laboratory deals with the elucidation of neural and hormonal mechanisms involved in the maintenance of body fluid and cardiovascular homeostasis. The regulation of body fluids involves both the determination of overall hydromineral balance and the control of appropriate distribution of salt and water in the body. The processes providing for the consistency of body fluid evoke behavioral, humoral, and cardiovascular effector systems. The unique character for which our laboratory has become widely recognized is its rigorous analysis of the role of behavior as a control of body fluid homeostasis and for studies which focus on the interaction of hormonal and cardiovascular systems with motivated (primary) behaviors. Specifically, we have a major emphasis on the role of ingestive behaviors (thirst, or the drinking of water; and sodium appetite, or the ingestion of sodium) which are important for the determination of overall fluid balance, and we study the interactions of environmental stimuli and the study of ongoing behaviors (e.g., ingestion; aggression, defense, etc.) with the cardiovascular system (i.e., heart rate, blood pressure, regional blood flow).

Development of the Character of Our Laboratory at Iowa

Our current research thrust evolved from an interest in the neuropsychology of thirst and salt appetite. The conceptual model which we adopted for the physiological analysis of this motivational process is analogous to that employed by sensory systems (e.g., vision, audition, etc.). That is, the types of questions that we ask are: (1) What are the effective stimuli which produce thirst and mobilize drinking behavior? In the case of classic sensory systems the stimuli are well known, but in our problem area much effort is directed at defining the nature of intrinsic (i.e., internal) stimuli that stimulate drinking. (2) What type of receptors is stimulated by such internal stimuli? (3) Where are these receptors located? (4) What are the biophysical and biochemical mechanisms involved in signaling pathways in these receptors? (5) What neuroanatomical pathways are activated when such receptors are stimulated?, and (6) What neurotransmitter systems are involved in mediating the communication between neurons within this motivational system? More specifically, we have conducted experiments designed to evaluate when the internal stimuli of increased osmolality and increased circulating levels of the hormone angiotensin II would serve as adequate stimuli for the induction of thirst. Our lab has played a major role in the definition of what critical regions within the central nervous system house receptors for these physiological stimuli. We have conducted significant studies in elucidating the projections of nerve tracks issuing from putative receptor sites. Finally, our group has made strides in evaluating the role of various transmitter and modulator systems associated with thirst and salt appetite.

Our Analysis of Thirst Mechanisms as a Model for the Analysis of Other Physiological Control Systems Related to Body Fluid Homeostasis

The stimuli responsible for the induction of thirst (i.e., increased angiotensin II levels and increased body fluid osmolality) act not only to increase water intake but also activate hormonal systems responsible for water retention as well. Thus, it became apparent to us that the model and techniques which we adopted for the analysis of salt and water intake could be applied to the analysis of these other complimentary physiological systems. Our decision to begin concurrent studies on the neural control of fluid retention as well as on the cardiovascular system is important for three reasons. First, basic alterations in either one of these intrinsic physiological systems can modify the behaviors of interest to

Research Discussion (continued);

us. For example, if fluid retention mechanisms are activated and the animal does not excrete as much water, drinking behavior is likely to be suppressed. The mechanism for the suppression or inhibition becomes an important consideration for our analyses. The second reason to study these parallel physiological processes is that under normal conditions it has been consistently demonstrated that fluid retention and distribution systems are mobilized when thirst is activated. Thus, it becomes possible to test whether an observation made in the process of conducting experiments on thirst is "physiologically valid" by examining the effects of the same manipulations on the complimentary physiological processes. The third reason for investigating the neural control of these complimentary physiological processes is that a working hypothesis can frequently be tested more easily (for example, under anesthesia) by studying either hormonal or cardiovascular endpoints because they are often easier to measure in a surgically manipulated animal in which behavior might be compromised. Therefore, results obtained from studying such physiological processes serve as a source of hypotheses that promote or encourage the development of better methods (e.g., surgical technique) that permit the development of more reliable techniques for collecting behavioral data. Thus, the study of complimentary physiological processes along with behavior increases our experimental and conceptual leverage and enhances our understanding of both behavior and physiology.

Significance of Program to Study of Pathological Processes

Over the past 40 years, our experiments investigating thirst, sodium appetite, the neuroendocrinology of water retention, and the central control of the cardiovascular system have stimulated interest among scientists of many disciplines because both behavioral and physiological processes are recognized to be significant in conditions where there is disordered regulation of body fluid balance or blood distribution, e.g., hypertension. From our basic studies attempting to localize and characterize receptors for dipsogenic (thirst) stimuli, we hypothesized that the same regions of the central nervous system may house receptors on which the identical stimuli act to promote body fluid expansion and to increase vasomotor tone. In collaboration with members of The University of Iowa Cardiovascular Center, we have been able to demonstrate that the major regions of the central nervous system, which we had implicated in the control of thirst, are necessary for the production and maintenance of many forms of experimental hypertension. Animals in which the receptive area for angiotensin is destroyed are "protected" against high blood pressure. This observation has provoked the development of a major research program to further elucidate the mechanisms and the basic neurobiology associated with this protection.

Cardiovascular-Behavioral Interactions

As a result of our developing interest in hypertension, we have directed a significant amount of our research interest over the past twelve to thirteen years to exploring the nature of interactions between the cardiovascular system and behavior in both normal and pathological states. In particular, we have been interested in exploring the neural and physiological mediators in models of hypertension and heart failure in which high blood pressure and heart disease is induced or exacerbated by exposing animals to physical or psychosocial stimuli which have been characterized as stressors. Second, we are pursuing experiments studying the effects of stressors administered early in life on the development of the autonomic controls of the circulatory system, adult behaviors (e.g., open field activity) and the level of adult blood pressure. Third, we are currently applying methodologies that will permit the characterization of several cardiovascular parameters in conjunction with ongoing behaviors. The ultimate aim of these latter experiments is to provide us a means to determine how the central nervous system functions to couple somatic (behavioral) and visceral (cardiovascular) responses and to determine whether

Research Discussion (continued);

repetitive activation of the neural substrates or the dissociation of the somatic and visceral responses can contribute to pathological conditions such as hypertension. Fourth, at the present time we are heavily involved in investigating brain mechanisms involved in states of orthostatic intolerance and

shock. Fifth, our most recent interest is investigating the role of the neuroendocrine and central neural systems responsible for high co-morbidity of depression and heart disease. These last five experimental areas require an application of refined behavioral, physiological and biochemical techniques. These later research directions we view as an emerging new discipline, experimental behavioral medicine.