Conference: Food Intake Regulation: Neuropeptides,
Circulating Factors and Genetics
Central Neuroanatomical Systems Involved in the
Regulation of Food Intake in Birds and Mammals¹
WAYNE J. KUENZEL **Tal Neuroanatomica**
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Department of Poultry Science, Unit *Pai* **Preuroanatomical Systems Involved in the

diation of Food Intake in Birds and Mammals¹
** *WAYNE J. KUENZEL***
** *Department of Poultry Science, University of Maryland, College Park, MD 20742*

Example 3 Found graduation
 RBSTRACT The neural regulation of food intake seems

to be quite similar in birds and mammals. The ven-

tromedial hypothalamic syndrome produced by lesions **THE SET ACT THE NET ALL SET A ABSTRACT** The neural regulation of food intake seems
to be quite similar in birds and mammals. The ven-
tromedial hypothalamic syndrome produced by lesions
within the mediobasal hypothalamus of both birds and
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tromedial hypothalamic syndrome produced by lesions
within the mediobasal hypothalamus of both birds and
mammals is composed of several independent physio-
logical and beh Interpreticular hypothalamic syndrome produced by lesions
within the mediobasal hypothalamus of both birds and
mammals is composed of several independent physio-
logical and behavioral changes. Other neural sites known
to within the mediobasal hypothalamus of both birds and
mammals is composed of several independent physio-
logical and behavioral changes. Other neural sites known
to be important in mammals for regulating food intake
need to mammals is composed of several independent physio-
logical and behavioral changes. Other neural sites known
to be important in mammals for regulating food intake
need to be examined in birds including the paraven-
tricular logical and behavioral changes. Other neural sites known
to be important in mammals for regulating food intake
need to be examined in birds including the paraven-
tricular nucleus, nucleus tractus solitarius and
parabrachi **food intake in and in avigating food intake**
 food intake
 **food to be examined in birds including the paraventricular nucleus, nucleus tractus solitarius and

parabrachial nucleus. Members of the opioid and pan-

creat** need to be examined in birds including the paraventricular nucleus, nucleus tractus solitarius and parabrachial nucleus. Members of the opioid and pancreatic polypeptide families are effective in stimulating food intake in tricular nucleus, nucleus tractus solitarius and
parabrachial nucleus. Members of the opioid and pan-
creatic polypeptide families are effective in stimulating
food intake in avian species. Both prolactin and growth
hormon parabrachial nucleus. Members of the opioid and pancreatic polypeptide families are effective in stimulating
food intake in avian species. Both prolactin and growth
hormone are also efficacious in stimulating food intake.
 creatic polypeptide families are effective in stimulating
food intake in avian species. Both prolactin and growth
hormone are also efficacious in stimulating food intake.
In contrast, cholecystokinin inhibits food intake w food intake in avian species. Both prolactin and growth
hormone are also efficacious in stimulating food intake.
In contrast, cholecystokinin inhibits food intake when
administered intracerebroventricularly. The autonomic
 hormone are also efficacious in stimulating food intake.
In contrast, cholecystokinin inhibits food intake when
administered intracerebroventricularly. The autonomic
and endocrine hypothesis developed to explain obesity in In contrast, cholecystokinin inhibits food intake when
administered intracerebroventricularly. The autonomic
and endocrine hypothesis developed to explain obesity in
mammals appears to be quite applicable to genetic
strain administered intracerebroventricularly. The autonomic
and endocrine hypothesis developed to explain obesity in
mammals appears to be quite applicable to genetic
strains of commercial birds selected for meat
production. Spe and endocrine hypothesis developed to explain obesity in
mammals appears to be quite applicable to genetic
strains of commercial birds selected for meat
production. Specifically the commercial broiler appears
to display an mammals appears to be quite applicable to genetic
strains of commercial birds selected for meat
production. Specifically the commercial broiler appears
to display an imbalance of the autonomic nervous
system. The parasympa strains of commercial birds selected for meat
production. Specifically the commercial broiler appears
to display an imbalance of the autonomic nervous
system. The parasympathetic nervous system
dominates as a consequence o to display an imbalance of
system. The parasympath
dominates as a consequence of
for growth rate. J. Nutr. 124
INDEXING KEY WORDS: *opinpathecte* nervols

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opioid peptides

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THE JOURNAL OF NUTRITION

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- hypothalamus · opioid peptides \bullet
- INDEXING KEY WORDS:
• hypothalamus opioid p
• neuropeptide Y cholecy
• autonomic nervous system *cholecystokinin* **EXING KEY WORDS:**
hypothalamus • opioid peptides
neuropeptide Y • cholecystokinin
autonomic nervous system
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• neuropeptide Y • cholecystokinin

• autonomic nervous system

The majority of information known about the

ural control of food intake in vertebrates has been

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is sixted to the state in the majority of information known about the neural control of food intake in vertebrates has been

obtained by experiments conducted with laboratory obtained by experiments conducted with laboratory

The majority of information known about the

obtained by experiments conducted with laboratory

rats. Therefore for the purpose of this review, the The majority of information known about the
neural control of food intake in vertebrates has been
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recent literature comp The majority of information known about the
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recent literature comp The majority of information known about the
neural control of food intake in vertebrates has been
obtained by experiments conducted with laboratory
rats. Therefore for the purpose of this review, the
recent literature comp have been presented to explain the neural regulation
obtained by experiments conducted with laboratory
rats. Therefore for the purpose of this review, the
recent literature completed in mammals was first
summarized to high obtained by experiments conducted with laboratom
rats. Therefore for the purpose of this review, the
recent literature completed in mammals was fir
summarized to highlight the main hypotheses th
have been presented to expl rats. Incretore for the purpose of this review, the
recent literature completed in mammals was first
summarized to highlight the main hypotheses that
have been presented to explain the neural regulation
of feeding. The neu summarized to highlight the main hypotheses that
have been presented to explain the neural regulation
of feeding. The neurotransmitters and neuromodu-
lators found in the brain that alter food intake as well
as major neura summarized to nightight the main hypotneses that
have been presented to explain the neural regulation
of feeding. The neurotransmitters and neuromodu-
lators found in the brain that alter food intake as well
as major neura mave been presented to explain the neural regulation
of feeding. The neurotransmitters and neuromodu-
lators found in the brain that alter food intake as well
as major neural pathways thought to influence
feeding behavior or reeding. The neurotransmitters and neuromodu-
lators found in the brain that alter food intake as well
as major neural pathways thought to influence
feeding behavior were then reviewed. The avian liter-
ature was review feeding behavior were then reviewed. The avian lites
ature was reviewed to ascertain neural structures an
neuromodulators shown to alter food intake. Finally
0022-3166/94 \$3.00 © 1994 American Institute of Nutrition.

Department of Poultry Science, University of Maryland, College Park, MD 20742
 ABSTRACT The neural regulation of food intake seems

to be quite similar in birds and mammals. The ven-
 $\begin{array}{|l|l|}\n\hline\n\text{ABSTRACT} & \text{The neural regulation of food intake seems to be$ f Maryland, College Park, MD 20742
general hypothesis is presented based largely on mam-
malian data that may be of use for designing future $\frac{8}{90}$ f Maryland, College Park, MD 20742
general hypothesis is presented based largely on mam-
malian data that may be of use for designing future
experiments with avian species in an attempt to r Maryland, College Park, MD 20742
general hypothesis is presented based largely on mam-
malian data that may be of use for designing future
experiments with avian species in an attempt to $\frac{a}{2}$
further the understand general hypothesis is presented based largely on mam-
malian data that may be of use for designing future
experiments with avian species in an attempt to
further the understanding of central nervous system
regulation of f general hypothesis is presented based malian data that may be of use f
experiments with avian species
further the understanding of cent
regulation of feeding in birds. Hanan data that hay be of use for designing future

xperiments with avian species in an attempt to

urther the understanding of central nervous system

egulation of feeding in birds.
 HYPOTHESES PROPOSED TO EXPLAIN

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THE REGULATION OF FOOD INTAKE

general hypothesis is presented based largely on mam-
malian data that may be of use for designing future
experiments with avian species in an attempt to
further the understanding of central nervous system
regulation of f lation of food intake can be categorized into one of **THE REGULATION OF FOOD INTAKE**
Theories introduced to explain the neural regulation of food intake can be categorized into one of two groups based upon 1) identification of the ana-
tomical structures in the brain that c THE REGULATION OF FOOD INTAKE
Theories introduced to explain the neural regu-
lation of food intake can be categorized into one of
two groups based upon 1) identification of the ana-
tomical structures in the brain that c Theories introduced to explain the neural regulation of food intake can be categorized into one of $\frac{3}{2}$
two groups based upon 1) identification of the ana-
tomical structures in the brain that can alter food
intake w Theories introduced to explain the neural regulation of food intake can be categorized into one of $\frac{99}{15}$
two groups based upon 1) identification of the anatomical structures in the brain that can alter food \geq in lation of food intake can be categorized into one of
two groups based upon 1) identification of the ana-
tomical structures in the brain that can alter food
intake when manipulated, and 2) changes in a nu-
trient, metabol two groups based upon 1) identification of the ana-
tomical structures in the brain that can alter food
intake when manipulated, and 2) changes in a nu-
trient, metabolite or ion. **Table 1** is a summary of
hypotheses base in take when manipulated, and 2) changes in a nutrient, metabolite or ion. **Table 1** is a summary of hypotheses based on the latter category. Emphasis in this review, however, will be on theories that identified a struc intake when manistrient, metabolite
hypotheses based of
this review, howev
tified a structure
nervous system.
Dual center hypotheses ent, metabolite or ion. **Table 1** is a summary of
potheses based on the latter category. Emphasis in
is review, however, will be on theories that iden-
ied a structure or structures within the central
ryous system.
Dual c

hypothesis explaining the neural regulation of food this review, however, will be on theories that iden-
tified a structure or structures within the central
mervous system.
Dual center hypothesis. An early and long-lived
hypothesis explaining the neural regulation of foo tified a structure or structures within the central
nervous system.
Dual center hypothesis. An early and long-lived
hypothesis explaining the neural regulation of food
intake, the dual center hypothesis, was introduced nervous system.
 Dual center hypothesis. An early and long-lived

hypothesis explaining the neural regulation of food

intake, the dual center hypothesis, was introduced by

Stellar (1954) shortly after the classic expe **Dual center hypothesis.** An early and long-lived $\frac{m}{6}$
hypothesis explaining the neural regulation of food $\frac{m}{6}$
intake, the dual center hypothesis, was introduced by
Stellar (1954) shortly after the classic expe hypothesis explaining the neural regulation of food
intake, the dual center hypothesis, was introduced by
Stellar (1954) shortly after the classic experiments of
Anand and Brobeck (1951). Based on electrolytic le-
sions ma intake, the dual center hypothesis, was introduced by $\frac{m}{50}$
Stellar (1954) shortly after the classic experiments of $\frac{m}{20}$
Anand and Brobeck (1951). Based on electrolytic le-
sions made within the diencephalon of Stellar (1954) shortly after the classic experiments of
Anand and Brobeck (1951). Based on electrolytic le-
sions made within the diencephalon of the rat, the
study showed that bilateral lesions of the ven-
tromedial hypo Anand and Brobeck (1951). Based on electrolytic lesions made within the diencephalon of the rat, the study showed that bilateral lesions of the ven-
tromedial hypothalamic area resulted in hyperphagia
and obesity (Hetheri sions made within the diencephalon of the rat, the
study showed that bilateral lesions of the ven-
tromedial hypothalamic area resulted in hyperphagia
and obesity (Hetherington and Ranson 1940) and
bilateral lesions of th

^{&#}x27;Presented as part of the 58th Annual Poultry Nutrition Con Ference: Food Intake Regulation: Neuropeptides, Circulating Factors and Genetics, given at the Experimental Biology '93
Factors and Genetics, given at the Experimental Biology '93
meeting, New Orleans, LA, March 28, 1993. ¹Presented as part of the 58th Annual Poultry Nutrition Conference: Food Intake Regulation: Neuropeptides, Circulating
Factors and Genetics, given at the Experimental Biology '93
meeting, New Orleans, LA, March 28, 1993. The Institute of the S8th Annual Poultry Nutrition Conference: Food Intake Regulation: Neuropeptides, Circulating
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Traditional hypotheses of food intake regulation based on changes in a specific nutrient, metabolite, ion,
changes in a specific nutrient, metabolite, ion,
neuromodulator or metabolic state **TABLE 1**
hypotheses of food intake regulation bas
neuromodulator or metabolic state
neuromodulator or metabolic state changes in a specific nutrient, metabolite, ion,
neuromodulator or metabolic state

Ionostatic Myers et al. 1972
Peptidergic Morley 1980
Brobeck 1951). The ventromedial hypothalamus,
termed a 'satiety' center, contained a set of neurons
that projected to and inhibited a lateral hypothalamic Brobeck 1951). The ventromedial hypothalamus, Brobeck 1951). The ventromedial hypothalamus,
termed a 'satiety' center, contained a set of neurons
that projected to and inhibited a lateral hypothalamic
area, the 'feeding' center.
Criticisms, however, arose concerning t

Criticisms, however, arose concerning the model, particularly in addressing the exact anatomical locus termed a 'satiety' center, contained a set of neurons
that projected to and inhibited a lateral hypothalamic
area, the 'feeding' center.
Criticisms, however, arose concerning the model,
particularly in addressing the exact that projected to and inhibited a lateral hypothalamic
area, the 'feeding' center.
Criticisms, however, arose concerning the model,
particularly in addressing the exact anatomical locus
mediating ventromedial hypothalamic area, the reeding center.

Criticisms, however, arose concerning the model,

particularly in addressing the exact anatomical locus

mediating ventromedial hypothalamic obesity. For

hy

example, using hypothalamic knife cu Criticisms, nowever, arose concerning the model,
particularly in addressing the exact anatomical locus
mediating ventromedial hypothalamic obesity. For
example, using hypothalamic knife cuts, the ven-
tromedial hypothalami particularly in addressing the exact anatomical locus
mediating ventromedial hypothalamic obesity. For
example, using hypothalamic knife cuts, the ven-
tromedial hypothalamic nucleus could be spared, and
gest
rats were sti mediating ventromedial hypothalamic obesity. For
example, using hypothalamic knife cuts, the ven-
tromedial hypothalamic nucleus could be spared, and
rats were still obese (Gold 1973). Fiber tracts that
needed to be severe example, using hypothalamic knife cuts, the ven-
tromedial hypothalamic nucleus could be spared, and
rats were still obese (Gold 1973). Fiber tracts that
needed to be severed to effect hyperphagia appeared to
involve a lon rats were still obese (Gold 1973). Fiber tracts that
needed to be severed to effect hyperphagia appeared to
involve a longitudinal neural pathway, not a direct
fiber projection between the ventromedial hypotha-
lamic nucle rats were still obese (Gold 1973). Fiber tracts that
needed to be severed to effect hyperphagia appeared to
involve a longitudinal neural pathway, not a direct
fiber projection between the ventromedial hypotha-
lamic nucle needed to be severed to effect hyperpring a appeared to
involve a longitudinal neural pathway, not a direct
fiber projection between the ventromedial hypotha-
lamic nucleus and lateral hypothalamic area (Gold
1973, Sclafan still became obese when the ventromedial hypotha-
still became obese when the ventromedial hypotha-
lamic nucleus and lateral hypothalamic area (Gold
1973, Sclafani and Grossman 1969). More impor-
tantly, ventromedial hypo their projection between the ventromedial hypotha-
lamic nucleus and lateral hypothalamic area (Gold
1973, Sclafani and Grossman 1969). More impor-
tantly, ventromedial hypothalamically lesioned rats
still became obese whe lamic nucleus and lateral hypothalamic area (Gold
1973, Sclafani and Grossman 1969). More impor-
tantly, ventromedial hypothalamically lesioned rats
still became obese when food restricted to prevent
hyperphagia (Brooks an 1973, Sclatani and Grossman 1969). More impor-
tantly, ventromedial hypothalamically lesioned rats
still became obese when food restricted to prevent
hyperphagia (Brooks and Lambert 1946, Han et al.
1965). Further researc tantly, ventromedial hypothalamically lesioned rats
still became obese when food restricted to prevent
hyperphagia (Brooks and Lambert 1946, Han et al.
1965). Further research resulted in the location of
additional neural roanatomical Structures Important for Altering Food
Intake). Further research resulted in the location of
additional neural sites that also had profound in-
fluence on the control of food intake (see Other Neu-
roanatomica hyperphagia (Brooks and Lambert 1946, Han et al.
1965). Further research resulted in the location of
additional neural sites that also had profound in-
fluence on the control of food intake (see Other Neu-
roanatomical Str 1965). Further research resulted in the location of
additional neural sites that also had profound in-
fluence on the control of food intake (see Other Neu-
roanatomical Structures Important for Altering Food
Intake). Fin fluence on the control of food intake (see Other Neu-

roanatomical Structures Important for Altering Food

Intake). Finally, the 'center' concept or compartmen-

talized view was challenged as inappropriate for the

manne roanatomical Struct
Intake). Finally, the
talized view was ch
manner in which the
tegrative or syste
(Morgane 1964).
Cephalic phase ake). Finally, the 'center' concept or compartmen-

ized view was challenged as inappropriate for the

inner in which the brain is organized, and an 'in-

rative or systems' approach was suggested

organe 1964).
 Cephalic talized view was challenged as inappropriate for the
manner in which the brain is organized, and an 'in-
tegrative or systems' approach was suggested
(Morgane 1964).
Cephalic phase hypothesis. The cephalic phase
hypothes

manner in which the brain is organized, and an 'in-
tegrative or systems' approach was suggested
(Morgane 1964).
Cephalic phase hypothesis. The cephalic phase
hypothesis superseded the dual-center hypothesis,
taking into tegrative or systems' approach was suggested

(Morgane 1964).
 Cephalic phase hypothesis. The cephalic phase

hypothesis superseded the dual-center hypothesis,

taking into account data accumulated on hypotha-

lamically (Morgane 1964).
 Cephalic phase hypothesis. The cephalic phase

hypothesis superseded the dual-center hypothesis,

taking into account data accumulated on hypotha-

lamically lesioned rats up through the mid-seventies
 Example 19 Compose the Compose Server S hypothesis superseded the dual-center hypothesis,
taking into account data accumulated on hypotha-
lamically lesioned rats up through the mid-seventies
(Powley 1977). The basis of the hypothesis was that
an animal senses t taking into account data accumulated on hypotha-
lamically lesioned rats up through the mid-seventies
(Powley 1977). The basis of the hypothesis was that
an animal senses that it is about to eat, not unlike the
Pavlovian r Example 1977). The basis of the hypothesis was that

an animal senses that it is about to eat, not unlike the

pavlovian response of an animal to the site of food.

Given the above, the hypothesis states that a primary

c (Powley 1977). The basis of the hypothesis was that
an animal senses that it is about to eat, not unlike the
Pavlovian response of an animal to the site of food.
Given the above, the hypothesis states that a primary
conseq an animal senses that it is about to eat, not unlike the
Pavlovian response of an animal to the site of food.
Given the above, the hypothesis states that a primary
consequence of ventromedial hypothalamus lesions is
an exa Pavlovian response of an animal to the site of tood.

Given the above, the hypothesis states that a primary

consequence of ventromedial hypothalamus lesions is

an exaggeration of the cephalic reflexes of digestion

(Pow Given the above, the hypothesis states that a primary
consequence of ventromedial hypothalamus lesions is
an exaggeration of the cephalic reflexes of digestion
(Powley 1977). In other words, the ventromedial
hypothalamic s consequence of ventromedial hypothalamus lesions is
an exaggeration of the cephalic reflexes of digestion
(Powley 1977). In other words, the ventromedial
hypothalamic syndrome is a series of augmented be-
havioral and phys lesions.

Brobeck 1951). The ventromedial hypothalamus,
termed a 'satiety' center, contained a set of neurons
termed a 'satiety' center, contained a set of neurons
that projected to and inhibited a lateral hypothalamic
area, the 'fe The hypothesis thereby explained the magnified EL
The hypothesis thereby explained the magnified
oropharyngeal contact of food exhibited by ven-
tromedial hypothalamically lesioned rats (Teitelbaum EL
The hypothesis thereby explained the magnified
oropharyngeal contact of food exhibited by ven-
tromedial hypothalamically lesioned rats (Teitelbaum
1955), finickiness (Mook and Blass 1968) and in-EL
The hypothesis thereby explained the magnified
oropharyngeal contact of food exhibited by ven-
tromedial hypothalamically lesioned rats (Teitelbaum
1955), finickiness (Mook and Blass 1968) and in-
creased gastric acid s The hypothesis thereby explained the magnified
oropharyngeal contact of food exhibited by ven-
tromedial hypothalamically lesioned rats (Teitelbaum
1955), finickiness (Mook and Blass 1968) and in-
creased gastric acid secr The mypothesis thereby explained the magnined
oropharyngeal contact of food exhibited by ven-
tromedial hypothalamically lesioned rats (Teitelbaum
1955), finickiness (Mook and Blass 1968) and in-
creased gastric acid secre oropharyngeal contact of food exhibited by ven-
tromedial hypothalamically lesioned rats (Teitelbaum
1955), finickiness (Mook and Blass 1968) and in-
creased gastric acid secretion (Powley 1977, Ridley
and Brooks 1965). It tromedial hypothalamically lesioned rats (Tettelbaum
1955), finickiness (Mook and Blass 1968) and in-
creased gastric acid secretion (Powley 1977, Ridley
and Brooks 1965). It is of interest that the cephalic
phase hypothes 1955), Innickiness (MOOK and Biass 1968) and Increased gastric acid secretion (Powley 1977, Ridley and Brooks 1965). It is of interest that the cephalic phase hypothesis was not intended to be restrictive; gastric and inte creased gastric acid secretion (Powiey 1977, Kidley
and Brooks 1965). It is of interest that the cephalic
phase hypothesis was not intended to be restrictive;
gastric and intestinal reflexes could be included for a
general and Brooks 1965). It is or interest that the cephalic
phase hypothesis was not intended to be restrictive;
gastric and intestinal reflexes could be included for a
general autonomic reflex model (Powley 1977). For
example, phase hypothesis was not intended to be restrictive;
gastric and intestinal reflexes could be included for a
general autonomic reflex model (Powley 1977). For
example, taste information, the major exteroceptive
stimulus f gastric and intestinal reflexes could be included for a
general autonomic reflex model (Powley 1977). For
example, taste information, the major exteroceptive
stimulus for cephalic responses, was suggested not to
be segrega general autonomic reflex model (Powley 1977). For
example, taste information, the major exteroceptive
stimulus for cephalic responses, was suggested not to
be segregated from visceral inputs because both
systems project an example, taste information, the major exteroceptive
stimulus for cephalic responses, was suggested not to
be segregated from visceral inputs because both
systems project anatomically to the same important
brainstem nucleu stimulus for cephalic responses, was suggested not to
be segregated from visceral inputs because both
systems project anatomically to the same important
brainstem nucleus, the nucleus tractus solitarius. It
was also known systems project anatomically to the
brainstem nucleus, the nucleus tract
was also known that ventromedia
obesity could be prevented if rats had
diaphragmatic vagotomy prior to h
sions (Powley and Opsahl 1974).
Autonomic a *Autonomic and endocrine hypotheses.* A more beat set of hypotheses was introduced shortly followed and endocrine hypotheses. A more beat set of hypotheses was introduced shortly fol-

systems project anatomically to the same important

brainstem nucleus, the nucleus tractus solitarius. It

was also known that ventromedial hypothalamic

dobesity could be prevented if rats had undergone sub-

diaphragmat was also known that ventromedial hypothalamic
obesity could be prevented if rats had undergone sub-
diaphragmatic vagotomy prior to hypothalamic le-
sions (Powley and Opsahl 1974).
Autonomic and endocrine hypotheses. A m obesity could be prevented if rats had undergone sub-
diaphragmatic vagotomy prior to hypothalamic le-
sions (Powley and Opsahl 1974).
Autonomic and endocrine hypotheses. A more
global set of hypotheses was introduced s mapplementic vagotomy prior to hypothalamic lessions (Powley and Opsahl 1974).
 Autonomic and endocrine hypotheses. A more global set of hypotheses was introduced shortly following the cephalic phase hypothesis. An auto **and endocrine hypotheses.** A more global set of hypotheses was introduced shortly following the cephalic phase hypothesis. An autonomic and phypothesis (Inoue and Bray 1979) and an autonomic and endocrine hypothesis (Bra Autonomic and emocrine hypotheses. A niote
global set of hypotheses was introduced shortly fol-
lowing the cephalic phase hypothesis. An autonomic
hypothesis (lnoue and Bray 1979) and an autonomic
and endocrine hypothesis lowing the cephalic phase hypothesis. An autonomic
hypothesis (Inoue and Bray 1979) and an autonomic
and endocrine hypothesis (Bray and York 1979) sug-
gested that the parasympathetic and sympathetic
nervous systems need t hypothesis (Inoue and Bray 1979) and an autonomic
and endocrine hypothesis (Bray and York 1979) sug-
gested that the parasympathetic and sympathetic
nervous systems need to be incorporated into a model
dealing with food in my
pothesis (mode and Bray 1979) and an autonomic
and endocrine hypothesis (Bray and York 1979) sug-
gested that the parasympathetic and sympathetic
nervous systems need to be incorporated into a model
dealing with food i and endocrine hypothesis (bray and TOR 1979) suggested that the parasympathetic and sympathetic nervous systems need to be incorporated into a model
dealing with food intake or obesity. These hypotheses proposed that reci gested that the parasympathetic and sympathetic
nervous systems need to be incorporated into a model
dealing with food intake or obesity. These hypotheses
proposed that reciprocal alterations occur in the
parasympathetic a mervous systems need to be incorporated into a model
dealing with food intake or obesity. These hypotheses
proposed that reciprocal alterations occur in the
parasympathetic and sympathetic nervous systems
following ventro dealing with lood intake of obesity. These hypotheses
proposed that reciprocal alterations occur in the
parasympathetic and sympathetic nervous systems
following ventromedial hypothalamus lesions so that
activity of the f proposed that reciprocal alterations occur in the

parasympathetic and sympathetic nervous systems

following ventromedial hypothalamus lesions so that

activity of the former is increased and that of the

latter is reduc parasympathetic and sympathetic nervous systems
following ventromedial hypothalamus lesions so that
activity of the former is increased and that of the
latter is reduced. A key change following ven-
tromedial hypothalamus following ventromedial hypothalamus lesions so tha
activity of the former is increased and that of the
latter is reduced. A key change following ven
tromedial hypothalamus lesions was hyperinsulemia
(Assimacopoulos-Jeanne activity of the former is increased and that of the
latter is reduced. A key change following ven-
tromedial hypothalamus lesions was hyperinsulemia
(Assimacopoulos-Jeannet and Jeanrenaud 1976). If
pancreatic β cells a Letter is reduced. A key change following ven-
tromedial hypothalamus lesions was hyperinsulemia
(Assimacopoulos-Jeannet and Jeanrenaud 1976). If
pancreatic β cells are first destroyed in rats by strep-
tozotocin, and tromedial hypothalamus lesions was hyperinsulemia

(Assimacopoulos-Jeannet and Jeanrenaud 1976). If

pancreatic β cells are first destroyed in rats by strep-

tozotocin, and fetal pancreatic tissue is then trans-

plan (Assimacopoulos-jeannet and jeanienaud 1976). If $\frac{2}{2}$
pancreatic β cells are first destroyed in rats by strep-
tozotocin, and fetal pancreatic tissue is then trans-
planted beneath the renal capsule, the operated tozotocin, and fetal pancreatic tissue is then trans-
planted beneath the renal capsule, the operated rats
recover from diabetes. If the same rats are then given
ventromedial hypothalamic lesions, the characteristic
hyperp tozotocin, and retai pancreatic tissue is then trans-
planted beneath the renal capsule, the operated rats
recover from diabetes. If the same rats are then given
ventromedial hypothalamic lesions, the characteristic
hyperp planted beneath the renal capsule, the operated rats
recover from diabetes. If the same rats are then given
ventromedial hypothalamic lesions, the characteristic
hyperphagia, obesity and hyperinsulemia are
prevented, sugge recover from diabetes. If the same rats are then given
ventromedial hypothalamic lesions, the characteristic $\frac{3}{8}$
hyperphagia, obesity and hyperinsulemia are $\frac{3}{8}$
prevented, suggesting that beta cells need to be ventromedial hypothalamic lesions, the characteristic $\frac{3}{88}$
hyperphagia, obesity and hyperinsulemia are $\frac{3}{88}$
prevented, suggesting that beta cells need to be inner-
vated in order to express the classical ventr myperphagia, obesity and nyperhisulemia are $\frac{1}{2}$
prevented, suggesting that beta cells need to be inner-
vated in order to express the classical ventromedial $\frac{10}{2}$
hypothalamic syndrome (Inoue et al. 1978). Beca prevented, suggesting that beta cells need to be inner-
vated in order to express the classical ventromedial
hypothalamic syndrome (Inoue et al. 1978). Because $\frac{3}{100}$
interruption of the vagus nerve below the diaphra vated in order to express the classical ventromedial by
hypothalamic syndrome (Inoue et al. 1978). Because
interruption of the vagus nerve below the diaphragm
prevented obesity following ventromedial hypotha-
lamic lesion mypothalamic syndrome (inoue et al. 1978). Because
interruption of the vagus nerve below the diaphragm
prevented obesity following ventromedial hypotha-
lamic lesions (Powley 1977), activity of the parasym-
pathetic nervou Interruption or
prevented obes
lamic lesions ()
pathetic nervou
ventromedial)
the obesity.
Another tes Evented obesity following ventromedial hypotha-
nic lesions (Powley 1977), activity of the parasym-
thetic nervous system could be enhanced following
ntromedial hypothalamic surgery accounting for
e obesity.
Another testab relations (Powiey 1977), activity of the parasym-
pathetic nervous system could be enhanced following
ventromedial hypothalamic surgery accounting for
the obesity.
Another testable thesis that stems from the
original auton

pathetic nervous system could be enhanced following
ventromedial hypothalamic surgery accounting for
the obesity.
Another testable thesis that stems from the
original autonomic and endocrine theories was
termed the Mona Li ventromedial hypothalamic surgery accounting for
the obesity.
Another testable thesis that stems from the
original autonomic and endocrine theories was
termed the Mona Lisa hypothesis (Bray 1991). The
acronym stands for Mo the obesity.

Another testable thesis that stems from the

original autonomic and endocrine theories was

termed the Mona Lisa hypothesis (Bray 1991). The

acronym stands for Most Obesities kNown Are Low

In Sympathetic Ac Another testable thesis that stems from the
original autonomic and endocrine theories was
termed the Mona Lisa hypothesis (Bray 1991). The
acronym stands for Most Obesities kNown Are Low
In Sympathetic Activity. Figure 1 i original autonomic and endocrine theories was
termed the Mona Lisa hypothesis (Bray 1991). The
acronym stands for Most Obesities kNown Are Low
In Sympathetic Activity. Figure 1 is a modification of
a schematic diagram from termed the Mona Lisa hypothesis (Bray 1991). The
acronym stands for Most Obesities kNown Are Low
In Sympathetic Activity. Figure 1 is a modification of
a schematic diagram from Bray (1991) showing a
reduction in sympatheti

result in an imbalance of the autonomic nervous system FIGURE 1 The autonomic and endocrine hypothesis.
Lesions within the ventromedial hypothalamus (VMH)
result in an imbalance of the autonomic nervous system
(A.N.S.) with an increase and decrease in the activity of the
FIG FIGURE 1 The autonomic and endocrine hypothesis.

Lesions within the ventromedial hypothalamus (VMH)

result in an imbalance of the autonomic nervous system

(A.N.S.) with an increase and decrease in the activity of the

v FIGURE 1 The autonomic and endocrine hypothesis.
Lesions within the ventromedial hypothalamus (VMH)
result in an imbalance of the autonomic nervous system
(A.N.S.) with an increase and decrease in the activity of the
vagus FIGURE 1 The autonomic and endocrine hypothesis.
Lesions within the ventromedial hypothalamus (VMH)
result in an imbalance of the autonomic nervous system
(A.N.S.) with an increase and decrease in the activity of the
vagus Fractic T The autonomic and chuochine hypothesis.
Lesions within the ventromedial hypothalamus (VMH)
result in an imbalance of the autonomic nervous system
(A.N.S.) with an increase and decrease in the activity of the
vagu Fresult in an imbalan
(A.N.S.) with an increvagus and sympather
CORT, corticosteron
secretion; NE, norep
from Bray 1991). **CONSUMERTY AND SET ON SET ON SET ON SET ON SET ON SET OF SET**

For Son, NE, norepinephrine (modified with permission

Islam (1991).
 **STRUCTURES IMPORTANT FOR

ALTERING FOOD INTAKE** _{1991;}
THER NEUROANATOMICAL
RUCTURES IMPORTANT FOR
ALTERING FOOD INTAKE *PARTICLE COLORES IMPO

PALTERING FOOD

Paraventricular nucleus

Anatomical location and in:*

ALIERING FOOD INTAKE
 Anatomical location and initial studies. The ven-
 Anatomical location and initial studies. The ven-

medial hypothalamus and lateral hypothalamic trouble **Paraventricular nucleus
Anatomical location and initial studies.** The ven-
tromedial hypothalamus and lateral hypothalamic
area were not the only neural structures found to alter **Paraventricular nucleus**
Anatomical location and initial studies. The ventromedial hypothalamus and lateral hypothalamic
area were not the only neural structures found to alter
food intake when experimentally manipulate Frame entity and interest and initial studies. The ven-

tromedial hypothalamus and lateral hypothalamic

area were not the only neural structures found to alter

food intake when experimentally manipulated. One

of the mo Anatomical location and initial studies. The ven-
tromedial hypothalamus and lateral hypothalamic
area were not the only neural structures found to alter
food intake when experimentally manipulated. One
of the most importa Anatomical location and initial studies. The ven-
tromedial hypothalamus and lateral hypothalamic
area were not the only neural structures found to alter
food intake when experimentally manipulated. One
of the most importa Increase the parallel and the parallel and the parameters are seen the parameters found to alter food intake when experimentally manipulated. One of the most important studied during the seventies and eighties was the para area were not the only neural structures found to alter
food intake when experimentally manipulated. One
of the most important studied during the seventies
and eighties was the paraventricular nucleus. The
location of the shood intake when experimentally manipulated. One
of the most important studied during the seventies
and eighties was the paraventricular nucleus. The
location of the paraventricular nucleus in the rat
brain is shown in **F** of the most important studied during the seventies
and eighties was the paraventricular nucleus. The
location of the paraventricular nucleus in the rat
brain is shown in **Figure 2**. Because it is most sen-
sitive for norad and eighties was the paraventricular nucleus. The
location of the paraventricular nucleus in the rat
brain is shown in **Figure 2**. Because it is most sen-
sitive for noradrenergic stimulation of food intake,
the paraventr Social of the paraventricular nucleus in the rat

brain is shown in **Figure 2**. Because it is most sen-

sitive for noradrenergic stimulation of food intake,

the paraventricular nucleus is a critical neural

structure (Le brain is shown in **Figure 2**. Because it is most sensitive for noradrenergic stimulation of food intake, the paraventricular nucleus is a critical neural structure (Leibowitz 1978a and 1978b). Bilateral lesions of the para strive for noradientergic stimulation of 1000 intake,
the paraventricular nucleus is a critical neural
structure (Leibowitz 1978a and 1978b). Bilateral le-
sions of the paraventricular nucleus also resulted in
hyperphagia the paraventricular nucleus is a critical neural
structure (Leibowitz 1978a and 1978b). Bilateral le-
sions of the paraventricular nucleus also resulted in
hyperphagia and obesity (Leibowitz et al. 1981). Neu-
roanatomical Structure (Leibowitz 1978a and 1978b). Bilateral le-
sions of the paraventricular nucleus also resulted in
hyperphagia and obesity (Leibowitz et al. 1981). Neu-
roanatomical studies have shown that the nucleus
comprises 8 sions of the paraventricular nucleus also resulted in
hyperphagia and obesity (Leibowitz et al. 1981). Neu-
roanatomical studies have shown that the nucleus
comprises 8 to 10 sub-nuclei (Swanson and Kuypers
1980). More imp median eminence and thereby a direct affect on the anterior pituitary. The magnocellular neurons of the anterior pituitary. The magnocellular neurons of the $\frac{1}{2}$ anterior pituitary. The magnocellular neurons of the $\$ roanatomical studies have shown that the nucleus

comprises 8 to 10 sub-nuclei (Swanson and Kuypers

1980). More importantly, the paraventricular nucleus

seems to have reciprocal connections with the

median eminence and comprises 8 to 10 sub-nuclei (Swanson and Kuypers
1980). More importantly, the paraventricular nucleus
seems to have reciprocal connections with the
median eminence and thereby a direct affect on the
anterior pituitary. Th posterior pituitary. The paraventricular nucleus

median eminence and thereby a direct affect on the

median eminence and thereby a direct affect on the

paraventricular nucleus project directly to the

posterior pituitary seems to have reciprocal connections with the
median eminence and thereby a direct affect on the
anterior pituitary. The magnocellular neurons of the
posterior pituitary. The paraventricular nucleus also
has a monosynaptic median eminence and thereby a direct affect on the
anterior pituitary. The magnocellular neurons of the
paraventricular nucleus project directly to the
posterior pituitary. The paraventricular nucleus also
has a monosynapt paraventricular nucleus project directly to the posterior pituitary. The paraventricular nucleus also has a monosynaptic connection to the nucleus tractus solitarius and dorsal motor nucleus of the vagus, and, therefore, c posterior pituitary. The paraventricular nucleus also
has a monosynaptic connection to the nucleus also
has a monosynaptic connection to the nucleus tractus
solitarius and dorsal motor nucleus of the vagus, and,
therefore, posterior pituitary. The paraventricular nucleus also
has a monosynaptic connection to the nucleus tractus
solitarius and dorsal motor nucleus of the vagus, and,
therefore, can directly affect the parasympathetic
pharavent has a monosynaptic connection to the nucleus tractu
solitarius and dorsal motor nucleus of the vagus, and
therefore, can directly affect the parasympathet
nervous system (Swanson and Sawchenko 1983). Th
paraventricular nuc solitarius and dorsal motor nucleus of the vagus, and,
therefore, can directly affect the parasympathetic
nervous system (Swanson and Sawchenko 1983). The
paraventricular nucleus has also been shown to
project to the peria

location and approximate size of the paraventricular nu-AHP

AHP
 EIGURE 2 Cross-section of a rat brain taken at interaural

plate 7.2 mm of Paxinos and Watson (1986) showing the

location and approximate size of the paraventricular nu-

cleus (PVN). Abbreviations used. AHP, AHP

FIGURE 2 Cross-section of a rat brain taken at interaural

plate 7.2 mm of Paxinos and Watson (1986) showing the

location and approximate size of the paraventricular nu-

cleus (PVN). Abbreviations used: AHP, anterio FIGURE 2 Cross-section of a rat brain taken at interaural plate 7.2 mm of Paxinos and Watson (1986) showing the location and approximate size of the paraventricular nucleus (PVN). Abbreviations used: AHP, anterior hypothal

cleus (PVN). Abbreviations used: AHP, anterior hypotha-
lamic area, posterior, AL, ansa lenticularis, F, fornix, FPM,
medial forebrain bundle, LH, lateral hypothalamic area.
sympathetic nervous system (Luiten et al. 1987) structure appears to be well situated and connected to
affect food intake as well as serve other physiological
affect food intake as well as serve other physiological and internal bundle, 217, 2001al 2, point and connected
sympathetic nervous system (Luiten et al. 1987). This
structure appears to be well situated and connected to
affect food intake as well as serve other physiological
a sympathetic nervous system
structure appears to be well s
affect food intake as well as
and behavioral functions.
Regulatory vs. metabolic mpathetic nervous system (Luiten et al. 1987). This ucture appears to be well situated and connected to exect food intake as well as serve other physiological dehavioral functions.
Regulatory vs. metabolic obesity. A clas sympathetic nervous system (Luiten et al. 1987). This
structure appears to be well situated and connected to
affect food intake as well as serve other physiological
and behavioral functions.
Regulatory vs. metabolic obes

FFM
 **FIGURE 2 Cross-section of a rat brain taken at interaural
plate 7.2 mm of Paxinos and Watson (1986) showing the
location and paproximate size of the paraventricular numerical
cleus [PVN]. Abhreviations used: AHP, a** structure appears to be well situated and connected to
affect food intake as well as serve other physiological
and behavioral functions.
Regulatory vs. metabolic obesity. A classic study
compared the metabolic and behav attect tood intake as well as serve other physiological
and behavioral functions.
Regulatory vs. metabolic obesity. A classic study
compared the metabolic and behavioral differences in
rats that sustained bilateral lesi and behavioral tunctions.
 Regulatory vs. metabolic obesity. A classic study

compared the metabolic and behavioral differences in

rats that sustained bilateral lesions of the ven-

tromedial hypothalamus vs. the parav Regulatory vs. metabolic obesity. A classic study
compared the metabolic and behavioral differences in
rats that sustained bilateral lesions of the ven-
tromedial hypothalamus vs. the paraventricular nu-
cleus (Weingarten compared the metabolic and behavioral differences in
rats that sustained bilateral lesions of the ven-
tromedial hypothalamus vs. the paraventricular nu-
cleus (Weingarten et al. 1985). Both groups showed
hyperphagia and s rats that sustained bilateral lesions of the ven-
tromedial hypothalamics vs. the paraventricular nu-
cleus (Weingarten et al. 1985). Both groups showed
hyperphagia and similar body weight gains. However,
at the end of th tromedial hypothalamus vs. the paraventricular nu-
cleus (Weingarten et al. 1985). Both groups showed
hyperphagia and similar body weight gains. However,
at the end of the first experiment, ventromedial
hypothalamically l cleus (Weingarten et al. 1985). Both groups showed
hyperphagia and similar body weight gains. However,
at the end of the first experiment, ventromedial
hypothalamically lesioned rats were fatter. When food
was freely avai hyperphagia and similar body weight gains. However,
at the end of the first experiment, ventromedial
hypothalamically lesioned rats were fatter. When food
was freely available, ventromedial hypothalamically
lesioned rats at the end of the first experiment, ventromedial $\frac{2}{5}$
hypothalamically lesioned rats were fatter. When food
was freely available, ventromedial hypothalamically $\frac{1}{50}$
lesioned rats also exhibited hyperinsulemia. hypothalamically lesioned rats were fatter. When food
was freely available, ventromedial hypothalamically
lesioned rats also exhibited hyperinsulemia. Further
experiments showed that when food intake was re-
stricted, the was freely available, ventromedial hypothalamically \overline{B}
lesioned rats also exhibited hyperinsulemia. Further
experiments showed that when food intake was re-
stricted, the paraventricular nucleus lesioned group
had t lesioned rats also exhibited hyperinsulemia. Further
experiments showed that when food intake was re-
stricted, the paraventricular nucleus lesioned group
had the same percentage of carcass fat as controls, but
the ventro experiments showed that when food intake was re-
stricted, the paraventricular nucleus lesioned group
had the same percentage of carcass fat as controls, but
the ventromedial hypothalamically lesioned group
had a signific stricted, the paraventricular nucleus lesioned group
had the same percentage of carcass fat as controls, but
the ventromedial hypothalamically lesioned group
had a significantly higher percentage of fat. In ad-
dition, ga had the same percentage of carcass fat as controls, but
the ventromedial hypothalamically lesioned group
had a significantly higher percentage of fat. In ad-
dition, gastric acid secretion was elevated only in
ventromedia the ventromedial hypothalamically lesioned group
had a significantly higher percentage of fat. In ad-
dition, gastric acid secretion was elevated only in
wentromedial hypothalamus lesioned rats. From these
data, it was co had a significantly higher percentage of tat. In ad-
dition, gastric acid secretion was elevated only in
ventromedial hypothalamus lesioned rats. From these
data, it was concluded that ventromedial hypothalam-
ically lesi dition, gastric acid secretion was elevated only in
ventromedial hypothalamus lesioned rats. From these
data, it was concluded that ventromedial hypothalam-
ically lesioned rats showed metabolic obesity and
paraventricular ventromedial hypothalamus lesioned rats. From these
data, it was concluded that ventromedial hypothalam-
ically lesioned rats showed metabolic obesity and
paraventricular nucleus lesioned rats showed
regulatory obesity (**T** data, it was concluded that ventromedial hypothalamically lesioned rats showed metabolic obesity and
paraventricular nucleus lesioned rats showed
regulatory obesity (**Table 2**; Weingarten et al. 1985).
In other words the p ically lesioned rats showed metabolic obesity and
paraventricular nucleus lesioned rats showed
regulatory obesity (**Table 2**; Weingarten et al. 1985).
In other words the primary disturbance of paraven-
tricular nucleus les paraventricular nucleus lesioned rats showed
regulatory obesity (**Table 2**; Weingarten et al. 1985).
In other words the primary disturbance of paraven-
tricular nucleus lesioned rats is hyperphagia, ven-
tromedial hypothal regulatory obesity (**Table 2**; Weingarten et al. 1985).
In other words the primary disturbance of paraventricular nucleus lesioned rats is hyperphagia; ven-
tromedial hypothalamically lesioned animals show
metabolic change phagia. Figure 11 and the probability controller that the medial hypothalamically lesioned animals show trabolic changes such as hyperinsulemia, which descondarily to obesity with or without hyper-
agia.
Ventromedial hypothalamic tromedial hypothalamically lesioned animals show
metabolic changes such as hyperinsulemia, which
lead secondarily to obesity with or without hyper-
phagia.
Ventromedial hypothalamic lesions: single versus
multiple indepe

metabolic changes such as hyperinsulemia, which
lead secondarily to obesity with or without hyper-
phagia.
Ventromedial hypothalamic lesions: single versus
multiple independent effects. A second fundamental
interpretat lead secondarily to obesity with or without hyper-
phagia.
Ventromedial hypothalamic lesions: single versus
multiple independent effects. A second fundamental
interpretation made in the Weingarten et al. (1985)
study w

TABLE 2
TABLE 2
TWo distinct disorders occur when the ventromedial
ypothalamus (VMH) or paraventricular nucleus (PVN)
*is bilaterally lesioned*¹ **hypothalamus (VMH)** or paraventricular nucleus (PVN)
 is bilaterally lesioned¹
 is bilaterally lesioned¹

'Weingarten et al. 1985.

Secretion

IWeingarten et al. 1985.

(dual center and cephalic phase) proposed a single,

primary etiological factor as responsible for the ven-Weingarten et al. 1985.

(dual center and cephalic phase) proposed a single,

primary etiological factor as responsible for the ven-

tromedial hypothalamic syndrome, which then (dual center and cephalic phase) proposed a single,
primary etiological factor as responsible for the ven-
tromedial hypothalamic syndrome, which then
resulted in secondary effects. Specifically the first (dual center and cephalic phase) proposed a single,
primary etiological factor as responsible for the ven-
tromedial hypothalamic syndrome, which then
resulted in secondary effects. Specifically the first
hypothesis champi (dual center and cephalic phase) proposed a single,
primary etiological factor as responsible for the ven-
tromedial hypothalamic syndrome, which then
resulted in secondary effects. Specifically the first
hypothesis champi (dual center and cephalic phase) proposed a single,
primary etiological factor as responsible for the ven-
tromedial hypothalamic syndrome, which then
resulted in secondary effects. Specifically the first
hypothesis champi primary etiological factor as responsible for the ven-
tromedial hypothalamic syndrome, which then
resulted in secondary effects. Specifically the first
hypothesis championed hyperphagia as the primary
etiological factor (tromedial hypothalamic syndrome, which then
resulted in secondary effects. Specifically the first
hypothesis championed hyperphagia as the primary
etiological factor (Brobeck et al. 1943) and the second
suggested an elevat resulted in secondary effects. Specifically the first
hypothesis championed hyperphagia as the primary
etiological factor (Brobeck et al. 1943) and the second
suggested an elevation of the cephalic phase of
digestion (Powl hypothesis championed hyperphagia as the primary
etiological factor (Brobeck et al. 1943) and the second
suggested an elevation of the cephalic phase of
digestion (Powley 1977). An alternative interpretation
is that the la etiological factor (Brobeck et al. 1943) and the second
suggested an elevation of the cephalic phase of
digestion (Powley 1977). An alternative interpretation
is that the large lesions or knife cuts directed toward
the ven suggested an elevation of the cephalic phase of
digestion (Powley 1977). An alternative interpretation
is that the large lesions or knife cuts directed toward
the ventromedial hypothalamus induced a series of
multiple, in digestion (Powley 1977). An alternative interpretation
is that the large lesions or knife cuts directed toward
the ventromedial hypothalamus induced a series of
multiple, independent disturbances, which, given the
appropri is that the large lesions or knife cuts directed toward
the ventromedial hypothalamus induced a series of
multiple, independent disturbances, which, given the
gaspropriate anatomical and behavioral analyses, could
be diss the ventromedial hypothalamus induced a series of
multiple, independent disturbances, which, given the
appropriate anatomical and behavioral analyses, could
be dissociated from one another (Fig. 3A, Weingarten
et al. 1985) multiple, independent disturbances, which, appropriate anatomical and behavioral analys
be dissociated from one another (Fig. 3A; We
et al. 1985). A similar interpretation was m
birds following ventromedial hypothalamic
wh **Parabrachial nucleus**
et al. 1985). A similar interposite following ventromedi
which will be presented lat
Parabrachial nucleus

Anatomical location and initial studies. The
 Anatomical location and initial studies. The
 Anatomical location and initial studies. The

rabrachial nucleus is another locus important in

a control of food inteks. It Parabrachial nucleus $\begin{array}{c|c}\n\textbf{Parabrachial nucleus} & \textbf{and} & \textbf{initial studies.} \\
\textbf{anational locations} & \textbf{and} & \textbf{initial studies.} \\
\textbf{parametrized} & \textbf{in} & \textbf{in} \\
\textbf{in} & \$ **Parabrachial nucleus**
Anatomical location and initial studies. The
parabrachial nucleus is another locus important in
the control of food intake. It is located in a cross-
section of brain tissue (Fig. 4) 0.28 mm anteri **Parabrachial nucleus**
 Anatomical location and initial studies. The

parabrachial nucleus is another locus important in

the control of food intake. It is located in a cross-

section of brain tissue (Fig. 4) 0.28 mm a **Anatomical location and initial studies.** The
parabrachial nucleus is another locus important in
the control of food intake. It is located in a cross-
section of brain tissue (Fig. 4) 0.28 mm anterior to the
interaural ze Anatomical location and initial studies. The
parabrachial nucleus is another locus important in
the control of food intake. It is located in a cross-
section of brain tissue (Fig. 4) 0.28 mm anterior to the
interaural zero parabrachial nucleus is another locus important in
the control of food intake. It is located in a cross-
section of brain tissue (Fig. 4) 0.28 mm anterior to the
interaural zero reference point of the stereotaxic atlas
of the control of food intake. It is located in a cross-
section of brain tissue (Fig. 4) 0.28 mm anterior to the
interaural zero reference point of the stereotaxic atlas
of the rat brain (Paxinos and Watson 1986). Bilateral
 section of brain tissue (Fig. 4) 0.28 mm anterior to the
interaural zero reference point of the stereotaxic atlas
of the rat brain (Paxinos and Watson 1986). Bilateral
lesions of the dorsolateral parabrachial nucleus ef-
f interaural zero reference point of the stereotaxic atlas
of the rat brain (Paxinos and Watson 1986). Bilateral
lesions of the dorsolateral parabrachial nucleus ef-
fected hyperphagia and obesity (Nagai et al. 1987). Of
rel of the rat brain (Paxinos and Watson 1986). Bilateral
lesions of the dorsolateral parabrachial nucleus ef-
fected hyperphagia and obesity (Nagai et al. 1987). Of
relevance is the finding that a major projection to the
core lesions of the dorsolateral parabrachial nucleus effected hyperphagia and obesity (Nagai et al. 1987). Of
relevance is the finding that a major projection to the
core of the ventromedial hypothalamus originates
from the do Fected hyperphagia and obesity (Nagai et al. 1987). Of
relevance is the finding that a major projection to the
core of the ventromedial hypothalamus originates
from the dorsolateral parabrachial region (Záborszky
et al. 19 relevance is the finding that a major projection to the
core of the ventromedial hypothalamus originates
from the dorsolateral parabrachial region (Záborszky
et al. 1984). More importantly, the ventromedial
hypothalamus ha core of the ventromedial hypothalamus originates
from the dorsolateral parabrachial region (Záborszky
et al. 1984). More importantly, the ventromedial
hypothalamus has been shown to have the highest
cholecystokinin levels from the dorsolateral parabrachial region (Záborszky
et al. 1984). More importantly, the ventromedial
hypothalamus has been shown to have the highest
cholecystokinin levels within the hypothalamus
(Beinfeld and Palkovits 1 et al. 1984). More importantly, the ventromedial
hypothalamus has been shown to have the highest
cholecystokinin levels within the hypothalamus
(Beinfeld and Palkovits 1981). When that projection
was severed, cholecystokin cholecystokinin levels within the hypothalamus

(Beinfeld and Palkovits 1981). When that projection

was severed, cholecystokinin disappeared almost to-

tally from the ventromedial hypothalamus

(Záborszky et al. 1984). R

APPROACHES TO ANALYSIS OF VMH LESIONS
A MARALS **APPROACHES TO**
A. <u>MAMMALS</u>
1. SINGLE PRIMARY PROACHES TO ANALYS
IAMMALS
1. SINGLE PRIMARY CAUSE:

FIGURE 3 A) Approaches to analysis of ventromedial
hypothalamic (VMH) lesions. Scheme developed by Weingarten et al. (1985) showing either one primary effect of
NMH lesions in moving either one primary effect of
NMH lesio (KUENZEL,1974)

FIGURE 3 A) Approaches to analysis of ventromedial

hypothalamic (VMH) lesions. Scheme developed by Wein-

garten et al. (1985) showing either one primary effect of

VMH lesions in mammals or multiple inde FIGURE 3 A) Approaches to analysis of ventromedial
hypothalamic (VMH) lesions. Scheme developed by Wein-
garten et al. (1985) showing either one primary effect of
VMH lesions in mammals or multiple independent effects.
eff **FIGURE 3** A) Approaches to analysis of ventromedial hypothalamic (VMH) lesions. Scheme developed by Weingarten et al. (1985) showing either one primary effect of VMH lesions in mammals or multiple independent effects. B) **FIGURE 3 A**) Approaches
hypothalamic (VMH) lesions.
garten et al. (1985) showing
VMH lesions in mammals or
B) Birds and mammals appear t
effects from VMH lesions. VMH lesions in mammals or multiple independent effects.
 B) Birds and mammals appear to have multiple, independent

effects from VMH lesions.

(cholecystokinin) known to inhibit food intake (**Table**

3; Gibbs and Smit

 $\frac{3}{2}$
B) Birds and mammals appear to have multiple, independent
effects from VMH lesions.
(cholecystokinin) known to inhibit food intake (**Table**
3; Gibbs and Smith 1992) could account for the hyper-
phagia shown by r encets nom vmn resions.
(cholecystokinin) known to inhibit food intake (**Table**
3; Gibbs and Smith 1992) could account for the hyper-
phagia shown by rats lesioned in the parabrachial
nucleus. Hyperinsulemia was observed i (cholecystokinin) known to inhibit food intake (**Table 3**; Gibbs and Smith 1992) could account for the hyper-
phagia shown by rats lesioned in the parabrachial 9
nucleus. Hyperinsulemia was observed in bilaterally $\frac{5}{5$ (cholecystokinin) known to inhibit food intake (**Table**
3; Gibbs and Smith 1992) could account for the hyper-
phagia shown by rats lesioned in the parabrachial
nucleus. Hyperinsulemia was observed in bilaterally
lesioned p (cholecystokinin) known to inhibit rood intake (**1able**
3, Gibbs and Smith 1992) could account for the hyper-
phagia shown by rats lesioned in the parabrachial $\frac{S}{2}$
nucleus. Hyperinsulemia was observed in bilaterally 3; Gibos and Smith 1992) could account for the hyper-
phagia shown by rats lesioned in the parabrachial $\frac{3}{8}$
nucleus. Hyperinsulemia was observed in bilaterally $\frac{3}{8}$
lesioned parabrachial nucleus rats after food lesioned parabrachial nucleus rats after food deprivation for 2 h (Nagai et al. 1987). More data are needed to determine whether parabrachial nucleus rats will show elevated G.A.S., hyperinsulemia, and obesity when food in lesioned parabrachial nucleus rats arter rood deprivation for 2 h (Nagai et al. 1987). More data are needed to determine whether parabrachial nucleus $\frac{8}{5}$ rats will show elevated G.A.S., hyperinsulemia, and obesity w vation for 2 h (Nagai et al. 1987). More data are
needed to determine whether parabrachial nucleus
rats will show elevated G.A.S., hyperinsulemia, and
obesity when food intake is restricted to control
levels in order to as In show elevated G.A.S., hyperinsulemia, and
when food intake is restricted to control
n order to assess whether parabrachial nucleus
ow a regulatory or metabolic-type of obesity.
COMPOUNDS ADMINISTERED
CENTRALLY—MAMMALS

CENTRALLY—MAMMALS COMPOUNDS ADMINISTERED
CENTRALLY—MAMMALS
Many compounds have been identified in neural

histochemical procedures were developed and phar-CENTRALLY—MAMMALS

Many compounds have been identified in neural

tissue since the 1950s. Biochemical assays and

histochemical procedures were developed and phar-

macological agents synthesized to characterize better Many compounds have been identified in neural
tissue since the 1950s. Biochemical assays and
histochemical procedures were developed and phar-
macological agents synthesized to characterize better
the neuroanatomical syste Many compounds have been identified in neural
tissue since the 1950s. Biochemical assays and
histochemical procedures were developed and phar-
macological agents synthesized to characterize better
the neuroanatomical syste

THE JOURNAL OF NUTRITION

Compounds given centrally to mammals that altered food intake

JN THE JOURNAL OF NUTRITION

(continued)

IN THE JOURNAL OF NUTRITION

TABLE 3 continued
 TABLE 3 continued
 TABLE 3 continued
 TABLE 4 communes

Solutarius; PFH, perifornical hypothalamus; PVN, pWN, DMN, Decreased McGowan et al. 1992

14 abbreviations used in this column: Amyg, amygdala; DMN, dorsomedial hypothalamus nucleus; GP, globus pallidus; ICV,

14 accrebrov XMN, PVN, DMN, Decreased MCGOV

arcuate nucleus

Abbreviations used in this column: Amyg, amygdala; DMN, dorsomedial hypothalamus

cerebroventricular; Intra-Hypo, intra-hypothalamic; LHy, lateral hypothalamic area; MPOA, r Abbreviations used in this column: Amyg, amygdala, DMN, dorsomedial hypothalamus nucleus, GP, globus pallidus, ICV, increrebroventricular; Intra-Hypo, intra-hypothalamic; LHy, lateral hypothalamic area; MPOA, medial preop Abbreviations used in this column: Amygraderacerebroventricular, Intra-Hypo, intra-hypothalasolitarius, PFH, periformical hypothalamus, PVN

²Higher doses of galanin were required to o³An intact area postrema, nucleus

solitarius; PFH, perifornical hypothalamus; PVN, paraventricular nucleus; Str, striatum; VMH, ventromedial hypothalamus area.
²Higher doses of galanin were required to obtain a significant increase in food intake.
³An

Smith et al. 1985, South and Ritter 1988).
⁴Cyclo-His-Pro = cyclo-histidyl-proline-diketopiperazine.
The following is a brief summary of the major neu-rochemical compounds that have been microinjected ⁴Cyclo-His-Pro = cyclo-histidyl-proline-diketopiperazine.
The following is a brief summary of the major neurochemical compounds that have been microinjected lieither within the cerebroventricular system or within The following is a brief summary of the major neu-
rochemical compounds that have been microinjected
either within the cerebroventricular system or within
specific neuroanatomical structures of the mam-The following is a brief summary of the major neu-
rochemical compounds that have been microinjected
either within the cerebroventricular system or within
specific neuroanatomical structures of the mam-
malian brain. (McLe The following is
rochemical comp
either within the
specific neuroan
malian brain.
Biogenic amin Fremical compounds that have been microinjected
 Biogenic amines. The first biogenic amine shown
 Biogenic amines. The first biogenic amine shown

have an affect upon food intake was

inter-France and compounds that have been microinjected

either within the cerebroventricular system or within

specific neuroanatomical structures of the mam-

malian brain.
 Biogenic amines. The first biogenic amine shown

t

either within the cerebroventricular system or within
specific neuroanatomical structures of the mam-
malian brain.
Biogenic amines. The first biogenic amine shown
to have an affect upon food intake was
inorepinephrine. Wh specific neuroanatomical structures of the mam-

malian brain.
 Biogenic amines. The first biogenic amine shown

to have an affect upon food intake was

interal periformical hypothalamus, it decreased food

lintake (Gros malian brain.
 Biogenic amines. The first biogenic amine shown

to have an affect upon food intake was

norepinephrine. When it was injected within the

lateral periformical hypothalamus, it decreased food

intake (Gross Biogenic amines. The first biogenic amine shown
to have an affect upon food intake was
inorepinephrine. When it was injected within the
lateral periformical hypothalamus, it decreased food
intake (Grossman 1962). Dopamine to have an affect upon food intake was
norepinephrine. When it was injected within the
lateral periformical hypothalamus, it decreased food
intake (Grossman 1962). Dopamine and epinephrine
likewise depressed feeding when i morepinephrine. When it was injected within the

lateral periformical hypothalamus, it decreased food

intake (Grossman 1962). Dopamine and epinephrine

likewise depressed feeding when injected into the

same neural area lateral periformical hypothalamus, it decreased food
intake (Grossman 1962). Dopamine and epinephrine
likewise depressed feeding when injected into the
same neural area (Leibowitz and Rossakis 1979a and
1979b). It was det likewise depressed feeding when injected into the same neural area (Leibowitz and Rossakis 1979a and 1979b). It was determined that the catecholamine and amphetamine suppression of food intake involved β -adrenergic rec likewise depressed teeding when injected into the
same neural area (Leibowitz and Rossakis 1979a and
1979b). It was determined that the catecholamine and
amphetamine suppression of food intake involved β -
adrenergic re same neural area (Leibowitz and Rossakis 1979a and
1979b). It was determined that the catecholamine and
amphetamine suppression of food intake involved β -
adrenergic receptors (Leibowitz 1976). In contrast,
food intake 1979b). It was determined that the catecholamine and
amphetamine suppression of food intake involved β -
adrenergic receptors (Leibowitz 1976). In contrast,
food intake was significantly increased upon
norepinephrine ad amphetamine suppression of food intake involved β -
adrenergic receptors (Leibowitz 1976). In contrast,
food intake was significantly increased upon
norepinephrine administration directly into the
paraventricular nucleu adrenergic receptors (Leibowitz 1976). In contrast,
food intake was significantly increased upon
norepinephrine administration directly into the
paraventricular nucleus (Leibowitz 1978a). α 2-
Noradrenergic receptors wer norepinephrine administration directly into the
paraventricular nucleus (Leibowitz 1978a). α 2-
Noradrenergic receptors were shown to be involved in
the response (Goldman et al. 1985). Serotonin consis-
tently decreased Noradrenergic receptors were shown to be involved in paraventricular nucleus (Leibowitz 1978a). α 2-
Noradrenergic receptors were shown to be involved in
the response (Goldman et al. 1985). Serotonin consis-
tently decreased food intake in mammals (Blundell
1984, Leibowitz Noradrenergic receptors were shown to be involved in
the response (Goldman et al. 1985). Serotonin consis-
tently decreased food intake in mammals (Blundell
1984, Leibowitz and Shor-Posner 1986). The principle
site of acti the response (Goldman et al. 1985). Serotonin consistently decreased food intake in mammals (Blundell 1984, Leibowitz and Shor-Posner 1986). The principle site of action of serotonin appears to be the paraventricular nucle tently decreased tood intake in mammals (Blundell
1984, Leibowitz and Shor-Posner 1986). The principle
site of action of serotonin appears to be the paraven-
tricular nucleus. Serotonin has been shown to an-
tagonize norep 1984, Leibowitz and Shor-Posner 1986). The principle
site of action of serotonin appears to be the paraven-
tricular nucleus. Serotonin has been shown to an-
tagonize norepinephrine-stimulated feeding, reduce
the size and site of action of serotonin appears to be the paraventricular nucleus. Serotonin has been shown to antagonize norepinephrine-stimulated feeding, reduce
the size and duration of meals, decrease the rate of
feeding, but not tricular nucleus. Serotonin h
tagonize norepinephrine-stim
the size and duration of mea
feeding, but not influence lat
frequency of meals (Leibowitz
see Table 3 for summary).
Neuropeptides that stime Figure 10. The propriet of meals, decrease the rate of
 Neuropeptides that stimulated feeding, reduce
 Neuropeptides that stimulate food intake. A

mber of peptides have been shown to stimulate
 Neuropeptides have be the size and duration of meals, decrease the rate of
feeding, but not influence latency to meal onset or
frequency of meals (Leibowitz and Shor-Posner 1986;
see Table 3 for summary).
Neuropeptides that stimulate food intak

frequency of meals (Leibowitz and Shor-Posner 1986;
frequency of meals (Leibowitz and Shor-Posner 1986;
see Table 3 for summary).
Neuropeptides that stimulate food intake. A
number of peptides have been shown to stimulate frequency of meals (Leibowitz and Shor-Posner 1986;
see Table 3 for summary).
Neuropeptides that stimulate food intake. A
number of peptides have been shown to stimulate
food intake, particularly those within the opioid a see Table 3 for summary).

Neuropeptides that stimulate food intake. A

number of peptides have been shown to stimulate

food intake, particularly those within the opioid and

pancreatic polypeptide families. β -endorphi Neuropeptides that stimulate food intake. A
number of peptides have been shown to stimulate
food intake, particularly those within the opioid and
pancreatic polypeptide families. β -endorphin injected
into the ventromedi mumber of peptides have been shown to stimulate
food intake, particularly those within the opioid and
pancreatic polypeptide families. β -endorphin injected
into the ventromedial hypothalamic region (Gran-
dison and Gui

Example 1982) increased feeding in rats. Long-acting analogues of leucine and methionine enkephalin, $\overline{[D- \frac{m}{2}]}$ Hor 1982) increased feeding in rats. Long-acting ana-
logues of leucine and methionine enkephalin, [D-
alanine²-D-leucine⁵]-enkephalin (Tepperman and Hor 1982) increased feeding in rats. Long-acting analogues of leucine and methionine enkephalin, [D-
alanine²-D-leucine⁵]-enkephalin (Tepperman and $\frac{Z}{Z}$
Hirst 1983) and [D-alanine²-methionine]-enkephalin Hor 1982) increased feeding in rats. Long-acting ana-
logues of leucine and methionine enkephalin, [D-
alanine²-D-leucine⁵]-enkephalin (Tepperman and
Hirst 1983) and [D-alanine²-methionine]-enkephalin
(McLean and Hoe Hor 1982) increased feeding in rats. Long-acting analogues of leucine and methionine enkephalin, [D-
alanine²-D-leucine⁵]-enkephalin (Tepperman and $\frac{Z}{Z}$
Hirst 1983) and [D-alanine²-methionine]-enkephalin
(McLea From 1982) increased reeding in rats. Long-acting analogues of leucine and methionine enkephalin, [D-
alanine²-D-leucine⁵]-enkephalin (Tepperman and $\frac{2}{5}$
Hirst 1983) and [D-alanine²-methionine]-enkephalin
(McLe interior Findmannian and methodic enterprising and
alanine²-D-leucine⁵]-enkephalin (Tepperman and
Hirst 1983) and [D-alanine²-methionine]-enkephalin
(McLean and Hoebel 1983, Morley et al. 1982), respec-
tively enhanc alanine²-D-leucine³]-enkephalin (Tepperman and $\frac{z}{\infty}$
Hirst 1983) and [D-alanine²-methionine]-enkephalin
(McLean and Hoebel 1983, Morley et al. 1982), respec-
tively enhance food intake. Dynorphin increases foo Hirst 1983) and [D-alanine²-methionine]-enkephalin

[McLean and Hoebel 1983, Morley et al. 1982], respec-

tively enhance food intake. Dynorphin increases food

intake in rats during the light phase of the diurnal

cycl Morlean and Hoeber 1983, Morley et al. 1982), respectively enhance food intake. Dynorphin increases food
intake in rats during the light phase of the diurnal
cycle (Morley and Levine 1981) and dynorphin (1-17)
appears to tively emiance food intake. Dynorphin increases food
intake in rats during the light phase of the diurnal
cycle (Morley and Levine 1981) and dynorphin (1-17)
appears to be more effective than dynorphin (1-13,
Morley and L

FIGURE 4 Cross-section of a rat brain taken at interaural FIGURE 4 Cross-section of a rat brain taken at interaural
plate 0.28 mm of Paxinos and Watson (1986) showing the
location and approximate size of the parabrachial nucleus
(PB). Abbreviations used: KF, Kölliker-Fuse nucleus FIGURE 4 Cross-section of a rat brain taken at interaural
plate 0.28 mm of Paxinos and Watson (1986) showing the
location and approximate size of the parabrachial nucleus
(PB). Abbreviations used: KF, Kölliker-Fuse nucleus **FIGURE 4** Cross-section of a raplate 0.28 mm of Paxinos and W.
location and approximate size of (PB). Abbreviations used: KF, Köloral pontine reticular nucleus.

CONFERENCE: FOOD INTAKE REGULATION
most sensitive brain loci for D-alanine²-methionine⁵- injection of mercapt
enkephalinamide induced feeding are: paraventricular A major ascending in CONFERENCE: FOOD I
most sensitive brain loci for D-alanine²-methionine⁵-
enkephalinamide induced feeding are: paraventricular
nucleus, dorsomedial hypothalamic nucleus, lateral CONFERENCE: FOOD II
most sensitive brain loci for D-alanine²-methionine⁵-
enkephalinamide induced feeding are: paraventricular
nucleus, dorsomedial hypothalamic nucleus, lateral
hypothalamic area, septum and amygdala (CONFERENCE: FOOD IN

most sensitive brain loci for D-alanine²-methionine⁵-

enkephalinamide induced feeding are: paraventricular

nucleus, dorsomedial hypothalamic nucleus, lateral

hypothalamic area, septum and amygda most sensitive brain lo
enkephalinamide induo
nucleus, dorsomedial
hypothalamic area, sep
al. 1988; Table 3).
Members of the par. Solution: Solution and Correct Control of the phalinamide induced feeding are: paraventricular

cleus, dorsomedial hypothalamic nucleus, lateral

pothalamic area, septum and amygdala (Stanley et

1988; Table 3).

Members o enkephalinamide induced feeding are: paraventriculanucleus, dorsomedial hypothalamic nucleus, lateral
hypothalamic area, septum and amygdala (Stanley et
al. 1988; Table 3).
Members of the pancreatic polypeptide family are

metal. 1988; Table 3).

Al. 1988; Table 3).

Members of the pancreatic polypeptide family are

also effective in stimulating food intake. Neu-

ropeptide Y, peptide YY and pancreatic polypeptide

are included in the family hypothalamic area, septum and amygdala (Stanley et
al. 1988; Table 3).
Members of the pancreatic polypeptide family are
also effective in stimulating food intake. Neu-
ropeptide Y, peptide YY and pancreatic polypeptide
are al. 1988; Table 3).

Members of the pancreatic polypeptide family are

also effective in stimulating food intake. Neu-

ropeptide Y, peptide YY and pancreatic polypeptide

are included in the family. Administration of neu-Members of the pancreatic polypeptide tamily are

also effective in stimulating food intake. Neu-

ropeptide Y, peptide YY and pancreatic polypeptide

are included in the family. Administration of neu-

ropeptide Y into th also effective in stimulating food intake. Neu-
ropeptide Y, peptide YY and pancreatic polypeptide
are included in the family. Administration of neu-
ropeptide Y into the cerebral ventricles (Clark et al.
1984, Levine and ropeptide Y, peptide YY and pancreatic polypeptide

are included in the family. Administration of neu-

ropeptide Y into the cerebral ventricles (Clark et al.

1984, Levine and Morley 1984) or directly into the

paraventri are included in the family. Administration of neuropeptide Y into the cerebral ventricles (Clark et al. 1984, Levine and Morley 1984) or directly into the paraventricular nucleus (Stanley and Leibowitz 1984) effected marke resulted Y into the cerebral ventricles (Clark et al. 1984, Levine and Morley 1984) or directly into the paraventricular nucleus (Stanley and Leibowitz 1984) effected marked hyperphagia. Chronic administration of neuropept 1984, Levine and Morley 1984) or directly into the
paraventricular nucleus (Stanley and Leibowitz 1984)
effected marked hyperphagia. Chronic administration
of neuropeptide Y into the paraventricular nucleus
resulted in obe paraventricular nucleus (Stanley and Leibowitz 1984)
effected marked hyperphagia. Chronic administration
of neuropeptide Y into the paraventricular nucleus
resulted in obesity (Stanley et al. 1986). The first
detailed stud effected marked hyperphagia. Chronic administration
of neuropeptide Y into the paraventricular nucleus
resulted in obesity (Stanley et al. 1986). The first
detailed study mapping the active sites for neu-
ropeptide Y showe of neuropeptide Y into the paraventricular nucleus

resulted in obesity (Stanley et al. 1986). The first

detailed study mapping the active sites for neu-

ropeptide Y showed that the paraventricular nucleus,

ventromedial resulted in obesity (Stanley et al. 1986). The first
detailed study mapping the active sites for neu-
ropeptide Y showed that the paraventricular nucleus,
ventromedial hypothalamus and lateral hypothalamic
area were all eq represention and lateral hypothalamic ventromedial hypothalamic area were all equally potent for effecting hyperphagia when a volume of 300 nL (8 or 78 pmol neuropeptide Y) were injected into each site (Stanley et al. 1985 ropeptide Y showed that the paraventricular nucleus,

ventromedial hypothalamus and lateral hypothalamic

area were all equally potent for effecting hyperphagia

when a volume of 300 nL (8 or 78 pmol neuropeptide

Y) were ventromedial hypothalamus and lateral hypothalamic
area were all equally potent for effecting hyperphagia
when a volume of 300 nL (8 or 78 pmol neuropeptide
Y) were injected into each site (Stanley et al. 1985).
When a new area were all equally potent for effecting hyperphagia

when a volume of 300 nL (8 or 78 pmol neuropeptide

Y) were injected into each site (Stanley et al. 1985).

When a new injector technique was developed where

only 10 when a volume of 300 nL [8 or /8 pmol neuropeptide Y] were injected into each site (Stanley et al. 1985).
When a new injector technique was developed where
only 10 nL [78 pmol neuropeptide Y] was deposited,
the most sensit X) were injected into each site (Stanley et al. 1985).

When a new injector technique was developed where

only 10 nL (78 pmol neuropeptide Y) was deposited,

the most sensitive site for stimulating food intake

was the p When a new injector technique was developed where

only 10 nL (78 pmol neuropeptide Y) was deposited,

the most sensitive site for stimulating food intake

was the periformical hypothalamus (Stanley et al.

1993). Peptide only 10 nL (78 pmol neuropeptide Y) was deposited,
the most sensitive site for stimulating food intake
was the periformical hypothalamus (Stanley et al.
1993). Peptide YY (Stanley et al. 1985b) and human
pancreatic polypep the most sensitive site for stimulating food intake
was the periformical hypothalamus (Stanley et al.
1993). Peptide YY (Stanley et al. 1985b) and human
pancreatic polypeptide (Clark et al. 1984) were found
to stimulate fo was the periformical hypothalamus (Stanley et al.

1993). Peptide YY (Stanley et al. 1985b) and human

pancreatic polypeptide (Clark et al. 1984) were found

to stimulate food intake when they were injected

to stimulate f 1993). Peptide YY (Stanley et al. 1985b) and human
pancreatic polypeptide (Clark et al. 1984) were found
to stimulate food intake when they were injected
within the paraventricular nucleus or in-
tracerebroventricularly, pancreatic polypeptide (Clark et al. 1984) were found
to stimulate food intake when they were injected
within the paraventricular nucleus or in-
tracerebroventricularly, respectively. No detailed
studies of the most sensit to stimulate tood intake when they were injected
within the paraventricular nucleus or in-
tracerebroventricularly, respectively. No detailed
studies of the most sensitive brain sites for peptide
YY or human pancreatic pol the only current member of the pancreatic poly tracerebroventricularly, respectively. No detailed
studies of the most sensitive brain sites for peptide
YY or human pancreatic polypeptide have been
mapped because it is believed that neuropeptide Y is
the only current me studies of the most sensitive brain sites for pepti
YY or human pancreatic polypeptide have be
mapped because it is believed that neuropeptide Y
the only current member of the pancreatic po
peptide family that is endogenou For human pancreatic polypeptide have been

upped because it is believed that neuropeptide Y is

et al. 19

et al. 19

pride family that is endogenous to rat and human

ins (Adrian et al. 1983, Allen et al. 1983).

Galanin mapped because it is believed that neuropeptide Y is
the only current member of the pancreatic poly-
peptide family that is endogenous to rat and human
brains (Adrian et al. 1983, Allen et al. 1983).
Galanin, a 29 amino ac

the only current member of the pancreatic poly-
peptide family that is endogenous to rat and human
brains (Adrian et al. 1983, Allen et al. 1983).
Galanin, a 29 amino acid peptide isolated from
porcine intestines (Tatemoto peptide family that is endogenous to rat and human
brains (Adrian et al. 1983, Allen et al. 1983).
Galanin, a 29 amino acid peptide isolated from
porcine intestines (Tatemoto et al. 1983) is another
neuropeptide that stimu brans (Adrian et al. 1983, Allen et al. 1983).

Galanin, a 29 amino acid peptide isolated from

porcine intestines (Tatemoto et al. 1983) is another

meuropeptide that stimulates food intake when

microinjected into the pa Galanin, a 29 amino acid peptide isolated from
porcine intestines (Tatemoto et al. 1983) is anothe
neuropeptide that stimulates food intake whe:
microinjected into the paraventricular nucleus (Kyi
kouli et al. 1986). Furth porcine intestines (Tatemoto et al. 1983) is another
neuropeptide that stimulates food intake when
microinjected into the paraventricular nucleus (Kyr-
kouli et al. 1986). Further studies have shown that in
addition to the potent intestines (Tatemotive at 1.2007) is another

neuroperide that simulates food intake when

microinjected into the paraventricular nucleus (Kyr-

kouli et al. 1986). Further studies have shown that in

addition to th microinjected into the paraventricular nucleus (Kyr-

kouli et al. 1986). Further studies have shown that in

addition to the paraventricular nucleus, the periven-

tricular area, third ventricle and amygdala were other

s kouli et al. 1986). Further studies have shown that in
addition to the paraventricular nucleus, the periven-
tricular area, third ventricle and amygdala were other
sites where galanin significantly increased food
intake. T addition to the paraventricular nucleus, the periventricular area, third ventricle and amygdala were other
sites where galanin significantly increased food
intake. The dorsomedial hypothalamic nucleus was
another effective tricular area, third ventricle and amygdala were other
sites where galanin significantly increased food
intake. The dorsomedial hypothalamic nucleus was
another effective site but required a higher dose (Kyr-
kouli et al. sites where galanin significantly increased food
intake. The dorsomedial hypothalamic nucleus was
another effective site but required a higher dose (Kyr-
kouli et al. 1990). Additional experiments have shown
that galanin p intake. The dorsomedial hypothalamic nucleus was
another effective site but required a higher dose (Kyr-
kouli et al. 1990). Additional experiments have shown
that galanin preferentially increases the ingestion of a
fat di another effective site but required a higher dose (Kyr-

kouli et al. 1990). Additional experiments have shown

that galanin preferentially increases the ingestion of a

fat diet, rather than carbohydrate or protein (Temp kouli et al. 1990). Additional experiments have shown
that galanin preferentially increases the ingestion of a
fat diet, rather than carbohydrate or protein (Tempel
and Leibowitz 1990, Tempel et al. 1988). When M40,
a pote that galanin preterentially increases the ingestion of a
fat diet, rather than carbohydrate or protein (Tempel
and Leibowitz 1990, Tempel et al. 1988). When M40,
a potent antagonist of galanin receptors was microin-
jected that diet, rather than carbohydrate or protein (Tempel

and Leibowitz 1990, Tempel et al. 1988). When M40,

a potent antagonist of galanin receptors was microin-

jected into the paraventricular nucleus, it was ef-

fectiv and Leibowitz 1990, Tempel et al. 1988). When M40,

a potent antagonist of galanin receptors was microin-

jected into the paraventricular nucleus, it was ef-

fective in reducing spontaneous ingestion of a fat diet

(Leib a potent antagonist of galanin receptors was microin-
jected into the paraventricular nucleus, it was ef-
fective in reducing spontaneous ingestion of a fat diet
(Leibowitz and Kim 1992). Mercaptoacetate, a drug
that block jected into the paraventricular nucleus, it was effective in reducing spontaneous ingestion of a fat diet (Eleibowitz and Kim 1992). Mercaptoacetate, a drug methat blocks fatty acid utilization, stimulates food intake. Mer fective in reducing spontaneous ingestion of a fat diet
(Leibowitz and Kim 1992). Mercaptoacetate, a drug
that blocks fatty acid utilization, stimulates food
intake. Mercaptoacetate-induced lipoprivic feeding is
mediated b

TAKE REGULATION 1361S
injection of mercaptoacetate (Calingasan et al. 1992).
A major ascending relay site from the area postrema-TAKE REGULATION 1361

injection of mercaptoacetate (Calingasan et al. 1992

A major ascending relay site from the area postrema-

nucleus tractus solitarius region is to the dors. TAKE REGULATION 1361S

injection of mercaptoacetate (Calingasan et al. 1992).

A major ascending relay site from the area postrema-

nucleus tractus solitarius region is to the dorsal

lateral parabrachial nucleus. Electro 13618

injection of mercaptoacetate (Calingasan et al. 1992).

A major ascending relay site from the area postrema-

nucleus tractus solitarius region is to the dorsal

lateral parabrachial nucleus. Electrolytic or ibotena injection of mercaptoacetate (Calingasan et al. 1992).
A major ascending relay site from the area postrema-
nucleus tractus solitarius region is to the dorsal
lateral parabrachial nucleus. Electrolytic or ibotenate
lesions injection of mercaptoacetate (Calingasan et al. 1992).
A major ascending relay site from the area postrema-
nucleus tractus solitarius region is to the dorsal
lateral parabrachial nucleus. Electrolytic or ibotenate
lesions nucleus tractus solitarius region is to the dorsal cleus tractus solitarius region is to the dorsal
eral parabrachial nucleus. Electrolytic or ibotenate
ions of dorsal lateral parabrachial nucleus or
intral parabrachial nucleus abolished lipoprivic
ding (Koegler et al. 199 lateral parabrachial nucleus. Electrolytic or ibotenate
lesions of dorsal lateral parabrachial nucleus or
central parabrachial nucleus abolished lipoprivic
feeding (Koegler et al. 1992).
Growth hormone releasing factor fro

lesions of dorsal lateral parabrachial nucleus or
central parabrachial nucleus abolished lipoprivic
feeding (Koegler et al. 1992).
Growth hormone releasing factor from human pan-
creatic tumours or rat hypothalamus was sho central parabrachial nucleus abolished lipoprivic
feeding (Koegler et al. 1992).
Growth hormone releasing factor from human pan-
creatic tumours or rat hypothalamus was shown to
stimulate food intake in hungry rats with in teeding (Koegler et al. 1992).

Growth hormone releasing factor from human pan-

creatic tumours or rat hypothalamus was shown to

stimulate food intake in hungry rats with in-

tracerebroventricular administration (Vaccar creatic tumours or rat hypothalamus was shown to
stimulate food intake in hungry rats with in-
tracerebroventricular administration (Vaccarino et al.
1985). Further work has shown that the sensitive site
for growth hormone stimulate food intake in hungry rats with in-
tracerebroventricular administration (Vaccarino et al.
1985). Further work has shown that the sensitive site
for growth hormone releasing factor is within the
medial preoptic a tracerebroventricular administration (Vaccarino et al.
1985). Further work has shown that the sensitive site
for growth hormone releasing factor is within the
medial preoptic area, not the paraventricular nucleus
Dickson a 1985). Further work has shown that the sensitive site
for growth hormone releasing factor is within the
medial preoptic area, not the paraventricular nucleus
(Dickson and Vaccarino 1990, Vaccarino and Hayward
1988). In con for growth hormone releasing factor is within the
medial preoptic area, not the paraventricular nucleus
(Dickson and Vaccarino 1990, Vaccarino and Hayward
1988). In contrast, Tanaka et al. (1991) have shown
that the brain medial preoptic area, not the paraventricular nucleus

(Dickson and Vaccarino 1990, Vaccarino and Hayward

1988). In contrast, Tanaka et al. (1991) have shown

that the brain locus for stimulating feeding is the

ventromed (Dickson and Vaccarino 1990, Vaccarino and Hayward
1988). In contrast, Tanaka et al. (1991) have shown
that the brain locus for stimulating feeding is the
ventromedial hypothalamus while no significant
effect was obtained 1988). In contrast, Tanaka et al. (1991) have shown
that the brain locus for stimulating feeding is the
ventromedial hypothalamus while no significant
effect was obtained by microinjecting growth
hormone releasing factor cleus. ntromedial hypothalamus while no significant

ect was obtained by microinjecting growth

rmone releasing factor into the medial preoptic

ca, lateral hypothalamic area or paraventricular nu-

us.
 Neuropeptides that suppr ethect was obtained by microinjecting growth
hormone releasing factor into the medial preoptic
area, lateral hypothalamic area or paraventricular nu-
cleus.
Neuropeptides that suppress food intake. The best
characterized p

creatic tumours or at hypothalamus was shown to

strainulate food intake in hungry rats with in-

tracerebroventricular administration (Vaccarino et al.

1985). Further work has shown that the sensitive site

for growth h more releasing factor into the medial preoptic
area, lateral hypothalamic area or paraventricular nucleus.
 Neuropeptides that suppress food intake. The best

characterized peptides that inhibit feeding in animals

are area, lateral hypothalamic area or paraventricular nucleus.
 Neuropeptides that suppress food intake. The best

characterized peptides that inhibit feeding in animals

are cholecystokinin (cholecystokinin-8) and

bombesi cleus.

Neuropeptides that suppress food intake. The best

characterized peptides that inhibit feeding in animals

are cholecystokinin (cholecystokinin-8) and

bombesin. Both are highly concentrated in the gas-

trointesti Neuropeptides that suppress food intake. The best
characterized peptides that inhibit feeding in animals
are cholecystokinin (cholecystokinin-8) and
bombesin. Both are highly concentrated in the gas-
trointestinal tract of characterized peptides that inhibit feeding in animals
are cholecystokinin (cholecystokinin-8) and
bombesin. Both are highly concentrated in the gas-
trointestinal tract of many species, and both suppress
feeding when admi are cholecystokinin (cholecystokinin-8) and
bombesin. Both are highly concentrated in the gas-
trointestinal tract of many species, and both suppress
feeding when administered either centrally or
peripherally. Intracerebro bombesin. Both are highly concentrated in the gas-
trointestinal tract of many species, and both suppress
feeding when administered either centrally or
peripherally. Intracerebroventricular administration
of cholecystokin trointestinal tract of many species, and both suppress

feeding when administered either centrally or

peripherally. Intracerebroventricular administration

of cholecystokinin has been shown to be highly ef-

fective for feeding when administered either centrally or

peripherally. Intracerebroventricular administration

of cholecystokinin has been shown to be highly ef-

fective for decreasing food intake (Levine and Morley

1981, Maddiso peripherally. Intracerebroventricular administration
of cholecystokinin has been shown to be highly ef-
fective for decreasing food intake (Levine and Morley
1981, Maddison 1977, Nemeroff et al. 1978, Telegdy
et al. 1984, of cholecystokinin has been shown to be highly effective for decreasing food intake (Levine and Morley 1981, Maddison 1977, Nemeroff et al. 1978, Telegdy et al. 1984, Willis et al. 1984a). Other work has shown that microin fective for decreasing food intake (Levine and Morley
1981, Maddison 1977, Nemeroff et al. 1978, Telegdy
et al. 1984, Willis et al. 1984a). Other work has shown
that microinjections of cholecystokinin into the
paraventricu 1981, Maddison 1977, Nemeroff et al. 1978, Telegdy
et al. 1984, Willis et al. 1984a). Other work has shown
that microinjections of cholecystokinin into the
paraventricular nucleus suppress the noradrenergic
feeding system et al. 1984, Willis et al. 1984a). Other work has shown
that microinjections of cholecystokinin into the
paraventricular nucleus suppress the noradrenergic
feeding system (McCaleb and Myers 1980). Peripheral
or central adm that microinjections of cholecystokinin into the
paraventricular nucleus suppress the noradrenergic
feeding system (McCaleb and Myers 1980). Peripheral
or central administration of the sulfated form of the
synthetic, C-ter paraventricular nucleus suppress the noradrenergic
feeding system (McCaleb and Myers 1980). Peripheral
or central administration of the sulfated form of the
synthetic, C-terminal, cholecystokinin-8 produced
satiety, wherea feeding system (McCaleb and Myers 1980). Peripheral
or central administration of the sulfated form of the
synthetic, C-terminal, cholecystokinin-8 produced
satiety, whereas the desulfated form did not (Gibbs et
al. 1973, or central administration of the sulfated form of the
synthetic, C-terminal, cholecystokinin 8 produced
satiety, whereas the desulfated form did not (Gibbs et
al. 1973, Zhang et al. 1986). The work suggested that
exogenous synthetic, C-terminal, cholecystokinin-8 produced
satiety, whereas the desulfated form did not (Gibbs et
al. 1973, Zhang et al. 1986). The work suggested that
exogenous cholecystokinin acts at the type-A
cholecystokinin re satiety, whereas the desulfated form did not (Gibbs et al. 1973, Zhang et al. 1986). The work suggested that exogenous cholecystokinin acts at the type-A cholecystokinin receptor (predominates in the periphery), rather tha al. 1973, Zhang et al. 1986). The work suggested that exogenous cholecystokinin acts at the type-A cholecystokinin receptor (predominates in the periphery), rather than at the type-B receptor (most prominent in the brain). exogenous cholecystokinin acts at the type-A
cholecystokinin receptor (predominates in the
periphery), rather than at the type-B receptor (most
prominent in the brain). The initial peripheral site of
action appears to be a cholecystokinin receptor (predominates in the
periphery), rather than at the type-B receptor (most
prominent in the brain). The initial peripheral site of
action appears to be afferent (Smith et al. 1985), cap-
saicin-sens periphery), rather than at the type-B receptor (most
prominent in the brain). The initial peripheral site of
action appears to be afferent (Smith et al. 1985), cap-
saicin-sensitive (South and Ritter 1988) neurons of
the a prominent in the brain). The initial peripheral site of
action appears to be afferent (Smith et al. 1985), cap-
saicin-sensitive (South and Ritter 1988) neurons of
the abdominal vagus. Vagal neurons are thought
necessary f action appears to be afferent (Smith et al. 1985), capsaicin-sensitive (South and Ritter 1988) neurons of
the abdominal vagus. Vagal neurons are thought
necessary for satiety caused by action of peripherally
injected chole saicin-sensitive (South and Ritter 1988) neurons of
the abdominal vagus. Vagal neurons are thought
necessary for satiety caused by action of peripherally
injected cholecystokinin. Ablation of the area pos-
trema and caudal the abdominal vagus. Vagal neurons are thought
necessary for satiety caused by action of peripherally
injected cholecystokinin. Ablation of the area pos-
trema and caudal and medial nucleus tractus
solitarius (site of the necessary for satiety caused by action of peripherally
injected cholecystokinin. Ablation of the area pos-
trema and caudal and medial nucleus tractus
solitarius (site of the first, central synaptic relay of
gastrointestin injected cholecystokinin. Ablation of the area pos-
trema and caudal and medial nucleus tractus
solitarius (site of the first, central synaptic relay of
gastrointestinal vagal afferent neurons) abolished the
effect of low solitarius (site of the first, central synaptic relay of
gastrointestinal vagal afferent neurons) abolished the
effect of low but not high doses of cholecystokinin
(Edwards et al. 1986). In contrast to the proposed
mechani gastrointestinal vagal afferent neurons) abolished the
effect of low but not high doses of cholecystokinin
(Edwards et al. 1986). In contrast to the proposed
mechanism of cholecystokinin's action, satiety ef-
fected by bom effect of low but not high doses of cholecystokinin
(Edwards et al. 1986). In contrast to the proposed
mechanism of cholecystokinin's action, satiety ef-
fected by bombesin is not compromised by complete
subdiaphragmatic v (Edwards et al. 1986). In contrast to the proposed mechanism of cholecystokinin's action, satiety effected by bombesin is not compromised by complete subdiaphragmatic vagotomy (Gibbs and Smith 1988). Both high thoracic spi

abolish the action of bombesin (Stuckey et al. 1985,
Ladenheim and Ritter 1988a). Similar to
cholecystokinin, fourth ventricular injection of 1362S

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abolish the action of bombesin (Stuckey et al. 1985,

Ladenheim and Ritter 1988a). Similar to lar

cholecystokinin, fourth ventricular injection of to Ladenheim and Ritter 1988a). Similar to lamic area (Egawa et al. 1990). Further work is needed cholecystokinin, fourth ventricular injection of to determine the effective brain loci that effect a bombesin suppressed ingest KUENZI

abolish the action of bombesin (Stuckey et al. 1985,

Ladenheim and Ritter 1988a). Similar to

cholecystokinin, fourth ventricular injection of

bombesin suppressed ingestive behaviors in rats

(Ladenheim and Ritte abolish the action of bombesin (Stuckey et al. 1985,

Ladenheim and Ritter 1988a). Similar to last

cholecystokinin, fourth ventricular injection of to

bombesin suppressed ingestive behaviors in rats

(Ladenheim and Ritte abolish the action of bombesin (Stuckey et al. 1985,
Ladenheim and Ritter 1988a). Similar to
cholecystokinin, fourth ventricular injection of
bombesin suppressed ingestive behaviors in rats
(Ladenheim and Ritter 1988b, Fly Ladenheim and Ritter 1988a). Similar to
cholecystokinin, fourth ventricular injection of
bombesin suppressed ingestive behaviors in rats
(Ladenheim and Ritter 1988b, Flynn 1989). In ad-
dition, lesions of the area postrema cholecystokinin, fourth ventricular injection of
bombesin suppressed ingestive behaviors in rats
(Ladenheim and Ritter 1988b, Flynn 1989). In ad-
dition, lesions of the area postrema and nucleus
tractus solitarius attenuat bombesin suppressed ingestive behaviors in rats

(Ladenheim and Ritter 1988b, Flynn 1989). In addition, lesions of the area postrema and nucleus

tractus solitarius attenuated the inhibitory effect of

peripherally adminis (Ladenheim and Ritter 1988b, Flynn 1989). In ad
dition, lesions of the area postrema and nucleus
tractus solitarius attenuated the inhibitory effect o
peripherally administered bombesin on food intake
(Ladenheim and Ritter dition, lesions of the area postrema and nucleus
tractus solitarius attenuated the inhibitory effect of
peripherally administered bombesin on food intake
potentional evidence for the importance of the dorsal hind-
brain fo tractus solitarius attenuated the inhibitory effect of
peripherally administered bombesin on food intake
(Ladenheim and Ritter 1989) thereby providing addi-
tional evidence for the importance of the dorsal hind-
brain for peripherally administered bombesin on tood intake

(Ladenheim and Ritter 1989) thereby providing addi-

tional evidence for the importance of the dorsal hind-

brain for housing afferents that effect satiety evoked

by bot (Ladenheim and Ritter 1989) thereby providing additional evidence for the importance of the dorsal hinds brain for housing afferents that effect satiety evoke by both peptides. Microinjection of bombesin into the nucleus t tional evidence for the importance of the dorsal hind-
brain for housing afferents that effect satiety evoked
by both peptides. Microinjection of bombesin into the
nucleus tractus solitarius (deBeaurepaire and
Suaudeau 198 by both peptides. Microinjection of bombesin into the

nucleus tractus solitarius (deBeaurepaire and

Suaudeau 1988, Johnston and Merali 1988), paraven-

tricular nucleus (Willis et al. 1984b) and lateral

hypothalamic are satiety. cleus tractus solitarius (deBeaurepaire and

audeau 1988, Johnston and Merali 1988), paraven-

cular nucleus (Willis et al. 1984b) and lateral

pothalamic area (Stuckey and Gibbs 1982) effected

iety.

Another group of pep Suaudeau 1988, Johnston and Merali 1988), paraventricular nucleus (Willis et al. 1984b) and lateral
hypothalamic area (Stuckey and Gibbs 1982) effected
satiety.
Another group of peptides, included in the
glucagon-secretin

tricular nucleus (Willis et al. 1984b) and lateral
hypothalamic area (Stuckey and Gibbs 1982) effected
satiety.
Another group of peptides, included in the
glucagon-secretin family, largely suppress food intake.
Members of mypothalamic area (Stuckey and Gibbs 1982) effected
satiety.
Another group of peptides, included in the
glucagon-secretin family, largely suppress food intake.
Members of the family are glucagon, secretin,
vasoactive intes satiety.

Another group of peptides, included in the

glucagon-secretin family, largely suppress food intake.

Members of the family are glucagon, secretin,

vasoactive intestinal polypeptide, PHI (peptide with

NH₂-term Another group of peptides, included in the
glucagon-secretin family, largely suppress food intake.
Members of the family are glucagon, secretin,
vasoactive intestinal polypeptide, PHI (peptide with
NH₂-terminal histidine glucagon-secretin family, largely suppress food intake.

Members of the family are glucagon, secretin, et

vasoactive intestinal polypeptide, PHI (peptide with

NH₂-terminal histidine and COOH-terminal

isoleucine amide) Members of the family are glucagon, secretin,

vasoactive intestinal polypeptide, PHI (peptide with

NH₂-terminal histidine and COOH-terminal

isoleucine amide), corticotropin releasing hormone,

savagine, and growth ho vasoactive intestinal polypeptide, PHI (peptide with
NH₂-terminal histidine and COOH-terminal
isoleucine amide), corticotropin releasing hormone,
savagine, and growth hormone-releasing factor. Only
the latter has been sh NH₂-terminal histidine and COOH-terminal
isoleucine amide), corticotropin-releasing-hormone,
savagine, and growth-hormone-releasing-factor. Only
the latter has been shown to increase food intake
following central adminis isoleucine amide), corticotropin releasing hormone,
savagine, and growth hormone-releasing factor. Only
the latter has been shown to increase food intake
following central administration (Table 3). In-
tracerebroventricula savagine, and growth hormone-releasing factor. Only
the latter has been shown to increase food intake
following central administration (Table 3). In-
tracerebroventricular injection of vasoactive intes-
tinal polypeptide (the latter has been shown to increase tood intake
following central administration (Table 3). In-
tracerebroventricular injection of vasoactive intes-
inal polypeptide (Woods et al. 1981), glucagon (In-
okuchi et al. 1984) ration (Table 3). In-

tracerebroventricular injection of vasoactive intes-

tinal polypeptide (Woods et al. 1981), glucagon (In-

okuchi et al. 1984) and savagine (Britton et al. 1984,

Gosnell et al. 1983) significantly tracerebroventricular injection of vasoactive intes-
tinal polypeptide (Woods et al. 1981), glucagon (In-
okuchi et al. 1984) and savagine (Britton et al. 1984,
Gosnell et al. 1983) significantly reduce food intake in
rats tinal polypeptide (Woods et al. 1981), glucagon (In-

okuchi et al. 1984) and savagine (Britton et al. 1984,

Gosnell et al. 1983) significantly reduce food intake in

rats (Table 3). Considerably more feeding studies hav okuchi et al. 1984) and savagine (Britton et al. 1984,

Gosnell et al. 1983) significantly reduce food intake in

rats (Table 3). Considerably more feeding studies have

been conducted with corticotropin releasing

hormone Gosnell et al. 1983) significantly reduce tood intake in
rats (Table 3). Considerably more feeding studies have
been conducted with corticotropin releasing
hormone. Although corticotropin releasing hormone
is best known as rats (Table 3). Considerably more reeding studies have
been conducted with corticotropin releasing
hormone. Although corticotropin releasing hormone
is best known as the primary hypothalamic peptide
for the release of pitu been conducted with corticotropin releasing
hormone. Although corticotropin releasing hormone
is best known as the primary hypothalamic peptide
for the release of pituitary adrenocorticotropic
hormone (Spiess et al. 1981) France Methodia and increased that it plays a role in food
transport and the periodic studies have suggested that it plays a role in food
tons into the ventromedial hypothalamus increased
for the release of pituitary adren is best known as the primary hypothalamic peptide
for the release of pituitary adrenocorticotropic
hormone (Spiess et al. 1981) and thereby is necessary
for mediating stress responses in animals, other
studies have suggest tor the release of pituitary adrenocorticotropic
hormone (Spiess et al. 1981) and thereby is necessary
for mediating stress responses in animals, other
studies have suggested that it plays a role in food
consumption (Morle hormone (Spiess et al. 1981) and thereby is necessary
for mediating stress responses in animals, other
studies have suggested that it plays a role in food
consumption (Morley et al. 1983). With in-
tracerebroventricular ad for mediating stress responses in animals, other
studies have suggested that it plays a role in food
consumption (Morley et al. 1983). With in-
tracerebroventricular administration, corticotropin
releasing hormone reduces consumption (Morley et al. 1983). With in-
tracerebroventricular administration, corticotropin
releasing hormone reduces food intake (Arase et al.
1988, Britton et al. 1984, Morley and Levine 1982)
and retains its hypophag consumption (Morley et al. 1983). With in-
tracerebroventricular administration, corticotropin
releasing hormone reduces food intake (Arase et al.
1988, Britton et al. 1984, Morley and Levine 1982)
and retains its hypopha tracerebroventricular administration, corticotropin
releasing hormone reduces food intake (Arase et al.
1988, Britton et al. 1984, Morley and Levine 1982)
and retains its hypophagic effects in hypophysec-
tomized animals (releasing hormone reduces tood intake (Arase et al.

1988, Britton et al. 1984, Morley and Levine 1982)

and retains its hypophagic effects in hypophysec-

tomized animals (Morley and Levine 1982). It is also

effective in 1988, Britton et al. 1984, Morley and Levine 1982)
and retains its hypophagic effects in hypophysec-
tomized animals (Morley and Levine 1982). It is also
effective in reducing food intake in obese rats (Arase
et al. 1989a, and retains its hypophagic effects in hypophysectomized animals (Morley and Levine 1982). It is also
effective in reducing food intake in obese rats (Arase
et al. 1989a, 1989b). Microinjection studies have
shown that corti tomized animals (Morley and Levine 1982). It is also
effective in reducing food intake in obese rats (Arase
et al. 1989a, 1989b). Microinjection studies have
shown that corticotropin releasing hormone decreases
feeding whe et al. 1989a, 1989b). Microinjection studies have
et al. 1989a, 1989b). Microinjection studies have
shown that corticotropin releasing hormone decreases
feeding when administered to the paraventricular nu-
cleus, but not t et al. 1989a, 1989b). Microinjection studies have
shown that corticotropin releasing hormone decreases
feeding when administered to the paraventricular nu-
cleus, but not the ventromedial hypothalamus,
striatum, lateral hy shown that corticotropin releasing hormone decreases
feeding when administered to the paraventricular nu-
cleus, but not the ventromedial hypothalamus,
striatum, lateral hypothalamic area or globus pallidus
(Krahn et al. 1 teeding when administered to the paraventricular nu-
cleus, but not the ventromedial hypothalamus,
striatum, lateral hypothalamic area or globus pallidus
(Krahn et al. 1984). When mapping studies were com-
pleted regarding cleus, but not the ventromedial hypothalamus,
striatum, lateral hypothalamic area or globus pallidus
(Krahn et al. 1984). When mapping studies were com-
pleted regarding the effect of corticotropin releasing
hormone on sti striatum, lateral hypothalamic area or globus pallidus

(Krahn et al. 1984). When mapping studies were com-

pleted regarding the effect of corticotropin releasing

hormone on stimulating sympathetic firing rate, the

medi (Krahn et al. 1984). When mapping studies were com-
pleted regarding the effect of corticotropin releasing
hormone on stimulating sympathetic firing rate, the
medial preoptic area was most effective. In contrast,
problem c pleted regarding the effect of corticotropin releasing
hormone on stimulating sympathetic firing rate, the
medial preoptic area was most effective. In contrast,
no change in sympathetic firing rate was observed
when cortic hormone on stimulating sympathetic firing rate, the
medial preoptic area was most effective. In contrast,
no change in sympathetic firing rate was observed
when corticotropin releasing hormone was injected
into the anterio

EL
somedial hypothalamic nucleus, or lateral hypotha-
lamic area (Egawa et al. 1990). Further work is needed EL
somedial hypothalamic nucleus, or lateral hypotha-
lamic area (Egawa et al. 1990). Further work is needed
to determine the effective brain loci that effect a EL
somedial hypothalamic nucleus, or lateral hypotha-
lamic area (Egawa et al. 1990). Further work is needed
to determine the effective brain loci that effect a
decrease in food intake from microadministration of EL
somedial hypothalamic nucleus, or lateral hypotha-
lamic area (Egawa et al. 1990). Further work is needed
to determine the effective brain loci that effect a
decrease in food intake from microadministration of
corticotr lamic area (Egawa et al. 1990). Further work is needed
to determine the effective brain loci that effect a
decrease in food intake from microadministration of
corticotropin releasing hormone.
The last peptide group covered

corticotropin releasing hormone.
The last peptide group covered in this review is
thyrotropin releasing hormone and its related com-The last peptide group covered in this review is
thyrotropin releasing hormone and its related com-
pound cyclo-histidyl-proline-diketopiperazine [cycloto determine the effective brain loci that effect a
decrease in food intake from microadministration of
corticotropin releasing hormone.
The last peptide group covered in this review is
thyrotropin releasing hormone and it decrease in food intake from microadministration of
corticotropin releasing hormone.
The last peptide group covered in this review is
thyrotropin releasing hormone and its related com-
pound cyclo-histidyl-proline-diketopi corticotropin releasing hormone.
The last peptide group covered in this review is
thyrotropin releasing hormone and its related com-
pound cyclo-histidyl-proline-diketopiperazine [cyclo-
(His-Pro)]. Vijayan and McCann (197 The last peptide group covered in this review is
thyrotropin releasing hormone and its related com-
pound cyclo-histidyl-proline-diketopiperazine [cyclo-
[His-Pro]]. Vijayan and McCann (1977) first showed
that intracerebro thyrotropin releasing hormone and its related com-
pound cyclo-histidyl-proline-diketopiperazine [cyclo-
(His-Pro)]. Vijayan and McCann (1977) first showed
that intracerebroventricular injection of thyrotropin
releasing ho pound cyclo-histidyl-proline-diketopiperazine [cyclo-
(His-Pro]]. Vijayan and McCann (1977) first showed
that intracerebroventricular injection of thyrotropin
releasing hormone decreased food intake. Other
studies confirm (His-Pro]]. Vijayan and McCann (1977) first showed
that intracerebroventricular injection of thyrotropin
releasing hormone decreased food intake. Other
studies confirmed that work but significantly higher
levels of thyrot that intracerebroventricular injection of thyrotropin
releasing hormone decreased food intake. Other
studies confirmed that work but significantly higher
levels of thyrotropin releasing hormone were required
(Lin et al. 1 releasing hormone decreased tood intake. Other
studies confirmed that work but significantly higher
levels of thyrotropin releasing hormone were required
[Lin et al. 1983], thyrotropin releasing hormone also
reduced food i studies confirmed that work but significantly higher
levels of thyrotropin releasing hormone were required
(Lin et al. 1983), thyrotropin releasing hormone also
reduced food intake in hypophysectomized animals
(Morley and revels of thyrotropin releasing hormone were required
(Lin et al. 1983); thyrotropin releasing hormone also
reduced food intake in hypophysectomized animals
(Morley and Levine 1980). Specific neural sites where
thyrotropin (Lin et al. 1983), thyrotropin releasing hormone also
reduced food intake in hypophysectomized animals
(Morley and Levine 1980). Specific neural sites where
thyrotropin releasing hormone administration
reduced food intake reduced tood intake in hypophysectomized animals and (Morley and Levine 1980). Specific neural sites where the thyrotropin releasing hormone administration reduced food intake were the ventromedial hypothalamus and lateral (Morley and Levine 1980). Specific neural sites where
thyrotropin releasing hormone administration
reduced food intake were the ventromedial
hypothalamus and lateral hypothalamic area (Suzuki
et al. 1982). The first publi thyrotropin releasing hormone administration
reduced food intake were the ventromedial
hypothalamus and lateral hypothalamic area (Suzuki
et al. 1982). The first published work of the anorectic
effect of cyclo-histidyl-pr reduced tood intake were the ventromedial s
hypothalamus and lateral hypothalamic area (Suzuki
et al. 1982). The first published work of the anorectic
effect of cyclo-histidyl-proline-diketopiperazine was
that of Morley e hypothalamus and lateral hypothalamic area (Suzuki

et al. 1982). The first published work of the anorectic

effect of cyclo-histidyl-proline-diketopiperazine was

that of Morley et al. (1981). Of interest is that this

c et al. 1982). The first published work of the anorectic
effect of cyclo-histidyl-proline-diketopiperazine was
that of Morley et al. (1981). Of interest is that this
compound is elevated in the hypothalamus of unfed
but no ect of cyclo-histidyl-proline-diketopiperazine was
at of Morley et al. (1981). Of interest is that this $\frac{8}{96}$
mpound is elevated in the hypothalamus of unfed
 \pm t not fully-fed rats (Mori et al. 1983) and may play that of Morley et al. (1981). Of interest is that this $\frac{3}{62}$
compound is elevated in the hypothalamus of unfed
but not fully-fed rats (Mori et al. 1983) and may play a
mino acids that affect food intake. Two amino
ac

compound is elevated in the hypothalamus of unfed
but not fully-fed rats (Mori et al. 1983) and may play a
role in macronutrient selection (Prasad et al. 1992).
Amino acids that affect food intake. Two amino
acids have but not fully-fed rats (Mori et al. 1983) and may play a
role in macronutrient selection (Prasad et al. 1992). $\frac{Z}{Z}$
Amino acids that affect food intake. Two amino
acids have been shown to have significant effects o role in macronutrient selection (Prasad et al. 1992). $\frac{z}{z}$
 Amino acids that affect food intake. Two amino

acids have been shown to have significant effects on

food intake, γ -aminobutyric acid (GABA) and

glut **Amino acids that affect food intake.** Two amino
acids have been shown to have significant effects on
food intake, γ -aminobutyric acid (GABA) and $\frac{2}{5}$
glutamate. Bicuculline methiodide, a putative GABA
antagonist, acids have been shown to have significant effects on
food intake, γ -aminobutyric acid (GABA) and
glutamate. Bicuculline methiodide, a putative GABA
antagonist, increased ingestion of sweet milk, when
administered into food intake, γ -aminobutyric acid (GABA) and \leq
glutamate. Bicuculline methiodide, a putative GABA intagonist, increased ingestion of sweet milk, when
administered into the lateral hypothalamic area and
suppressed fe glutamate. Bicuculline methiodide, a putative GABA
antagonist, increased ingestion of sweet milk, when
administered into the lateral hypothalamic area and
suppressed feeding when injected into the ven-
tromedial hypothalam antagonist, increased ingestion of sweet milk, when
administered into the lateral hypothalamic area and
suppressed feeding when injected into the ven-
tromedial hypothalamus. Conversely, GABA injec-
tions into the ventrome administered into the lateral hypothalamic area and
suppressed feeding when injected into the ven-
tromedial hypothalamus. Conversely, GABA injec-
tions into the ventromedial hypothalamus increased
intake (Kelly et al. 19 suppressed feeding when injected into the ven-
tromedial hypothalamus. Conversely, GABA injec-
tions into the ventromedial hypothalamus increased
intake (Kelly et al. 1977). The GABA agonist mus-
cimol effected an increase tromedial hypothalamus. Conversely, GABA injections into the ventromedial hypothalamus increased
intake (Kelly et al. 1977). The GABA agonist mus-
cimol effected an increase in food intake when in-
jected into the paraven tions into the ventromedial hypothalamus increased
intake (Kelly et al. 1977). The GABA agonist mus-
cimol effected an increase in food intake when in-
jected into the paraventricular nucleus while bi-
cuculline methiodid intake (Kelly et al. 1977). The GABA agonist must
cimol effected an increase in food intake when ir
jected into the paraventricular nucleus while b
cuculline methiodide suppressed feeding (Kelly an
Grossman 1979). Intracer cimol effected an increase in food intake when in-
jected into the paraventricular nucleus while bi-
cuculline methiodide suppressed feeding (Kelly and $\frac{2}{2}$
Grossman 1979). Intracerebroventricular injections of
GABA jected into the paraventricular nucleus while bi-
cuculline methiodide suppressed feeding (Kelly and
Grossman 1979). Intracerebroventricular injections of
GABA or ethanolamine-O-sulphate (GABA-trans-
aminase inhibitor) gav cuculline methiodide suppressed feeding (Kelly and $\frac{Z}{Z}$
Grossman 1979). Intracerebroventricular injections of $\frac{Z}{Z}$
GABA or ethanolamine-O-sulphate (GABA-trans-
aminase inhibitor) gave different results from in-
 Grossman 1979). Intracerebroventricular injections of

GABA or ethanolamine-O-sulphate (GABA-trans-

aminase inhibitor) gave different results from in-

tracerebroventricular administration of muscimol

(GABA receptor-ago GABA or ethanolamine-O-sulphate (GABA-trans-

aminase inhibitor) gave different results from in-

tracerebroventricular administration of muscimol
 $\frac{1}{10}$

(GABA receptor-agonist). The former decreased while

the latt aminase inhibitor) gave different results from in-
tracerebroventricular administration of muscimol
(GABA receptor-agonist). The former decreased while
the latter increased food intake (Olgiati et al. 1980).
Additional stu tracerebroventricular administration of muscimol

(GABA receptor-agonist). The former decreased while

the latter increased food intake (Olgiati et al. 1980). $\frac{\infty}{\infty}$

Additional studies have shown that muscimol in-
 (GABA receptor-agonist). The former decreased while
the latter increased food intake (Olgiati et al. 1980). $\frac{8}{9}$
Additional studies have shown that muscimol in-
jected into the midbrain raphe nucleus or ventral
tegme the latter increased food intake (Olgiati et al. 1980). $\frac{1}{60}$
Additional studies have shown that muscimol in-
jected into the midbrain raphe nucleus or ventral
tegmental area increases food intake (Klitenick and
Wirt Additional studies have shown that muscimol in-
jected into the midbrain raphe nucleus or ventral
tegmental area increases food intake (Klitenick and
Wirtshafter 1988). Intra-raphe injections of two ex-
citatory amino acid jected into the midbrain raphe nucleus or ventral
tegmental area increases food intake (Klitenick and
Wirtshafter 1988). Intra-raphe injections of two ex-
citatory amino acid antagonists, kynurenic acid and
2-amino-5-phosp tegmental area increases food in
Wirtshafter 1988). Intra-raphe in
citatory amino acid antagonists,
2-amino-5-phosphonovaleric acid
pendent increases in food and
shafter and Trifunovic 1988).
Recent work has shown that a rtshafter 1988). Intra-raphe injections of two ex-
atory amino acid antagonists, kynurenic acid and
imino-5-phosphonovaleric acid resulted in dose-de-
ndent increases in food and water intake (Wirt-
after and Trifunovic 19 citatory amino acid antagonists, kynurenic acid and
2-amino-5-phosphonovaleric acid resulted in dose-de-
pendent increases in food and water intake (Wirt-
shafter and Trifunovic 1988).
Recent work has shown that an excitat

2-amino-5-phosphonovaleric acid resulted in dose-de-
pendent increases in food and water intake (Wirt-
shafter and Trifunovic 1988).
Recent work has shown that an excitatory amino
acid, glutamate can stimulate food intake pendent increases in food and water intake (Wirt-
shafter and Trifunovic 1988).
Recent work has shown that an excitatory amino
acid, glutamate can stimulate food intake in rats. The
primary site mediating glutamate-induced after and Trifunovic 1988).

Recent work has shown that an excitatory amino

id, glutamate can stimulate food intake in rats. The

mary site mediating glutamate-induced eating is

a lateral hypothalamic area (Stanley et al Recent work has shown that an excitatory amino
acid, glutamate can stimulate food intake in rats. The
primary site mediating glutamate-induced eating is
the lateral hypothalamic area (Stanley et al. 1993).
Hormones given acid, glutamate can stimulate food intake in rats. The
primary site mediating glutamate-induced eating is
the lateral hypothalamic area (Stanley et al. 1993).
Hormones given centrally (insulin). Hatfield et al.
(1974) we

IN THE JOURNAL OF NUTRITION

CONFERENCE: FOOD INT
feeding in normal and diabetic rats. Large doses of
intracerebroventricular administered insulin also CONFERENCE: FOOD INTA
feeding in normal and diabetic rats. Large doses of
intracerebroventricular administered insulin also
reduced food intake (Brief and Davis 1984, Plata-CONFERENCE: FOC
feeding in normal and diabetic rats. Large doses of
intracerebroventricular administered insulin also
reduced food intake (Brief and Davis 1984, Plata-
Salaman and Oomura 1986, Woods et al. 1979) CONFERENCE: FOOD IN

feeding in normal and diabetic rats. Large doses of

intracerebroventricular administered insulin also

reduced food intake (Brief and Davis 1984, Plata-

Salaman and Oomura 1986, Woods et al. 1979).
 feeding in normal and diabetic rats. Large doses of
intracerebroventricular administered insulin also
reduced food intake (Brief and Davis 1984, Plata-
Salaman and Oomura 1986, Woods et al. 1979).
Bilateral injection of in teeding in normal and diabetic rats. Large doses of
intracerebroventricular administered insulin also
reduced food intake (Brief and Davis 1984, Plata-
Salaman and Oomura 1986, Woods et al. 1979).
Bilateral injection of in intracerebroventricular administered insulin also
reduced food intake (Brief and Davis 1984, Plata-
Salaman and Oomura 1986, Woods et al. 1979).
Bilateral injection of insulin antibodies into the ven-
tromedial hypothalamu reduced food intake (Brief and Davis 1984, Plata-Salaman and Oomura 1986, Woods et al. 1979).
Bilateral injection of insulin antibodies into the ven-
tromedial hypothalamus increased food intake
(Strubbe and Mein 1977). Mc Salaman and Oomura 1986, Woods et al. 1979).
Bilateral injection of insulin antibodies into the ven-
tromedial hypothalamus increased food intake
(Strubbe and Mein 1977). McGowan et al. (1992)
reported that the most sensit Bilateral injection of insulin antibodies into the ven-
tromedial hypothalamus increased food intake
(Strubbe and Mein 1977). McGowan et al. (1992)
reported that the most sensitive sites within the ven-
tromedial hypothala tromedial hypothalamus increased food intak
(Strubbe and Mein 1977). McGowan et al. (1992)
reported that the most sensitive sites within the ventromedial hypothalamic area that decreased foo
intake from small insulin injec (Strubbe and Mein 1977). McGowan et al. (1992)
reported that the most sensitive sites within the ven-
tromedial hypothalamic area that decreased food
intake from small insulin injections included portions
of the ventromedi tromedial hypothalamic area that decreased food
intake from small insulin injections included portions
of the ventromedial hypothalamic nucleus, paraven-
tricular nucleus, dorsomedial hypothalamic nucleus
and arcuate nucle tromedial hypothalamic area that decreased food
intake from small insulin injections included portions
of the ventromedial hypothalamic nucleus, paraven-
tricular nucleus, dorsomedial hypothalamic nucleus
and arcuate nucle Intake from small insulin injections included portions
of the ventromedial hypothalamic nucleus, paraven-
tricular nucleus, dorsomedial hypothalamic nucleus
and arcuate nucleus. They also confirmed that in-
jection of anti and arcuate nucleus. They also confirmed that injection of antibodies to insulin within the ventromedial hypothalamus increased food intake and body weight. The method is to insulin within the ven-

edial hypothalamus increased food intake and

weight.

NEURAL REGULATION OF FOOD

INTAKE IN BIRDS **INTAKE IN BIRDS**

NEURAL REGULATION OF FOOD

INTAKE IN BIRDS

To date, the hypotheses that have been proposed to

plain the neural regulation of food intake in ver-

div **EXPLAIN REGULATION OF FOOD INTAKE IN BIRDS**
To date, the hypotheses that have been proposed to explain the neural regulation of food intake in vertebrates have been derived from those developed for INTAKE IN BIRDS
To date, the hypotheses that have been proposed to
explain the neural regulation of food intake in ver-
tebrates have been derived from those developed for
mammals. Because the majority of neural sites in-To date, the hypotheses that have been proposed to
explain the neural regulation of food intake in ver-
tebrates have been derived from those developed for
mammals. Because the majority of neural sites in-
volved in the co To date, the hypotheses that have been proposed to
explain the neural regulation of food intake in ver-
tebrates have been derived from those developed for
mammals. Because the majority of neural sites in-
volved in the co To date, the hypotheses that have been proposed to
explain the neural regulation of food intake in ver-
tebrates have been derived from those developed for
mammals. Because the majority of neural sites in-
volved in the co explain the neural regulation of food intake in ver-
tebrates have been derived from those developed for
mammals. Because the majority of neural sites in-
volved in the control of food intake in mammals are
located in lowe tebrates have been derived from those developed for
mammals. Because the majority of neural sites in-
volved in the control of food intake in mammals are
located in lower brain levels (medulla oblongata, pons
and diencepha mammals. Because the majority of neural sites in-
volved in the control of food intake in mammals are
located in lower brain levels (medulla oblongata, pons
and diencephalon) and the process of feeding is such a
basic requ volved in the control of food intake in mammals are
located in lower brain levels (medulla oblongata, pons
and diencephalon) and the process of feeding is such a
basic requirement for survival of all animals, it makes
sens Front and diencephalo and the process of feeding is such a

and diencephalon and the process of feeding is such a

basic requirement for survival of all animals, it makes

basic requirement for survival of all animals, it and diencephalon) and the process of teeding is such a
basic requirement for survival of all animals, it makes
sense that the regulation of appetite in birds and
mammals should share fundamental mechanisms.
The main idea o basic requirement for survival of all animals, it makes
sense that the regulation of appetite in birds and
mammals should share fundamental mechanisms.
The main idea of this section will be that the auto-
nomic and endocri Sense that the regulation of appetite in birds and

mammals should share fundamental mechanisms.

The main idea of this section will be that the auto-

nomic and endocrine hypothesis proposed for

regulating feeding in mam mammals should share fundamental mechanisms.
The main idea of this section will be that the auto-
nomic and endocrine hypothesis proposed for
regulating feeding in mammals (Bray and York 1979,
Inoue and Bray 1979) is quite The main idea of this section will be that the auto-
nomic and endocrine hypothesis proposed for
regulating feeding in mammals (Bray and York 1979,
Inoue and Bray 1979) is quite relevant to explain the
neural control of f well. The this end of the this end of the this end of the this end of the this end neuroanatomical structures identified
that alter food intake, compounds administered cen-
that alter food intake, compounds administered centhat alter food intake and body weight changes

not only in domestic avian species but wild ones as

well.

To this end neuroanatomical structures identified

that alter food intake, compounds administered cen-

trally and

neural control of food intake and body weight changes

not only in domestic avian species but wild ones as

well.

To this end neuroanatomical structures identified

that alter food intake, compounds administered cen-

tra not only in domestic avian species but wild ones as
well.
To this end neuroanatomical structures identified
that alter food intake, compounds administered cen-
trally and neural pathways associated with the auto-
nomic ner viewed. *A* Nova *Intake*, compounds administed conditional difference dend neural pathways associated with the auto
nervous system in avian species will be represent that a system in avian species will be represented.

**STRUCTURES IMPORTANT FOR

STRUCTURES IMPORTANT FOR

STRUCTURES IMPORTANT FOR

STRUCTURES IMPORTANT FOR VIAN NEUROANATOMICAL
RUCTURES IMPORTANT FOR
ALTERING FOOD INTAKE** AVIAN NEUROANATOMICAL
STRUCTURES IMPORTANT FOR
ALTERING FOOD INTAKE
Electrolytic lesions of the ventromedial
hypothalamus of Leghorn chickens (Lepkovsky and

STRUCTURES IMPORTANT FOR

ALTERING FOOD INTAKE

Electrolytic lesions of the ventromedial

hypothalamus of Leghorn chickens (Lepkovsky and analytical Passive and Tassuda 1966, Snapir et al. 1973, Sonoda 1983) and of 10 SINUCTURES IMPORTAITE POR

ALTERING FOOD INTAKE

Electrolytic lesions of the ventromedial

hypothalamus of Leghorn chickens (Lepkovsky and

Yasuda 1966, Snapir et al. 1973, Sonoda 1983) and of

the ventromedial hypothalami ALTERING FOOD INTAKE

Electrolytic lesions of the ventromedial

hypothalamus of Leghorn chickens (Lepkovsky and

Yasuda 1966, Snapir et al. 1973, Sonoda 1983) and of

the ventromedial hypothalamic nucleus including the

in Electrolytic lesions of the ventromedial
hypothalamus of Leghorn chickens (Lepkovsky and
Yasuda 1966, Snapir et al. 1973, Sonoda 1983) and of
the ventromedial hypothalamic nucleus including the
inferior hypothalamic nucleu Electrolytic lesions of the ventromedia
hypothalamus of Leghorn chickens (Lepkovsky and
Yasuda 1966, Snapir et al. 1973, Sonoda 1983) and o
the ventromedial hypothalamic nucleus including the
inferior hypothalamic nucleus hypothalamus of Leghorn chickens (Lepkovsky and
Yasuda 1966, Snapir et al. 1973, Sonoda 1983) and of
the ventromedial hypothalamic nucleus including the
inferior hypothalamic nucleus of White-throated
Sparrows (Kuenzel and Yasuda 1966, Snapir et al. 1973, Sonoda 1983) and of
the ventromedial hypothalamic nucleus including the
inferior hypothalamic nucleus of White-throated
Sparrows (Kuenzel and Helms 1967) effected hyper-
phagia and obesity.

VMN

FIGURE 5 Cross-section of a chick brain taken from

atlas plate A 7.4 mm of Kuenzel and Masson (1988) showing

the location and approximate size of the paraventricular

clusical PVN. The PVNa shows its size when stain FIGURE 5 Cross-section of a chick brain taken from
atlas plate A 7.4 mm of Kuenzel and Masson (1988) showing
the location and approximate size of the paraventricular
nucleus (PVN). The PVNa shows its size when stained with **FIGURE 5** Cross-section of a chick brain taken from
atlas plate A 7.4 mm of Kuenzel and Masson (1988) showing
the location and approximate size of the paraventricular
nucleus (PVN). The PVNa shows its size when stained w **FIGURE 5** Cross-section of a chick brain taken from
atlas plate A 7.4 mm of Kuenzel and Masson (1988) showing
the location and approximate size of the paraventricular
nucleus (PVN). The PVNa shows its size when stained w the location and approximate size of the paraventricular
nucleus (PVN). The PVNa shows its size when stained with
classical nissl stains. The PVNb is much larger and sweeps
out toward the lateral portion of the thalamus wh mucleus (PVN). The PVNa shows its size when stained with
classical nissl stains. The PVNa shows its size when stained with
classical nissl stains. The PVNb is much larger and sweeps
out toward the lateral portion of the th classical rissl stains. The PVNb is much larger and sweeps
out toward the lateral portion of the thalamus when sec-
tions are immunostained with an antibody to arginine
vasotocin. Abbreviations used: AId, v, the dorsal an out toward the lateral portion of the thalamus when sec-
tions are immunostained with an antibody to arginine
vasotocin. Abbreviations used: AId, v, the dorsal and ventral
divisions of the archistriatum; AL, ansa lenticul vasotocin. Abbreviations used: AId, v, the dorsal
divisions of the archistriatum; AL, ansa lentia
lateral forebrain bundle; LHy, lateral hypotha
OM, occipitomesencephalic tract; QF, quintof
RSd, dorsal, superior reticular

FILM IS CONSET ON THE TRANSFALL THE SERVICE AND AN INTERNATION THE AND AN ANNEX INTERNATION THE PAVA BOND AND SURFALL CHIRAL DISSISTIONS are imm OM, occipitomesencephalic tract; QF, quintorrontal tract;
RSd, dorsal, superior reticular nucleus; Tn, nucleus taeniae;
VMN, ventromedial hypothalamic nucleus.
migratory birds were disrupted by the ventromedial
hypothalami KSU, uorsai, superior fericular hucleus; 1 n, nucleus taemae;

VMN, ventromedial hypothalamic nucleus.

migratory birds were disrupted by the ventromedial

hypothalamic lesions including water intake, gonadal

development, development, Zugunruhe [migratory birds were disrupted by the ventromedial
hypothalamic lesions including water intake, gonadal
development, Zugunruhe [migratory behavior (noc-
turnal activity) shown by birds in cages], a migratory birds were disrupted by the ventromedial
hypothalamic lesions including water intake, gonadal
development, Zugunruhe [migratory behavior (noc-
turnal activity) shown by birds in cages], and molt
(Kuenzel 1974, K migratory birds were disrupted by the ventromedial
hypothalamic lesions including water intake, gonadal
development, Zugunruhe [migratory behavior (noc-
turnal activity) shown by birds in cages], and molt
(Kuenzel 1974, K migratory birds were disrupted by the ventromedial $\frac{c}{\leq}$
hypothalamic lesions including water intake, gonadal $\frac{c}{\leq}$
development, Zugunruhe [migratory behavior (noc-
turnal activity) shown by birds in cages], a hypothalamic lesions including water intake, gonadal \leq
development, Zugunruhe [migratory behavior (noc-
turnal activity) shown by birds in cages], and molt
[Kuenzel 1974, Kuenzel and Helms 1970]. The most
common occur development, Zugunruhe (migratory behavior (noc-
turnal activity) shown by birds in cages), and molt
(Kuenzel 1974, Kuenzel and Helms 1970). The most
common occurrence from ventromedial hypothalamic
lesions was hyperphagia turnal activity) shown by birds in cages], and molt
(Kuenzel 1974, Kuenzel and Helms 1970). The most
common occurrence from ventromedial hypothalamic $\frac{C}{C}$
lesions was hyperphagia, obesity, polydipsia, no
gonadal deve (Kuenzel 1974, Kuenzel and Helms 1970). The most $\frac{2}{5}$ common occurrence from ventromedial hypothalamic lesions was hyperphagia, obesity, polydipsia, no gonadal development, no Zugunruhe and no molt, $\frac{2}{7}$ however common occurrence from ventromedial hypothalamic
lesions was hyperphagia, obesity, polydipsia, no
gonadal development, no Zugunruhe and no molt,
however a number of combinations of different effects
were obtained. The ove lesions was hyperphagia, obesity, polydipsia, no $\frac{m}{20}$
gonadal development, no Zugunruhe and no molt, $\frac{m}{20}$
however a number of combinations of different effects
were obtained. The overall conclusion of the stud gonadal development, no Zugunruhe and no molt,
however a number of combinations of different effects
were obtained. The overall conclusion of the study
was that in birds the ventromedial hypothalamic syn-
drome involves th however a number of combinations of different effects
were obtained. The overall conclusion of the study
was that in birds the ventromedial hypothalamic syn-
drome involves the disruption of a number of physio-
logical an were obtained. The overall conclusion of the study
was that in birds the ventromedial hypothalamic syn-
drome involves the disruption of a number of physio-
logical and behavioral events, the neural control
mechanisms for was that in birds the ventromedial hypothalamic syn-
drome involves the disruption of a number of physio-
logical and behavioral events, the neural control $\frac{8}{6}$
mechanisms for each being anatomically separable $\frac{8}{$ drome involves the disruption of a number of physio-
logical and behavioral events, the neural control
mechanisms for each being anatomically separable
 $\frac{18}{10}$
(Kuenzel 1974). It is of interest that Weingarten et al.
 logical and behaviour
mechanisms for ea
(Kuenzel 1974). It is
(1985) suggested tha
sions in rats induce
as well (Fig. 3).
No published s Echanisms for each being anatomically separable $\overline{\circ}$
uenzel 1974). It is of interest that Weingarten et al.
985) suggested that ventromedial hypothalamic le-
ns in rats induce a set of independent disturbances
well (F (Kuenzel 1974). It is of interest that Weingarten et al.
(1985) suggested that ventromedial hypothalamic le-
sions in rats induce a set of independent disturbances
as well (Fig. 3).
No published studies were found where th

(1985) suggested that ventromedial hypothalamic lesions in rats induce a set of independent disturbances
as well (Fig. 3).
No published studies were found where the
paraventricular nucleus or parabrachial nucleus was
lesio sions in rats induce a set of independent disturbances
as well (Fig. 3).
No published studies were found where the
paraventricular nucleus or parabrachial nucleus was
lesioned in any avian species. The paraventricular
nucl as well (Fig. 3).

No published studies were found where the

paraventricular nucleus or parabrachial nucleus was

lesioned in any avian species. The paraventricular

nucleus is a large, hypothalamic nucleus in chicks

and No published studies were found where the
paraventricular nucleus or parabrachial nucleus was
lesioned in any avian species. The paraventricular
nucleus is a large, hypothalamic nucleus in chicks
and, similar to that in ra paraventricular nucleus or parabrachial nucleus was
lesioned in any avian species. The paraventricular
nucleus is a large, hypothalamic nucleus in chicks
and, similar to that in rats, can be divided into at least
10 sub-nu lesioned in any avian species. The paraventricular
nucleus is a large, hypothalamic nucleus in chicks
and, similar to that in rats, can be divided into at least
10 sub-nuclei (unpublished data). When brain slices
are immun nucleus is a large, hypothalamic nucleus in chicks
and, similar to that in rats, can be divided into at least
10 sub-nuclei (unpublished data). When brain slices
are immunostained with an antibody to arginine
vasotocin (eq and, similar to that in rats, can be divided into at least
10 sub-nuclei (unpublished data). When brain slices
are immunostained with an antibody to arginine
vasotocin (equivalent to vasopressin in mammals),
the paraventri 10 sub-nuclei (unpublished data). When brain slices
are immunostained with an antibody to arginine
vasotocin (equivalent to vasopressin in mammals),
the paraventricular nucleus extends from the tradi-
tional midline nucleu

1364S

important connections to the nucleus tractus

solitarius and dorsal motor nucleus of the vagus 1364S

important connections to the nucleus tractus

solitarius and dorsal motor nucleus of the vagus

(Arends et al. 1988, Berk and Finkelstein 1983), (Arends et al. 1988)

(Arends et al. 1988, Berk and Finkelstein 1983),

(Arends et al. 1988, Berk and Finkelstein 1983),

(Arends et al. 1988, Berk and Finkelstein 1983),

parabrachial nucleus (Wild et al. 1990) and median KUENZEI

important connections to the nucleus tractus

solitarius and dorsal motor nucleus of the vagus

(Arends et al. 1988, Berk and Finkelstein 1983),

parabrachial nucleus (Wild et al. 1990) and median

eminence (Kiss important connections to the nucleus tractus
solitarius and dorsal motor nucleus of the vagus
(Arends et al. 1988, Berk and Finkelstein 1983),
parabrachial nucleus (Wild et al. 1990) and median
eminence (Kiss 1988, Korf 19 mportant connections to the nucleus tractus

solitarius and dorsal motor nucleus of the vagus

(Arends et al. 1988, Berk and Finkelstein 1983),

parabrachial nucleus (Wild et al. 1990) and median

eminence (Kiss 1988, Korf solitarius and dorsal motor nucleus of the vagus

(Arends et al. 1988, Berk and Finkelstein 1983),

parabrachial nucleus (Wild et al. 1990) and median

eminence (Kiss 1988, Korf 1984) warrants study of its

effect on food (Arends et al. 1988, Berk and Finkelstein 1983),
parabrachial nucleus (Wild et al. 1990) and median
eminence (Kiss 1988, Korf 1984) warrants study of its
effect on food intake. No studies have been completed
where ventrome eminence (Kiss 1988, Korf 1984) warrants study of its

effect on food intake. No studies have been completed

where ventromedial hypothalamic lesions have been

compared to paraventricular nucleus lesions in birds

to dete regulatory obesity. Sonoda (1984) warrants study of its
effect on food intake. No studies have been completed
where ventromedial hypothalamic lesions have been
compared to paraventricular nucleus lesions in birds
to determ effect on food intake. No studies have been completed
where ventromedial hypothalamic lesions have been
compared to paraventricular nucleus lesions in birds
to determine whether there is a metabolic vs. a
regulatory obesit where ventromedial hypothalamic lesions have been
compared to paraventricular nucleus lesions in birds
to determine whether there is a metabolic vs. a
regulatory obesity. Sonoda (1983) has demonstrated
that insulin levels compared to paraventricular nucleus lesions in birds
to determine whether there is a metabolic vs. a
regulatory obesity. Sonoda (1983) has demonstrated
that insulin levels are higher than controls in ven-
tromedial hypotha to determine whether there is a metabolic vs. a
regulatory obesity. Sonoda (1983) has demonstrated
that insulin levels are higher than controls in ven-
tromedial hypothalamically lesioned chickens.
Nineteen days following regulatory obesity. Sonoda (1983) has demonstrated
that insulin levels are higher than controls in ven-
tromedial hypothalamically lesioned chickens.
Nineteen days following surgery, when food was
withheld from ventromedia that insulin levels are higher than controls in ven-
tromedial hypothalamically lesioned chickens.
Nineteen days following surgery, when food was
withheld from ventromedial hypothalamically le-
sioned birds and controls, v tromedial hypothalamically lesioned chickens.

insulfunction days following surgery, when food was

withheld from ventromedial hypothalamically le-

sioned birds and controls, ventromedial hypothalami-

cally lesioned bird withheld from ventromedial hypothalamically le-
sioned birds and controls, ventromedial hypothalami-
cally lesioned birds had significantly elevated plasma
insulin suggesting that they, like mammals, may
show metabolic obe the the the method is and controlled hypothalamically le-

level birds and controls, ventromedial hypothalami-

ly lesioned birds had significantly elevated plasma

ulin suggesting that they, like mammals, may

ow metaboli sioned birds and controls, ventromedial hypothalamically lesioned birds had significantly elevated plasma
insulin suggesting that they, like mammals, may
show metabolic obesity.
Lateral hypothalamic lesions have been perfo

cally lesioned birds had significantly elevated plasma

insulin suggesting that they, like mammals, may

show metabolic obesity.

Lateral hypothalamic lesions have been performed

in chickens (Feldman et al. 1957), White-t Insulin suggesting that they, like mammals, may

show metabolic obesity.

Lateral hypothalamic lesions have been performed

in chickens (Feldman et al. 1957), White-throated

Sparrows (Kuenzel 1972), pigeons (Ziegler 1976, show metabolic obesity.

Lateral hypothalamic lesions have been performed

in chickens (Feldman et al. 1957), White-throated

Sparrows (Kuenzel 1972), pigeons (Ziegler 1976,

Ziegler and Karten 1973, Zeigler et al. 1969) a Lateral hypothalamic lesions have been performed
in chickens (Feldman et al. 1957), White-throated
Sparrows (Kuenzel 1972), pigeons (Ziegler 1976,
Ziegler and Karten 1973, Zeigler et al. 1969) and
broiler chicks (Kuenzel 1 in chickens (Feldman et al. 1957), White-throated

Sparrows (Kuenzel 1972), pigeons (Ziegler 1976, Ward Viegler and Karten 1973, Zeigler et al. 1969) and

broiler chicks (Kuenzel 1982). Results were consistent in

in that Sparrows (Kuenzel 1972), pigeons (Ziegler 1976,

Ziegler and Karten 1973, Zeigler et al. 1969) and

broiler chicks (Kuenzel 1982). Results were consistent

in that lesions in and around the lateral hypothalamic

area effec Ziegler and Karten 1973, Zeigler et al. 1969) and
broiler chicks (Kuenzel 1982). Results were consistent
in that lesions in and around the lateral hypothalamic
area effected reduced food intake. A marked
difference between broiler chicks (Kuenzel 1982). Results were consisten
in that lesions in and around the lateral hypothalamic
area effected reduced food intake. A markec
difference between birds and mammals, however, was
that the aphagic r In that lesions in and around the lateral hypothalamic

area effected reduced food intake. A marked li

difference between birds and mammals, however, was

that the aphagic response was not permanent. Large,

bilateral les area effected reduced food intake. A marked
difference between birds and mammals, however, was
that the aphagic response was not permanent. Large,
bilateral lesions involving the ansa lenticularis, quin-
tofrontal tract an difference between birds and mammals, however, was
that the aphagic response was not permanent. Large,
bilateral lesions involving the ansa lenticularis, quin-
tofrontal tract and lateral hypothalamic area were
most effect that the aphagic response was not permanent. Large bilateral lesions involving the ansa lenticularis, quintofrontal tract and lateral hypothalamic area were most effective for reducing food intake but it usuall returned to bilateral lesions involving the ansa lenticularis, quin-
tofrontal tract and lateral hypothalamic area were
most effective for reducing food intake but it usually
hare
turned to preoperative levels within 1 wk (Kuenzel
198 to trontal tract and lateral hypothalamic area were
most effective for reducing food intake but it usually
returned to preoperative levels within 1 wk (Kuenzel
1982). Similar results were obtained when stimulus-
bound feed most effective for reducing food intake but it usually

returned to preoperative levels within 1 wk (Kuenzel

1982). Similar results were obtained when stimulus-

bound feeding was attempted in fowl. More than 625

sites w returned to preoperative levels within 1 wk (Kuenzel

1982). Similar results were obtained when stimulus-

bound feeding was attempted in fowl. More than 625

sites were implanted with electrodes for electrical

brain stim 1982). Similar results were obtained when stimulus-
bound feeding was attempted in fowl. More than 625
sites were implanted with electrodes for electrical
brain stimulation in conscious animals. No positive
brain loci wer bound feeding was attempted in fowl. More than 625
sites were implanted with electrodes for electrical
brain stimulation in conscious animals. No positive
brain loci were identified for stimulus-bound food
intake (Tweeton sites were implanted with electrodes for electrical
brain stimulation in conscious animals. No positive
brain loci were identified for stimulus-bound food
intake (Tweeton et al. 1973). With respect to the
function of the l brain stimulation in conscious animals. No positive
brain loci were identified for stimulus-bound food
intake (Tweeton et al. 1973). With respect to the
function of the lateral hypothalamic area, either birds
are different The Compact of the lateral hypothalamic area, either birds
arent from mammals, or the equivalent neural
s not been identified to date in avian species.
COMPOUNDS ADMINISTERED
CENTRALLY IN BIRDS In the factor in approximation area, either bin
from mammals, or the equivalent neur-
been identified to date in avian specie
MPOUNDS ADMINISTERED
CENTRALLY IN BIRDS

COMPOUNDS ADMINISTERED

CENTRALLY IN BIRDS

The first compounds given centrally to birds were

genic amines. In broiler-type chickens (selected for **COMPOUNDS ADMINISTERED
CENTRALLY IN BIRDS**
The first compounds given centrally to birds were
biogenic amines. In broiler-type chickens (selected for
growth rate and meat production), intracerebroven-COMPOGINDS ADMINISTERED
CENTRALLY IN BIRDS
The first compounds given centrally to birds we
biogenic amines. In broiler-type chickens (selected f
growth rate and meat production), intracerebrove
tricularly injected epinephr The first compounds given centrally to birds were
biogenic amines. In broiler-type chickens (selected for
growth rate and meat production), intracerebroven-
tricularly injected epinephrine significantly increased
food inta The first compounds given centrally to birds were
biogenic amines. In broiler-type chickens (selected for
growth rate and meat production), intracerebroven-
tricularly injected epinephrine significantly increased
food inta The first compounds given centrally to birds were
biogenic amines. In broiler-type chickens (selected for
growth rate and meat production), intracerebroven-
tricularly injected epinephrine significantly increased
food inta biogenic amines. In broiler-type chickens (selected for
growth rate and meat production), intracerebroven-
tricularly injected epinephrine significantly increased
food intake (Denbow et al. 1981). In contrast, no
significa growth rate and meat production), intracerebrov
tricularly injected epinephrine significantly increas
food intake (Denbow et al. 1981). In contrast,
significant change was noted when Leghorn chief
(strain selected for tabl tricularly injected epinephrine significantly increased
food intake (Denbow et al. 1981). In contrast, no
significant change was noted when Leghorn chicks
(strain selected for table egg production) were given
epinephrine (food intake (Denbow et al. 1981). In contrast, no
significant change was noted when Leghorn chicks
(strain selected for table egg production) were given
epinephrine (Denbow et al. 1983). Intracerebroventric-
ularly injecte significant change was noted when Leghorn chicks
(strain selected for table egg production) were given
epinephrine (Denbow et al. 1983). Intracerebroventric-
ularly injected norepinephrine had an overriding nar-
coleptic e (strain selected for table egg production) were given
epinephrine (Denbow et al. 1983). Intracerebroventric-
ularly injected norepinephrine had an overriding nar-
coleptic effect on birds that masked other behaviors.
When epinephrine (Denbow et al. 1983). Intracerebroventric-
ularly injected norepinephrine had an overriding nar-
coleptic effect on birds that masked other behaviors.
When it was injected into specific brain loci at lower
dose

EL
medial preoptic area, anterior medial hypothalamic
nucleus, paraventricular nucleus or medial septal nu-EL
medial preoptic area, anterior medial hypothalamic
nucleus, paraventricular nucleus or medial septal nu-
cleus of broiler chicks increased food intake (Denbow EL
medial preoptic area, anterior medial hypothalamic
nucleus, paraventricular nucleus or medial septal nu-
cleus of broiler chicks increased food intake (Denbow
and Sheppard 1993). Norepinephrine administered EL
medial preoptic area, anterior medial hypothalamic
nucleus, paraventricular nucleus or medial septal nu-
cleus of broiler chicks increased food intake (Denbow
and Sheppard 1993). Norepinephrine administered
near the lat medial preoptic area, anterior medial hypothalamic
nucleus, paraventricular nucleus or medial septal nu-
cleus of broiler chicks increased food intake (Denbow
and Sheppard 1993). Norepinephrine administered
near the latera medial preoptic area, anterior medial hypothalamic
nucleus, paraventricular nucleus or medial septal nu-
cleus of broiler chicks increased food intake (Denbow
and Sheppard 1993). Norepinephrine administered
near the latera nucleus, paraventricular nucleus or medial septal nucleus of broiler chicks increased food intake (Denbow
and Sheppard 1993). Norepinephrine administered
near the lateral septal organ [a circumventricular
organ in birds (K cleus of broiler chicks increased tood intake (Denbow
and Sheppard 1993). Norepinephrine administered
near the lateral septal organ [a circumventricular
organ in birds (Kuenzel and Blähser 1994, Kuenzel and
vanTienhoven 19 and Sheppard 1993). Norepinephrine administered
near the lateral septal organ [a circumventricular
organ in birds (Kuenzel and Blähser 1994, Kuenzel and
vanTienhoven 1982)], within the dorsal, superior
reticular nucleus or near the lateral septal organ [a circumventricular
organ in birds (Kuenzel and Blähser 1994, Kuenzel and
vanTienhoven 1982)], within the dorsal, superior
reticular nucleus or lateral forebrain bundle resulted
in significan organ in birds (Kuenzel and Blähser 1994, Kuenzel and
van Tienhoven 1982)], within the dorsal, superior
reticular nucleus or lateral forebrain bundle resulted
in significant reductions in feeding (Denbow and
Sheppard 1993) van Tienhoven 1982)], within the dorsal, superior
reticular nucleus or lateral forebrain bundle resulted
in significant reductions in feeding (Denbow and
Sheppard 1993). When 6-hydroxydopamin was given
intracerebroventricu reticular nucleus or lateral forebrain bundle resulted
in significant reductions in feeding (Denbow and
Sheppard 1993). When 6-hydroxydopamin was given
intracerebroventricularly to broiler chicks, food
intake was greatly r in significant reductions in feeding (Denbow a
Sheppard 1993). When 6-hydroxydopamin was giv
intracerebroventricularly to broiler chicks, fo
intake was greatly reduced as was striatal, hypoth
lamic and brainstem norepineph Sheppard 1993). When 6-hydroxydopamin was given
intracerebroventricularly to broiler chicks, food
intake was greatly reduced as was striatal, hypotha-
lamic and brainstem norepinephrine and striatal $\frac{1}{5}$
dopamine (Ku intracerebroventricularly to broiler chicks, tood
intake was greatly reduced as was striatal, hypotha-
lamic and brainstem norepinephrine and striatal $\frac{1}{5}$
dopamine (Kuenzel et al. 1987b). Intracerebroven-
tricular s In take was greatly reduced as was striatal, hypotha-
lamic and brainstem norepinephrine and striatal g
dopamine (Kuenzel et al. 1987b). Intracerebroven-
tricular serotonin also significantly reduced food
intake in fully lamic and brainstem
dopamine (Kuenzel et
tricular serotonin als
intake in fully fed bracausing satiety in broil
al. 1982; Table 4).
A diverse group of p tricular serotonin also significantly reduced food
intake in fully fed broilers, but was ineffective for
causing satiety in broilers unfed for 24 h (Denbow et
al. 1982; **Table 4**).
A diverse group of peptides, amino acids

lamine and brannstrom noreprine from the development and striated
dopamine (Kuenzel et al. 1987b). Intracerebroven-
include from also significantly reduced food
intake in fully fed broilers, but was ineffective for
and in intake in fully fed broilers, but was ineffective for $\frac{8}{3}$
causing satiety in broilers unfed for 24 h (Denbow et al. 1982; **Table 4**).
A diverse group of peptides, amino acids, and hor-
mones has been shown to stimul causing satiety in broilers unted tor 24 h (Denbow et al. 1982; Table 4).

A diverse group of peptides, amino acids, and hor-

mones has been shown to stimulate food intake.

Within the opioid peptide family, β -endorph al. 1982; Table 4).

A diverse group of peptides, amino acids, and hor-

mones has been shown to stimulate food intake.

Within the opioid peptide family, β -endorphin (os-

trich) was first shown in pigeons to stimulat A diverse group of peptides, amino acids, and hor-
mones has been shown to stimulate food intake.
Within the opioid peptide family, β -endorphin (os-
trich) was first shown in pigeons to stimulate food
intake with intra mones has been shown to stimulate food intake.
Within the opioid peptide family, β -endorphin (os-
trich) was first shown in pigeons to stimulate food and
intake with intracerebroventricular injection $\frac{a}{b}$
(Deviche Within the opioid peptide family, β -endorphin (os-
trich) was first shown in pigeons to stimulate food
intake with intracerebroventricular injection
(Deviche and Schepers 1984). β -Endorphin (human)
likewise stimulat trich) was first shown in pigeons to stimulate food
intake with intracerebroventricular injection
 $\frac{a}{T}$
(Deviche and Schepers 1984). β -Endorphin (human)
 $\frac{p}{Z}$
intervise stimulates food intake in both broiler-ty intake with intracerebroventricular injection $\frac{a}{b}$
(Deviche and Schepers 1984). β -Endorphin (human)
likewise stimulates food intake in both broiler-type
and egg-layer type chicks (McCormack and Denbow,
1988). Meth (Deviche and Schepers 1984). β -Endorphin (human)
likewise stimulates food intake in both broiler-type
and egg-layer type chicks (McCormack and Denbow, $\frac{1}{\alpha}$
1988). Methionine-enkephalin stimulates food intake
in b likewise stimulates food intake in both broiler-type $\frac{6}{6}$
and egg-layer type chicks (McCormack and Denbow, $\frac{6}{5}$
1988). Methionine-enkephalin stimulates food intake
in broiler-type chicks (McCormack and Denbow
19 and egg-layer type chicks (McCormack and Denbow,

1988). Methionine-enkephalin stimulates food intake

in broiler-type chicks (McCormack and Denbow

1989). Within the pancreatic polypeptide family, three

have been shown 1988). Methionine-enkephalin stimulates tood intake
in broiler-type chicks (McCormack and Denbow
1989). Within the pancreatic polypeptide family, three
have been shown to significantly increase food intake.
Neuropeptide Y in broiler-type chicks (McCormack and Denbow

1989). Within the pancreatic polypeptide family, three

have been shown to significantly increase food intake.

Neuropeptide Y and peptide YY given in-

tracerebroventricularl 1989). Within the pancreatic polypeptide family, three
have been shown to significantly increase food intake.
Neuropeptide Y and peptide YY given in-
tracerebroventricularly to broiler chicks had a robust
effect on food i have been shown to significantly increase tood intake.

Neuropeptide Y and peptide YY given in-

tracerebroventricularly to broiler chicks had a robust

effect on food intake [Kuenzel et al. 1987a]. Following

neuropeptid Neuropeptide Y and peptide YY given in-

tracerebroventricularly to broiler chicks had a robust

effect on food intake (Kuenzel et al. 1987a). Following

neuropeptide Y injections, significant increases in

plasma insulin tracerebroventricularly to broiler chicks had a robust

effect on food intake (Kuenzel et al. 1987a). Following

meuropeptide Y injections, significant increases in

plasma insulin were obtained with no changes in

glucag effect on food intake (Kuenzel et al. 1987a). Following
neuropeptide Y injections, significant increases in
plasma insulin were obtained with no changes in $\frac{2}{5}$
glucagon or plasma glucose (Kuenzel and McMurtry
1988). neuropeptide Y injections, significant increases in $\frac{m}{2}$
plasma insulin were obtained with no changes in $\frac{m}{6}$
glucagon or plasma glucose (Kuenzel and McMurtry $\frac{m}{60}$
1988). Avian pancreatic polypeptide injec plasma insulin were obtained with no changes in $\frac{2}{5}$
glucagon or plasma glucose (Kuenzel and McMurtry $\frac{1}{5}$
1988). Avian pancreatic polypeptide injected into
Leghorn hens also was effective for increasing food
in glucagon or plasma glucose (Kuenzel and McMurtry

1988). Avian pancreatic polypeptide injected into

Leghorn hens also was effective for increasing food

intake (Denbow et al. 1988). As can be seen in Table

4, the only p 1988). Avian pancreatic polypeptide injected into $\frac{30}{2}$
Leghorn hens also was effective for increasing food $\frac{30}{2}$
intake (Denbow et al. 1988). As can be seen in Table $\frac{9}{2}$
4, the only peptide applied centra Leghorn hens also was effective for increasing food $\frac{\pi}{8}$
intake (Denbow et al. 1988). As can be seen in Table $\frac{9}{5}$
4, the only peptide applied centrally to date in birds $\frac{8}{5}$
that decreases food intake is c Intake (Denbow et al. 1988). As can be seen in Table
4, the only peptide applied centrally to date in birds
that decreases food intake is cholecystokinin. In-
tracerebroventricular injection of cholecystokinin-8
reduced f 4, the only peptide applied centrally to date in birds
that decreases food intake is cholecystokinin. In-
tracerebroventricular injection of cholecystokinin-8
reduced food intake in broilers (Denbow and Myers
1982). It wo that decreases food intake is cholecystokinin. In-
tracerebroventricular injection of cholecystokinin-8
reduced food intake in broilers (Denbow and Myers
1982). It would be of interest to determine whether
any of the othe tracerebroventricular injection of cholecystokinin-8
reduced food intake in broilers (Denbow and Myers $\frac{10}{9}$
1982). It would be of interest to determine whether
any of the other peptides that have been effective in
r reduced tood intake in broilers (Denbow and Myers $\frac{3}{5}$ 1982). It would be of interest to determine whether any of the other peptides that have been effective in reducing food intake in mammals (Table 3), particularly 1982). It would be of interest to determine whether
any of the other peptides that have been effective in
reducing food intake in mammals (Table 3), particu-
larly bombesin and members of the glucagon-secretin
family, like any of the other peptides that have been effective in
reducing food intake in mammals (Table 3), particu-
larly bombesin and members of the glucagon-secretin
family, likewise reduce food intake. The author only
uncovered o reducing food intake in mammais (Table 3), particu-
larly bombesin and members of the glucagon-secretin
family, likewise reduce food intake. The author only
uncovered one study addressing central adminis-
tration of an ami family, likewise reduce food intake. The author only uncovered one study addressing central administration of an amino acid. Intracerebroventricular injection of muscimol, a GABA agonist, increased food intake in turkey he tration of an amino acid. Intracerebroventricular inuncovered one study addressing central adminis-
tration of an amino acid. Intracerebroventricular in-
jection of muscimol, a GABA agonist, increased food
intake in turkey hens (Denbow 1991).
The author was unable to find p

tration of an amino acid. Intracerebroventricular in-
jection of muscimol, a GABA agonist, increased food
intake in turkey hens (Denbow 1991).
The author was unable to find published infor-
mation on central injections of jection of muscimol, a GABA agonist, increased food
intake in turkey hens (Denbow 1991).
The author was unable to find published infor-
mation on central injections of insulin in birds. The
lack of supporting evidence for intake in turkey hens (Denbow 1991).
The author was unable to find published infor-
mation on central injections of insulin in birds. The
lack of supporting evidence for a glucostatic control
of food intake in birds could The author was unable to find published infor-
mation on central injections of insulin in birds. The
lack of supporting evidence for a glucostatic control
of food intake in birds could be one reason for the
paucity of info

THE JOURNAL OF NUTRITION

Compounds administered centrally that alter food intake in birds1

norepinephrine.
³Photostimulated.

4Non-photostimulated, light:dark cycle (8 h:16 h).

following goldthioglucose or 2-deoxyglucose adminis-
following goldthioglucose or 2-deoxyglucose adminis-
tration, and birds are resistant to drugs that cause ⁴Non-photostimulated, light:dark cycle $(8 \text{ h:}16 \text{ h})$.

following goldthioglucose or 2-deoxyglucose administration, and birds are resistant to drugs that cause

diabetes, e.g., alloxan (Simon 1989). Of particular infollowing goldthioglucose or 2-deoxyglucose adminis-
tration, and birds are resistant to drugs that cause
diabetes, e.g., alloxan (Simon 1989). Of particular in-
terest, however, is the increase in food intake shown following goldthioglucose or 2-deoxyglucose adminis-
tration, and birds are resistant to drugs that cause
diabetes, e.g., alloxan (Simon 1989). Of particular in-
terest, however, is the increase in food intake shown
in rin following goldthioglucose or 2-deoxyglucose adminis-
tration, and birds are resistant to drugs that cause
diabetes, e.g., alloxan (Simon 1989). Of particular in-
terest, however, is the increase in food intake shown
in rin moleculowing goldthoglucose or 2-deoxyglucose administration, and birds are resistant to drugs that cause
diabetes, e.g., alloxan (Simon 1989). Of particular in-
terest, however, is the increase in food intake shown fo
in tration, and birds are resistant to drugs that cause

diabetes, e.g., alloxan (Simon 1989). Of particular in-

terest, however, is the increase in food intake shown

in ring doves following intracerebroventricular ad-

min diabetes, e.g., alloxan (simon 1989). Of particular in-
terest, however, is the increase in food intake shown
in ring doves following intracerebroventricular ad-
ministration of prolactin (Buntin 1989) and growth
hormone (in ring doves following intracerebroventricular administration of prolactin (Buntin 1989) and growth hormone (Buntin and Figge 1988). In a mapping study addressing the most sensitive brain sites for eliciting an orexigenic in ring doves following intracerebroventricular ad-

ministration of prolactin (Buntin 1989) and growth

hormone (Buntin and Figge 1988). In a mapping study

peladdressing the most sensitive brain sites for eliciting

an o ministration of prolactin (Buntin 1989) and growth
hormone (Buntin and Figge 1988). In a mapping study
addressing the most sensitive brain sites for eliciting
an orexigenic effect of prolactin, the ventromedial
hypothalamu normone (Buntin and Figge 1988). In a mapping study
addressing the most sensitive brain sites for eliciting
an orexigenic effect of prolactin, the ventromedial
al.
hypothalamus and medial preoptic area were found to
be the

have a high density of prolactin binding sites in ring
dove brains (Fechner and Buntin 1989). Because have a high density of prolactin binding sites in ring
dove brains (Fechner and Buntin 1989). Because
mammals have been shown to increase food intake have a high density of prolactin binding sites in ring
dove brains (Fechner and Buntin 1989). Because
mammals have been shown to increase food intake
following administration of growth hormone releasing have a high density of prolactin binding sites in ring
dove brains (Fechner and Buntin 1989). Because
mammals have been shown to increase food intake
following administration of growth hormone releasing
factor into the ven have a high density of prolactin binding sites in ring
dove brains (Fechner and Buntin 1989). Because
mammals have been shown to increase food intake
following administration of growth hormone releasing
factor into the ven dove brains (Fechner and Buntin 1989). Because
mammals have been shown to increase food intake
following administration of growth hormone releasing
factor into the ventromedial hypothalamus or medial
preoptic area (Table 3 dove brains (rechner and buntin 1989). Because
mammals have been shown to increase food intake
following administration of growth hormone releasing
factor into the ventromedial hypothalamus or medial
preoptic area (Table 3 mammais nave been shown to increase rood intake
following administration of growth hormone releasing
factor into the ventromedial hypothalamus or medial
preoptic area (Table 3), and vasoactive intestinal poly-
peptide has rollowing administration of growth hormone releasing
factor into the ventromedial hypothalamus or medial
preoptic area (Table 3), and vasoactive intestinal poly-
peptide has been shown to be a potent releaser of
prolactin ractor into the ventromedial hypothalamus or medial
preoptic area (Table 3), and vasoactive intestinal poly-
peptide has been shown to be a potent releaser of
prolactin in birds (Opel and Proudman 1988, Sharp et
al. 1989), preoptic area (1 able 3), and vasoactive intestinal poly-
peptide has been shown to be a potent releaser of
prolactin in birds (Opel and Proudman 1988, Sharp et
al. 1989), both vasoactive intestinal polypeptide and
growth peptiae nas been shown to be a potent releaser of
prolactin in birds (Opel and Proudman 1988, Sharp et
al. 1989), both vasoactive intestinal polypeptide and
growth hormone releasing factor would be worth in-
vestigating in

PROURE 6 A sagittal-section of a chick brain taken from
 PROURE 6 A sagittal-section of a chick brain taken from
 PROURE 6 A sagittal-section of a chick brain taken from
 PROPERTY ASSES
 PROPERTY ASSAUTE ACTES
 FIGURE 6 A sagittal-section of a chick brain taken from atlas plate L 0.2 mm of Kuenzel and Masson (1988) showing projections that have been documented in the avian brain that can affect the sympathetic (S.N.S.) and par **FIGURE 6 A sagittal-section of a chick brain taken from**
atlas plate L 0.2 mm of Kuenzel and Masson (1988) showing
projections that have been documented in the avian brain
that can affect the sympathetic (S.N.S.) and para **FIGURE 6 A sagittal-section of a chick brain taken from**
atlas plate L 0.2 mm of Kuenzel and Masson (1988) showing
projections that have been documented in the avian brain
that can affect the sympathetic (S.N.S.) and para atlas plate L 0.2 mm of Kuenzel and Masson (1988) showing
projections that have been documented in the avian brain
that can affect the sympathetic (S.N.S.) and parasym-
pathetic (P.N.S.) nervous systems. Abbreviations used that can affect the sympathetic (S.N.S.) and parasym-
pathetic (P.N.S.) nervous systems. Abbreviations used: Ac,
nucleus accumbens; GCt, central grey; IML, intermedi-
olateral; IN, infundibular nucleus; LHy, lateral hypoth pathetic (P.N.S.) nervous systems. Abbreviations used: Ac,
nucleus accumbens; GCt, central grey; IML, intermedi-
olateral; IN, infundibular nucleus; LHy, lateral hypotha-
lamic area; LoC, locus ceruleus; LSO, lateral septa nucleus accumbens; GCt, central grey; IML, intermedi-
olateral; IN, infundibular nucleus; LHy, lateral hypotha-
lamic area; LoC, locus ceruleus; LSO, lateral septal organ;
ME, median eminence; n IX-X, glossopharyngeal and olateral; IN, infundibular nucleus; LHy, lateral hypotha-
lamic area; LoC, locus ceruleus; LSO, lateral septal organ;
ME, median eminence; n IX-X, glossopharyngeal and dorsal
motor nucleus of vagus; nST, bed nucleus stria motor interess or vagas; no r, bed interess stra terminalis;
nTS, nucleus tractus solitarius; PB, parabrachial nucleus;
PVN, paraventricular nucleus; SCE, stratum celluare ex-
ternum; TPc, substantia nigra.
administration

administration to the ventromedial hypothalamus or medial preoptic area.

ministration to the ventromedial hypothalamus or

dial preoptic area.

NEUROANATOMICAL PATHWAYS OF

NPORTANCE IN BIRDS WITH RESPECT

TO THE AITONOMIC AND Mathematical preoptic area.

In picture of the ventromedial hypothalamus or

in picture in picture

IMPORTANCE IN BIRDS WITH RESPECT

IMPORTANCE IN BIRDS WITH RESPECT

TO THE AUTONOMIC AND

ENDOCEINE HYPOTHESIS THE REAL PATHWAYS

TROPIC ATES

TANCE IN BIRDS WITH RESPECT

TO THE AUTONOMIC AND

ENDOCRINE HYPOTHESIS NEUROANATOMICAL PATHWAYS OF

IMPORTANCE IN BIRDS WITH RESPECT

TO THE AUTONOMIC AND

ENDOCRINE HYPOTHESIS

The most critical neuroanatomical sites in NEUROANATOMICAL PATHWAYS OF

MPORTANCE IN BIRDS WITH RESPECT

TO THE AUTONOMIC AND

ENDOCRINE HYPOTHESIS

The most critical neuroanatomical sites in

mmals for regulating food intake include the ven-

medial hypothalamic a

IMPORTANCE IN BIRDS WITH RESPECT

TO THE AUTONOMIC AND

ENDOCRINE HYPOTHESIS

The most critical neuroanatomical sites in

The most critical neuroanatomical sites in

tromedial hypothalamic area (ventromedial hypotha-

lami TO THE AUTONOMIC AND
ENDOCRINE HYPOTHESIS
The most critical neuroanatomical sites in
mammals for regulating food intake include the ventromedial hypothalamic nucleus,
lamic nucleus, dorsomedial hypothalamic nucleus ENDOCRINE HYPOTHESIS

The most critical neuroanatomical sites in

mammals for regulating food intake include the ven-

tromedial hypothalamic area (ventromedial hypotha-

lamic nucleus, dorsomedial hypothalamic nucleus

an ENDOCKINE INFOTIESIS
The most critical neuroanatomical sites in
mammals for regulating food intake include the ven-
tromedial hypothalamic nucleus, dorsomedial hypothalamic nucleus
and infundibular nucleus), lateral hypoth The most critical neuroanatomical sites in
mammals for regulating food intake include the ven-
tromedial hypothalamic area (ventromedial hypotha-
lamic nucleus, dorsomedial hypothalamic nucleus
and infundibular nucleus), l The most critical neuroanatomical sites in

mammals for regulating food intake include the ven-

tromedial hypothalamic area (ventromedial hypotha-

lamic nucleus, dorsomedial hypothalamic nucleus

and infundibular nucleu mammais for regulating food intake include the ven-
tromedial hypothalamic area (ventromedial hypotha-
lamic nucleus, dorsomedial hypothalamic nucleus
and infundibular nucleus), lateral hypothalamic area,
paraventricular n romealal hypothalamic area (ventromealal hypothalamic nucleus
and infundibular nucleus), lateral hypothalamic area,
paraventricular nucleus, perifornical hypothalamus,
nucleus tractus solitarius, and parabrachial nucleus. The schematic diagram in Figure 6 is obtained. Note that the lateral hypothalamic area, the schematic diagram in Figure 6 is obtained. Note that the lateral hypothalamic area projects to the nucleus tractus solitarius (Ber and intundibular nucleus), lateral hypothalamic area,
paraventricular nucleus, periformical hypothalamus,
nucleus tractus solitarius, and parabrachial nucleus. If
the equivalent avian structures are then grouped ac-
cordi paraventricular nucleus, performical hypothalamus,
nucleus tractus solitarius, and parabrachial nucleus. If
the equivalent avian structures are then grouped ac-
cording to known projections within the avian brain,
the sche the equivalent avian structures are then grouped according to known projections within the avian brain, the schematic diagram in Figure 6 is obtained. Note that the lateral hypothalamic area projects to the nucleus tractus 1988). This structure contains the parasym-

1988). This structure contains the projects to the properties of the vagus (hoted as n.IX-X in Fig. 6; Arends et al.

1988). This structure contains the preganglionic neurons th mat the lateral hypothalamic area projects to the
nucleus tractus solitarius (Berk 1987) and the nucleus
tractus solitarius projects to the dorsal motor nucleus
of the vagus (noted as n.IX-X in Fig. 6; Arends et al.
1988). mucieus tractus solitarius projects to the dorsal motor nucleus
tractus solitarius projects to the dorsal motor nucleus
of the vagus (noted as n.IX-X in Fig. 6; Arends et al.
1988). This structure contains the preganglioni tractus solitarius projects to the dorsal motor nucleus

of the vagus (noted as n.IX-X in Fig. 6; Arends et al.

1988). This structure contains the preganglionic

parthetic nervous system. The mammal has a similar

project of the vagus inoted as n.LX-X in Fig. 6; Arends et al.
1988). This structure contains the preganglionic
neurons that are a major component of the parasym-
pathetic nervous system. The mammal has a similar
projection and th Fracture contains the pregangionic

neurons that are a major component of the parasym-

pathetic nervous system. The mammal has a similar

projection and the components have been shown to

be part of the parasympathetic ne neurons that are a major component of the parasym-
pathetic nervous system. The mammal has a similar
projection and the components have been shown to
be part of the parasympathetic nervous system
(Luiten et al. 1987). In c

mammals the central grey projects to the intermedi-
olateral column that houses preganglionic neurons EL

mammals the central grey projects to the intermedi-

olateral column that houses preganglionic neurons

serving as major components of the sympathetic

nervous system. The dashed line in the diagram indi EL
mammals the central grey projects to the intermedi-
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nervous system. The dashed line in the diagram indi-
cates that more n serving as major components of the sympathetic
nervous system. The dashed line in the diagram indi-
cates that more neuroanatomical studies are needed
to verify the projection in birds. Hence the ven-EL
mammals the central grey projects to the intermedi-
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cates that more neuroanatomical studies are needed
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tromedial hypothalami tromedial hypothalamic area, composed of the ven-
tromedial hypothalamic nucleus and infundibular nu-
cleus is part of the sympathetic nervous system
(Luiten et al. 1987). The original ventromedial
hypothalamus lesion stud cates that more heuroanatomical studies are needed
to verify the projection in birds. Hence the ven-
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tromedial hypothalamic area, composed of the ven-
tromedial hypothalamic nucleus and infundibular nu-
cleus is part of the sympathetic nervous system
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(Luiten et al. 1987). The original ventromedial
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(Luiten et al. 1987). The original ventromedial

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hypothalamic area lesions effected a loss of body
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both parasympathetic and sympathetic connections.
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1981). It would be of interest to bilaterally lesion the

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The auton subhucclei (unpublished data) and also projects to the

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it is a critical nucleus to focus upon in avian brains.

The autonomic and endocrine hypothesis appears

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to be very applicable to some of the avian strains
found in the commercial poultry industry. For many
years turkeys and particularly broilers have been ge-
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netically selected for meat production. In selecting for
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selected for appetite. Broiler breeders are a good ex-
ample o metically selected for meat production. In selecting for
growth rate, birds have also been inadvertently
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ample of birds that have a voracious appetite and a
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ample of birds that have a voracious appetite and a
tendency to become obese. Their offspring are
generally hypoactive selected for appetite. Broller breeders are a good example of birds that have a voracious appetite and a tendency to become obese. Their offspring are generally hypoactive and have a lower basal metabolic rate (Kuenzel an ample of birds that have a voracious appetite and a
tendency to become obese. Their offspring are
generally hypoactive and have a lower basal meta-
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to that of ventrom bonc rate (Kuenzel and Kuenzel 1977) and a rapid
growth rate. In other words, broilers show a domi-
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to that of ventromedial hypothalamically-lesioned
rats and in some obe growth rate. In other words, orollers show a domi-
nance of the parasympathetic nervous system similar
to that of ventromedial hypothalamically-lesioned
rats and in some obese humans. It will be important
in the future for strains.

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tring the figures and P. Bokman for typing the The author wishes to thank M. Masson for completing the figures and P. Bokman for typing the

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EXERCTION 1. Roberts, G. W., Crow, T. J., Tatemoto, K. & Polak, J. M.

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