Psych 2220A Lecture 10

- Is it true only humans have language?
  - Evidence in non-human primates and other animals
  - Evolution of language
- How specialized are our brains for language?
  - Domain-general and domain-specific mechanisms
  - Language disorders
  - Reading
- How is language processed in the brain?
  - Wernicke-Geschwind model
  - Cognitive neurosciences approachLearning outcomes

<u>What is language?</u>

- Systematic and conventional use of sounds (or signs or written symbols) for the purpose of communication or self expression
  - Complex communication system
  - Intentionality
  - Arbitrary symbols/reference
  - Grammar/syntax
  - Cultural or social transmission
- If language is unique to humans, then we are the only ones that should exhibit these behaviours/abilities

<u>Language in non-human primates</u>

- Can acquire aspects of language
- No syntax or reference
- Why can't chimpanzees acquire language?
  - Don't have innate language acquisition device
  - Vocal limitation
  - Reference problem
  - Cultural/social transmission

<u>Are humans the only animals that exhibit these behaviours/abilities?</u>

- Complex communication system
  - Honey bee (*apis mellifera*)
- Grammar/syntax and Arbitrary symbols/reference in Black-capped chick-a-dee call notes
  - Cultural transmission:
    - Predisposed to learn own-species song
    - Abnormal song regains normal song characteristics over generations
    - Learned dialects

- Many other animals share characteristics that we use to define human language
- So what aspect of language is truly unique to humans?
- If we are the only ones to exhibit language, how did this ability evolve?

Psych 2220A Lecture 10

How did the brain evolve to process language?
- Adaptation
  - New brain areas
  - Specialized, not shared with non-linguistic ancestors
- Descent with modification
  - Remodeling and reuse of existing rudimentary structures
  - Share some similarities with non-linguistic ancestors

Revising the nature-nurture debate
- Language acquisition and use requires both nature and nurture
- **Domain-general and domain-specific** theories of language differ on views of how specialized our innate learning mechanisms are:
  - Have we evolved specialized brain areas only for learning and processing language?
  - Or have we found ways to adapt our pre-existing abilities for use with language?

Brains for language: specialized or coopted?
- **Domain-specific mechanisms**
  - Complex cognitive abilities are supported by specialized, evolutionarily adaptive learning devices
  - **Modularity:** mind as a bundle of many special-purpose modules
- Linguistic theory
  - Primary role of left hemisphere is language
- **Domain-general mechanisms**
  - Complex cognitive abilities arise from combinations of general, basic cognitive processes
  - General capacities support language acquisition and use
    - Symbolic representation, memory, pattern analysis
- Language is a secondary function of these general processes
  - Analytic-synthetic theory
  - Motor theory

Examples
- **Domain-specific**
  - Dual-process theory
    - Rule learning; **Rule**: past tense, add “ed” visit --> visited
    - Memory; **Memory**: irregular forms, go --> went
- **Domain-general**
  - Neural networks & computational models
    - Decentralized interconnected network of simple units (neurons)

Support for domain-specific mechanisms
- Universal grammar
- Language invention
- Werker & Tees, 1984

Psych 2220A Lecture 10

- Innate language acquisition device
  - Infants show language processing skills not likely due to experience

Support for domain-general mechanisms
- "Why, what weekend were you guys gonna be there?"
  - Statistical learning; Saffran et al 1996
- Areas that are more active during singing than speaking
  - Singing – humming (speaking)
- Shared neural networks between language and music; Besson & Schon, 2006

Language disorders
- Language disorders may tell us more about domain specificity of language
  - **Specific language impairment (SLI)**
  - **Dyslexia**
  - **Aphasia**

Specific language impairment (SLI)
- Abnormal grammar without obvious sensory/motor impairment or IQ deficit
- KE family in Birmingham: 16/30 members with severe SLI
- Genetic mutation?
  - FOXP2 is a regulatory gene on chromosome 7 that affects transcription of a variety of other genes
  - Mutations at FOXP2 affect neural development and communication in other species

Dyslexia
- Impairment in reading without apparent visual, motor, or intelligence deficits
- Developmental dyslexia
  - Evident when child learns to read
  - **Surface:** lexical, cannot pronounce words based on memory
  - **Deep:** phonological, cannot spell out words phonetically
- Lexical knowledge is equivalent to vocabulary; phonemes are the smallest meaningful units of sounds in a language

SLI & Dyslexia
- Impaired innate grammar mechanism or impaired information-processing ability?
- SLI: deficit in processing of rapid, sequential information, phonological salience? (Jonaisse & Seidenberg, 1998).
- Developmental dyslexia may be caused by deficits in phonological awareness (and mapping)
- Plasticity
  - Changes in behaviour can be cause or consequence of dyslexia
  - Differences in brain activation of beginning vs proficient readers

Psych 2220A Lecture 10

Aphasia

- Broca's aphasia
  - Affects production
- Wernicke's aphasia
  - Affects comprehension

Does Broca and Wernicke's aphasia support domain-specific mechanisms?

- Broca's aphasics also have difficulty comprehending complex sentences with potential ambiguity
  - *The apple that the boy is eating is red.*
  - *The boy that the girl is chasing is tall.*
- Difficulty primarily in linking disparate parts of sentences
- Singing helps speech production in some patients with Broca's aphasia

Reading

- Reading and writing are relatively recent inventions.
- Improbable that our brains have evolved to read writing.
- Visual word form area
  - Left midfusiform gyrus
  - More strongly activated when viewing words than other objects - including complex line drawings, digits, and unknown characters (Dehaene & Cohen 2011)
  - Localized activation reproducible across cultures, reading direction, types of script
  - But also activated in tasks that do not engage visual word form processing such as naming colours & pictures, repeating auditory words (Price & Devlin 2003)
- **Cortical recycling hypothesis:** VWFA becomes functionally specialized (but not completely specific) for words because it is competent at recognizing line configurations and junctions (Dehaene & Cohen 2011)

- The debate regarding language as domain-specific or domain-general is still ongoing
  - Existence of disorders specific to language suggests language is special
  - However, many of the deficits in these disorders can also be explained by general non-language specific impairments (e.g. mapping sounds to letters in dyslexics)
- Language is concretely represented in the brain
  - Language activates certain areas in the brain
  - Damage to specific language-related brain areas can cause relatively predictable language problems

Language processing in the brain

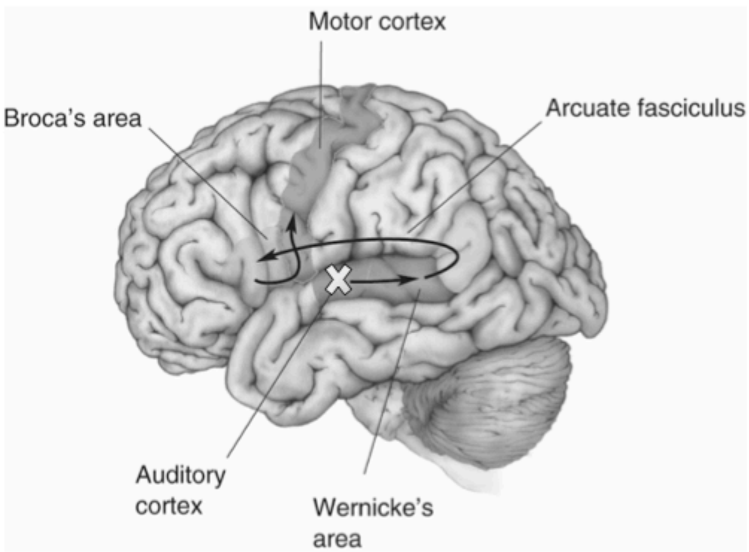
- We will discuss two ideas of how language is processed:
  - Wernicke-Geschwind model
  - Cognitive neurosciences approach

Psych 2220A Lecture 10

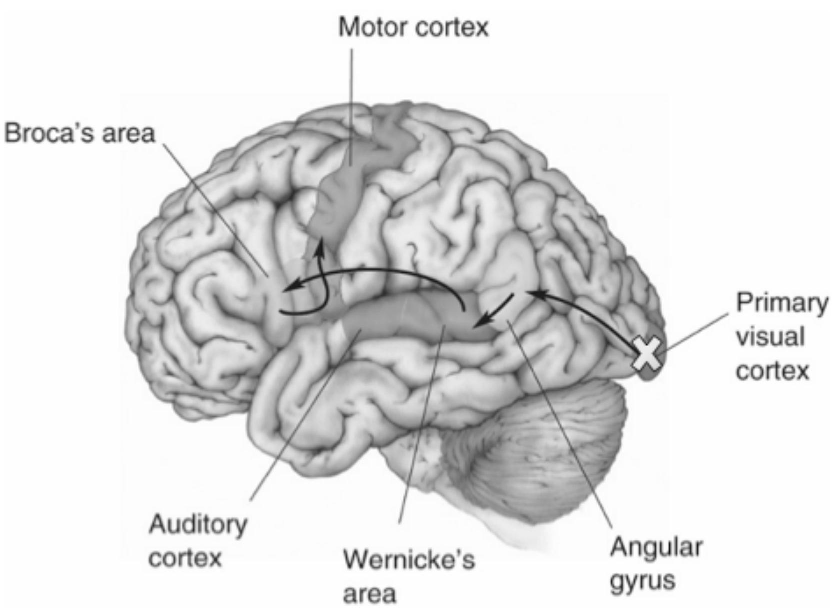
• Can you tell which one takes a domain-specific view and which one takes a domain-general view of language?

Wernicke-Geschwind model
• Serially-connected brain regions
• Localization centers for perception, comprehension, production
• Each region performs a specific function, thus damage to specific regions should have predictable effects

Joining in on a conversation



Reading Aloud



Psych 2220A Lecture 10

<u>Problems with the Wernicke-Geschwind model</u>
- Proposes localization of language perception, production, and comprehension
- What is involved in these abilities?
  - Attention
  - Planning
  - Inhibitory processes
  - Long-term memory
  - Short-term memory
  - Decoding noise (e.g. variations in voice/accents or handwriting)

<u>More problems with the Wernicke- Geschwind model</u>
- Words do not have to be transformed into pseudo- auditory response
  - Project to Broca's without stopping at angular gyrus
- Damage or leisons resulting in aphasia are not restricted to Broca's or Wernicke's area
  - Subcortical structures
- Cortical plasticity and recovery of function
- Comprehension deficits in Broca's aphasia and production deficits in Wernicke's aphasia
- Individual differences in locations of cortical language areas

<u>Mapping language-related areas</u>
- Penfield & Roberts 1959
  - Electrical stimulation of different brain areas to see which ones affected language
- Large individual differences
- Not limited to areas in Wernicke-Geschwind model

Decentralized language areas
- Cognitive neuroscience approach
  - Small, widespread, individual variation, multiple functions
- Attention
- Planning
- Inhibitory processes
- Long-term memory
- Short-term memory
- Decoding noise

	Wernicke-Geschwind model	**Cognitive neuroscience approach**
Level of analysis	Perception, production, comprehension	Phonology, syntax, semantics
Neural correlates	Connected large, homogenous language module areas	Combined activity of several, specialized, small, widely dispersed areas
Language specific?	Yes	No

Psych 2220A Lecture 10

• Language-like abilities and intelligence in an African Grey Parrot
http://www.youtube.com/watch?v=cO6XuVlcEO4

Brain organization
• Mammalian brain structure not necessary for complex cognitive processes and behaviours.

Localization of function
• Both Wernicke-Geschwind and cognitive neuroscience- based theories propose localization of language functions
• But they disagree on the degree and specificity of localization
• What are the advantages/disadvantages of smaller, but widespread and decentralized functional areas?
  • Isn't this redundant compared to larger, centralized areas for processing?
  • (is redundancy a bad thing?)
  • How and why would have it evolved this way?

Hippocampus and Memory
• Object recognition may be subserved primarily by perirhinal cortex (last week)
• Substantial evidence implicates hippocampus as important for spatial cognition

Hippocampus and Spatial Cognition
• Bilateral lesions of hippocampus disrupts performance on spatial tasks
• E.g. Morris Water Maze and Radial Arm Maze

Radial Arm Maze
• Hippocampus lesions disrupt both working memory and reference memory aspects of performance

Hippocampal place cells
• Selective response of hippocampal cells to specific locations
• Individual neurons have receptive place fields

Place cells
• By manipulating the navigational environment it has been show that place cell firing depends on where the rat thinks it is, rather than where it actually is

Entorhinal Grid Cells
• **Grid cells** – entorhinal neurons with an array of place fields
– Receptive fields produce a grid-like pattern
• Other cell types in entorhinal cortex
– Head-direction cells
– Border cells
• These cells may project to hippocampal place cells

Psych 2220A Lecture 10

Comparative studies

• Many species store food in multiple cache sites and retrieve food using spatial memory
• The hippocampus is larger, relative to the size of the telencephalon, in food- storing birds than in non-food-storing birds, over a wide range of telencephalon size.

Food caching

• Hippocampus lesions disrupt cache retrieval in food-storing birds
– Do not affect motivation to store food
– Do not affect colour discrimination task

Hippocampal function?

• Declarative memory?
• Spatial memory?
• Function still not clear
• Olton: important for reduction of interference
• Eichenbaum: relational memory

Navigation

• Spatial map approach replaced by spatial relationships

Relational Memory

• Hippocampus may function to encode relational components of memory across and within modalities
• Hippocampal 'place' cells can act as place cells, odour cells, or place-odour cells
• Hippocampus lesions disrupt odour-place discrimination
• Relational memory may be integral part of declarative or episodic memory (what- where-when)

Where Are Memories Stored?

• Each memory may be stored diffusely throughout the brain structures that were involved in its formation
• Some structures have particular roles in storage of memories
– Hippocampus – spatial location
– Perirhinal cortex – object recognition
– Mediodorsal nucleus – Korsakoff's symptoms
– Basal forebrain – Alzheimer's symptoms

Where Are Memories Stored?

• Damage to a variety of structures results in memory deficits
• **Inferotemporal cortex**
– Visual perception of objects
– Changes in activity seen with visual recall
• Amygdala
– Emotional learning
– Lesions of the amygdalae disrupt fear learning

Psych 2220A Lecture 10

• **Prefrontal cortex**
– Temporal order of events and working memory
– Tasks involving a series of responses
– Different part of prefrontal cortex may mediate different types of working memory
  • Some evidence from functional brain imaging studies
• **Cerebellum and striatum**
– Cerebellum
  • Stores memories of sensorimotor skills
– Striatum
  • Stimulus response associations
• Growing evidence for role in other memories as well

<u>Synaptic Mechanisms of Learning and Memory</u>
• Molecular events that appear to underlie learning and memory
– Hebb
  • Changes in synaptic efficiency are the basis of LTM
– Long-term potentiation (LTP)
  • Synapses are effectively made stronger by repeated stimulation
  • "fire together, wire together"

<u>Long-Term Potentiation (LTP)</u>
• LTP is consistent with the synaptic changes hypothesized by Hebb
– LTP can last for many weeks
– LTP only occurs if presynaptic firing is followed by postsynaptic firing
• Hebb's postulate for learning
– Co-occurrence of firings in pre- and postsynaptic neurons necessary for learning and memory

<u>LTP as a Neural Mechanism of Learning and Memory</u>
• Elicited by high frequency electrical stimulation of presynaptic neuron; mimics normal neural activity
• LTP effects are greatest in brain areas involved in learning and memory
• Learning can produce LTP-like changes
• Drugs that impact learning often have parallel effects on LTP
• Much indirect evidence supports a role for LTP in learning and memory
• LTP can be viewed as a three-part process:
– Induction (learning)
– Maintenance (memory)
– Expression (recall)

<u>Induction of LTP: Learning</u>
• Most commonly studied where NMDA glutamate receptors are prominent
• NMDA receptors do not respond maximally unless glutamate binds **and** the neuron is already partially depolarized
• Ca2+ channels do not open fully unless both conditions are met

Psych 2220A Lecture 10

• Ca2+ influx only occurs if there is the co-occurrence that is needed for LTP, leading to the binding of glutamate at an NMDA receptor that is already depolarized
• Ca2+ influx may activate protein kinases that induces changes causing LTP

Maintenance and Expression of LTP: Storage and Recall
• Pre- and postsynaptic changes
• LTP is only seen in synapses where it was induced
• Protein-synthesis (structural changes) underlies long-term changes
• LTP begins in the postsynaptic neuron, which signals the presynaptic neuron
• Astrocytes (not just neurons) also involved in LTP
• How are presynaptic and postsynaptic changes coordinated?
• Nitric oxide synthesized in postsynaptic neurons in response to Ca2+ influx may diffuse back to presynaptic neurons
• Structural changes are now a well- established consequence of LTP

Variability of LTP
• Most LTP research has focused on NMDA-receptor-mediated LTP in the hippocampus, but LTP is mediated by different mechanisms elsewhere
• LTD (long-term depression) also exists
• Much of LTP and the neural basis of memory is still a mystery

Biopsychology of Memory
• Section 11.9
• Infantile amnesia
• Smart drugs

Infantile Amnesia
• We remember virtually nothing of infancy (explicit memory)
• However children do have implicit memories of infancy
– Children show skin conductance response to pre-school classmates even if they weren't explicitly recalled
– 3 year olds improve on incomplete pictures test

Smart drugs
• Variety of drugs that claim to be nootropics or cognitive enhancers
• Clinical data is weak in most cases
• Effects on performance may be due to attention rather than memory

Neural Mechanisms of Fear Conditioning
• Fear conditioning
– Pair a neutral stimulus (e.g., a tone) with an aversive stimulus (e.g., a shock)
– Present the tone later and the animal will show a conditioned fear response
• Usually a defensive behavior

Psych 2220A Lecture 10

Amygdala and Fear Conditioning
• Lesions of the amygdala block fear conditioning
• The amygdala receives input from all sensory systems
– Appears to be responsible for adding emotional significance to another stimulus
– Amygdala projects to brainstem regions that control emotional behavior output

Amygdala
• Can lead to behavioral response without cortical processing

Contextual Fear Conditioning and the Hippocampus
• Pair an aversive stimulus with the context instead of with a discrete stimulus
– Hippocampus is linked to spatial memory
– Effect of bilateral hippocampal lesions on contextual fear conditioning
  • Before training – prevents conditioning
  • Shortly after training – blocks retention of conditioning

Amygdala Complex and Fear Conditioning
• Current synthesis of findings indicates that the lateral amygdala is most critical in conditioned fear
In addition, conditioned fear is suppressed by the prefrontal cortex inhibiting the lateral amygdala
• The hippocampus mediates conditioned fear learning by informing the lateral amygdala about the context of the fear- related event

Learning and Drug Tolerance --> Text section 15.1, 15.2

Drug Tolerance
• Decreased sensitivity to a drug as a consequence of exposure to it
– Shift in the dose-response curve to right
• **Cross tolerance** – exposure to one drug can produce tolerance to similar drugs
– Example: alcohol and benzodiazepines
• Tolerance often develops to some effects and not others
• Metabolic
– Less drug is getting to the site of action
• Functional
– Decreased responsiveness at the site of action, fewer receptors, decreased efficiency of binding at receptors, receptors less responsive

Drug Withdrawal Effects and Physical Dependence
• Seen when drug use is terminated
• Symptoms are the opposite of the drug's effects
• Body has made changes to compensate for drug's presence – functions normally with the drug present
• Severity varies with drug and pattern of use

Psych 2220A Lecture 10

Role of Learning in Drug Tolerance
• Contingent drug tolerance
– Tolerance only develops to drug effects that are experienced
• Conditioned drug tolerance
– Maximal tolerance effects are seen in the environment in which a drug is usually taken
• Conditioned withdrawal effects
– Withdrawal elicited by drug-related cues

Conditioned Tolerance and Withdrawal
• Situational specificity of drug tolerance is well documented
• Addicts are more likely to overdose in unfamiliar surroundings
• Exteroceptive or interoceptive cues associated with drug-taking become conditioned stimuli that elicit conditioned compensatory responses, producing tolerance prior to drug use or withdrawal in the absence of the drug