**Innateness and Epigenesis**

Brain structure (physical) and function - innately pre-specified or developing in response to the environment (epigenesis).

Genes always interact with the environment, so a strong definition of innate is not useful. Instead, psychologists use it to mean ‘affected by factors intrinsic to the developing organism rather than external ones’.

**Genetic determinism** - pre-specified blueprint which imposes itself on the developing child; minimally affected by the environment.

**Epigenesis** - development occurs as a result of a child developing in an environment which affects the way in which the brain develops.

**Prenatal and postnatal brain development**

Human sequence of development is similar to other mammals - but the timescale is greater. This means:

(i) Prolonged period of postnatal development - so the brain develops outside of the limited womb environment.

(ii) More delayed development => larger relative volume of the later developing brain. i.e. cerebral cortex (cc) and prefrontal cortex (pfc) - the site of high level cognitive abilities (Milner, Fuster, Goldman-Rakic ...)

Significant that the part of the brain which undergoes most pre-natal development is that associated with high level cognitive abilities - executive function.

**Prenatal brain development**

Neurons are born (cell division), migrate (inside-out in the formation of the brain - newest cells in the cc) and differentiate: (i) branching and growing of dendrites to pick up signals from other neurons (ii) Myelination - if delayed, a delay in development can occur.

At the same time, regressive events occur; **Oppenheim** found that 20-50% of neurons die - errors in cell division, temporary neurons or surplus to requirements.

Very specific pattern of branching of dendrites and synapses is apparent in adults - 10^11 neurons, each with 10^3 connections. Useful connections remain; unused connections are pruned. The results of this process are not encoded in the human genome - not enough space.

**Postnatal brain development**

Most remarkable sign is the increase in size and complexity of the dendritic tree and synapses. **Huttenlocher** reports a steady increase in several regions of the cc - e.g. parts of the cortex have 50% more synapses at 12 m.o. pfc density of synapses peaks at 24 m.o. There is a decrease in the overall number of connections between nerve cells from age 1 onwards - causing the specialisation of function of areas of the cortex.

**Plasticity**

Inherent property of the developing brain. If one area is damaged, another can take over. The brain is far less plastic in adults, when specialisation has already occurred.

**Increasing specialisation of structure**

One view of how this happens is ‘selectionism’ - **Changeux**

Decrease in synaptic connectivity is a function of a type of Darwinian selection. Frequently used neural circuits are preserved; infrequently used ones die out.

Evidence to support this view comes from **Lewkowicz and Turkewitz**. Newborn preference for bright or dim visual stimulus affected by prior auditory stimulus, suggesting cross-modal transfer, and **Stetri**, cross-modal transfer of tactile and visual information is present at 0;3 but not at 0;5.

**Johnson** - adult experience of synaesthesia may result from a failure to lose neural connectivity.

**Functional specialisation of the cortex: modules**

Neuropsychology has found that the majority of adults have similar perceptual, motor and cognitive functions associated with the same regions of the cortex. These functional units are known as modules.

**Innate modularity vs modularisation**

**Neural modules** - physical structural units of brain.

**Cognitive modules** - hypothetical constructs which may not exist.

[Module in these notes now = cognitive module]

**Fodor** - innately specified modularity

**Karmiloff-Smith** - modularisation.

**Fodor**

Nativist standpoint. Innate capacity to process information is present. Environment influences only over the course of evolutionary time (phylogenetic) - not over the life of a single individual (ontogenetic).

Model of the mind with 3 major components:

- **Transducers** - organs which process sensory info.
- **Input systems** - specialised processing
- **Central systems** - higher cognitive functions such as problem solving, abstract thought ...

**Modules** = input systems. For Fodor, higher cognitive functions are not modular.
Input systems are at the heart of theory - they are:

(i) Domain specific - limited set of stimuli processed
(ii) Encapsulated - operate in isolation
(iii) Mandatory - processing is unconscious
(iv) Rapid - very efficient processing

Karmiloff-Smith

Accepts basic notion of encapsulated cognitive modules but argues they are not innate. If a system is more highly specified it lacks flexibility and creativity - seen in humans.

Instead proposes modules are a product of development. There are a few innately specified constraints - predispositions - which are epigenetic in origin. The mind therefore becomes modular as a result of development.

Wider research evidence for Fodor / Karmiloff-Smith

Rakic - differentiation can be explained in terms of a molecular and genetic specification - like all the other organs in the body - supports Fodor.

However, balance of evidence (O'Leary, Elman, Katz & Shatz, Johnson ...) suggests differentiation occurs as part of an epigenetic system - an interaction between molecular, genetic and environmental factors.

Petersen et al - PET - study of native English speaking adults. Found left visual cortex only responded to English words or pseudo words (not other patterns of letters). Argues it is implausible to suggest they are programmed to develop an area of the brain which only responds to letters grouped together into English (like) words.

Neville and Mills - ERP - processing of known words narrows to an area of the left temporal lobe when vocabulary reaches 200 (not age dependent).

Self-organisation

Is a process that could explain how gene->environment interaction proposed by Karmiloff-Smith and others operates. Occurs in all stages of brain development (Johnson, Keslo).

Self-organisation=structure emerging in response to a system's interactions with an environment. Karmiloff-Smith is therefore arguing that as modules are produced because of brain development, the brain is self-organising.

Hebb rule - two adjacent neurons that are repeatedly activated results in lower synaptic resistance between them, increasing the probability that activation in one will lead to activation in the other. Fits with the concept of 'selectionism'.

Other instances of self-organisation include snowflakes, clouds - complex patterns that appear 'designed' but follow simple rules in their construction; and neural networks.

Language

Johnson - the most 'biologically special' of all human abilities.

An instinct for language?

Pinker - arguments for the innateness of language. Does not use the universality of language as a reason - as universal systems are not necessarily innate in origin. Instead, his four arguments are:

(i) The existence of pidgins that turn into creoles in a single generation. Children could not have been exposed to grammar (pidgin lacks it) but 'cannot help' creating grammatically well-formed creoles from them. A similar argument applies to sign languages.

(ii) Poverty of input - children could not possibly figure out all possible sentences and grammatical rules from what they hear - yet they have no difficulty in forming increasingly complex constructions.

(iii) Use of auxiliary verbs is common to many languages - as if people independently produced morse code or typewriter keyboard layouts in the same way.

(iv) There is an identifiable seat for language in the brain - the left hemisphere in normally developing individuals.

Evidence from developmental cognitive neuroscience

Asks are there any areas that are critical for language? The debate is sometimes framed in terms of equipotentiality - i.e. left & right hemispheres have an equal potential for developing language. Evidence in favour of this would undermine nativist perspective.

In adults there are specialised regions which process language, but evidence from comparing fMRI imaging results of hearing and deaf participants (Neville et al) shows different parts and hemispheres process language.

Evidence suggests there are constraints on the organisation of the neural system that support language - but nature of, timing of, sensory & language experience affect where it develops.

Additional evidence comes from study of focal lesions in 3-9 y.o (Reilly et al):

(i) Functional recovery from a focal lesion is an ongoing process in childhood
(ii) Functions affected by the lesion are taken over by other areas of the cortex.

Factorial experimental design shows LHD and RHD children by age 9-10 have similar grammatical and narrative skills as each other and a control sample.

Stiles and Thal - longitudinal study of children < 6m.0. with lesions - language delays occur regardless of damaged hemisphere; most significant delay is in RHD cases (LH is where language usually develops).
Note that none of this rules out the pre-wiring of language - just that other areas are plastic enough to take the function over if needed.

Nativist perspective is therefore supported to some extent by cognitive neuroscience. But the fact that early damage can be recovered through plasticity suggests the general properties of brain structure and connectivity are suited to language, without need for pre-specification.

Therefore, in typical and atypical development, it looks as if cognitive modules are a product of development, supporting the view of Karmiloff-Smith and other epigenetic theorists.

**Emergence of specialised cortical function:**

*Prefrontal cortex example*

Pfc is 1/3rd of surface of the brain. Seat of higher cognitive functions *(Milner, Fuster, Goldman-Rakic)* - executive function. Shows the most prolonged period of postnatal development *(Huttenlocher)*. There is disagreement as to how its development relates to cognitive development.

**Relating structural change to cognitive development**

One approach is to try to see if the emergence of a particular cognitive ability relates to a change in brain structure.

Diamond and Goldman-Rakic - research with monkeys. They argue that the pfc between 6-12m.o. accounts for the transition to understanding object permanence and object retrieval (A not B) Piagetian tasks. This is because young monkeys < 9m.o. and adult monkeys with lesions to the DLPC fail the task.

In humans, evidence from EEG *(Fox and Bell)* show increased response from pfc correlate to ability to respond correctly.

Children with PKU (reduced dopamine levels) are also less effective on A not B and object permanence tasks - though to involve the pfc.

**Pfc and the acquisition of new skills**

*Thatcher* proposes an alternative perspective. The pfc plays a role in organising other areas of the cortex when new skills and knowledge are being acquired. As the new skill / knowledge is achieved, pfc involvement decreases and the newly specialised areas of the brain take over.

Support from *Johnson et al* - infants with localised damage to the cortex on a visual attention task. Only infants with damage to pfc impaired. However, in an adult, only those with damage to the parietal regions are impaired.

**Conclusion**

Bridging the gap between physical brain structure and function is a major challenge. Connectionist computer modelling is one other tool that is used to attempt to do this.