the other hand had unchanging rhythms. This therefore explains why not all shift workers experience negative effects and demonstrates an impressive ability of our biological rhythms to adapt to changing environmental circumstances.

**Lifespan Changes** –

**Infancy** – Babies only have two sleep stages, active and quiet. These cycles are short lasting only 50 to 60 minutes for the first 9 months (Jenni et al) At birth 50% of sleep is active with the infant falling straight into this stage.

**Childhood/Adeolescence** – At the age of 5 children resemble similar EEG patterns to adults but sleep for 12 hours a day. At this stage parasomnias become apparent. From 5-12 years of age sleep time drops between 9 and 12 hours; this includes 25% REM. Children sleep deeply indicating thalamocortical maturation. During puberty; sexual and pituitary hormones are released in bursts during SWS. REM decreases and total sleep time drops to 8-9 hours.

**Adulthood** – From 18-30 individuals experience less sleep and it is not relatively deep sleep. This results in 53% of adults experiencing daytime sleepiness. Women suffer a loss of hormones during the menopause stage (45-60 years) as do men during the andropause (45-60 years). As a result also suffering from effects of sleep deprivation, adults experience poorer quality of sleep. Sleep lasts about 7 hours, stage 4 is minimal and REM remains about 25%.

**Old Age** – Older people wake up about 6 times a night which encourages daytime napping. Older people may experience phase advance, REM decreases to about 20%; stage two to 60% and stage 4 to 5%. Borbely et al found that 60% of 65-83 year olds reported frequent daytime napping.

**Evaluation:**

- There are validity issues with parental reports of infants sleep patterns. Babies do not sleep through the night; their sleep patterns are short at 50-60 minutes resulting in a higher likelihood of night time arousal. When parents state their babies sleep through the night in actual fact they are not aware that their baby has roused during the night. As parents do not understand sleep patterns in infants it is clear there are validity (methodology) issues when it comes to reports in infant sleep cycle research.

- There are positive applications from research conducted on Infants sleep patterns. Babies predisposed to SIDS active sleep is seen as a higher risk. A study of babies who late died of SIDS found they were less likely to arouse from active sleep. (Kato) Active sleep is associated with more frequent episodes of heart irregularities and sleep apnea; they are potentially life threatening if the baby doesn’t wake. Therefore, knowing babies are more likely to die from SIDS if they do not arouse easily from active sleep has allowed a decrease in SIDS to occur resulting in positive applications from the research conducted.
Sleep Revision  

- demonstrating NREM is important to energy conservation. This therefore demonstrates a difference between REM and NREM sleep that has evolved over time.

- Research to support sleep as an evolutionary process more specifically energy conservation has been flawed by Capellini. Capellini et al argued previous research was flawed as the methods used to collect data on sleep in different animals were not standardised and therefore comparisons between species were meaningless. The study was shown to only focus on mammals as aquatic mammals have different sleep patterns and as a result the data cannot be generalised to different species. This therefore demonstrates flaws on the energy conservation hypothesis of sleep.

Sleep Disorders; Insomnia

Insomnia is a sleep disorder characterised by poor quality and or quantity of sleep, despite adequate opportunity to sleep. This leads to daytime functional impairment. Insomnia can be classified in a number of ways including:

**Transient**: inability to sleep lasts for less than one week (causes such as jet lag, noise etc)

**Acute**: problem lasts for less than one month (perhaps an issue with stress)

**Chronic**: inability to sleep persists for over one month and may be present for many years.

Insomnia can be categorised into primary and secondary insomnia:

Primary Insomnia is the difficulty initiating or maintaining sleep or non-restorative sleep for at least 1 month. Secondary Insomnia is caused by a direct secondary factor. Examples of such causes include sleep apnoea, restless legs syndrome (RLS), circadian rhythm disorders due to night shiftwork, and various medical, substance use, and emotional problems.

Brain chemicals such as neurotransmitters are a factor that can lead to insomnia. A reduction in the neurotransmitter GABA is associated with insomnia. The neurotransmitter is known to help brain regions shut down by reducing electrical activity, and several common sleeping pills work by helping nerve receptors link to the chemical more efficiently.

- However there are many methodological problems with sleep studies such as Smith et al's. Sleep studies have been criticised for lacking ecological validity because they are conducted in lab experiments. This means that participants would not sleep in their normal beds and this change in environment could affect sleeping patterns and researchers do not know the extent to which the results are affected. The results from those lab experiments cannot be generalised to normal sleep as a result as they lack mundane realism.

- It is important to distinguish between primary and secondary insomnia because of the implications for the treatments. If insomnia is a symptom of another disorder then it is important to treat the disorder rather than the insomnia. However for some cases it may make sense to treat the insomnia regardless for example does depression cause insomnia or does insomnia cause depression. A study of 15,000 Europeans found that insomnia more often preceded rather than followed cases of mood disorders. Therefore treating insomnia regardless of primary or secondary effects may be a useful treatment.
and homeostasis in the hypothalamus. Research has uncovered a link between this neurotransmitter and narcolepsy. (Sakuri) Normally there are 10000-20000 hypocretin-producing cells in the hypothalamus but in many narcoleptics a large number of these cells are missing, resulting in low levels of hypocretin.

- The role of hypocretin can be supported by evidence from narcoleptic dogs. They were found to have a mutation in a gene on chromosome 12. This mutation had the effect of disrupting the process of hypocretin (Lin et al.) These findings have been confirmed in humans; lower levels of hypocretin were found in the brain (Nishino et al.) This therefore supports the role of hypocretin as a factor affecting narcolepsy.

- There is evidence to suggest narcolepsy is not inherited. Mignot found that narcolepsy does not run in families and in cases where one twin has the disorder, it has not been found in the other twin. Therefore it could be more likely that lower levels of hypocretin are due to brain injury, infection, diet, stress or an autoimmune attack. This therefore suggests that narcolepsy is not an inherited sleep disorder and other factors contribute to the explanation.

- REDUCTIONIST- Only biological, other approaches need to be considered.

**Sleep Disorders; Sleep Walking**

Sleepwalking is considered a parasomnia. Parasomnias are a group of sleeping disorders which involve unusual activities during sleep. These may include:

**Incomplete Arousal**

Sleep walking is considered an arousal disorder; a person sleep walking is partly awake in the sense that they are engaged in activities normally associated with a waking state however they’re also asleep. They are in deep sleep; SWS, meaning it is very difficult to rouse them in this state. Brain activity recording show a mixture of delta waves showing SWS and higher frequency beta waves showing the awake state. So it looks as if the sleep walking occurs when a person in deep sleep is awakened but arousal of the brain is incomplete.

**Why Children?**

Many explanations have been offered to explain why sleepwalking is more common in childhood. One possibility is that it happens because children have more SWS than adults. A recent suggestion by Oliviero is that the system that normally inhibits motor activity in SWS is not sufficiently developed in children and it also may be underdeveloped in adults. This was demonstrated in a study reported by Oliviero that examined the motor excitability of adult sleepwalkers during wakefulness. Compared to normal controls, the sleepwalkers had signs of immaturity in the relevant neural circuits.

**Risk Factors**