The effects of shifting to and from Daylight Saving Time

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Abstract

Two times a year 1.5 billion people undergo a transition of a one-hour shift of their clocks. This happens in spring and fall and is called daylight saving time. The main purpose of introducing daylight saving time is energy saving. Daylight saving time disturbs the circadian clock and therefore it shifts the activity pattern. There are many effects of the clock shift, it increases acute myocardial infarcts, alertness on traffic and the sleep quality. In this thesis these effects of daylight saving time will be investigated and discussed.

It is concluded that daylight saving time has an impact on the incidence of acute myocardial infarct, the spring shift increases the incidence but the autumn shift decreases the incidence of acute myocardial infarcts. The number of traffic jams and car crashes seems to decrease when shifting to daylight saving time and increases when shifting back. Daylight saving time disturbs the sleep quality, sleep becomes less efficient and more fragmented. Daylight saving time does induce these effects as described above, however to investigate the effects of daylight saving time in more detail more research is necessary.

Table of contents

Introduction

Two times a year 1.5 billion people will be exposed to a one-hour shift of their clocks (Janszky and Ljung, 2008). In spring they will lose one hour and in fall they will gain one hour. These transitions were introduced so the energy consumption could be reduced. In spring, time shifts to Daylight Saving Time (DST) and in fall it shifts back to the original time.

Shifting one hour could disturb the circadian clock (Kantermann et al., 2007). The human circadian clock is an important mechanism to entrain to a Zeitgeber such as sunlight. Therefore the clock can inform us when to wake, digest, eat, sleep etc. Such rhythms can entrain to strong cues (e.g. light/dark cycle), but they are endogenous because they persist in constant conditions (e.g. constant light or darkness) (Roenneberg et al., 2007). A one-hour shift can disturb the circadian clock and can induce dramatic effects (Kantermann et al., 2007) such as increased myocardial infarcts. It is known that these effects can last for a week due to a one-hour shift (Janszky and Ljung, 2008).

In this thesis I want to investigate what the effects are of DST on the human body. I want to discuss the effects on Acute Myocardial Infarcts (AMI) and the effect on traffic jams and car crashes. Also the effect of DST on sleep quality will be discussed in this thesis. The hypothesis is that the number of AMI will increase when shifting into DST and decrease when shifting out of DST because of the loss and gain of one hour and that the number of car crashes and traffic jams will increase when shifting into DST and decrease when shifting out of DST. These effects may be due to sleep deprivation and sleep gain, respectively. As a consequence, the sleep quality will be more disrupted when shifting into DST compared to shifting back.

What is daylight saving time?

Daylight saving time is advancing the clock in spring for one hour till the end of October. These shifts are a yearly phenomenon. DST has been introduced due to the fact that the morning light was not used efficiently in summer. With the shift to DST there is more sunlight in the evening. So the advantage is that there is less need for artificial light in the evening e.g. candles and electric lighting.

In ancient times, people already adapted their time to the sunlight per day. An hour in their summer could have been 74 minutes, and an hour in their winter could have been 44 minutes. Back then these hours per day were measured by ancient water clocks (Harrison, 2013). However, in medieval times an hour was set to 60 minutes all year round. The main problem was that during winter sunset was rather early. Benjamin Franklin (1784) was the first one who came up with an idea to introduce DST. He said: "Early to bed, and early to rise, makes a man healthy, wealthy and wise" (Manser and Pickering, 2009). He wanted to introduce this so there was less need for candle lighting. A New Zealander, called George Venon Hudson, first introduced modern DST (George, 1993). He wanted this so he could collect insects after his shift-work. His idea was to shift the clock 2 hours.

Daylight saving time was first introduced in Germany in 1916 during World War 1. The reason was to conserve coal during wartime. Allies of Britain,

European neutrals followed soon. After World War 2 DST was abandoned. Europe introduced DST again between 1973-1985 because of the energy crisis of the seventies. Every year in Europe DST starts the last Sunday of March and it lasts until the last Sunday of October. This has been generalized for whole Europe in 1981 (Prerau, 2005).

Daylight saving time and acute myocardial infarcts

The incidence of Acute Myocardial Infarct (AMI) is highly correlated with the shift to and from DST (Janszky et al., 2012).

It is already known that, independently of DST, the highest incidence of AMI is on Monday for every week (Willich et al., 1994). So every week there is a higher chance of getting an AMI on Monday, there is even a difference between working and non-working subjects. Non-working subjects show no significant difference over a week, however the working subjects do show a significant difference over a week especially a peak on Mondays (*see figure 1)*.

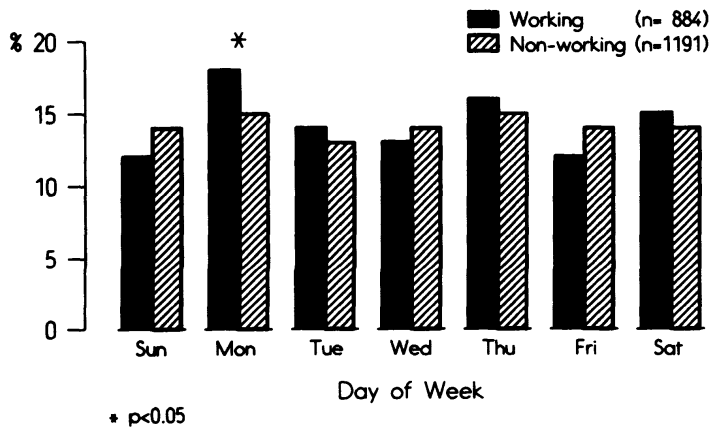

Fig.1 – AMI on work status
The incidence of AMI during a week between non-working (n=1191) and working subjects (n=884). The working subjects show a significant peak on Monday whereas the non-working subjects are rather equally distributed over the week (Willich et al., 1994).

These effects could be due to social jetlag. Social jetlag is a phenomenon where the midpoint of sleep on working days is shifted from the free days. This is due to the fact that in weekends people are more likely to get to bed later, whereas on working days they get up early. So the sleep duration between Sunday and Monday is often little, therefore the Mondays are the worst days to start working (Wittmann et al., 2006).

Hospitals in Sweden keep track of everyone who is diagnosed with AMI. All the information is stored in the national Swedish Myocardial Infarction Register. This registry provides high quality information about AMI since 1987. Janzsky et al (2008) used this register to investigate what the effect is of DST on AMI. DST could be seen as a kind of large-scale natural experiment. On top of the weekly recurrence of early activity on Mondays, an extra hour is imposed and in fall an hour is lost. The incidence of AMI in the spring and autumn shift is shown in figure 2A and 2B. The incidence ratio is calculated by dividing the incidences of the seven days by the mean incidences of two weeks before and after the transition. The incidence for the first 3 workdays after the spring shift is significantly enlarged and the incidence for the whole week was increased by a factor of 1.051. In contrast to the autumn shift where the incidence decreased by a factor of 0.985, but only for the first weekday the incidence ratio differed

significantly (Janszky and Ljung, 2008). During these transitions the social jetlag is more altered due to the one-hour shifts.

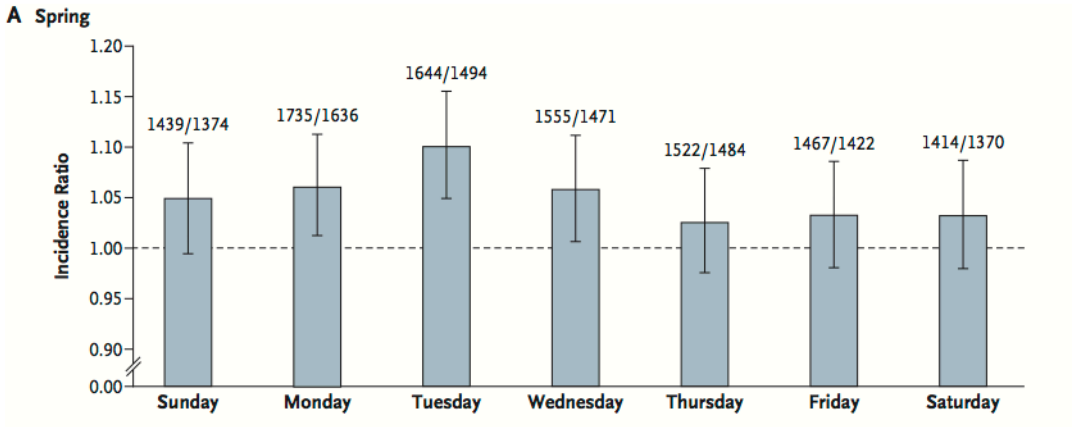

Fig. 2A – Spring shift
The shift to DST. On the days after the shift there is a higher chance of getting an AMI. There is a slightly higher peak on Tuesday (Janszky et al., 2008).

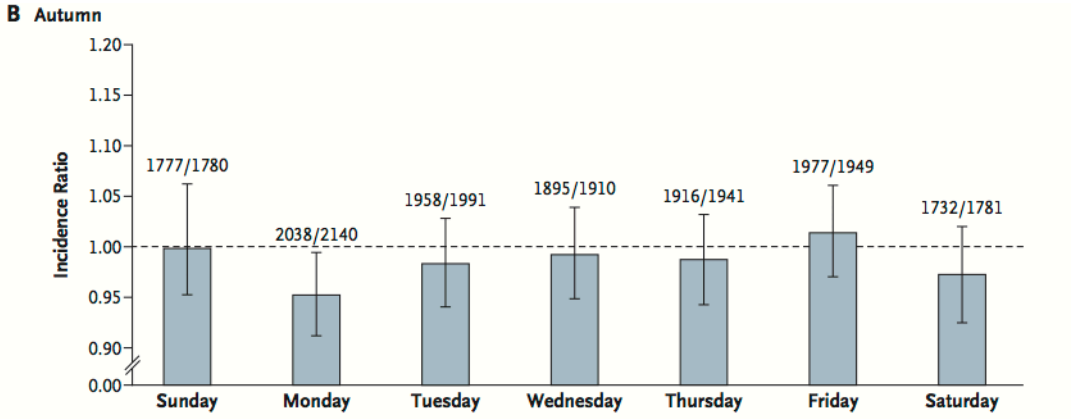

Fig. 2B – Autumn shift
The autumn shift is the shift from DST. There is a slightly lower incidence on Monday after the transition (Janszky et al., 2008).

In figure 2A & 2B the incidence ratio of AMI is plotted over the week while in figure 1 the distribution of getting an AMI is plotted over the week. In figure 3A & 3B the distribution of AMI is plotted for the transition into and out of DST. Figure 3A & 3B are transposed from figure 2A & 2B.

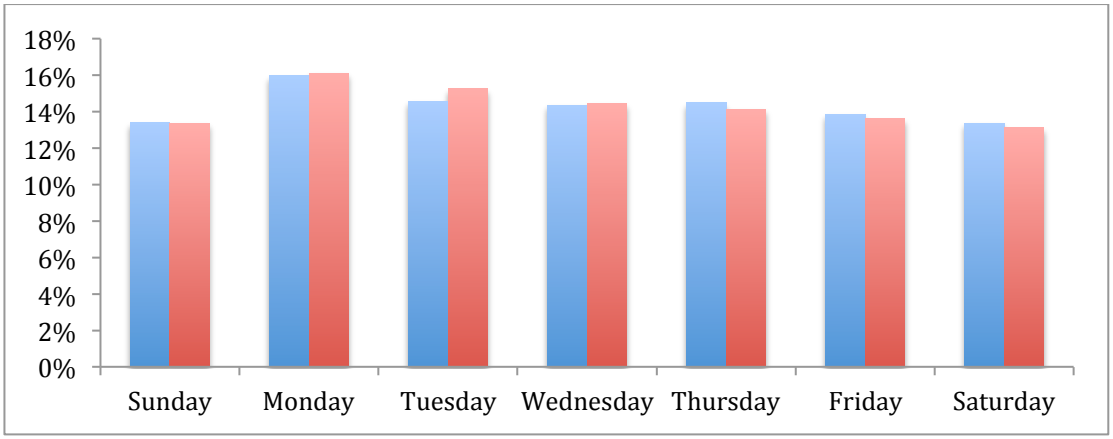

Fig. 3A – The distribution of AMI over the week during the spring shift
The distribution of getting an AMI is plotted over the week when shifting into DST. As shown in figure 2A on Tuesday is the highest peak and it decreases at the end of the week. The blue bars represent the average of AMIs for two weeks before the transition and the red bar represents the average of AMIs for two weeks after the transition.

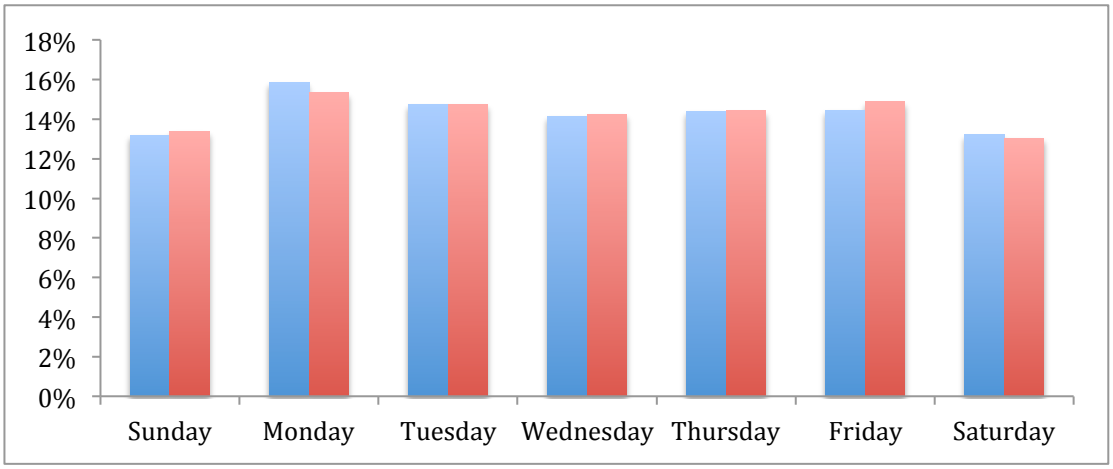

Fig. 3B – The distribution of AMI over the week during the autumn shift
The distribution of getting an AMI is plotted over the week when shifting out of DST. As shown in figure 2B on Monday the incidence is lower. The blue bars represent the average of AMIs for two weeks before the transition and the red bars represent the average of AMIs two weeks after the transition.

In figure 2A & 2B the incidence ratio is calculated and there is a difference between the days. However in figure 3A & 3B the distribution of AMIs is calculated over the week and there seems to be no significant difference between the days, except for the Tuesday after the transition into DST and for the Monday after the transition out of DST.

Physiologies of sleep deprivation and clock shifting

Physiology of sleep deprivation

But how could a shift of one hour induce a higher chance of getting an AMI? There are two effects of one-hour shifting the clock and that is sleep deprivation and a shift of sleep against the biological clock.

The autumn effect could be due to a gain of sleep and therefore it has a positive effect on the cardiovascular system. This is exactly the opposite for the

spring shift. The most plausible explanation for an increase in AMI in spring is that the sympathetic nervous system is more active during the shift and therefore increases cytokine-C levels (Meier-Ewert et al., 2004, Spiegel et al., 1999). Experimental sleep deprivation studies have shown that leukocytes (Dinges et al., 1994, Born et al., 1997) and interleukin (IL)-6 (Shearer et al., 2001) increase significantly during sleep deprivation. This means that sleep deprivation causes an inflammatory reaction and if this persist and even becomes chronic then it could lead to cardiovascular diseases (Meier-Ewert et al., 2004). C-reactive protein (CRP) is regulated by the cytokines leukocytes and interleukin. Therefore, CRP is the marker of increased levels of these leukocytes (Morley and Kushner, 1982).

Meier-Ewert et al (2004) investigated the effects of sleep deprivation on the CRP levels. In their experiment they made two experimental groups, a partial sleep deprived group (PSD) and a total sleep deprived group (TSD) (*see figure 4)*.

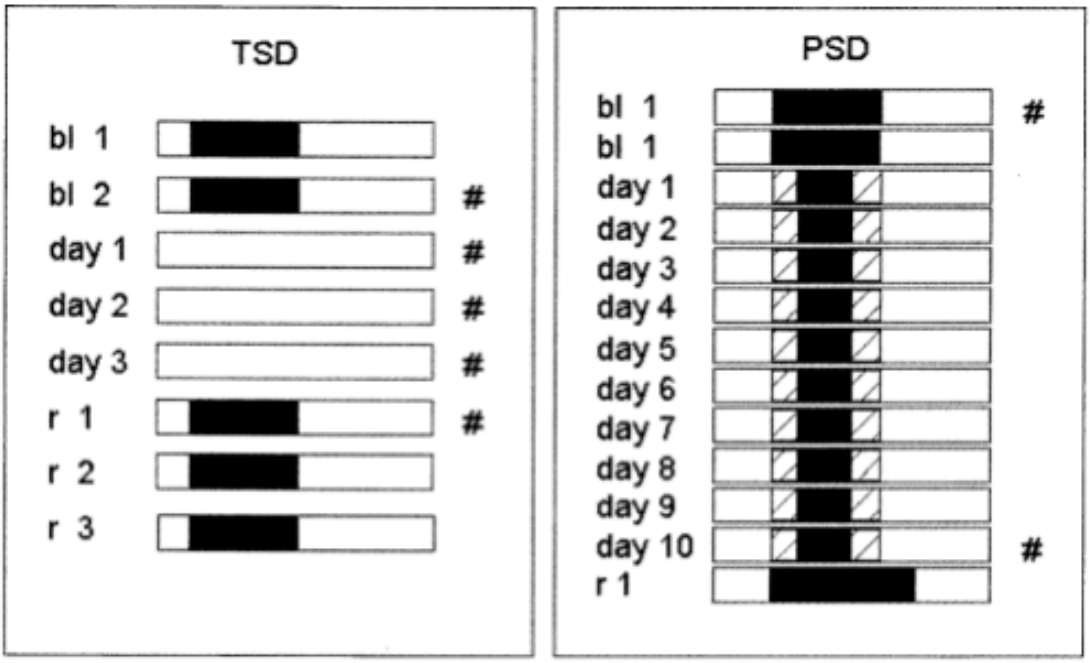

Fig. 4 – Schematic overview of the experimental setup
The total sleep deprived group is shown on the left and it underwent two baseline days, 3 total sleep deprived days and then 3 recovery days.
The partiel sleep deprived group is shown on the right and it underwent two baseline days, 10 partiel sleep deprived days and 1 recovery day (Meier-Ewert et al., 2004).

During this experiment CRP levels, blood pressure (systolic and diastolic) and heart rate have been measured. The results of this experiment are shown in figure 5A and 5B. CRP is significantly increased in both groups. Table 1 & 2 shows the results of blood pressure and heart rate are for both groups on the experimental days.

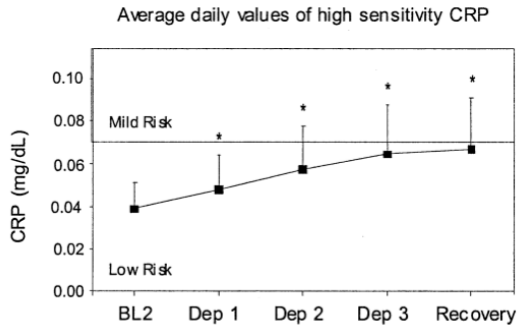

Fig. 5A – Average CRP values of TSD
The average CRP values of the TSD group. There is a significant increase of CRP levels till the recovery day (n=10) (Meier-Ewert et al., 2004).

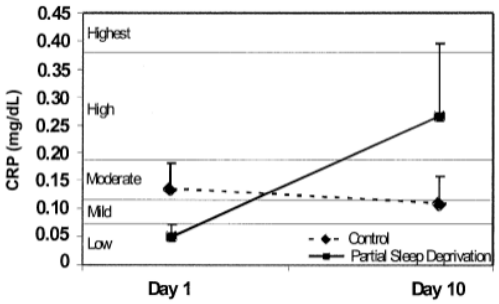

Fig. 5B – Average CRP values of PSD
The average CRP values of the PSD group. There is a significant increase of CRP levels between the first and last experimental day (n=4, squares; n=5, diamonds) (Meier-Ewert et al., 2004).

Baseline	BL (n = 8)	Dep 1 (n = 8)	Dep 2 (n = 7)	Dep 3 (n = 8)	Recovery 1 (n = 7)	Significance
Heart rate (beats/min)	63.0 ± 3.5	65.1 ± 3.7	70.1 ± 3.3	70.9 ± 2.8	69.7 ± 2.9	$F(4,7) = 3.3$, $p < 0.10$
Systolic BP (mm Hg)	121.1 ± 3.8	122.5 ± 2.7	125.4 ± 6.4	128.9 ± 3.0	130.0 ± 4.2	$F(4,7) = 313.3$, $p < 0.0001$
Diastolic BP (mm Hg)	68.1 ± 2.9	70.0 ± 2.4	72.0 ± 4.0	72.9 ± 2.8	71.4 ± 3.6	$F(4,7) = 3.4$, $p < 0.10$

Data are presented as the mean value ± SEM.
BL = baseline; BP = blood pressure; Dep = deprivation (day).

Table 1 – Heart rate and blood pressure during total sleep deprivation
The heart rate and blood pressure increases significantly with more sleep deprivation (Meier-Ewert et al., 2004).

Baseline	BL	Dep 10	Condition by Day Interaction	BL vs. Day 10
HR in sleep (beats/min)	75.0 ± 4.1	79.5 ± 7.3	$F(1,7) = 5.89$, $p < 0.05$	NS
HR in PSD (beats/min)	75.2 ± 2.6	97.0 ± 4.8		$t(7) = 4.59$, $p < 0.01$
SBP in sleep (mm Hg)	127.8 ± 6.2	137.3 ± 3.5	NS	
SBP in PSD (mm Hg)	113.6 ± 6.7	135.2 ± 9.4		
DBP in sleep (mm Hg)	73.5 ± 7.4	86 ± 2.9	NS	
DBP in PSD (mm Hg)	68.6 ± 3.0	85.4 ± 4.8		

Data are presented as the mean value ± SEM.
DBP = diastolic blood pressure; HR = heart rate; PSD = partial sleep deprivation; SBP = systolic blood pressure; other abbreviations as in Table 1.

Table 2 – Heart rate and blood pressure during partial sleep deprivation
The heart rate and blood pressure also increases significantly over more sleep deprivation (Meier-Ewert et al., 2004).

In both experimental groups (TSD, PSD) there is a significant increase in CRP which indicates a high concentration of plasma levels of leukocytes (Morley and Kushner, 1982) (*see figure 5A & 5B)*. Also the blood pressure and heart rate increased significantly over the experimental days (*see table 1 & 2).*

Physiology of clock shifting

The circadian clock entrains to light to a 24 hour cycle. When shifting to or from DST the shifts days become respectively 23 and 25 hours. How does this affect the physiology of the molecular circadian clock?

The SupraChiasmatic Nuclei (SCN) generates human circadian rhythmicity. The SCN entrains to the light-dark cycle and therefore our rhythm is based on a 24-hour cycle *(see figure 6)*. The SCN synchronises its rhythm to an external cue from the environment (Zeitgeber) such as sunlight (Golombek and Rosenstein, 2010).

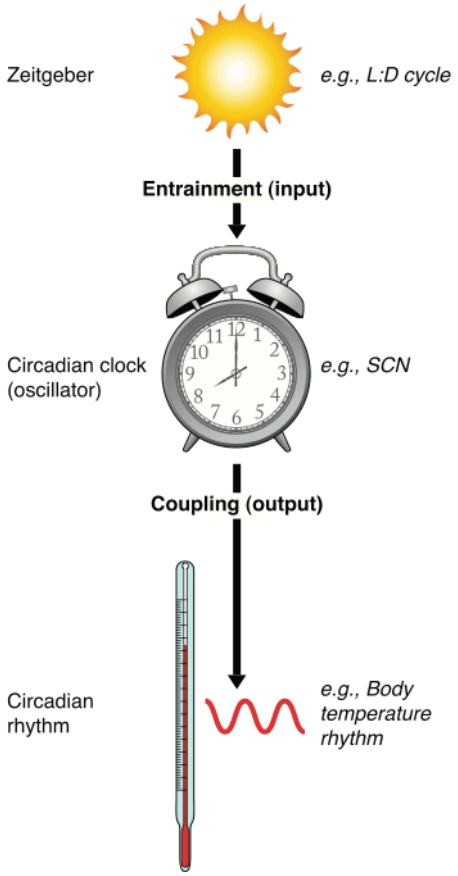

Fig. 6 – Entrainment
The human circadian clock receives light from the sun (Zeitgeber) and uses its rhythm for synchronising the circadian clock (SCN). The output of the clock governs overt biological rhythms such as body temperature rhythm (Golombek and Rosenstein et al., 2010).

Light reaches the SCN through the retinohypothalamic tract. Light enters the retina of the eye and activates photoreceptors in ganglion cells. These ganglion cells directly project on the SCN. The signal reaches the ventral part of the SCN by secreting the neurotransmitters glutamate and PACAP (pituitary adenyl- ate cyclase-activating polypeptide) (Golombek and Rosenstein, 2010) *(see figure 7)*.

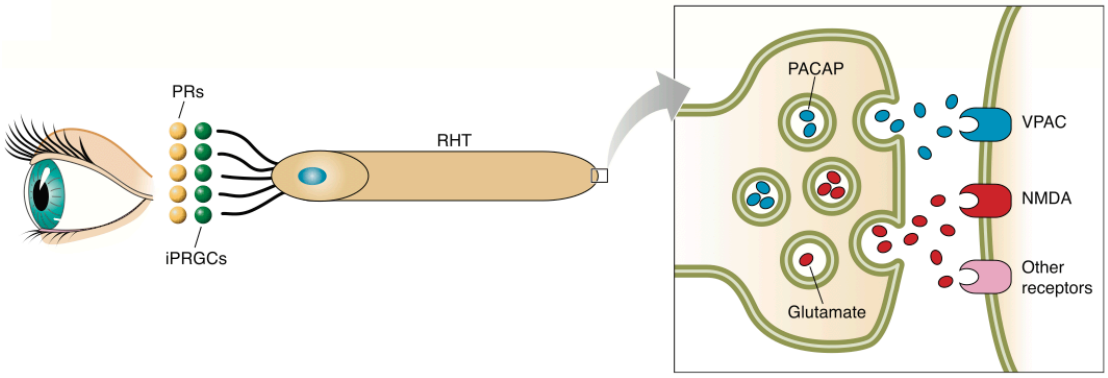

Fig. 7 – Retinohypothalamic tract
A schematic overview of the retinohypothalamic tract. Light enters the retina and acitivating photoreceptor ganglion cells (PRCGs), those cells innervating the ventral part of the SCN mediated by glutamate and PACAP (Golombek and Rosenstein et al., 2010).

The SCN synchronises the clock with the light-dark cycle. The circadian clock does this entrainment by clock genes. The clock contains of seven clock genes that transcripts over 24 hours *clock, bmal1, per1, per2, cry1, cry2 and per3.* These genes uses negative feedback loops in order to transcript each clock gene and therefore generating a circadian rhythm (Lowrey and Takahashi, 2004). After 24 hours it starts over again *(see figure 8A)*.

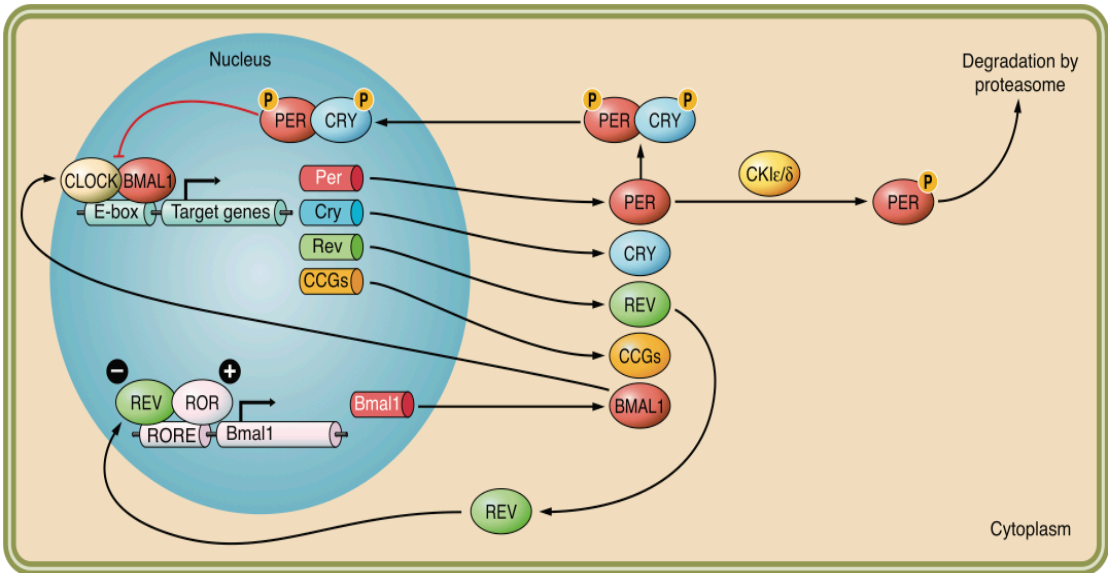

Fig. 8A – Schematic overview of the molecular clock
In mammals there are two core clock genes Clock and Bmal1. Clock and Bmal1 heterodimerize in the cytoplasm to form a complex that can activate genes containing an E-box promotor region. The PERs, CRYs and other proteins form a heteromultimeric complex that directly inhibit the transcriptional activity of CLOCK:BMAL1 complex, which indirectly lowers the Per and Cry RNA levels. Also another core clock gene is involved in the circadian clock namely REV-ERBα, this gene is also activated by the CLOCK:BMAL1 complex. REV-ERBα inhibits the transcription of Bmal1 and perhaps also other clock genes such as Cry1 and Clock. REV-ERBα binds to the retinoic acid-related orphan receptor response elements (RORE) of the Bmal1 promotor region. Therefore REV-ERBα indirectly suppresses its own transcription. Also the CRY-PER complex inhibits the transcription of REV-ERBα. So these processes annul REV-ERBα-mediated inhibition of Bmal1 such that BMAL1 accumulates at a certain time that it can heterodimerize again with Clock, and initiate a new transcription round (Golombek and Rosenstein et al., 2010).

The molecular clock generates a morning and an evening peak, induced by clock genes. These clock genes are *per1, per2, cry1, and cry2. Cry1 and per1* are responsible for the morning peak, whereas *Cry2 and per2* are responsible for the evening peak. This mechanism contains negative feedback loops (Daan et al., 2001) *(see figure 8B)*.

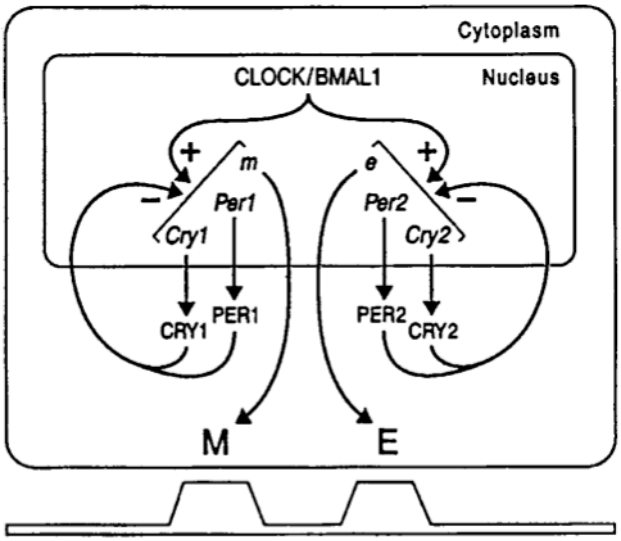

Fig – 8B Morning and Evening peaks
The clock genes *per1 and cry1* are activated by the CLOCK:BMAL1 complex in the morning. Therefore it generates a morning peak, the levels of CRY1 and PER1 decreases again due to a negative feedback loop. The same happens for the *per2 and cry2* clock genes. PER2 and CRY2 are therefore responsible for the evening peak (Daan et al.,2001).

The clock genes make the body aware of time, so it can for instance induce sleepiness in the evening and wakefulness in the morning. However it can also induce time when to digest and therefore knowing when to eat. The effect of DST is time is shifted for one hour, so the SCN generates all the rhythms one hour earlier or later. Therefore it can be concluded that the effects of DST on the physiology is shifting all the rhythms that are generated by the SCN. The molecular clock needs to adapt to one-hour shift and therefore it takes several days for adaptation.

Effects of DST on car crashes and traffic jams

Shifting to and from daylight saving time do not only affect the risk for acute myocardial infarct but it can also affect car crashes and traffic jams (Huang and Levinson, 2010). People suffer from a one-hour sleep deprivation on the first day after shifting to DST, therefore it can be expected that they may not function optimal in traffic and may cause more accidents. However, many studies found that the number of traffic accidents decreases after shifting to DST (Ward and White, 1994, Adams et al., 2005), while other studies found that shifting to DST increases car crashes due to increased sleepiness (Leger, 1994, Coren, 1996). Two studies even found that there was not a significant effect of DST on traffic accidents (Lambe and Cummings, 2000, Yeung et al., 1994). The decrease in traffic jams could be explained by the fact that during rush hour there is more sunlight available, however the increase in traffic accidents could be explained by the fact that due to sleep deprivation people are less alert.

All those studies looked at the whole day of traffic accidents. During a day the number of traffic accidents may differ from dawn to dusk. Taking the whole day into account it is not sufficient to understand the effects of DST of traffic accidents over a day (Huang and Levinson, 2010). Huang et al, (2010) investigated the number of car crashes in Minnesota per time stamp *(see figure 9A & 9B).* DST may change the traffic flow pattern near dawn and dusk, which can impact further crashes.

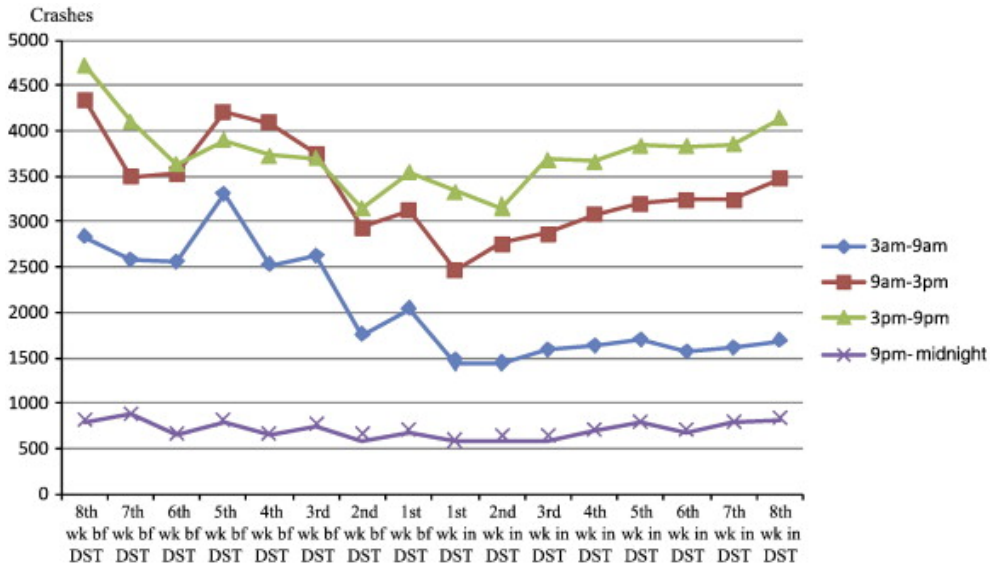

Fig. 9A– The number of crashes to DST
The number of crashes to DST for 8 weeks before till 8 weeks after the transition. The crashes are counted per 3-h bin, namely 3am-9am; 9am-3pm; 3pm-9pm and 9pm-midnight (Huang et al., 2010).

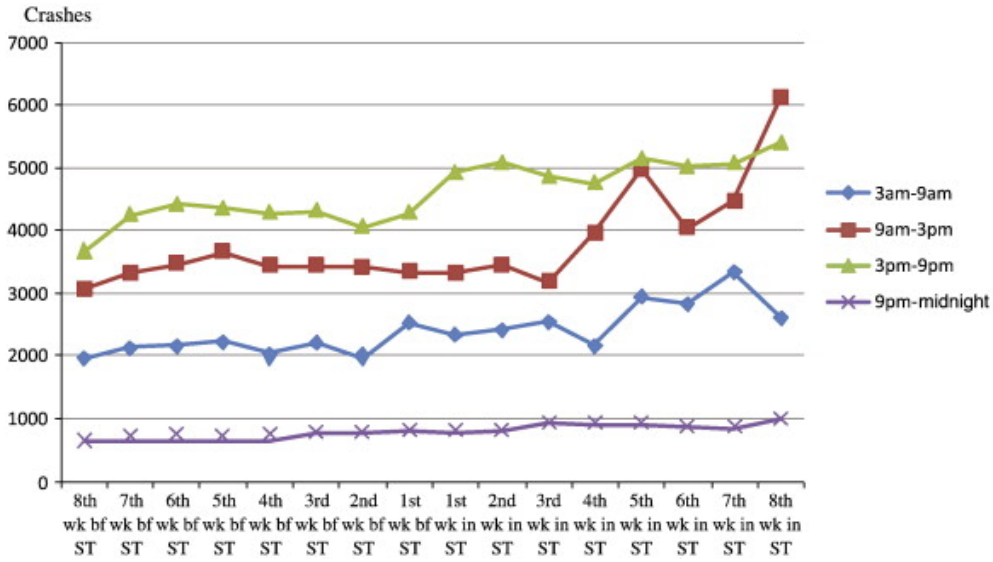

Fig. 9B – The number of crashes from DST
The number of crashes from DST for 8 weeks before till 8 weeks after the transition. The crashes are binned per 3-h interval, namely 3am-9am; 9am-3pm; 3pm-9pm and 9pm-midnight (Huang et al., 2010).

The number of crashes, when shifting to DST, decreases in the first week of DST (*see figure 9A).* However at the end of the 8 weeks after shifting to DST the number of car crashes slightly increases. However the average of the last 8 weeks in DST compared with the first 8 weeks, the number of crashes significantly decreases after the transition into DST (*see figure 9A).* However, the number of crashes, when shifting from DST, increases after the transition (*see figure 9B)*. This is especially the case for 3pm – 9pm, whereas 3am - 9am has lower crashes. Remarkable is that the increase of crashes starts at the 5th week after the transition, this could be due to the fact that around that period snow starts to fall in Minnesota.

For investigating how people function after the transition into DST it is interesting to investigate whether the number traffic accidents increases but also whether the number of traffic jams increases. Since there is no publication about traffic jams during the transition to or from DST, for this research traffic jam data were requested from the VID (Verkeers Informatie Dienst). Every 5 minutes the VID keeps track of the number and the length of the traffic jams in the Netherlands. The data covers the period from 2007 till 2012, and length and number of traffic jams was selected for the weeks before, during and after the transitions to and from DST. And the days of the weeks were compared to each other, so the Mondays, Tuesdays etc. The data of shifting to DST is shown in figure 10A, and the data of shifting out of DST is shown in figure 10B.

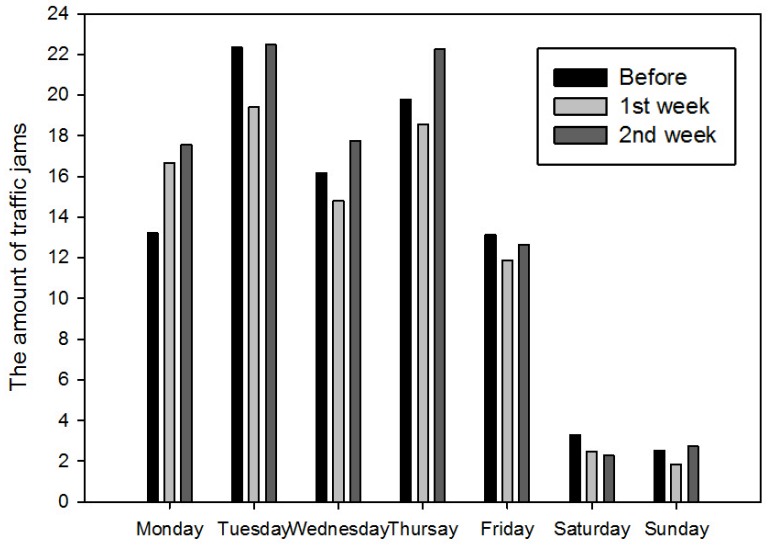

Fig. 10A – The number of traffic jams for the transition into DST
For each workday the number of traffic jams is plotted for the week before, during (1st) and after (2nd) the transition.

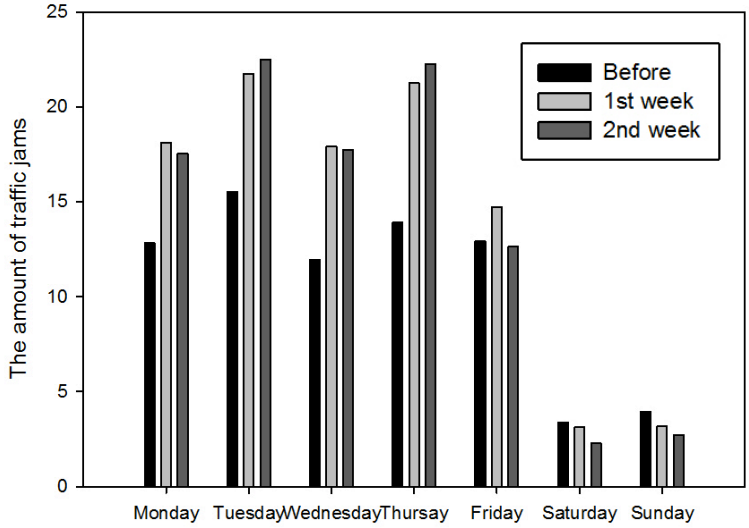

Fig. 10B – The number of traffic jams for the transition from DST
For each workday the number of traffic jams is plotted for the week before, during (1st) and after (2nd) the transition.

When shifting to DST the number of traffic jams decreases for the first week during DST compared with the week before the transition *(see figure 10A)*. This is especially the case on Tuesday. On Monday after the transition the number of traffic jams increases. The second week after the transition into DST the number of traffic jams is somewhat equal compared to the week before the transition (*see figure 10A)*.

The shift from DST is accompanied by an increase in the number of traffic jams *(see figure 10B)*. The increase in the number of traffic jams lasts for two weeks after the transition for every day, except for the Friday, Saturday and Sunday.

The decrease in the number of traffic jams and accidents could be explained due to the fact that after shifting to DST more light is present during rush hour. Therefore accidents could be avoided. However two weeks after the transition the number of traffic jams and accidents seems to shift back to the values before the transition. The same effect is seen when shifting out of DST. The traffic jams and accidents increases the first week after the transition but then decreases again. This could indicate that people have to get used to the transitions and that is why the number of traffic jams and accidents shifts back to the values before the transition.

Effects of DST on sleep quality

The effects of DST do not only include AMI and traffic accidents but it also affects the rest-activity cycle of humans (Lahti et al., 2006). The sleep efficiency and the sleep fragmentation is altered during the transitions (Lahti et al., 2006). Due to less sleep efficiency and increased fragmentation people are less rested.

Lahti et al, (2006) have recorded for nine subjects their sleep efficiency and sleep fragmentation during the transition into and from DST. They recorded their activity pattern by using an accelerometer or actiwatch. The nine subjects consist of 8 females and one male between 20-40 years old. The recordings for each transition were one week before and four weeks after. These recordings for

the transition out of DST were made in fall 2005, the recordings for the transition into DST was made in spring 2006.

These transitions affect different chronotypes. A chronotype is different for each individual and this can either be an early (morning) type or a late (evening) type (*see figure 11)*. The chronotype depends on the midpoint of sleep that can be measured from the MCTQ (Munich ChronoType Questionnaire)(Wittmann et al., 2006). The later the midpoint of sleep is the later the chronotype is. The earlier the midpoint of sleep is, the earlier the chronotype is.

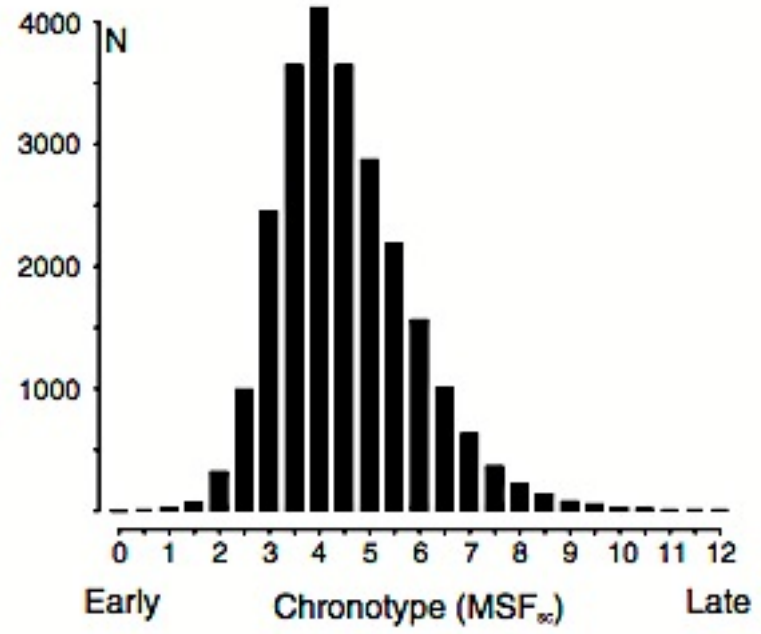

Fig. 11 – Chronotypes
The chronotypes are distinguished by the midpoint of sleep on free days. A midpoint of sleep of 0 is at midnight. The later the midpoint of sleep, the later the chronotype (Wittmann et al., 2006).

For both transitions the results are shown in figure 12A and 12B. In both graphs sleep efficiency (SE) and fragmentation index (FI) are plotted. This has been done for morning type (Morn) and evening type (Eve) persons.

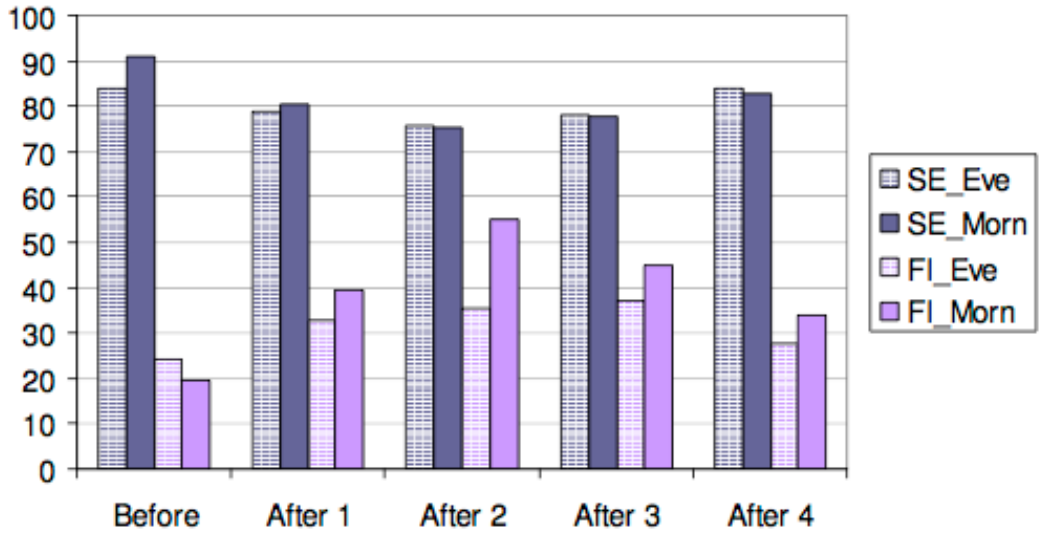

Fig. 12A – Sleep efficiency and fragmentation index in Spring
The SE is decreased but not significantly after the transition ($p<0.161$) for both morning and evening types. The fragmentation index is significantly increased after the transition ($p<0.019$) (Lahti et al., 2006).

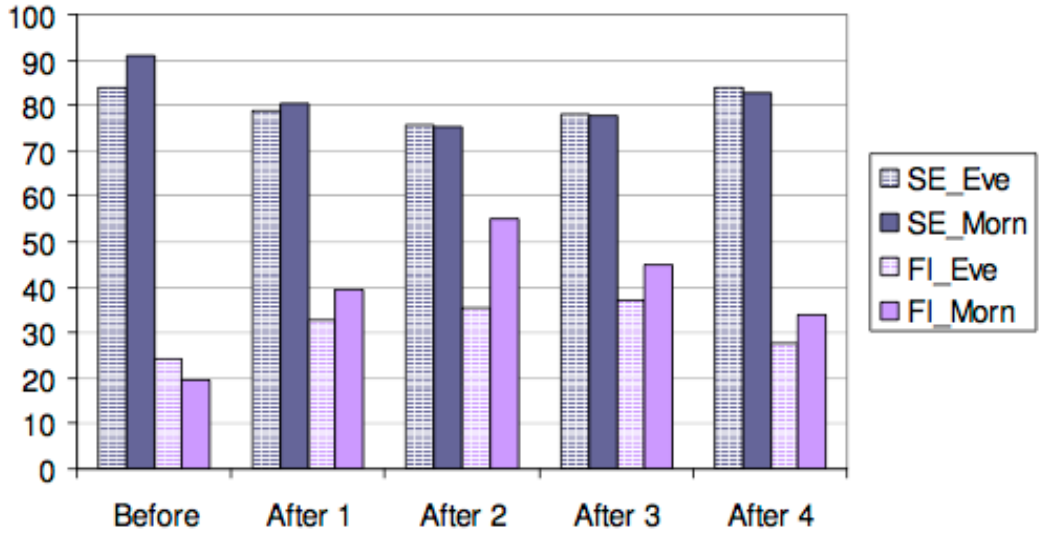

Fig. 12B – Sleep efficiency and fragmentation index in Fall
The SE is significantly decreased after the transition from DST ($p<0.006$), for both morning and evening time. The fragmentation index is significantly increased after the transition ($p<0.003$), also for both the morning and evening type (Lahti et al., 2010).

The sleep quality is clearly altered after the transition compared to the week before the transition *(see figure 12A & 12B)*. Sleep efficiency is only significantly decreased in the transition from DST *(see figure 12B)* in contrast to the transition into DST *(see figure 12A)*. There is no difference between the morning and the evening types, however the morning types have higher sleep efficiency on the week before the transition.

The fragmentation index is significantly higher in both transitions for at least 4 weeks after the transitions *(see figure 12A & 12B)*. This is especially the case for the morning types in both transitions.

Discussion & Conclusion

In this thesis the research question was to investigate the effects of DST on AMI, traffic jams, traffic accidents and activity-rest cycles. The effect of DST on AMI seems to be a negative effect (Janszky et al., 2012). The incidence of AMI increases during the transition into DST but decreases during the transition out of DST *(see figure 2A &2B)*. An explanation for this phenomenon could be that misalignment on cardiovascular health (Janszky and Ljung, 2008) is due to sleep deprivation. During the fall transition the sleep pattern gained one hour therefore the incidence of AMI decreases.

Nowadays people are chronically sleep deprived, they used to sleep for 9 hours but now the average sleep length is 7.5 hours (Spiegel et al., 1999). Therefore it is interesting to examine whether prolonged sleep has a positive effect on DST so it would be beneficial for people who are particularly susceptible for the shift on Monday. Monday is the day with the most stress and social jetlag due to the big difference between midpoint of sleep of Sunday and

Monday. It is not sufficient to prolong sleep duration on the transition day but also to reduce the social jetlag between free days and working days (Witte et al., 2005). Therefore the effects of social jetlag between Sunday and Monday during the transition such as AMI will be reduced.

One explanation of why the incidence of AMI increases during the transition into DST is because the activity of the sympathetic nervous system is increased *(see figure 5A & 5B)* (Meier-Ewert et al., 2004). This mechanism has been supported by the results of total sleep deprivation (TSD) and partial sleep deprivation (PSD) experiments. CRP levels, which are induced by leukocytes, are highly increased (60%), by both types of sleep deprivation without any signals of inflammation (Meier-Ewert et al., 2004). Normally CRP and leukocytes levels increases when there is an inflammation, but inflammation is not present during sleep deprivation. The results are consistent with other studies where the comparison is made between daytime sleepiness and sleep deprivation. The symptoms of sleep deprivation could be due to an increase in systemic IL-6 concentration (Vgontzas et al., 2000, Vgontzas et al., 1997). Also the molecular clock of the SCN has to adapt to the time shift. For synchronizing the molecular clock it takes several days due to the fact that the clock genes have to transcript at the proper time in 24 hours.

The effect of DST on traffic jams and traffic accidents is not in line with the hypothesis. As shown in figure 9A and 10A, where the transition into DST is shown for the traffic accidents and traffic jams, the number of accidents and traffic jams is decreasing just after the transition. For the traffic jams this happens on the Tuesday after the transition. It was thought that the decrease of the number of car accidents is due to more light during the rush hours. However, the car crashes have been measured per time unit over the transition for eight weeks whereas I looked at the average of all traffic jams over the transition for three weeks. Therefore the effect of DST per time unit over a day could not be seen for the number of traffic jams. When shifting from DST in fall the hypothesis was that the number of car crashes and traffic jams should decrease because of prolonged sleep. However, the number of car crashes and traffic jams is increasing. For the car crashes this happens 5 weeks after the transition *(see figure 9B)*. This could be due to the fact that around that time snow is beginning to fall in Minnesota, where the number of crashes has been measured. But a similar effect has been seen for the number of traffic jams in the Netherlands *(see figure 10B)*. The most plausible explanation for this phenomenon is that during the transition out of DST there is less light during rush hour because of the one-hour shift. It can be concluded that the effect of DST on car crashes and traffic jams, is not due to sleep deprivation but due to gain or loss of light during rush hour. However, if this is the case the number of car crashes and traffic jams should increase every week after the transition out of DST because because there is less light during rush hour. This has not been seen yet, so to investigate this effect of DST on car crashes and traffic jams more research is necessary.

The sleep quality is altered during the transitions into and out of DST (Lahti et al., 2006). As shown in figure 8A and 8B sleep efficiency (SE) and fragmentation index (FI) are altered during the transitions. During the transition into DST the SE is not significantly decreased however this is the case during the transition out of DST. In both transitions the FI is significantly increased, this increase indicates that the sleep pattern is disrupted. A distinction has been made

between morning and evening types. Morning types seem to have a higher FI than evening types. This is the case for both transitions whereas the SE shows no difference between both types. In this research only nine subjects have been used to measure the sleep quality. The sample size should be increase for a better understanding of the effects of DST on rest-activity cycles.

In this thesis my research question was to investigate what the effect of daylight saving time is on the incidence of AMI, on the number of car crashes and traffic jams and on the sleep quality. So the increase in AMI suggests an increase in stress levels induced by the transition into DST. Sleep quality suffers from both transitions equally and could be involved in increasing stress levels. This is especially the case for the FI. Car crashes and traffic jams decreases when shifting into DST and increases when shifting out of DST, this could be explained due to the gain of more light during the evening rush hour during the transition into DST. However more research has to be done to provide more detail on the causation of the effects of DST on traffic jams and accidents.

Acknowledgements

I would like to thank prof. dr. DGM Beersma for his input and supervision. Also I would like to thank Jeroen Wunnink for providing me the data from the Verkeers Informatie Dienst (VID) for the number of traffic jams in the Netherlands.

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