

Daylight Savings Time Transitions and the Incidence Rate of Unipolar Depressive Episodes

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Background: Daylight savings time transitions affect approximately 1.6 billion people worldwide. Prior studies have documented associations between daylight savings time transitions and adverse health outcomes, but it remains unknown whether they also cause an increase in the incidence rate of depressive episodes. This seems likely because daylight savings time transitions affect circadian rhythms, which are implicated in the etiology of depressive disorder. Therefore, we investigated the effects of daylight savings time transitions on the incidence rate of unipolar depressive episodes.

Methods: Using time series intervention analysis of nationwide data from the Danish Psychiatric Central Research Register from 1995 to 2012, we compared the observed trend in the incidence rate of hospital contacts for unipolar depressive episodes after the transitions to and from summer time to the predicted trend in the incidence rate.

Results: The analyses were based on 185,419 hospital contacts for unipolar depression and showed that the transition from summer time to standard time were associated with an 11% increase (95% CI = 7%, 15%) in the incidence rate of unipolar depressive episodes that dissipated over approximately 10 weeks. The transition from standard time to summer time was not associated with a parallel change in the incidence rate of unipolar depressive episodes.

Conclusion: This study shows that the transition from summer time to standard time was associated with an increase in the incidence rate of unipolar depressive episodes. Distress associated with the sudden advancement of sunset, marking the coming of a long period of short

days, may explain this finding. See video abstract at, <http://links.lww.com/EDE/B179>.

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Daylight savings time refers to the practice of shifting clocks 1 hour forward in the spring (from standard time to summer time) and shifting clocks 1 hour backwards in the fall (from summer time back to standard time). As the name indicates, the purpose of these time transitions is to maximize the exposure to daylight during the period in which people are active. Daylight savings time has been introduced in more than 70 countries worldwide and affects more than 1.6 billion people.

Because sunlight is the major factor controlling human circadian rhythms,^{1,2} and because the daylight savings time transitions interferes with the timing of sunlight, it has been studied whether the transitions disrupt circadian rhythms. This appears to be the case.^{3,4} Because the stability of circadian rhythms is essential to a range of biological processes in humans, prior studies have examined if the transitions have adverse health effects. Indeed, it has been demonstrated that daylight savings time transitions are associated with disturbed sleep,^{5–7} cardiovascular function,^{8–11} and possibly an increased tendency for fatal accidents.^{12,13}

It is plausible that daylight savings time transitions would also increase the incidence of mood disorder episodes because disruption of circadian rhythms has been implicated in the etiology of mood disorders.^{14–17} However, to our knowledge, the impact of daylight savings time transitions on mood disorders has only rarely been studied. Shapiro and colleagues¹⁸ found no association of the transitions with admissions for mood disorders (defined broadly to cover any diagnosis, “which may include an affective element”) based on their analysis of admission data from Edinburgh, Scotland. Similarly, Lahti and colleagues¹⁹ found no association of daylight savings time transitions with the incidence of manic episodes based on analysis of population-wide data from the Finnish Hospital Discharge Register. Notably, neither Shapiro et al.¹⁸ nor Lahti et al.¹⁹ studied the effect of daylight savings time transitions on unipolar depressive episodes alone. Thus, if the transitions only affect unipolar depression and not bipolar disorder, this may have gone undetected in prior studies. Therefore, we investigated the effect of daylight savings time transitions on the incidence of

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unipolar depressive episodes based on time series intervention analysis^{20,21} of longitudinal data from a nationwide hospital registry. To allow for comparison with results from prior studies, we conducted an identical time series intervention analysis for episodes of bipolar disorder.

METHODS

Source of Data

The study used data drawn from the Danish Psychiatric Central Research Register (DPCRR), which has been used for research purposes for decades.²² The DPCRR contains data on all patient contacts with psychiatric inpatient-, outpatient-, and psychiatric emergency room services from 1995 onwards for all individuals residing in—or visiting the Kingdom of Denmark (Denmark, Greenland, and The Faroe Islands).²³ In this period, the International Classification of Disease-10th revision (ICD-10)²⁴ was used as diagnostic reference. We based our analyses on primary diagnoses of unipolar depressive episodes (ICD-10 diagnoses: F32 and F33) and bipolar disorder (ICD-10 diagnoses: F30 and F31) registered in the DPCRR between January 1, 1995 and December 31, 2012 following acute contacts with psychiatric services. We therefore excluded all contacts that were labeled as “nonacute” or “unknown status,” as well as all outpatient contacts. This was done to avoid misclassification of contacts that were planned/scheduled before the daylight savings time transitions, but carried out and registered in the DPCRR after the transitions. However, acute contacts by individuals undergoing outpatient treatment were included in the study. Permission to use data from the DPCRR for the present study was granted by the State Serum Institute, Statistics Denmark, and the Danish Data Protection Agency. Ethical review board approval is not required for Danish register-based studies of this kind.

Establishing the Time Series

Using the full DPCRR data from 1995 to 2012, we generated two time series counting the total number of daily acute contacts for unipolar depressive episodes and bipolar disorder. The daily series exhibited large variances due to extreme day-to-day fluctuations and were rather noisy. As is common in the literature,^{25–27} we therefore aggregated the data to the next “natural” temporal level (weeks). This greatly reduced the variance and facilitated a simpler modeling of the series (the variance was reduced by a factor of 3 for depression—from 153 to 50—and close to 6 for bipolar disorder—from 34 to 6, while the means remained the same). After aggregation, each observation consisted of the average daily number of diagnoses in a given week of the year (subsequently, we refer to this as the “incidence rate”). This resulted in 940 weekly observations of average incidence rates. The time series were constructed such that each week began on a Monday, ensuring that each of the first postintervention weeks would begin immediately after the Sunday of the time transitions, which

occurred once in the fall and once in the spring of every year throughout the series (see Table 3 in eAppendix 1; <http://links.lww.com/EDE/B124> for the exact dates). To stabilize variance we finally logged both time series. We then conducted the intervention analysis using all of the Sundays with time transitions in the autumn as intervention dates for the stimulus of summer time to standard time transition and all of the Sundays with transitions in the spring as intervention dates for the stimulus of standard time to summer time. For both types of transitions, this yielded 18 intervention dates, all of which were included in the model simultaneously.

Statistical Analysis

The analysis followed the transfer function approach given in Cryer and Chan²⁰ and originally introduced by Box and Tiao.²¹ This approach generally views the time series under examination as consisting of two parts: One being the natural or unperturbed process, which represents how the time series would have progressed in the absence of any intervention, and the other being the perturbed process, which represents the actual values of the time series after the specified intervention has acted upon it. The key idea is that if the intervention timing is exogenous to the series itself, one can use preintervention data to estimate the unperturbed series and then obtain an unbiased estimate of the effect of the intervention by comparing these estimates to the observed postintervention trend. This comparison is performed by estimating the parameters of a transfer function of the intervention timing.

Figure 1 illustrates the basic principles of this approach using simulated data.

As the incidence rates in our data were repeatedly affected by the biannual time transitions, we had no clear preintervention period on which to estimate a model for the unperturbed series. Instead, we estimated it using the entire time series. We modeled the unperturbed series as an autoregressive integrated moving average process (ARIMA) and the effects of the time transitions (i.e., the difference between the unperturbed series and the perturbed process) as transfer functions of a binary variable that took on a value of one in the first week immediately after each of the transitions. As part of the Box and Tiao²¹ procedure, we inspected the time series for seasonality, using the Canova-Hansen and the Osborn-Chui-Smith-Birchhall test. Although neither of these indicated any substantial seasonality, we re-estimated a number of models correcting for any remaining seasonality with Fourier series and controls for substantive seasonal phenomena, such as weather and hours of daylight. None of the results deviated from those presented below. For a detailed description of these additional models and the transfer function intervention analysis method, see eAppendix 1 (<http://links.lww.com/EDE/B124>).

Having completed the Box and Tiao²¹ procedure, we performed a series of placebo-like tests for the incidence rates of unipolar depressive episodes. These tests involved

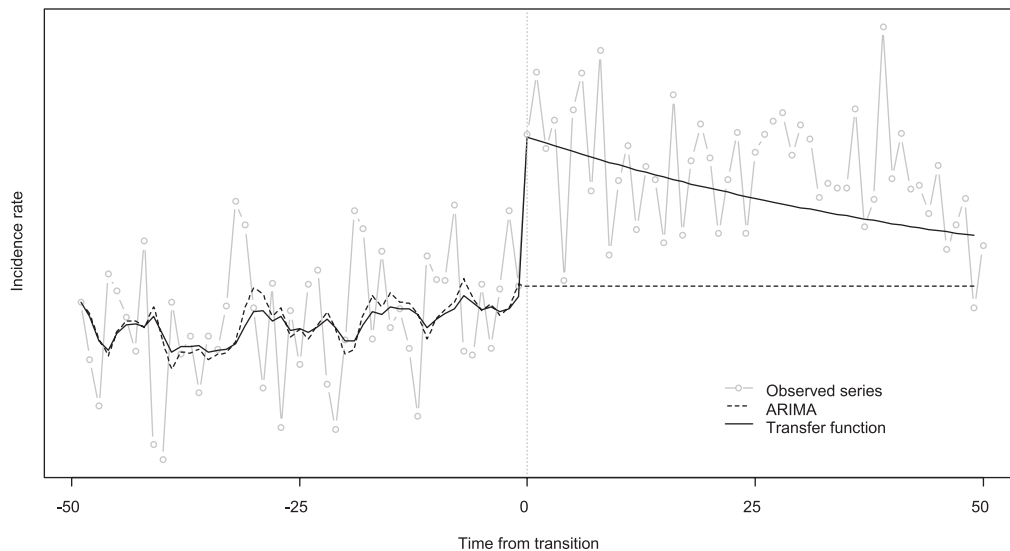


FIGURE 1. Illustration of dynamic forecasts of incidence rates based on simulated data. Using a window of 100 periods around a hypothetical time transition, this figure plots simulated rates of a given disorder of interest (e.g., unipolar depression) along with predictions from both a transfer function model and from a pure autoregressive integrated moving average (ARIMA) model. Up until the first period after the transition (time = 0), the predictions are one-step, meaning that the models use data from all relevant previous periods in predicting the value of the series in a given period. After the transition, the predictions become dynamic and are thus based solely on pretransition data and the parameters of the transfer function. The dashed line represents the expectations of what would have occurred had there not been a transition, and the solid line represent our expectations given that we know that a transition occurred. The difference between the solid and the dashed lines is the estimated effect of the time transition on the simulated incidence rate.

estimating the same intervention model as in the main analysis, but doing so with the intervention time moved to different dates than the actual time transitions. The logic behind this was simply that if the estimated difference right after the transition date was indeed a consequence of the transitions themselves, we should not be able to estimate a similar difference at a large number of other, unrelated dates. These tests are also described in detail in eAppendix 1 (<http://links.lww.com/EDE/B124>). The dataset used in the study and the code implementing the analyses outlined above are provided in eAppendix 2 (<http://links.lww.com/EDE/B125>) and eAppendix 3 (<http://links.lww.com/EDE/B126>), respectively.

RESULTS

Between January 1, 1995 and December 31, 2012, 2,719,311 contacts were made to Danish psychiatric hospital services. Of these, 1,271,061 were nonacute, of unknown status, or covered outpatient activity, leaving the time series used in the analysis with a total of 1,448,250 contacts. Of these acute contacts, 185,419 resulted in a primary diagnosis of a unipolar depressive episode, while 92,180 resulted in a primary diagnosis of bipolar disorder. Table 1 shows the diagnostic distribution within these two categories, and Figure 2 shows the mean daily number of cases of unipolar depressive episodes and bipolar disorder for each year in the study period (1995–2012).

Following the Box and Tiao²¹ procedure described above (see eAppendix 1 for further details; <http://links.lww.com/EDE/B124>), we used these data to estimate the following model:

$$\Delta^1 y_t = m_t + c_t + \phi_1 B^1 \Delta^1 y_t + \theta_1 B^1 \epsilon_t + \epsilon_t,$$

where y is the time series being modeled, t indexes time, ϵ is a white noise error term, Δ^1 denotes the first difference such that $\Delta^1 x_t = x_t - x_{t-1}$, and m_t and c_t are transfer functions of the daylight savings time interventions. B denotes the backshift operator, such that $B^k y_t = y_{t-k}$. m_t is specified as $m_t = \omega W_t + \delta B^1 m_t$, where W is a binary variable that takes on a value of 1 in the first week after each daylight savings time transition to standard time and zero everywhere else. c_t is the exact analog for transitions to daylight savings time, with $c_t = \lambda D_t + \pi B^1 c_t$ and D being a binary variable that takes on a value of 1 in the first week after each transition to daylight savings time. Below, we refer to W and D as moving average terms of order zero (MA0) and $B^1 m$ and $B^1 c$ as autoregressive terms of order 1 (AR1). The coefficients of the former give the immediate change in the incidence rates following a daylight savings time transition, and the coefficients of the latter give the decay in this change over time. The estimates are presented in Table 2.

The MA0 coefficient for standard time indicates that the transition from summer time to standard time was associated with a substantial 11% increase (95% CI = 7%, 15%) in the

TABLE 1. The Diagnostic Distribution Within the Unipolar Depression (A) and Bipolar Disorder (B) Categories in Denmark, 1995–2012

ICD-10 Diagnoses	Diagnoses per Day ^a	Min	1st q.	2nd q.	3rd q.	Max	%
A							
F32–F33 Total	28.2	2	18	27	37	87	
F32.0 Mild depressive episode	1.9	0	1	2	3	11	6.8
F32.1 Moderate depressive episode	6.0	0	3	6	8	22	21.2
F32.2 Severe depressive episode w/o psychotic symptoms	2.7	0	1	2	4	16	9.6
F32.3 Severe depressive episode w. psychotic symptoms	1.4	0	0	1	2	8	4.9
F32.8 Depressive episodes (other)	0.1	0	0	0	0	4	0.4
F32.9 Depressive episodes (unspecified)	2.4	0	1	2	4	15	8.4
F33.0 Recurrent depressive disorder (mild)	1.3	0	0	1	2	9	4.7
F33.1 Recurrent depressive disorder (moderate)	5.2	0	3	5	7	21	18.5
F33.2 Recurrent depressive disorder (severe w/o psychotic symptoms)	2.7	0	1	2	4	18	9.5
F33.3 Recurrent depressive disorder (severe w. psychotic symptoms)	1.4	0	0	1	2	11	5.0
F33.4 Recurrent depressive disorder (remission)	0.8	0	0	0	1	7	2.8
F33.8 Recurrent depressive disorder (other)	0.1	0	0	0	0	3	0.3
F33.9 Recurrent depressive disorder (unspecified)	1.8	0	0	1	3	14	6.3
B							
F30–F31 (total)	14.0	0	10	14	18	52	
F30.0 Hypomania	0.2	0	0	0	0	5	1.1
F30.1 Mania w/o psychotic symptoms	0.2	0	0	0	0	4	1.5
F30.2 Mania w. psychotic symptoms	0.3	0	0	0	0	4	1.9
F30.8 Mania (other)	0.0	0	0	0	0	2	0.1
F30.9 Mania (unspecified)	0.1	0	0	0	0	3	0.8
F31.0 Bipolar affective disorder (hypomania)	1.5	0	0	1	2	9	10.6
F31.1 Bipolar affective disorder (Mania w/o psychotic symptoms)	1.5	0	0	1	2	11	11.0
F31.2 Bipolar affective disorder (mania w. psychotic symptoms)	1.8	0	1	2	3	10	12.6
F31.3 Bipolar affective disorder (mild to mod. depression)	2.1	0	1	2	3	12	14.8
F31.4 Bipolar affective disorder (severe depression w/o psychotic symptoms)	1.0	0	0	1	2	11	6.8
F31.5 Bipolar affective disorder (severe depression w. psychotic symptoms)	0.5	0	0	0	1	6	3.6
F31.6 Bipolar affective disorder (mixed)	1.3	0	0	1	2	10	9.1
F31.7 Bipolar affective disorder (remission)	1.3	0	0	1	2	9	9.5
F31.8 Bipolar affective disorder (other)	0.1	0	0	0	0	4	1.0
F31.9 Bipolar affective disorder (unspecified)	2.0	0	1	2	3	12	14.4

^aThere are 6,575 days in each time series and “diagnoses per day” refer to the mean daily number of each diagnosis. Furthermore, the minimal (min), 1st quantile (1st q.), median (2nd q.), 3rd quantile (3rd q.), and maximal (max) number of daily diagnoses within the study period are reported. Finally, the proportion (%) of each diagnoses of the total number of contacts is reported. Note that the proportions do not sum up to 100% as a few contacts have not been registered with diagnostic specification (decimals) beyond the overall categorization (i.e., F30, F31, F32, F33).

incidence rate of hospital contacts for unipolar depressive episodes. Figure 3 shows that the effect dissipated over approximately 10 weeks.

In contrast, the estimated effect on the incidence rate of hospital contacts for bipolar disorder at this transition was negligible, and its 95% confidence interval stretched far both above and below zero. Similarly, we found small and imprecise estimates for the estimated effect of the transition from standard time to summer time on the incidence rates of both unipolar depressive episodes and bipolar disorder. The results of the “placebo” tests are shown in eAppendix 1 (<http://links.lww.com/EDE/B124>). Once

seasonality was accounted for, only a single placebo intervention yielded a positive estimate whose 95% confidence did not overlap zero, while another yielded a negative estimate with an upper bound on its confidence interval barely below zero. Given that other events, taking place at other points in time over the course of the year, may also influence incidence rates, it is not clear how many placebo interventions with confidence intervals not overlapping zero one should expect. However, with 95% confidence intervals, between one and two is very close to what we could expect due to pure chance, as that made up about 5.6% to 11.1% of the 18 placebo interventions that were carried out. To test

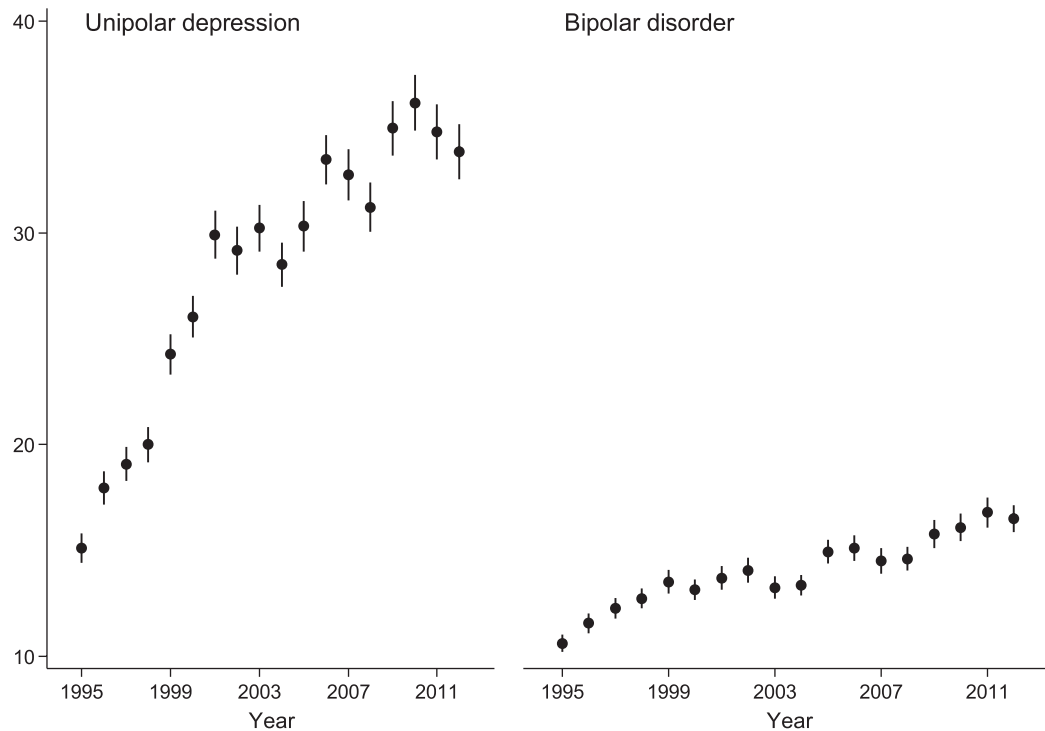


FIGURE 2. The mean daily number of unipolar depressive episodes and episodes of bipolar disorder for each year in the study period (1995–2012). The vertical bars represent 95% confidence intervals.

this more formally, we simulated distributions for each of the estimates and then ranked each draw by estimate size, allowing for rank-order tests of interventions. The results of these tests are presented in eAppendix 1 (<http://links.lww.com/EDE/B124>), and they strongly indicate that the positive change observed at the true intervention was not an artifact of noise. In sum, the placebo tests lend further credibility to the interpretation that the positive change in incidence rate was indeed due to the daylight savings time transitions.

DISCUSSION

By means of time series intervention analysis, this study compared the observed trends in the incidence rates of hospital contacts for unipolar depressive episodes and bipolar disorder after the daylight savings time transitions to and from summer time to the predicted trends in these incidence rates. Our results indicate that the transition from summer time to standard time is associated with an increase in the incidence of unipolar depressive episodes. The placebo tests substantiated that this effect was not merely an artifact of random noise in the time series.

To our knowledge, this is the first time that daylight savings time transitions have been associated with increased incidence of mood disorders. Prior studies of this association^{18,19} have not stratified on type of mood disorder (unipolar depression versus bipolar disorder), which may explain why the link between the transition from summer time to standard time and

increased incidence of unipolar depressive episodes has previously gone undetected.

The exact mechanism underlying the association between daylight savings time transition and the incidence of unipolar depressive episodes cannot be determined based on the data at hand. However, the fact that the association was only observed at the transition from summer time to standard time (and not from standard time to summer time) indicates that it is unlikely to be caused by the 1-hour time-shifts (and the resulting disruption of circadian rhythms) per se, but rather represents a specific consequence of the turning back of clocks in the fall.

So what happens at this particular transition that may explain the change in the incidence of depression? Figure 4 shows the timing of sunrise and sunset at the transition from summer time to standard time in Denmark on the last Sunday in October (European Union standard). On the Saturday leading up to the daylight savings time transition, the sun rises at approximately 8 am and sets at 6 pm. On the Sunday of the transition, the sun rises at approximately 7 am and sets at 5 pm. The fall daylight savings time transition thus entails increased exposure to sunlight in the morning and decreased exposure to sunlight in the evening. As depression can be treated effectively by bright light therapy in the morning, especially in individuals with a phase-delayed circadian misalignment,^{28–33} who are overrepresented among those with unipolar depression,^{17,28,34} we would expect increased exposure to sunlight in the morning to decrease the incidence of unipolar

depressive episodes. Instead, we observed the opposite and can therefore rule out this explanation.

This observation means that other mechanisms than those outlined above must be at play to explain the increased

TABLE 2. The Association of Daylight Saving Time Transitions with the Incidence Rate of Mood Disorders^a

	Dependent Variables in Logs 95% Confidence Intervals Below the Estimates	
	Unipolar Depression	Bipolar Disorder
	ARIMA (1, 1, 1)	ARIMA (1, 1, 1)
Summer time (AR1)	0.01 (−2.21, 2.23)	−0.31 (−1.53, 0.91)
Summer time (MA0)	0.02 (−0.04, 0.07)	−0.04 (−0.10, 0.02)
Standard time (AR1)	0.76 (0.64, 0.88)	−0.96 (−1.05, −0.87)
Standard time (MA0)	0.11 (0.07, 0.15)	−0.02 (−0.04, 0.01)
Obs.	939	939
Log likelihood	660.05	505.81
AIC	−1,308.1	−999.63

^aThe table contains the coefficients of the moving average (MA0) and autoregressive (AR1) terms from a series of transfer function models of order (1, 0) with autoregressive integrated moving average (ARIMA) specifications of the unperturbed processes. Each MA0 term multiplied by 100% provides an approximate estimate of the immediate increase in the incidence rate after the time transitions. The AR1 term shows how much of the estimated increase in each week that persisted into the following week. The order of the ARIMA model for each series is given in the column header and the coefficients are omitted from the table body. The first number in parenthesis following “ARIMA” gives the number of AR terms, the second gives the number of differences taken, and the third gives the number of MA terms. For each order of differencing an observation in the beginning of the time series is lost.
AIC indicates akaike information criterion.

incidence in unipolar depressive episodes at the transition from summer time to standard time. One possible explanation is that the sudden advancement of sunset from 6 pm to 5 pm (Figure 4), which in Denmark marks the coming of a long period of very short days, has a negative psychological impact on individuals prone to depression, and pushes them over the threshold to develop manifest depression. Furthermore, individuals having previously developed depression in the winter (as part of seasonal affective disorder) may perceive the transition from summer time to standard time as an omen of a new depression to come, which could have a depressogenic effect in itself. Some may argue that we should then observe an opposite effect, that is, a reduction in the incidence rate of unipolar depressive episodes, at the transition from standard time to summer time in the spring. However, the absence of such an effect could be explained by a valence-specific cognitive bias (i.e., an inclination to focus on negative events or emotions rather than positive ones), which is a well-known feature of unipolar depression.^{35–37}

Limitations of this study warrant mention. First and foremost, in time series intervention analysis, the interventions should ideally take the form of random shocks to ensure exogeneity to the time series itself. While the daylight savings time transitions are fixed such that the timing of the interventions could not have been influenced by any determinants of the incidence rates being studied, their timing is still known in advance. This means that our estimates could have been biased by people altering their behavior in anticipation of the transitions. However, if individuals develop unipolar depressive episodes in response to upcoming transition from summer time to standard time because of the actual transition, this would bias the estimated parameter towards zero, because we would then be comparing postintervention incidence rates to already increased

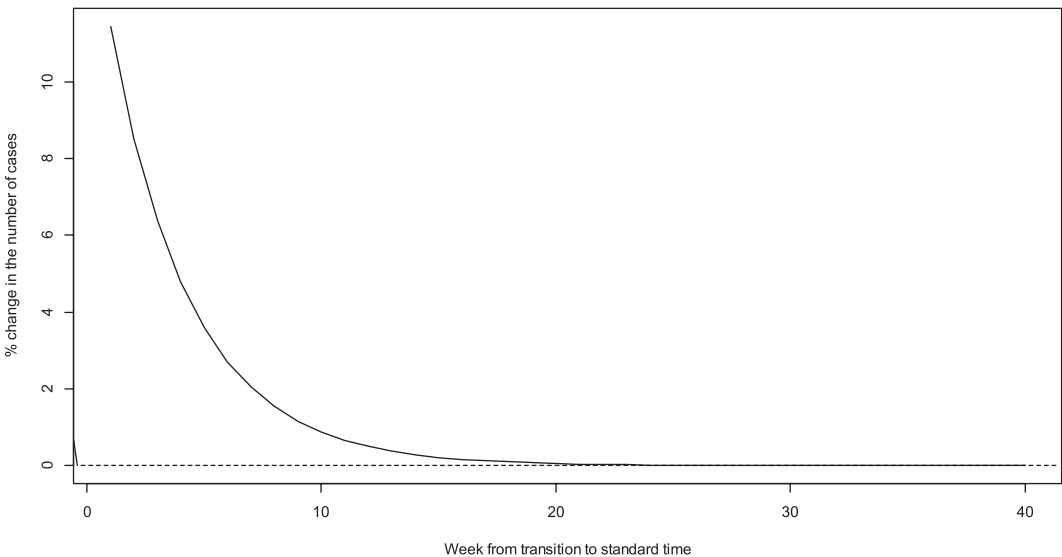
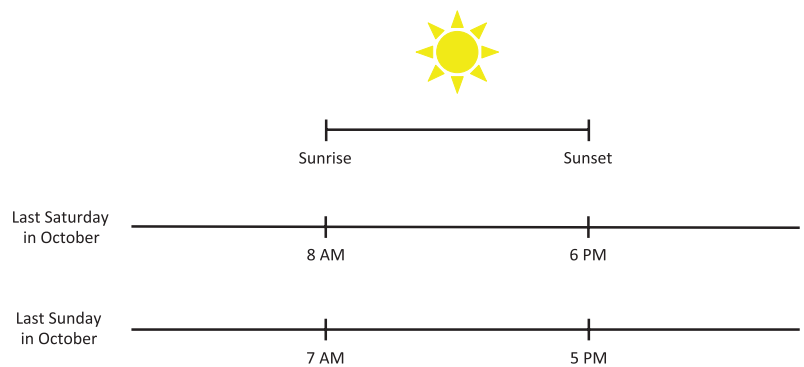


FIGURE 3. The estimated depressogenic effect of the transition from summer time to standard time. This figure shows the percent extra cases of unipolar depression in the weeks following the transition from summer time to standard time.

FIGURE 4. The timing of sunrise and sunset at the transition from summer time to standard time in Denmark on the last Sunday in October (European Union standard). Figure is available in color online.



preintervention incidence rates. If anything, the observed effect of the daylight savings time transition from summer time to standard time could therefore be considered a lower-bound estimate, and the actual effect of this transition on unipolar depression could be even more pronounced than reported here.

Another limitation is that our study is based on data from the Danish Psychiatric Central Research Register (DPCRR). Most importantly in this regard, the diagnoses recorded in the DPCRR are not necessarily based on formal research-based criteria as in most clinical studies, but are assigned as part of everyday clinical practice by the treating psychiatrist. While the DPCRR diagnoses are high in ecologic validity (i.e., real-world consequences), all diagnostic categories in the DPCRR have not been subjected to clinical validation.²³ However, the clinical validity of both unipolar depression³⁸ and bipolar disorder³⁹ diagnoses in the register has been established successfully. Furthermore, as reported elsewhere,^{40,41} the cases of unipolar depression and bipolar disorder considered in this study are not representative of all cases in Denmark. Some individuals suffering from unipolar depression or bipolar disorder (usually less severe cases) are treated by general practitioners or private practicing psychiatrists, who do not report diagnoses to the DPCRR.²³ Therefore, the results of this study are only fully representative for unipolar depression and bipolar disorder treated at psychiatric hospital services in the Kingdom of Denmark. At the same time, our exclusive focus on a specific type of outcome only implies that the consequences of daylight savings time transitions for unipolar depression are most likely even more pronounced in absolute terms as some of the affected individuals are not treated in hospitals.

In conclusion, this is to our knowledge the first study to show that the transition from summer time to standard time is associated with an increase in the incidence rate of unipolar depressive episodes. The fact that the association was only observed at the transition from summer time to standard time (and not also vice versa) indicates that it is unlikely to be caused by the 1-hour time-shift per se, but rather represents a specific consequence of the turning back of clocks in the fall. This is counterintuitive from a chronobiological perspective as the earlier sunrise after this transition should decrease

the likelihood of developing a unipolar depressive episode. Instead, we believe that the observed association is primarily related to the psychological distress associated with the sudden advancement of sunset from 6 pm to 5 pm, which marks the coming of winter and a long period of short days.

REFERENCES

1. Roenneberg T, Kantermann T, Juda M, Vetter C, Allebrandt KV. Light and the human circadian clock. *Handb Exp Pharmacol*. 2013;217:311–331.
2. Schulz P, Steimer T. Neurobiology of circadian systems. *CNS Drugs*. 2009;23(suppl 2):3–13.
3. Kantermann T, Juda M, Mewes M, Roenneberg T. The human circadian clock's seasonal adjustment is disrupted by daylight saving time. *Curr Biol*. 2007;17:1996–2000.
4. Monk TH, Folkard S. Adjusting to the changes to and from daylight saving time. *Nature*. 1976;261:688–689.
5. Schneider AM, Randler C. Daytime sleepiness during transition into daylight saving time in adolescents: are owls higher at risk? *Sleep Med*. 2009;10:1047–1050.
6. Tonetti L, Erbacci A, Fabbri M, Martoni M, Natale V. Effects of transitions into and out of daylight saving time on the quality of the sleep/wake cycle: an actigraphic study in healthy university students. *Chronobiol Int*. 2013;30:1218–1222.
7. Toth Quintilham MC, Adamowicz T, Pereira EF, Pedrazzoli M, Louzada FM. Does the transition into daylight saving time really cause partial sleep deprivation? *Ann Hum Biol*. 2014;41:554–560.
8. Jiddou MR, Pica M, Boura J, Qu L, Franklin BA. Incidence of myocardial infarction with shifts to and from daylight savings time. *Am J Cardiol*. 2013;111:631–635.
9. Sandhu A, Seth M, Gurm HS. Daylight savings time and myocardial infarction. *Open Heart*. 2014;1:e000019.
10. Janszky I, Ahnve S, Ljung R, et al. Daylight saving time shifts and incidence of acute myocardial infarction—Swedish Register of Information and Knowledge About Swedish Heart Intensive Care Admissions (RIKS-HIA). *Sleep Med*. 2012;13:237–242.
11. Janszky I, Ljung R. Shifts to and from daylight saving time and incidence of myocardial infarction. *N Engl J Med*. 2008;359:1966–1968.
12. Hicks GJ, Davis JW, Hicks RA. Fatal alcohol-related traffic crashes increase subsequent to changes to and from daylight savings time. *Percept Mot Skills*. 1998;86(3 pt 1):879–882.
13. Varughese J, Allen RP. Fatal accidents following changes in daylight savings time: the American experience. *Sleep Med*. 2001;2:31–36.
14. Pinho M, Sehmbe M, Cudney LE, et al. The association between biological rhythms, depression, and functioning in bipolar disorder: a large multi-center study. *Acta Psychiatr Scand*. 2016;133:102–108.
15. Malhi GS, Kuiper S. Chronobiology of mood disorders. *Acta Psychiatr Scand Suppl*. 2013;444:2–15.
16. Bechtel W. Circadian rhythms and mood disorders: are the phenomena and mechanisms causally related? *Front Psychiatry*. 2015;6:118.
17. Emens J, Lewy A, Kinzie JM, Arntz D, Rough J. Circadian misalignment in major depressive disorder. *Psychiatry Res*. 2009;168:259–261.

18. Shapiro CM, Blake F, Fossey E, Adams B. Daylight saving time in psychiatric illness. *J Affect Disord.* 1990;19:177–181.
19. Lahti TA, Haukka J, Lönnqvist J, Partonen T. Daylight saving time transitions and hospital treatments due to accidents or manic episodes. *BMC Public Health.* 2008;8:74.
20. Cryer J, Chan K. *Time Series Analysis: With Applications in R.* New York, NY: Springer; 2010.
21. Box GEP, Tiao GC. Intervention analysis with applications to economic and environmental problems. *J Amer Statist Assoc.* 1975;349:70–79.
22. Munk-Jørgensen P, Østergaard SD. Register-based studies of mental disorders. *Scand J Public Health.* 2011;39(7 suppl):170–174.
23. Mors O, Perto GP, Mortensen PB. The Danish Psychiatric Central Research Register. *Scand J Public Health.* 2011;39(7 suppl):54–57.
24. World Health Organization. The ICD-10 Classification of Mental and Behavioural Disorders. Diagnostic criteria for research. WHO: Geneva; 1993.
25. Finn, R. Weekly vs monthly forecasting in the supply chain. *Logist Transport Focus.* 2004;6:22–28.
26. Hotta LK, Valls Pereira PL, Otta R. Effect of outliers on forecasting temporally aggregated flow variables. *Sociedad de Estadística e Investigación Operativa Test.* 2004;13:371–402.
27. Jin Y, Brent W, Travis T, Matthew W. Forecasting with temporally aggregated demand signals in a retail supply chain. *J Bus Logist.* 2015;36:199–211.
28. Lewy AJ, Sack RL, Singer CM, White DM. The phase shift hypothesis for bright light's therapeutic mechanism of action: theoretical considerations and experimental evidence. *Psychopharmacol Bull.* 1987;23:349–353.
29. Lewy AJ, Bauer VK, Cutler NL, et al. Morning vs evening light treatment of patients with winter depression. *Arch Gen Psychiatry.* 1998;55:890–896.
30. Lievever R, Van Someren EJ, Nielen MM, Uitdehaag BM, Smit JH, Hoogendijk WJ. Bright light treatment in elderly patients with nonseasonal major depressive disorder: a randomized placebo-controlled trial. *Arch Gen Psychiatry.* 2011;68:61–70.
31. Sack RL, Lewy AJ, White DM, Singer CM, Fireman MJ, Vandiver R. Morning vs evening light treatment for winter depression. Evidence that the therapeutic effects of light are mediated by circadian phase shifts. *Arch Gen Psychiatry.* 1990;47:343–351.
32. Terman JS, Terman M, Schlager D, et al. Efficacy of brief, intense light exposure for treatment of winter depression. *Psychopharmacol Bull.* 1990;26:3–11.
33. Lam RW, Levitt AJ, Levitan RD, et al. Efficacy of bright light treatment, fluoxetine, and the combination in patients with nonseasonal major depressive disorder: a randomized clinical trial. *JAMA Psychiatry.* 2015:1–9.
34. Lewy AJ, Lefler BJ, Emens JS, Bauer VK. The circadian basis of winter depression. *Proc Natl Acad Sci U S A.* 2006;103:7414–7419.
35. Kilford EJ, Foulkes L, Potter R, Collishaw S, Thapar A, Rice F. Affective bias and current, past and future adolescent depression: a familial high risk study. *J Affect Disord.* 2015;174:265–271.
36. Beck AT. The evolution of the cognitive model of depression and its neurobiological correlates. *Am J Psychiatry.* 2008;165:969–977.
37. Soltani S, Newman K, Quigley L, Fernandez A, Dobson K, Sears C. Temporal changes in attention to sad and happy faces distinguish currently and remitted depressed individuals from never depressed individuals. *Psychiatry Res.* 2015;230:454–463.
38. Bock C, Bukh JD, Vinberg M, Gether U, Kessing LV. Validity of the diagnosis of a single depressive episode in a case register. *Clin Pract Epidemiol Ment Health.* 2009;5:4.
39. Kessing L. Validity of diagnoses and other clinical register data in patients with affective disorder. *Eur Psychiatry.* 1998;13:392–398.
40. Østergaard SD, Waltoft BL, Mortensen PB, Mors O. Environmental and familial risk factors for psychotic and non-psychotic severe depression. *J Affect Disord.* 2013;147:232–240.
41. Østergaard SD, Straszek S, Petrides G, et al. Risk factors for conversion from unipolar psychotic depression to bipolar disorder. *Bipolar Disord.* 2014;16:180–189.

Good morning,

The clock is ticking, literally, for the Legislative Assembly of Ontario to take action and abolish the “fall-back” practice where the province of Ontario will be shifting back to standard time.

While I appreciate some of the considerations raised with the Legislative Assembly of Ontario for the abolition of the standard time to take place, I strongly believe that the motion abolish the standard time is timely. The correlation of mental health (most specifically as it relates to “SAD”) and the shift back to standard time during the winter are already known facts (refers to a 2016 study on Daylight Savings Time Transitions and the Incidence Rate of Unipolar Depressive Episodes); adding the already taxing situation that Ontarians have been facing for the past eight months (a year that has already been difficult for most on the mental health front (and physical health as a matter of fact)) could have devastating effects.

With the current restrictions being into effects in most metropolitan regions of Ontario, the well being and mental health of the Ontario population is already being impacted, again, by COVID-19. While most of us are trying to remain optimistic, it may not appear that a vaccine will be available for some months now, indicating that we will be facing most if not all of the winter with a COVID-19 pandemic at its strongest – and the “cancellation” of the Holiday season family gathering(s) being almost inevitable at this time, the mental wellness of Ontarians (and more broadly of Canadians) will continue to be negatively impacted.

In the same vein as the Ontario government pressing Ontarians to be vaccinated against the flu this winter, abolishing the shifting back to standard time can be another step to help the health of Ontarians, and ultimately win the fight against COVID-19. Every action counts, whether the impact will be direct against the spread of COVID-19, or indirect in support the physical and mental health of the population.

Thank you for raising this motion, and I trust the Legislative Assembly of Ontario to do the right thing.

With thanks,

Jonathan, an-Ottawa-South-resident-who-also-happens-to-be-a-federal-public-servant-working-with-the-Ottawa-Gatineau-reality