# Longitudinal Study of Moderate Weight Change and Sleep-Disordered Breathing

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**Context** Excess body weight is positively associated with sleep-disordered breathing (SDB), a prevalent condition in the US general population. No large study has been conducted of the longitudinal association between SDB and change in weight.

**Objective** To measure the independent longitudinal association between weight change and change in SDB severity.

**Design** Population-based, prospective cohort study conducted from July 1989 to January 2000.

**Setting and Participants** Six hundred ninety randomly selected employed Wisconsin residents (mean age at baseline, 46 years; 56% male) who were evaluated twice at 4-year intervals for SDB.

**Main Outcome Measures** Percentage change in the apnea-hypopnea index (AHI; apnea events + hypopnea events per hour of sleep) and odds of developing moderate-to-severe SDB (defined by an AHI ≥15 events per hour of sleep), with respect to change in weight.

**Results** Relative to stable weight, a 10% weight gain predicted an approximate 32% (95% confidence interval [CI], 20%-45%) increase in the AHI. A 10% weight loss predicted a 26% (95% CI, 18%-34%) decrease in the AHI. A 10% increase in weight predicted a 6-fold (95% CI, 2.2-17.0) increase in the odds of developing moderate-to-severe SDB.

**Conclusions** Our data indicate that clinical and public health programs that result in even modest weight control are likely to be effective in managing SDB and reducing new occurrence of SDB.

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SLEEP-DISORDERED BREATHING (SDB), a condition characterized by repeated episodes of apnea and hypopnea events during sleep, is highly prevalent among adults in the United States and other Western countries.[1-6] The high prevalence has raised concerns of the public health burden of SDB because of demonstrated cross-sectional and retrospective associations between SDB and behavioral[7-15] and cardiovascular[16-23] morbidity. Recently, indicators of even mild SDB have been significantly related to hypertension, cardiovascular disease, and mortality in population-based prospective studies.[24-27] Although nightly use of continuous positive airway pressure can prevent apnea and hypopnea events, this therapy poses too high a life-long patient burden to be practical for mild or asymptomatic SDB. Thus, risk-factor modification may be the most feasible way to reduce the prevalence of SDB on a large scale.

Obesity, a strong correlate of SDB, is extremely prevalent in the United States and is increasing to epidemic proportions in the general population.[28-30] Obesity has been hypothesized to alter breathing during sleep via multiple mechanisms, including alteration of upper airway structure and function and disturbance of the relation between respiratory drive and load compensation.[31] If obesity is causally related to SDB, weight loss and the prevention of weight gain may offer the best hope for reducing the occurrence and severity of SDB and its related morbidity. Consequently, there is a pressing need to quantify the effect of weight change on SDB. Most previous studies linking obesity and SDB have used cross-sectional convenience samples of patients from sleep-disorders clinics[32-40] or cross-sectional population-based samples.[3-6,22,41-44] Several small studies, most lacking control groups, have found marked reductions in indicators of SDB following surgical[45-49] or diet-related weight loss[49-58] in obese patients. There is, however, a paucity of research relating weight gain to SDB incidence and progression, and little is known about the role of weight change in SDB across the spectrum of mild-to-severe SDB.

To date, there has been no large population-based study of the longitudinal association of change in weight and SDB. Longitudinal information is especially crucial in preclinical, asymptomatic people with mild-to-moderate SDB who are most likely to benefit from noninvasive and preventive weight control strategies. Our longitudinal study was designed to measure the degree to which weight gain is associated with increased SDB severity and

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**Table 1.** Summary of Key Variables for Eligible Baseline Participants Invited for a Follow-up Study and Participants in the Follow-up Study*

| Variable | Invited Participants Baseline (n = 948) | Follow-up Participants | |
|---|---|---|---|
| | | Baseline (n = 690) | Follow-up (n = 690) |
| Age, mean (SD), y | 45 (8) | 46 (7) | 50 (7) |
| Male, No. (%) | 542 (57) | 385 (56) | 385 (56) |
| AHI, events/hour | | | |
| Mean (SD) | 4.5 (9.8) | 4.1 (9.1) | 5.5 (10.8) |
| Median | 1.1 | 1.1 | 1.6 |
| No. (%) | | | |
| <5 | 755 (80) | 554 (80) | 495 (72) |
| 5-<15 | 120 (13) | 90 (13) | 127 (18) |
| ≥15 | 73 (8) | 46 (7) | 68 (10) |
| Weight, mean (SD), kg | 86 (20) | 85 (19) | 88 (20) |
| BMI, mean (SD), kg/m$^2$ | 29 (6) | 29 (6) | 30 (7) |
| Neck girth, mean (SD), cm | 38 (4) | 38 (4) | 38 (4) |
| Waist girth-to-hip girth ratio, mean (SD) | 0.89 (0.09) | 0.89 (0.09) | 0.89 (0.09) |
| Skinfold total, mean (SD), mm† | 80 (32) | 81 (32) | 106 (45) |
| Hypertensive, No. (%)‡ | 276 (29) | 195 (28) | 207 (30) |
| Smoker, No. (%) | 181 (19) | 120 (17) | 112 (16) |
| Alcohol, mean (SD), drinks/wk | 4 (7) | 4 (7) | 4 (5) |

*AHI indicates apnea-hypopnea index; BMI, body mass index.
†Sum of biceps, triceps, subscapular, and suprailiac.
‡Blood pressure ≥140/90 mm Hg or current use of antihypertensive medications.

weight loss with decreased SDB severity. This study uses a sample of participants from the Wisconsin Sleep Cohort Study (WSCS), a continuing prospective study of the natural history of SDB in middle-aged adults.[4]

## METHODS

### Participants

Participants in the WSCS are continuously recruited from a stratified random sample of adult men and women employed in a diverse set of job classifications at 5 State of Wisconsin agencies. A detailed description of the sample construction has been previously published.[4] Participants completed a baseline overnight protocol that included nocturnal polysomnography and other tests. Approximately 4 years later, baseline participants were invited for follow-up studies.

Criteria precluding WSCS participation included pregnancy, unstable or decompensated cardiopulmonary disease, airway cancers, and recent upper respiratory tract surgery. In addition, for this report, participants were excluded if, at baseline or follow-up, they had sleep studies with unusable physiologic parameters or less than 4 hours of sleep time (n=42), medical treatment for SDB (n=20), or physician-diagnosed stroke or cardiovascular disease (n=56). Finally, participants who experienced weight change in excess of 20% of baseline body weight (n=28) were excluded from the analyses.

As of January 2000, there were 948 eligible participants with a completed baseline study who were invited for a 4-year follow-up study. Of these, 690 completed a follow-up study (a 72.8% follow-up rate), 242 declined (25.5%), and 16 could not be contacted (undelivered mail or died, 1.7%). TABLE 1 provides baseline and follow-up key descriptive statistics for all eligible persons invited to participate in the follow-up study and for participants actually used for this analysis.

### Data Collection

Protocols and informed consent documents for WSCS were approved by the institutional review board of the University of Wisconsin Medical School, Madison. Baseline and 4-year follow-up overnight protocols were conducted at the University of Wisconsin General Clinical Research Center using rooms designed to mimic the decor of typical bedrooms. Participants arrived for overnight studies in the early evening. Sleep technicians obtained written informed consent, administered health history and lifestyle questionnaires, and measured blood pressure and body habitus parameters.

Body habitus measures, including height and weight without shoes; waist, neck, and hip girths; and biceps, triceps, subscapular, and suprailiac skinfolds, were measured using standard procedures.[59] Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters. Information on medical history, smoking, alcohol use, education, age, and other sociodemographic factors was obtained by interview and questionnaire.

Following body habitus assessment, technicians affixed polysomnography leads to participants and performed calibrations. An 18-channel polysomnography recording system (Polygraph model 78; Grass Instruments, Quincy, Mass) assessed sleep state, respiratory, and cardiac parameters. Sleep state parameters were determined by electroencephalography, electro-oculography, and chin electromyography. These leads were used to score sleep stage for each 30-second epoch of the polysomnographic record, using conventional criteria.[60] Measurement of arterial oxyhemoglobin saturation, oral and nasal airflow, nasal air pressure, and thoracic cage and abdominal respiratory motion were used to assess SDB events. Oxyhemoglobin saturation was recorded continuously using pulse oximetry (Ohmeda 3740, Englewood, Colo). Stalk-mounted thermocouples (ProTec, Hendersonville, Tenn) were used to detect oral and nasal airflow. A pressure transducer (Validyne Engineering Corp, Northridge, Calif) continuously measured air pressure at the nares via nasal prongs. Respiratory inductance plethysmography (Respitrace; Ambulatory Monitoring, Ardsley, NY) continuously recorded thoracic cage and abdominal excur-

sions. Sleep state and respiratory event scorings were performed by trained sleep technicians and reviewed by an expert polysomnographer.

Each 30-second epoch of the polysomnographic records was visually inspected and scored for abnormal breathing events. Cessation of airflow lasting 10 or more seconds defined an apnea event. A discernible reduction in the sum of thoracic cage plus abdomen respiratory inductance plethysmography amplitude associated with a 4% or greater reduction in oxyhemoglobin saturation defined a hypopnea event. The mean number of apnea events plus hypopnea events per hour of objectively measured sleep defined the apnea-hypopnea index (AHI), our summary parameter of SDB.

### Statistical Analysis

Descriptive and regression analyses were performed with SAS software, releases 6.12 and 8.00 (SAS Institute Inc, Cary, NC). Two types of models were used to measure the relation between weight change and change in SDB severity. Both approaches are detailed below.

Multiple linear regression models were used to assess the association between change in the AHI and weight change while controlling for potential confounding variables. These models were implemented by regressing the log of the ratio of follow-up AHI divided by baseline AHI (ie, $\log_e[AHI_2+1/AHI_1+1]$, the dependent variable) on the log of the ratio of follow-up weight divided by baseline weight (ie, $\log_e[weight_2/weight_1]$, the primary independent variable). $\text{Log}_e([AHI_2+1]/[AHI_1+1])$, as opposed to other measures of change in the AHI, followed an approximately normal distribution in the WSCS population. The resulting coefficient of $\log_e(weight_2/weight_1)$ can be interpreted as approximately the predicted percentage change in AHI related to a 1% weight change. The addition of the constant (1) to both the baseline and follow-up AHI measures was necessary because some participants had an AHI equal to zero. We refer to this model as the "progression" model, although reductions as well as increases in AHI values may be predicted.

Conditional (intrasubject) logistic regression modeling was used to estimate the increased likelihood of developing moderate-to-severe SDB (defined as AHI $\geq$15 events/h) associated with percentage weight change. We refer to this as the "incidence" model. Crossing the 15 events/h cutoff in either direction is accommodated by the model, allowing the model to account for an association of both weight gain and loss with changing SDB classification. The conditional model implicitly controls for fixed intraperson characteristics, such as sex and genetic profile.

The following were investigated as interacting and confounding factors in linear regression models, and, when appropriate, in the conditional logistic regression models: sex; baseline values of age, smoking habits (never, ever, and current-use status and cigarette packs per week), alcohol use (usual weekly consumption and amount consumed 24 hours prior to sleep study), menopausal status, body habitus (BMI; weight, height, and skinfold measurements; neck, hip, and waist girths; and waist-to-hip girth ratio), levels of education and physical activity; and 4-year change in smoking habits, alcohol use, menopausal status, and body habitus. Covariates, which substantially altered (>10% change) the regression coefficient for $\log_e(weight_2/weight_1)$ in the progression model or the coefficient for percentage weight change in the incidence model, were retained in final models. Interactions between the covariates and weight change were tested for statistical significance. The statistical significance (2-tailed $P<.05$ for main effects and $P<.01$ for interactions) of linear regression coefficients was assessed by $t$ tests. Conditional logistic regression coefficients were tested using the Wald $\chi^2$ statistic.[61] Regression diagnostics were performed to assess model fit and adequacy of compliance with modeling assumptions.

Intrasubject variability and measurement error in the AHI prevented meaningful assessment of whether the association of weight change and change in the AHI varied according to the baseline level of AHI. To address this problem, a supplemental analysis was performed using data from 215 participants who had completed baseline, 4-year, and 8-year follow-up sleep laboratory studies. Here, baseline and 4-year follow-up studies were averaged to produce a new "baseline" measured with less error than the AHI based on a single assessment. Using this new baseline AHI variable, we found no evidence for an interaction between baseline AHI and weight change ($P>.50$ for interaction term). That is, the relation between percentage weight change and percentage AHI change appears to be independent of baseline AHI. Thus, we expect that the regression model results presented here are valid across the range of baseline AHI values analyzed in this study.

## RESULTS

At baseline, unadjusted means (SDs) of AHI were 7.4 (13.1) events/h in obese participants (BMI$\geq$30 kg/m$^2$, n=268), 2.6 (4.5) events/h in overweight participants (25$\leq$BMI$<$30 kg/m$^2$, n=241), and 1.2 (2.4) events/h in normal weight participants (BMI$<$25 kg/m$^2$, n=181). During 4 years of follow-up, study participants gained a mean (SD) of 2.4 (5.7) kg. The mean (SD) change in AHI was+1.4 (8.7) events/h. Change in AHI, unadjusted for covariates, was related in a dose-response fashion to change in weight (FIGURE). Of 644 participants who did not have moderate-to-severe SDB at baseline ($AHI_1<15$ events/h), 39 did have moderate-to-severe SDB ($AHI_2\geq15$ events/h) at follow-up. These participants experienced a mean 3.9 (6.8) kg weight increase. Of 46 participants with moderate-to-severe SDB at baseline, 17 fell below 15 events/h at follow-up and experienced a mean 3.1 (6.2) kg weight loss. Forty-three participants experienced no change in the AHI (both $AHI_1$ and $AHI_2=0$). These participants experienced a mean 2.2 (4.9) kg increase in weight, compared with a mean weight increase of 4.0 (6.9)

kg in participants who experienced any increase in the AHI from baseline to follow-up.

The SDB progression model is summarized in TABLE 2. Adjusting for sex, baseline age and BMI, and change in smoking habits, weight change was positively related to change in the AHI. For small weight increments or decrements, each percentage change in weight was associated with an approximate mean 3% change in the AHI. For example, a person who experiences a 10% weight gain is expected to have an approximate 32% increase in AHI beyond the AHI increase that would be expected to occur if weight remained stable. Weight loss was associated with analogous predicted reductions in the AHI.

**Figure.** Mean Change in the Apnea-Hypopnea Index AHI by Weight Change Category

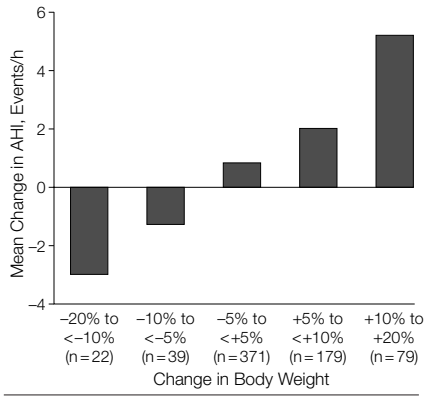


**Table 2.** Estimated Percent Change in the AHI for Selected Decrements and Increments of Percent Body Weight*

| Percent Change in Weight (vs No Change) | Estimated Percent Change in AHI (95% Confidence Interval)† |
|---|---|
| −20 | −48 (−58 to −35) |
| −10 | −26 (−34 to −18) |
| −5 | −14 (−18 to −9) |
| +5 | +15 (+10 to +21) |
| +10 | +32 (+20 to +45) |
| +20 | +70 (+42 to +104) |

*AHI indicates apnea-hypopnea index. Adjusted for sex, change in cigarette packs/wk, baseline body mass index (kg/m$^2$), and baseline age. All $P<.001$.
†In addition to change expected if weight remained stable.

Regression estimates were not materially altered by adjustment for menopausal status, physical activity, alcohol use, or education level, and these variables were not retained in the final progression model. Change in cigarette packs smoked per week did not materially change the association between weight change and AHI change. However, change in smoking habits was retained in final models because smoking cessation was associated with weight gain in this study, and smoking was positively related to increased SDB severity in a previous cross-sectional analysis from the WSCS.[62] Baseline values and changes in skinfold thicknesses; neck, waist, and hip girths; and waist-to-hip girth ratio were not significant predictors of change in the AHI independent of the variables included in the presented model. However, if substituted for the weight change variable in the progression model, change in BMI ($P<.001$), neck girth ($P<.001$), waist girth ($P<.001$), and total skinfold thickness ($P=.05$) were positively associated with change in the AHI. Baseline BMI was a significant predictor of AHI change ($P=.01$), independent of weight change. The regression coefficient (SE) of baseline BMI was 0.013 (0.005), indicating an expected increase of approximately 1% in the AHI for each increment of 1 kg/m$^2$ in baseline BMI. No interaction terms between weight change and any other examined covariates, including baseline weight, were statistically significant.

Conditional logistic regression was used to estimate the within-participant relation between percentage weight gain and the odds of developing moderate-to-severe SDB. TABLE 3 provides odds ratios and confidence intervals for weight increases of 5%, 10%, and 20%, adjusting for changes in cigarette use. Adults experiencing a 10% weight gain were estimated to have 6 times the odds of being newly classified as having moderate-to-severe SDB at follow-up (AHI≥15 events/h) compared with those with stable body weight. For persons with AHI greater than 15 events/h at baseline, these odds ratios can be interpreted as the relative odds of reducing the AHI below 15 events/h associated with weight loss. Since the conditional logistic approach models intrasubject changes in the AHI, fixed characteristics, such as sex, are implicitly accounted for. There were no significant interactions between weight change and examined covariates.

## COMMENT

In persons with SDB, we found a relation between weight gain and increased SDB severity. In persons who initially had mild or no SDB, we found weight gain predicted the development of moderate-to-severe SDB. Weight loss was associated with a reduced SDB severity and likelihood of developing SDB. These results were independent of many potential confounding factors, such as age, baseline body habitus measures, and change in smoking habits.

This prospective study benefited from a unique combination of features. It used a large population-based sample that provided more precise and generalizable results than previous clinic-based studies of weight loss and severe SDB in patients who were morbidly obese. Unlike those studies, this study was able to assess the relation between weight gain and SDB. This is an important advantage for public health interpretation of the study because of the increasing prevalence of obesity in

**Table 3.** Conditional Logistic Regression Coefficients and Odds Ratios* for Development of Moderate-to-Severe Sleep-Disordered Breathing (SDB) (AHI≥15 Events/h) for Selected Increments of Weight Gain

| Percent Gain in Weight (vs No Gain) | Regression Coefficient (SE) | Estimated Odds Ratio for Moderate-to-Severe SDB (95% Confidence Interval) |
|---|---|---|
| 5 | 0.9 (0.3) | 2.5 (1.5 to 4.1) |
| 10 | 1.8 (0.5) | 6.0 (2.2 to 17.0) |
| 20 | 3.6 (1.1) | 36.6 (4.6 to >50) |

*Adjusted for change in cigarette packs/wk. AHI indicates apnea-hypopnea index. All $P<.001$.

the United States.[28-30] This study also benefits from high-quality laboratory-based polysomnographic assessment of SDB, currently the diagnostic gold standard for SDB.

Our results are largely consistent with other research examining excess weight and its relation to SDB. Cross-sectional clinic-[32-40] and population-based[3-6,22,41-44] investigations typically find significant correlations. Five small ($n \leq 15$) uncontrolled studies of surgical weight loss[45-49] in patients who were severely obese found mean weight loss ranging from 25% to 50% of baseline weight yielded 70% to 98% mean reductions in indices of SDB. Eight small ($n < 30$) uncontrolled studies of dietary weight loss[49-55,58] in obese patients found that a range of 10% to 20% mean weight losses yielded mean 30% to 75% reductions in indices of SDB. Two controlled dietary weight loss studies[56,57] found mean weight losses of 9% and 17% yielded mean AHI decreases of 47% and 61%, respectively. Two small ($n \leq 55$) longitudinal studies of SDB change in patients with sleep apnea[63,64] found no statistically significant correlations between change in AHI and change in BMI. These null findings may be because of insufficient statistical power. Together, our longitudinal results, those from cross-sectional and weight-loss studies by other investigators, and biological plausibility provide evidence consistent with a causal link between excess body weight and SDB.

A variety of body habitus measures, including neck morphology[3,5,6,32-35,38,43,65] (neck girth or neck fat distribution), general obesity[37,39,43] (BMI and skinfold measurements), and central obesity[36,39,40,43] (waist-to-hip ratio, waist girth, and abdominal visceral adiposity) have been cross-sectionally associated with SDB. Accordingly, we investigated changes in neck girth, waist-to-hip ratio, skinfold measurements, and BMI, as well as percentage body weight, as prospective predictors of SDB. We found that changes in percentage body weight predicted changes in AHI as well as those other measures and that our models were not substantially improved by the addition of other body habitus parameters. We chose to focus on weight change as the measure of change in body habitus, as it is a common and easily measured parameter.

Study limitations include incomplete follow-up of the eligible baseline sample. Twenty-seven percent of the baseline sample (258/948 baseline participants) either refused or were not reachable for follow-up. If the relation between body weight and SDB is substantially different in the entire baseline sample and the 690 participants examined for this study, we would be concerned about a bias in our results because of incomplete follow-up. As a check for such a discrepancy, we used linear regression to examine the baseline cross-sectional associations of log(AHI + 1) and log (weight), controlling for height, age, sex, current cigarette smoking status, and alcoholic drinks per week in the 2 samples. The baseline coefficient (SE) for log(weight) in this study's sample is 2.0 (0.2). The corresponding coefficient in the entire eligible, invited baseline sample is 2.1 (0.1). Here we find no substantial difference in the relation of SDB and weight in the samples. Although this does not rule out a longitudinal bias, it reduces concern that incomplete follow-up compromises our findings.

There are a few additional issues regarding our results that merit discussion. First, because few individuals in the sleep cohort experienced large percentage changes in weight, we do not recommend generalizing our findings to very large weight changes. Supplementary semiparametric spline modeling of our data indicated that within the range of ±20% weight change, the association of weight change and SDB change (as characterized by our progression model) was well described by a linear function. However, the relation at greater weight change plateaued. Unfortunately, the number of participants with extreme weight change proved too few to carefully characterize associations that involved more than 20% weight change. Thus, these participants were excluded, and the findings presented in this report should not be extrapolated beyond 20% weight change.

Second, 43 (6%) of the participants had baseline and follow-up AHI equal to zero. This minority may represent persons with normal nocturnal breathing that is resistant to perturbation in the presence of weight change or other disturbance. Our progression model does not readily accommodate such persons.

Third, there is a substantial amount of variability in AHI change that is not accounted for in our final models. The residual variability is due to both factors other than weight change that impact SDB and measurement error in assessing SDB.

Fourth, we found no statistically significant evidence that the association between percentage weight change and SDB depended strongly on baseline habitus. However, in normal weight participants, the mean AHI was low and weight loss uncommon. Thus, we could not rigorously address the association of weight loss and reduced SDB severity in the normal weight participants with SDB.

Fifth, it is plausible that excess body weight acts either over time, by accelerating the progression of SDB, or acutely by rapidly modulating SDB through, for example, increased resistance to airflow via fat deposition in the proximity of the upper airway. With our study design, we were unable to determine to what extent one or both of these processes might be occurring. However, we found change in weight and baseline habitus (BMI in our progression model) independently predicted change in SDB severity, indicating that both an accelerated progression and short-term response might occur. Weight-loss studies that have demonstrated a reduction in SDB severity have tended to be short term, also suggesting that at least some of the response of SDB to excess weight is incurred almost simultaneously with weight change.

Finally, we do not know the causes of weight variations in participants whose weight did change and thus cannot specify the relative importance of weight change due to alterations in energy intake, physical activity, or metabolism. These last 2 issues point to the need for future longer-term follow-up studies to examine the relation between body habitus and SDB over decades, focusing on the effects of diet, exercise, and other related medical and lifestyle factors.

Obesity is a growing worldwide health problem, and its strong association with SDB is likely to be causal. It follows that the incidence of SDB will continue to grow in prominence and that clinical and public health strategies using weight control will be attractive approaches to the treatment of SDB. Our findings have important clinical implications for overweight patients with mild-to-moderate SDB who are poor candidates for nasal continuous positive airway pressure therapy. Weight loss may be appropriate as an alternative strategy for reduction in the severity and progression of SDB and for improvement in daytime symptoms. Furthermore, overweight people without overt clinical manifestations of SDB now have another incentive to lose weight or at least not to gain additional weight. Finally, these findings emphasize the importance of preventing weight gain in normal weight persons to avoid the development or progression of SDB.

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Besides learning to see, there is another art to be learned—*not to see* what is not.
—Maria Mitchell (1818-1889)