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Original Article

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Revisiting chronic low back pain: evidence that it is not non-specific

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Abstract

Context: There is a common symptom pattern with most chronic low back pain (CLBP), suggesting that there is a common underlying etiology, belying the term "nonspecific." Many studies of CLBP and its treatment have been conducted with the assumption of nonspecificity, and as a result, treatment has not been focused, thus there has not been a significant change in CLBP prevalence over the past several decades. It is the thesis of this study to show that there is an underlying, specific cause of CLBP and that the presumption that CLBP is nonspecific is misdirected. The lumbosacropelvic (LSP) region, including the sacroiliac joint (SIJ), is part of a neuromusculoskeletal (NMSK) feedback system, and it is proposed here that CLBP is the result of a change in the feedback (afferent) aspect in that system.

Objectives: The objectives of this study are to show that CLBP presents as a pattern of symptoms that actually represents the final common pathway for a dysfunctional LSP joint system. Rather than being "nonspecific," the majority of CLBP has an underlying cause that is quite specific and predictable.

Methods: A total of 252 patients were seen for CLBP, 67% of whom were diagnosed with an SIJ dysfunction. The presence of pain was recorded from seven structures most closely associated with CLBP. The conditional probabilities of having each pain generator given a SIJ dysfunction and an SIJ dysfunction given the presence of the pain generator were estimated, and associations were analyzed utilizing chi-square tests. Phi coefficients and odds ratios were utilized to quantify the strength of the association. The multivariable logistic regression model was fit to relate the presence or absence of the SIJ dysfunction to the seven pain generators.

Results: The associations between SIJ dysfunction and each pain generator were statistically significant. Phi coefficients indicated moderate strengths of these bivariate associations. Iliolumbar ligament (ILL) and psoas muscle (PSM) were significant predictors of SIJ dysfunction in the multivariable model.

Conclusions: Seven pain generators had a strong association with SIJ dysfunction. This empirical clinical evidence supports our hypothesis that LSP system dysfunction, as evidenced by SIJ dysfunction, is a common source of symptom patterning associated with CLBP and is most likely the causal element. This is evidence that most CLBP is not "nonspecific" but rather the result of changes made by the NMSK control system for the LSP region.

Keywords: chronic low back pain; sacroiliac joint; system feedback

Chronic low back pain (CLBP) is commonly referred to as "nonspecific"; however, this paper proposes a novel perspective that CLBP has a single specific cause. It has been observed that several pain generators are commonly associated with CLBP. We hypothesize that joint dysfunction or restriction, specifically the sacroiliac joint (SIJ), is a major factor in lumbosacropelvic (LSP) control system changes that lead to CLBP. The SIJ is the synovial joint that connects the spine's sacrum to the ilium, which is the upper portion of the innominate or hip bone. Numerous strong ligaments support and maintain SIJ function, which transmits dynamic forces through the pelvis from the hip joint to the lumbar spine. Additional elements can become painful in the surrounding ligamentous and muscle structures. Our pilot data into the clinical presentation reveal a correlation between the presence of SIJ restriction and many of these pain generators. We conclude that the

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SIJ plays a major role in CNS control of the system around that joint, as observed in the arthrogenic muscle inhibition (AMI) phenomenon [1]. We hypothesize that a correlation exists between the presence of an SIJ restriction and many of these pain generators, and we suggest a specific factor contributing to CLBP.

This perspective is consistent with the Osteopathic Medicine Principle of homeostasis, that is, incorporating a feedback control system that senses and regulates the neuromusculoskeletal (NMSK) system. It is proposed that CLBP is a syndrome and is most likely the result of perturbations within the NMSK control system in the LSP region. CLBP is the second most common cause of disability in US adults [2] and the most common reason for lost workdays [3]. A population-based study in North Carolina from 1992 to 2006 reported that prevalence has increased from 3.9 to 10.2% over the past several decades and that focus should be placed on "causality and self-management" to address the issue [4]. The confounding issue may be the misunderstanding of what is causing CLBP. The current, general perspective of nonspecific CLBP is that it is multifactorial, multifaceted, and relates to a disorder of the lumbar spine. A 2018 paper stated, "Low back pain is usually nonspecific or mechanical. Mechanical low back pain arises intrinsically from the spine, intervertebral disks, or surrounding soft tissues" [5].

Alternatively, others state that 85% of CLBP cases do not come from a spinal, spinal disc, or nerve injury disorder but rather from a wide variety of musculoskeletal (MSK) issues, thus "nonspecific" low back pain [6]. A 2015 study of 320 patients at walk-in orthopedic clinics in Japan reports that they identified the cause of CLBP in 79% of cases by conducting extensive examinations by orthopedic specialists [7]. Unfortunately, the researchers hypothesized the causes of CLBP based on clinical examination without treatment confirmation. Many new lines of scientific and clinical investigation are being undertaken to study low back pain but have not yet translated into practical solutions, particularly for people with CLBP [8].

A Veterans Administration (VA) research paper, published in 2020, proposed that CLBP is a syndrome rather than a nonspecific pain condition [9]. That is, CLBP is a common pathway for a combination of contributing factors, including spinal degeneration. The common presence of pain generators, such as SIJ syndrome, leg length discrepancy, iliotibial (tendon) band (ITB) syndrome, and greater trochanteric pain syndrome with back pain, support this theory. No central mechanism or cause was proposed [9]. The model proposed is a similar concept to the idea that CLBP is a syndrome, a commonly occurring group of symptoms with a common cause.

As with most physiological systems in the body, whether regulation of blood pressure or heart rate, the NMSK system is also a control system. The concept of joint dysfunction affecting muscle as part of an NMSK control system is well studied in the peripheral joints. We propose that it also applies to the LSP region. When a peripheral joint is dysfunctional, surrounding muscles and tissues are inhibited or facilitated in a sensory feedback loop. Inhibited muscles are affected at the synaptic level of afferent nerves in a phenomenon called AMI [1].

Methods

Data source

Following Institutional Review Board approval (MSU IRB 17–675), a retrospective chart review was conducted in 2018 by the author (CN) of new patients during a randomly selected year and start month (June 2013) in 8 years of private practice. The author (CN) is Board Certified by the American Osteopathic Board of Neuromusculoskeletal Medicine (AOBNMM). The year and month were randomly selected by utilizing a random date generator (Random.org) for the interval between January 1, 2008 and December 31, 2016. The reviewed charts were the records embedded for patients in the practice's Electronic Medical Records (EMR) software (MacPractice, Inc., Lincoln, NE). The clinic was operated as a solo practice with a single provider. The patient inclusion criteria included new patients who were treated at the clinic during the selected year (2013) and met the inclusion/exclusion criteria. The inclusion criteria were patients 18 years and older, who are ambulatory, and who could understand and follow instructions. The exclusion criteria were patients with spinal or neurological disorders, spinal fractures within the past year, or severe osteoporosis, scoliosis, or osteoarthritis in the spine or hip joints. The patients must not have a history or diagnosis of retroperitoneal tumors or lymphadenopathy or a diagnosis of lumbar spine radiculopathy verified by imaging, electromyography and nerve conduction velocity (EMG/NCV) testing, as well as matching symptoms. A patient with a spinal surgery completed within the past year was also rejected. Rhizotomies, epidural injections, and spinal cord stimulator implants were acceptable if completed at least 3 months prior to the beginning of the study. To qualify for the diagnosis of CLBP, the patient must have been diagnosed with "nonspecific" CLBP, which consists of having not been excluded by the above criteria, having symptoms of CLBP for more than 3 months, and being within the ages of 18 and 95. Other than the notations concerning the pain-generating tissues associated with CLBP discussed below, only anonymous demographic information (sex, age, and insurance type) was extracted.

Measures – methodology

The data recorded included the presence or absence of SIJ restriction and tenderness in seven bilateral structures within the LSP region known to be the principal pain generators most closely associated with CLBP: iliolumbar ligament (ILL), psoas muscle (PSM), long dorsal

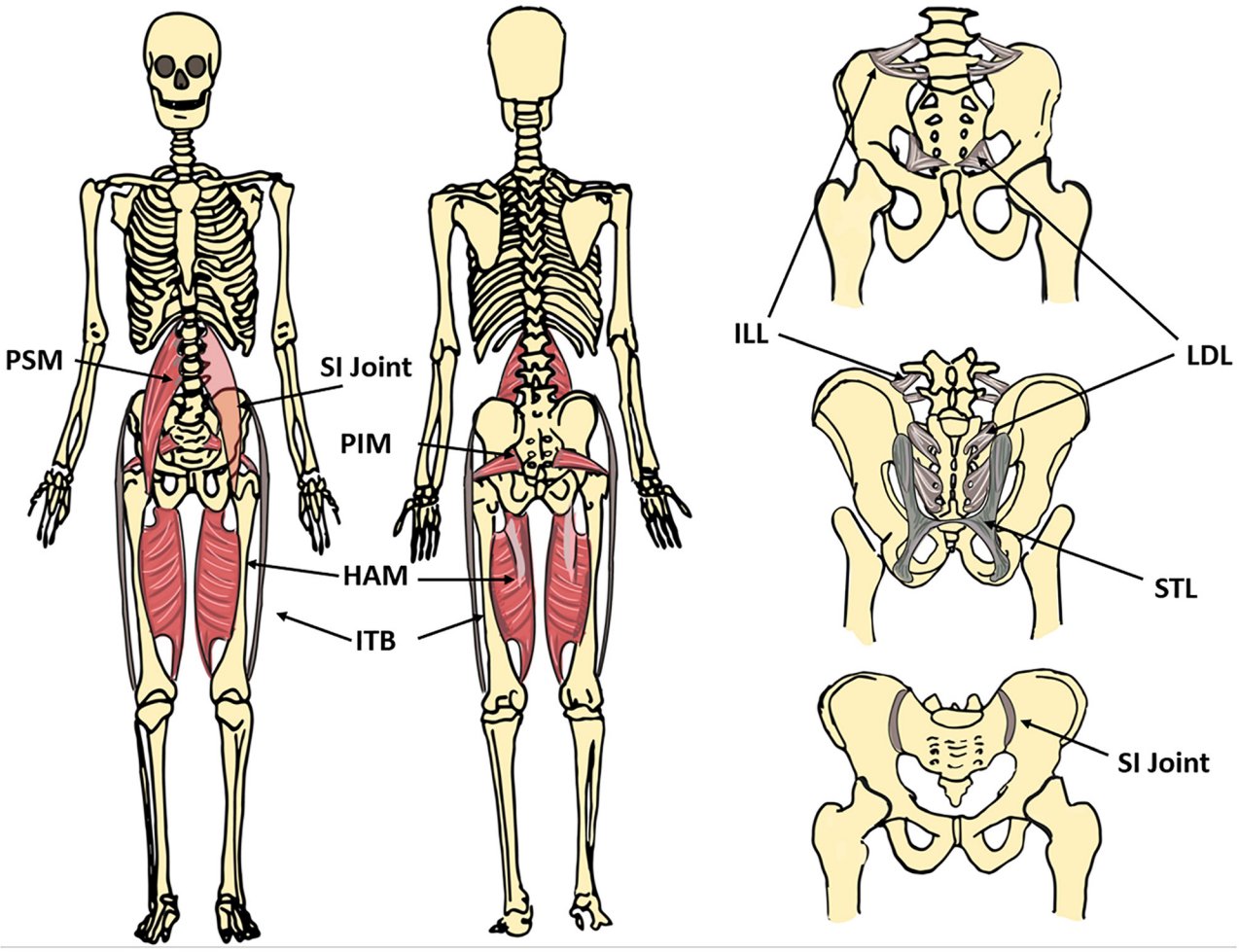

Figure 1: The sacroiliac (SI) joint and the lumbosacropelvic (LSP) region. The LSP region most closely associated with chronic low back pain (CLBP) includes: iliolumbar ligament (ILL), psoas muscle (PSM), long dorsal ligament (LDL), sacrotuberous ligament (STL), piriformis muscle (PIM), iliotibial (tendon) band (ITB), adductor magnus muscle (hip adductor muscle) (HAM). This area is comprised of the principal pain generators. CLBP, chronic low back pain; HAM, adductor magnus muscle (hip adductor muscle); ILL, iliolumbar ligament; ITB, iliotibial (tendon) band; LDL, long dorsal ligament; LSP, lumbosacropelvic; PIM, piriformis muscle; PSM, psoas muscle; SI, sacroiliac; STL, sacrotuberous ligament.

ligament (LDL), sacrotuberous ligament (STL), piriformis muscle (PIM), iliotibial (tendon) band (ITB), and adductor magnus muscle (hip adductor muscle [HAM]) (Figure 1). SIJ restriction was determined by utilizing three techniques: standing and seated flexion tests and pelvic rocking [10]. Restriction of a joint is often called a joint dysfunction [11].

Once SIJ dysfunction was confirmed, the presence of each pain generator was determined by direct palpation of each of the LSP region structures with patient response. The presence or absence of pain was recorded as a 0 for no pain and 1 for any pain. Muscle palpation was done at the approximate middle of the muscle belly. For ligaments and ITB, tenderness anywhere along its length was considered positive. A pain scale was not utilized because a longitudinal study of outcomes to treatment was not an objective of this study, so relative change was not considered. The presence or absence of pain, as verbalized by the patient in response to direct palpation by the provider, was the only criterion utilized and recorded at the patient initial visit.

Results

The seven binary variables indicating the presence or absence of SIJ dysfunction and specific pain generator tissue problems (ILL, PSM, PRM, ITB, HAM, STL, and LDL) were analyzed in a sample of 252 patients records. Descriptive statistics were utilized to summarize patient age and sex. First, the associations between each pain

Table 1: Summary of the association between each pain generator and an SIJ dysfunction in a sample of 252 patients.

Pain generator	n (%)	P (pain generator given SIJ)	P (SIJ given pain generator)	Kappa	Percent agreement	Percent positive agreement	Percent negative agreement	Adjusted odds ratio for SI joint dysfunction (95% confidence interval) from multivariable model
ILL	121 (48%)	0.64	0.88	0.41	70%	74%	66%	2.79 (1.28, 6.08)
PSM	140 (56%)	0.72	0.86	0.46	74%	78%	67%	3.93 (1.90, 8.17)
PIM	43 (17%)	0.25	0.95	0.17	50%	39%	57%	–
ITB	57 (23%)	0.32	0.93	0.21	54%	48%	58%	4.35 (1.38, 13.75)
HAM	26 (10%)	0.16	1.00	0.11	44%	33%	38%	–
STL	73 (29%)	0.39	0.89	0.24	57%	54%	59%	2.39 (0.98, 5.82)
LDL	4 (2%)	0.02	1.00	0.02	36%	5%	51%	–

Because of the low conditional counts, PIM, HAM, and LDL were not included in the subsequent multivariable modeling. HAM, adductor magnus muscle (hip adductor muscle); ILL, iliolumbar ligament; ITB, iliotibial (tendon) band; LDL, long dorsal ligament; PIM, piriformis muscle; PSM, psoas muscle; STL, sacrotuberous ligament.

generator and the SIJ dysfunction was analyzed utilizing chi-square tests. The kappa statistic was utilized to quantify the level of agreement according to the classification system proposed by Landis and Koch: <0 – no agreement, 0–0.20 – slight agreement, 0.21–0.40 – fair agreement, 0.41–0.60 – moderate agreement, 0.61–0.80 – substantial agreement, and 0.81–1.00 – almost perfect agreement. Because kappa depends on the prevalence of the problem (which was not high for some of the individual pain generators) and no single number summary fully describes the agreement or disagreement, the percentages for positive and negative agreement were also computed. The positive agreement proportion was calculated as the number of cases in which a pain generator was present with a SIJ dysfunction divided by the average number of positive cases in the two places. Similarly, the negative agreement proportion was calculated as the ratio of the number of cases in which the pain generator and SIJ dysfunction were absent. The proportions were expressed as percentages and summarized in Table 1, along with the conditional probabilities of having each pain generator given SIJ, and the conditional probabilities of SIJ given the presence of the pain generator.

Second, to account for the associations among multiple pain generators, they were entered into the multivariable logistic regression model with the outcome of SIJ dysfunction (yes/no). Adjusted odds ratios for SIJ dysfunction and their 95% confidence intervals were estimated for each pain generator from the multivariable model.

Finally, to explore another way to summarize multiple pain generators, seven variables to indicate each of them were subjected to the exploratory factor analysis (EFA) with categorical indicators. Eigenvalues greater than 1 were utilized to determine the summary factors, and summary counts were calculated for each factor. These counts were then evaluated as predictors of SIJ dysfunction utilizing the logistic regression model.

The age range of the patients was 18–92 years, with a mean age of 47.2 (standard deviation [SD] 18.8). There were more males (144, 57%) than females (108, 43%) in the sample; their mean ages were not different statistically, with the mean for the males being 45.8 (SD 17.8) and the mean for the females being 50.5 (SD 19.8). The interquartile range was 29.5 for males, 36.3 for females, and 33 combined.

The SIJ dysfunction was found among 166 (66%) patients. All bivariate associations of each pain generator with SIJ were statistically significant (all $p<0.01$, Table 1), with the exception of the LDL. The odds ratios ranged from 2.39 to 4.35 (Table 1). In the multivariable logistic regression relating the log odds of the SIJ dysfunction, ILL, PSM, and ITB were all significant predictors, whereas STL was not (Table 1). The wide confidence interval for the ITB odds ratio reflects the previously mentioned low conditional count. The predictive ability of the model was very good, with a c-statistic (area under the receiver operating characteristic curve) of 0.83.

The EFA resulted in two factors. Two of the pain generators, ILL and PSM, loaded on the first factor, whereas the other five pain generators loaded on the second factor. Based on these results, two summary counts were created: the count of ILL and PSM pain, and the count of the other five pain generators. Both counts were significantly related to the SIJ dysfunction over and above each other in the multivariable logistic regression model. For the first count, the odds ratio of the SIJ dysfunction was 3.16, 95% CI (2.10, 4.75), $p<0.01$. For the second count, the odds ratio of the SIJ dysfunction was 2.52, 95% CI (1.52, 4.19), $p<0.01$. The predictive ability of the model was very good, with a c-statistic of 0.84.

Discussion

We hypothesized that SIJ dysfunction is a major factor in LSP control system changes leading to CLBP, similar to the AMI phenomenon. Data analysis in this retrospective study supports this hypothesis as well as the idea that the CLBP syndrome is the result of a perturbance in the NMSK system controlling the LSP region. The concept of CLBP as an outcome of a control system response is novel but consistent with the Osteopathic Medicine tenets. The body is capable of self-regulation, and all body systems are regulated, including the NMSK system. Structure and function are reciprocally interrelated, and by viewing CLBP as the response of a control system, there can be more rational CLBP treatment.

The result shows seven MSK pain generators with a strong association in the presence of SIJ dysfunction. This notion was first documented by Vladimir Janda in the 1960's in Morris et al. [12] Janda observed that joint dysfunction would be reflected in quality of the muscles and nervous system. He focused primarily on locomotor system functional stability rather than MSK mobility. Greenman and DeStefano [10] expressed the same idea in more Osteopathic terms, suggesting that arthrogenic changes in the periphery will cause a central response. The same idea was expressed in an engineering systems sense as a stability issue with a change in afferent feedback (proprioception) in a control system which alters the controller's effect on the controlled system [13, 14]. These engineering studies correlate to the underlying foundation for CLBP diagnosis and treatment with a NMSK control system involved in the changes and common symptoms in CLBP patients.

Arthrogenic-related changes in peripheral skeletal muscle as a control system has been described in the literature as AMI. AMI as described, enforces a cycle of atrophy and weakness in surrounding extensor muscles [1]. In general, with joint restriction, receptors act upon inhibitory interneurons to decrease motor neuron recruitment within surrounding muscle, leading to decreased usage of the joint's extensor muscles. Afferent information is sent through sensory neurons embedded in the joints. Most joint sensory information occurs via mechanoreceptors, which respond to stimuli such as tension, and proprioceptors that determine position in space. In a functional joint, receptors send excitatory information through an afferent pathway that travels through surrounding muscles to the spinal cord. From the CNS, efferent information travels to muscles, and motor neuron recruitment (and contraction) can occur. However, with joint injury or dysfunction, there is an increase in afferent activity where both presynaptic and postsynaptic inhibition prevent motor neuron recruitment in the surrounding extensor muscles. During postsynaptic inhibition, a signal reaches a synapse and an ion channel is opened. The membrane becomes hyperpolarized and an action potential cannot occur.

In the case of AMI, the inhibitory neurotransmitter utilized is gamma-aminobutyric acid. Presynaptic inhibition can also occur during AMI, which involves a decrease in neurotransmitter release at the synapse. In a review by Stokes and Young, the researchers describe the role of type II fibers in AMI involving high threshold motor units. It was demonstrated that patient's muscles experiencing AMI have preferential atrophy of these fibers [15]. Many motor neurons are inhibited and their inactive nature ultimately leads to atrophy of type II fibers. Hopkins and Ingersoll additionally describe the specific relationship between AMI and surrounding muscles. In a study of sheep, when ACL motor neurons were stimulated, extensor muscles were inhibited and flexor muscles were facilitated [1].

Sacroiliac joint dysfunction and AMI

The concept described for peripheral joints as discussed above also apply to central joints as well because they share similar NMSK anatomy. The analogous extensors for the SIJ are the gluteus muscle group of the LSP region. This is in agreement with observations by Janda that the hip extensors are inhibited in CLBP [12]. Hossain and Nokes [16] describe this exact phenomenon: muscle wasting in the gluteus maximus muscles in patients with SIJ dysfunction. Biomechanically, the SIJs are in the load path of and involved in the distribution and equilibration of upper body weight, ground reaction forces transmitted from the lower extremities, and most of the inertial forces generated during motion of the body. Muscles and ligaments with the LSP make this transfer of load possible, and if one element is not working properly, the whole LSP system can become dysfunctional and present as CLBP. Impairment in the LSP system is accompanied by atrophy and weakness of the gluteus maximus and extensor muscles [16]. Gluteus maximus muscle fibers activate and stabilize the SIJ joint. If the SIJ is dysfunctional, these fibers show atrophy.

Hungerford et al. [17] studied activation of trunk and hip muscles in patients with SIJ dysfunction. They studied 14 male subjects diagnosed with SIJ dysfunction and 14 control subjects similar in gender, age, and health. Electromyographic sensors were placed superficially on

seven hip and trunk muscles, including the obliquus internus abdominis, tensor fascia latae, multifidus, gluteus maximus, gluteus medius, biceps femoris, and adductor longus. The sensors measured muscle activity during hip flexion of participants. Results showed a delay in contraction of the obliquus internus abdominis, multifidus, and gluteus maximus muscles in subjects with SIJ dysfunction [17]. This delay was due to a lack of motor neuron deployment in these muscles, primarily the gluteus maximus extensors of the SIJ.

The mechanism of gluteus maximus wasting due to SIJ dysfunction is analogous to quadriceps weakness and atrophy that occurs after knee trauma [14]. This is a novel theory that buttresses the central thesis of this paper: joint dysfunction in the LSP region, most likely the SIJ, is the cause of the AMI-like response that manifests as the syndrome called "nonspecific CLBP."

The principal flexor muscles for the SIJ are the psoas major muscles because they pass just anterior to the SIJs. There are other ancillary muscles, including the rectus femoris and iliacus that act to flex the hip. These are also a commonly but lessor named pain generator in the CLBP syndrome. The fact that these muscles are facilitated in CLBP parallels the finding in the AMI study where the joint flexors are facilitated in joint injury [1]. The term "compensation" has been utilized in the past to describe the changes in these muscles, but the term was specifically avoided here because of its lack of specificity. That is, "compensation" offers no insight as to the mechanism(s) involved. In large part, the mechanism for such changes in muscle in response to joint dysfunction is what this paper is attempting to explore.

Limitations

The limitations of this study include its observational retrospective nature, and the conclusions are limited to associations and not causal relationships.

Conclusions

The relatedness of the SIJ to the low back demonstrates how the altered structure impairs the function and targets the proximal cause of most of the symptoms of CLBP. Understanding this relationship and defining CLBP as a syndrome caused by an AMI-like reflex with a specific cause, rather than being "nonspecific," has important positive implications in the diagnosis and rational treatment of CLBP and how it is taught in all provider schools. It may serve to reduce the multiple, erroneous referrals and failed procedures that CLBP patients endure today.

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