ABSTRACT

CIMBALNIK, A. M. Relationship among indices of training and the incidence of illnesses and injuries in elite athletes. MS in Adult Fitness/Cardiac Rehabilitation, December 2001, 59pp. (C. Foster)

Members of the U.S. Speedskating Team (n = 5; 1 male, 4 females) were monitored during training in order to evaluate the relationship between training characteristics and the incidence of illnesses, injuries, and complaints. A daily training log-questionnaire along with questions regarding illness, injury, muscular aches and pains, and state of mental well-being was completed. Training load, training monotony, and training strain were computed using the session Rating of Perceived Exertion (RPE) method. The product of the weekly muscular aches and pains and weekly state of mental well being defined the complaint index. A low incidence of illnesses was found in relation to training load or strain. A general relationship between training load and the complaint index with weak to moderate correlations ($r = 0.35 - 0.71$) was found. There was no evidence for a threshold effect of the complaint index in relation to either training load or strain. The present data suggest that prospective studies with good data recovery might be a productive process in terms of evaluating negative training outcomes.

RELATIONSHIP AMONG INDICES OF TRAINING AND THE INCIDENCE OF ILLNESSES AND INJURIES IN ELITE ATHLETES

A MANUSCRIPT STYLE THESIS PRESENTED

TO

THE GRADUATE FACULTY

UNIVERSITY OF WISCONSIN-LA CROSSE

IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR THE

MASTER OF SCIENCE DEGREE

BY

AMY M. CIMBALNIK

DECEMBER 2001

COLLEGE OF HEALTH, PHYSICAL EDUCATION, RECREATION AND TEACHER EDUCATION

UNIVERSITY OF WISCONSIN-LA CROSSE

THESIS FINAL ORAL DEFENSE FORM

Candidate: Amy M. Cimbalnik

We recommend acceptance of this thesis in partial fulfillment of this candidate's requirements for the degree:

Master of Science in Adult Fitness/Cardiac Rehabilitation

The candidate has successfully completed the thesis final oral defense.

	5/16/01
Thesis Committee Chairperson Signature	**Date**
	5-16-01
Thesis Committee Member Signature	**Date**
	5/16/01
Thesis Committee Member Signature	**Date**

This thesis is approved by the College of Health, Physical Education, Recreation and Teacher Education.

	8/29/01
Associate Dean, College of Health, Physical Education, Recreation and Teacher Education	**Date**
	8/29/01
Dean of Graduate Studies	**Date**

ACKNOWLEDGEMENTS

I would like to take this opportunity to express my great appreciation to those who have assisted me along the way with this great endeavor. I would like to extend my greatest appreciation to Dr. Carl Foster, my thesis chair. I admire you for your patience, expertise, and wisdom. The extra time that you spent advising, guiding, and proofreading is greatly appreciated. Thank you for making this an enjoyable learning experience, and most of all thank you for always making me laugh.

I would like to thank Dr. Glenn Brice and Scott Doberstein for the advice, time, and effort you provided in helping me prepare my thesis. Your keen eyes are greatly appreciated.

A special thanks goes to my roommate, Shana VanWychen. We did it! I cannot believe that this year has come and gone. I have enjoyed all of our late night talks and all of our long walks. Thank you for being there for me when I needed you the most. This year wouldn't have been the same without you. To the rest of my classmates, thank you for the great memories.

A final thanks is to my family. To my parents, thank you for the never ending support and confidence you have always had in me. Through the most stressful times, you were only a phone call away. Thank you for being such good listeners, and thank you for always being there for me. To my brother, Andy, thank you for always believing in me, and to my fiance, Keith, thank you for all of your support and encouragement.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF APPENDICES

INTRODUCTION

The goal of every athlete and coach is to achieve optimal athletic performance. In an attempt to optimize performance and to maximize athletic ability, athletes tend to train beyond a comfortable level (11). Unfortunately, there is a thin line between improved performance and deterioration (11). Banister and colleagues (3, 10) have proposed a model which describes these adaptations to stress. If the athlete trains hard and is allowed enough recovery time, performance should improve. If an athlete does not receive sufficient recovery time, a decrease in performance may occur. When an athlete performs unfavorably in training and competitions, the automatic reaction of both coach and athlete is to train harder (7). Progressively harder training often leads to overtraining syndrome (OTS).

OTS is a complex condition wherein an athlete is training strenuously, yet performance deteriorates (11). This is usually accompanied by a variety of biochemical and physiological alterations as well as changes in mood/behavior. Lehman et al. (9) define OTS as an imbalance between training and recovery, exercise and exercise capacity, and stress and stress tolerance.

Presently, there is no accepted global hypothesis for the genesis of OTS. Fry et al. (8) reported a large number of symptoms associated with overtraining. These symptoms have been grouped according to psychological processing, physiological performance, immunological, and biochemical parameters. It is obvious that OTS cannot

be defined by one symptom, but rather as a cluster of multiple symptoms in a variety of combinations (11).

Illness and injury are undesired conditions that often arise throughout an elite athlete's career. Evidence suggests an increased incidence of illness is associated with impairment of the immune system caused by intense exercise (11). Several hypotheses have been proposed to account for the role of injuries in OTS. Some researchers believe that injuries cause OTS while others believe that OTS causes injuries.

Smith (11) suggested that repeated micro-trauma to the muscular, skeletal, and/or joint system frequently initiates OTS. Local inflammatory factors (e.g., cytokines) are released as a result of exercise-induced musculoskeletal trauma. Local acute inflammation becomes chronic with continued high-volume, high-intensity exercise coupled with limited rest. Smith (11) hypothesized that the central cause of OTS is chronic local inflammation which leads to systemic inflammation.

Foster and Lehman (7) suggested that the lack of day-to-day variation in training could initiate OTS. This is known as the monotony theory of overtraining. As a result, the daily "sameness" of intense training will make the athlete more prone to injury due to the excessive stress.

Overtraining syndrome has been difficult to study because there are no experimental models of the condition. Secondly, evaluation of responses to training have been difficult because, until recently, there has been no method for adequately quantitating training load (4). Recent development of the session Rating of Perceived Exertion (RPE) method developed by Foster (6) allows for monitoring of training. Thirty

minutes following a training session, the athlete is asked to rate the global intensity of the entire session. The intensity score (RPE) is multiplied by the session duration to determine the training load. The training monotony of the session is determined by dividing the daily mean load by the standard deviation within a week. Training "monotony" can be defined as an index of the lack of training variability. Observations suggest that some index of training load can predict the development of OTS (2). Therefore, the product of training load and training monotony can be used as an index of the "training strain" which may be an indicator of the likelihood of negative adaptations to training.

Foster (4) suggested that the incidence of banal infections and injuries is related to training above individually identifiable thresholds of training strain. However, these data were collected over a relatively brief period of time and in sub-elite athletes. Data needs to be collected in elite athletes over longer periods of time. Therefore, the purpose of this study was to make preliminary observations of the relationships among indices of training and the incidence of illnesses, minor injuries, and complaints in elite speed skaters.

METHODS

The subjects for this study were elite members of the all around long distance and sprint training groups of the U.S. Speedskating Team (1 male, 4 females). Olympic and World Championship medalists, current and former world record holders, and members of the U.S. Junior National Team were included in the sample. Demographic and physical characteristics were taken from the U.S. Speed Skating registry. The athletes trained either in Milwaukee, WI or Park City/Salt Lake City, UT. The study was approved by the Institutional Review Board for the Protection of Human Subjects at the University of Wisconsin La Crosse and all subjects provided written informed consent prior to participation.

Table 1. Subject Characteristics (mean ± standard deviation)

Variable	Male (n=1)	Female (n=4)
Age (years)	23	24 ± 1
Height (cm)	185	166 ± 3
Weight (kg)	80	61 ± 2
% Body fat	8	16 ± 1
VO_2max (ml/kg/min)	67	60 ± 3

A well-validated system of monitoring training was used with the elite speed skating team during both the dry land and on-ice training during the 2000-2001 year (4). The workouts were monitored using the session RPE method (6). Thirty minutes after each training session, each athlete rated the overall intensity of each segment of the training session using the category ratio Rating of Perceived Exertion (0-10) scale developed by Borg (1). The duration of the entire training session, including warm-up, cool down, and recovery intervals along with comments on details about illnesses, injuries, muscular aches and pains, and state of mental well-being were recorded on a daily training log/questionnaire. Each athlete determined the level of muscular aches and pains by using the Complaint Index that ranges from 1-5, with 1 representing that muscles feel great and 5 representing the question "Do I have to get out of bed?" Additionally, the state of mental well-being of each athlete was determined using a 1-5 scale, with 1 meaning "I feel great, goals are attainable, bring on the Dutch" and 5 being "Why am I wasting my time with this stuff?" The athletes were requested to transmit the training log/questionnaires through e-mail to the research investigator on a daily basis.

The training load of each session was calculated by multiplying the intensity score (RPE) by the duration of the training session. When multiple training sessions were performed on any given day, the training load was summated for that day to determine a daily training load. Summating training loads during each week created weekly training loads. In addition, the daily mean training load as well as the standard deviation of training load was calculated each week. The daily mean was divided by the standard deviation in order to determine training "monotony." Training "monotony" is an index of

training variability over the period of a week (4). "Strain" was calculated as the product of the weekly training load and monotony.

The software for analyzing these data represents a simple adaptation of the Excel spreadsheet. Simple plots of the number of weeks versus weekly training load and weekly training strain were constructed. The incidence of illness was noted and plotted together with the indices of weekly training load and weekly training strain. The correspondence between spikes in the indices of training and subsequent illness was noted. Another area that was evaluated was the weekly training load and weekly training strain versus the weekly complaint index. The product of the weekly muscular aches and pains in the evening and weekly state of mental well-being defined the weekly complaint index. Weekly training load and weekly training strain were plotted versus the weekly complaint index. Regression statistics were used to determine if there was a relationship between either training load and training strain and the complaint index.

RESULTS

It was a challenge to obtain continuous data from each athlete throughout the study. Often periods of two to four weeks would pass with no data being returned. Several options were attempted to receive the data, but many of the athletes were traveling quite frequently which may have caused problems with prompt data return. This challenge of obtaining continuous data has contributed to the results of this study.

Illnesses

With our approach of correlating the incidence of illnesses with indices of training, 4 illnesses of 12 (33%) could be explained by a preceding spike in training load. For the training strain, 3 illnesses of 12 (25%) could be explained by a preceding spike in training. Data for each subject are presented in Figures 1-10. The circles represent weekly training load and the squares represent time of illness. We attributed an illness to an unusually heavy training load (or strain) if the week immediately preceding the illness was $\geq$ 1sd above the mean training load (or strain).

Complaint index

There was a weak positive relationship between the training load and the complaint index (Appendix A 1-5) and the training strain and the complaint index (Appendix A 6-10) with weak to moderate correlations ($r = 0.35 - 0.71$). It was found that as a group there was a weak positive relationship between the training load and the complaint index (Figure 11) and the training strain and the complaint index (Figure 12). A pattern of an increase in training load accompanied by an increase in complaints was

similar among the athletes. There was no evidence for a threshold effect of the complaint index in relation to either training load (Appendix A 1 - 5) or strain (Appendix A 6 - 10).

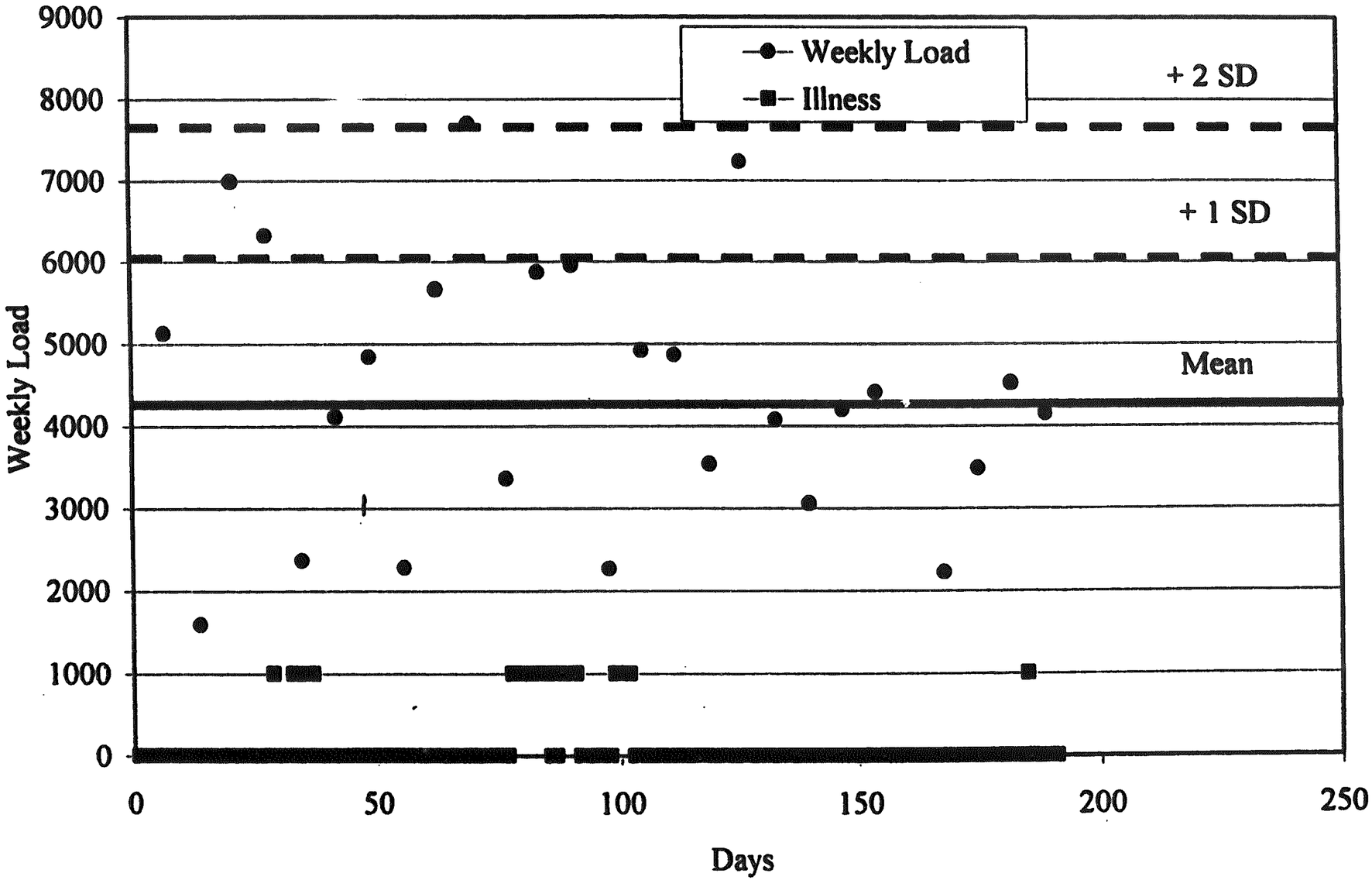

Figure 1. Weekly load and illness for subject 1

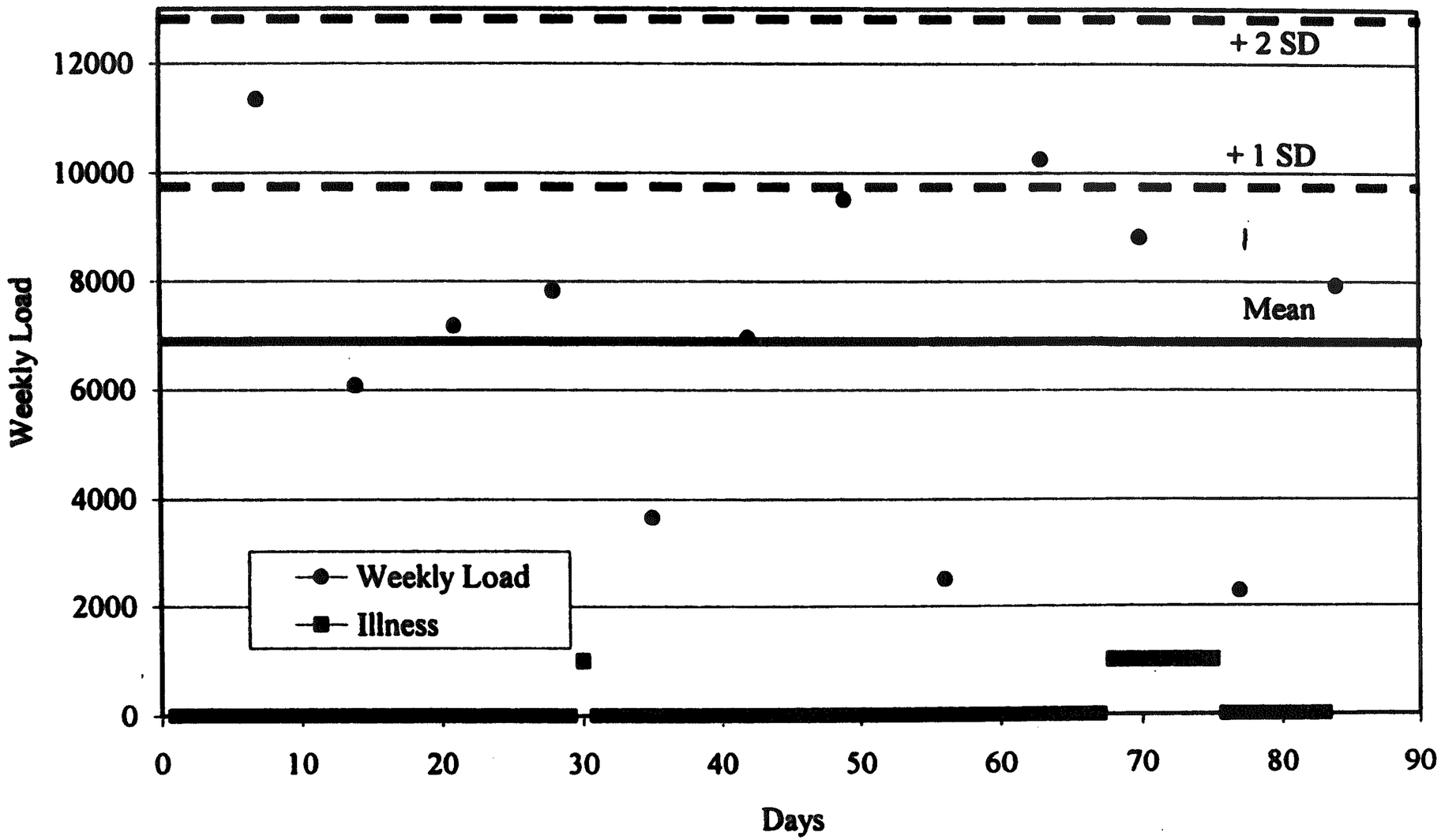

Figure 2. Weekly load and illness for subject 2

Subject 3

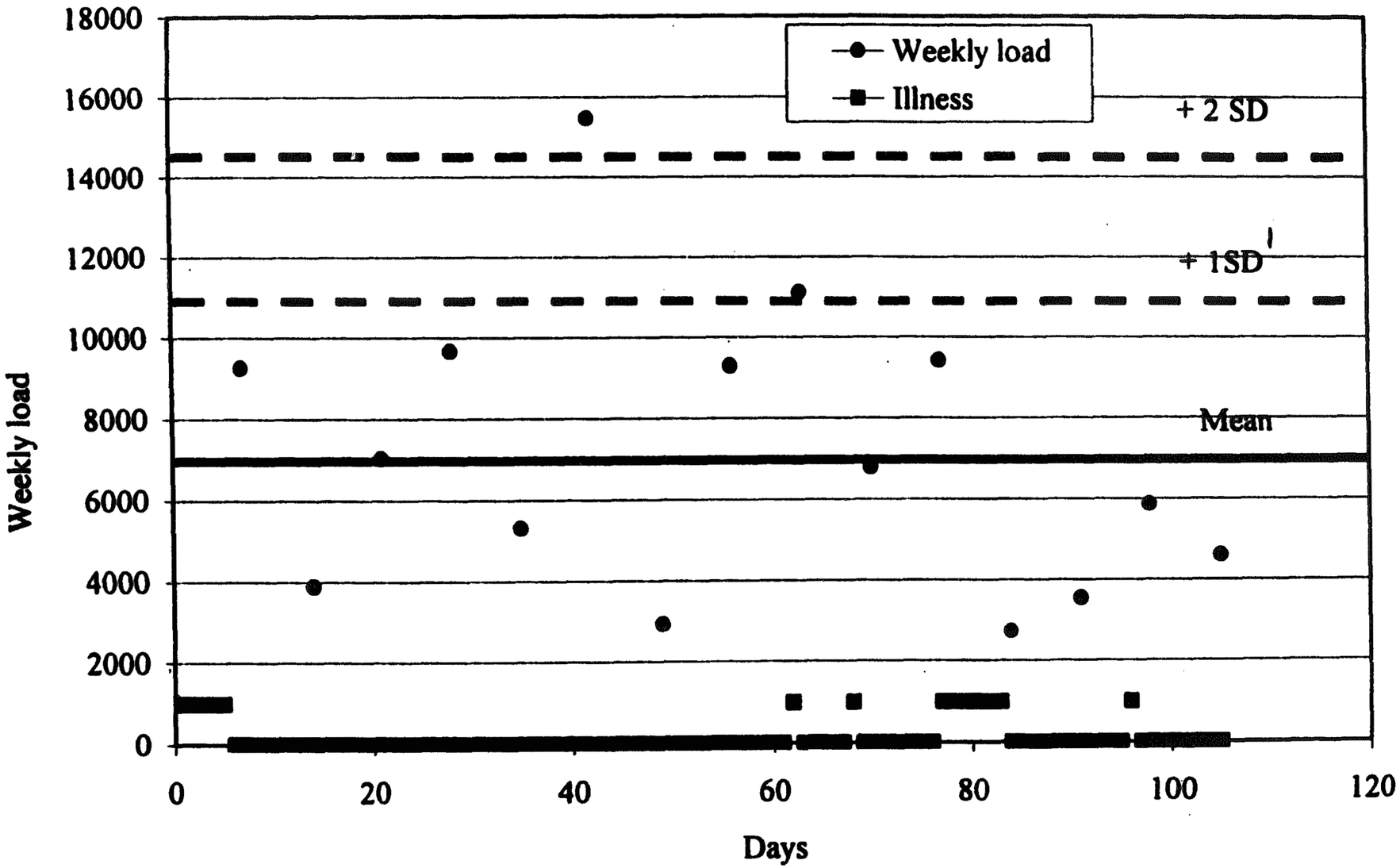

Figure 3. Weekly load and illness for subject 3

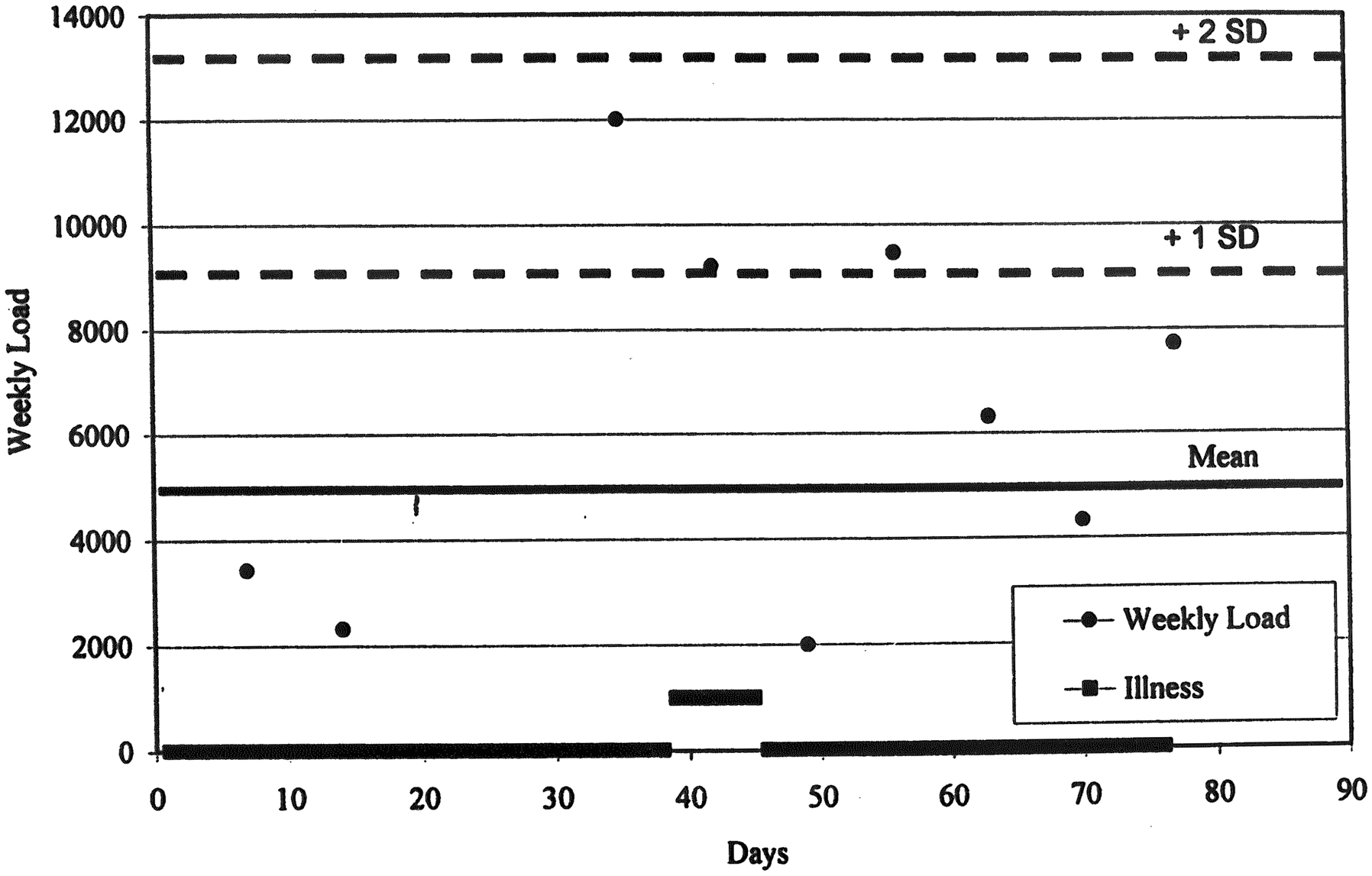

Figure 4. Weekly load and illness for subject 4

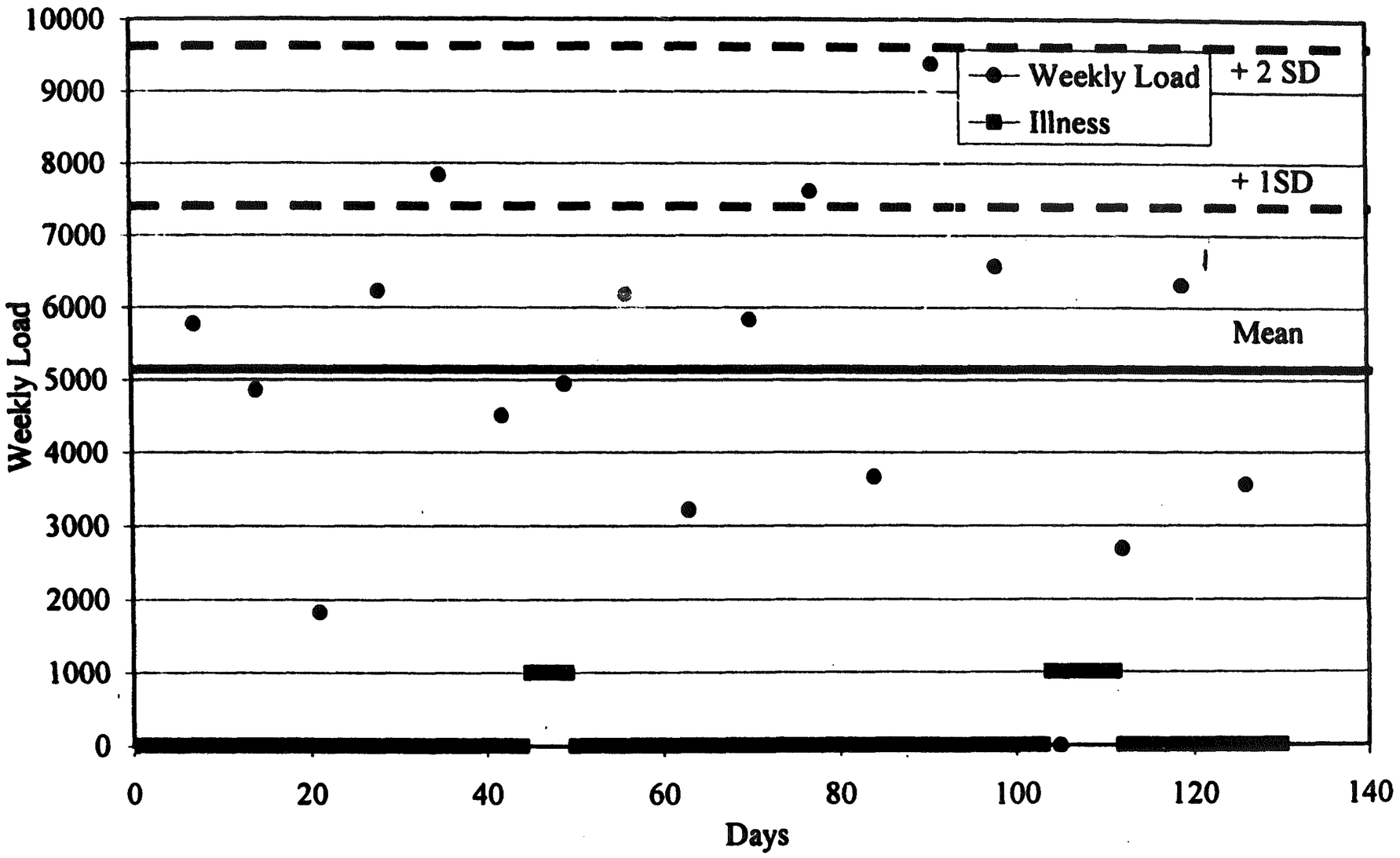

Figure 5. Weekly load and illness for subject 5

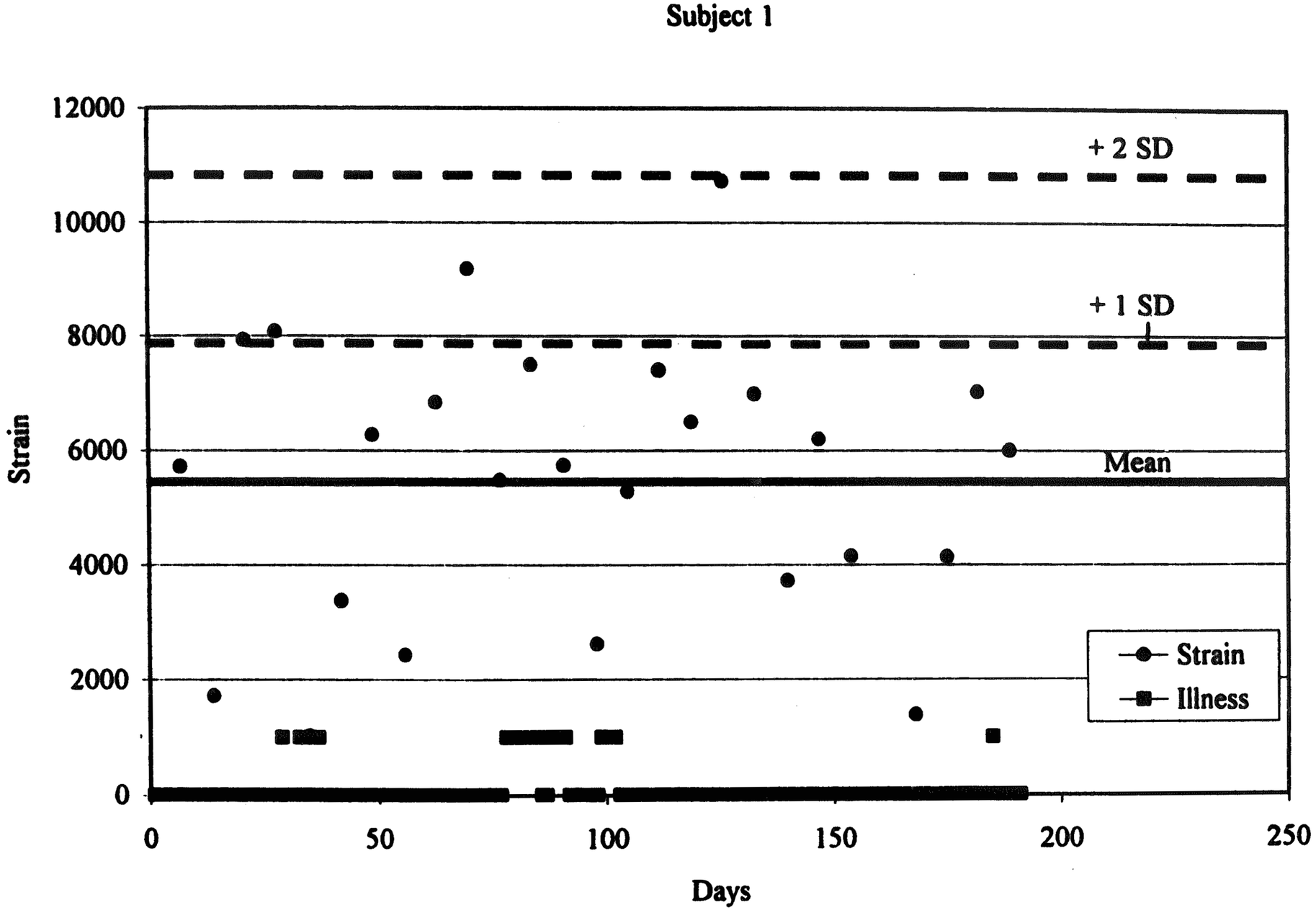

Figure 6. Weekly strain and illness for subject 1

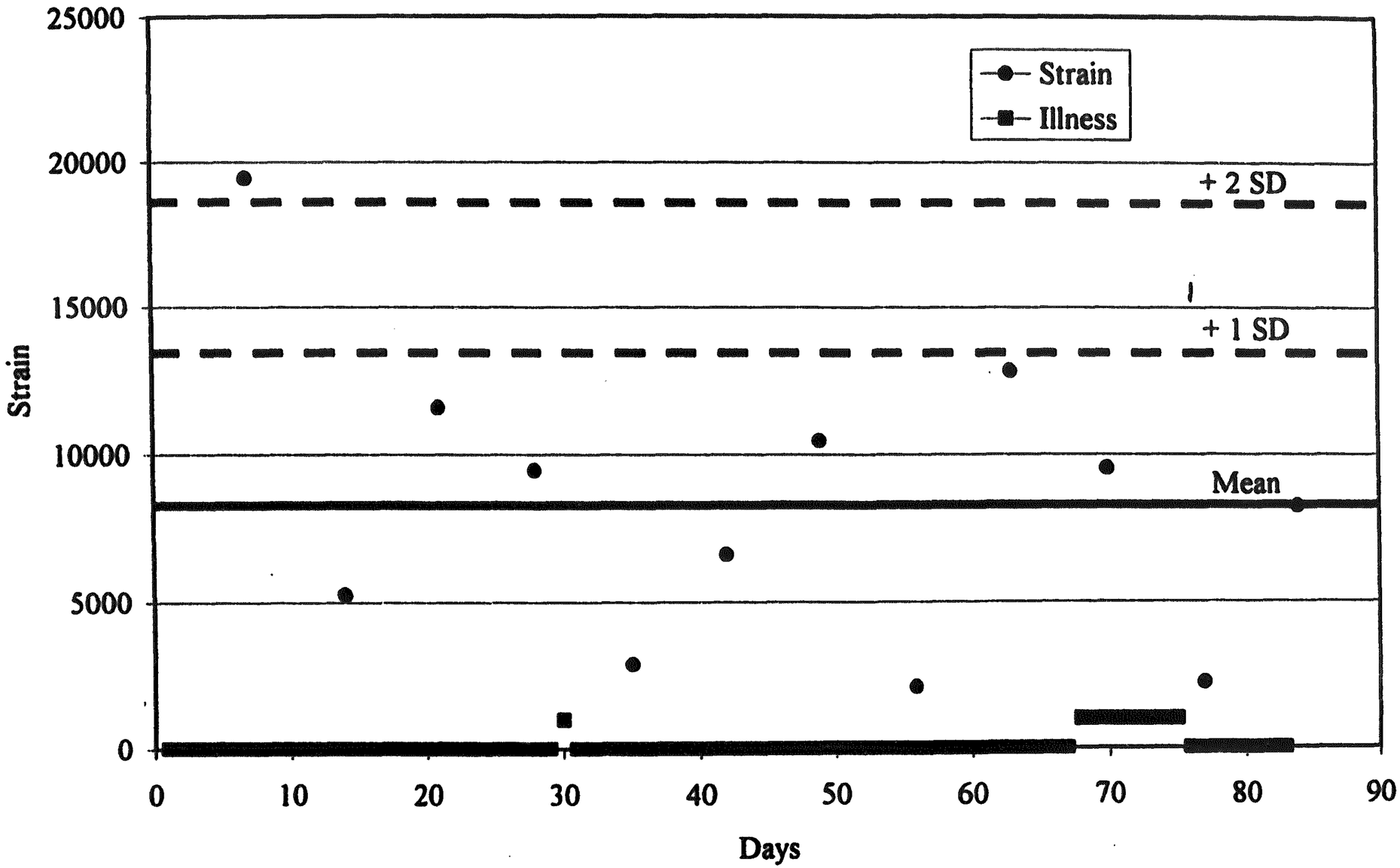

Figure 7. Weekly strain and illness for subject 2

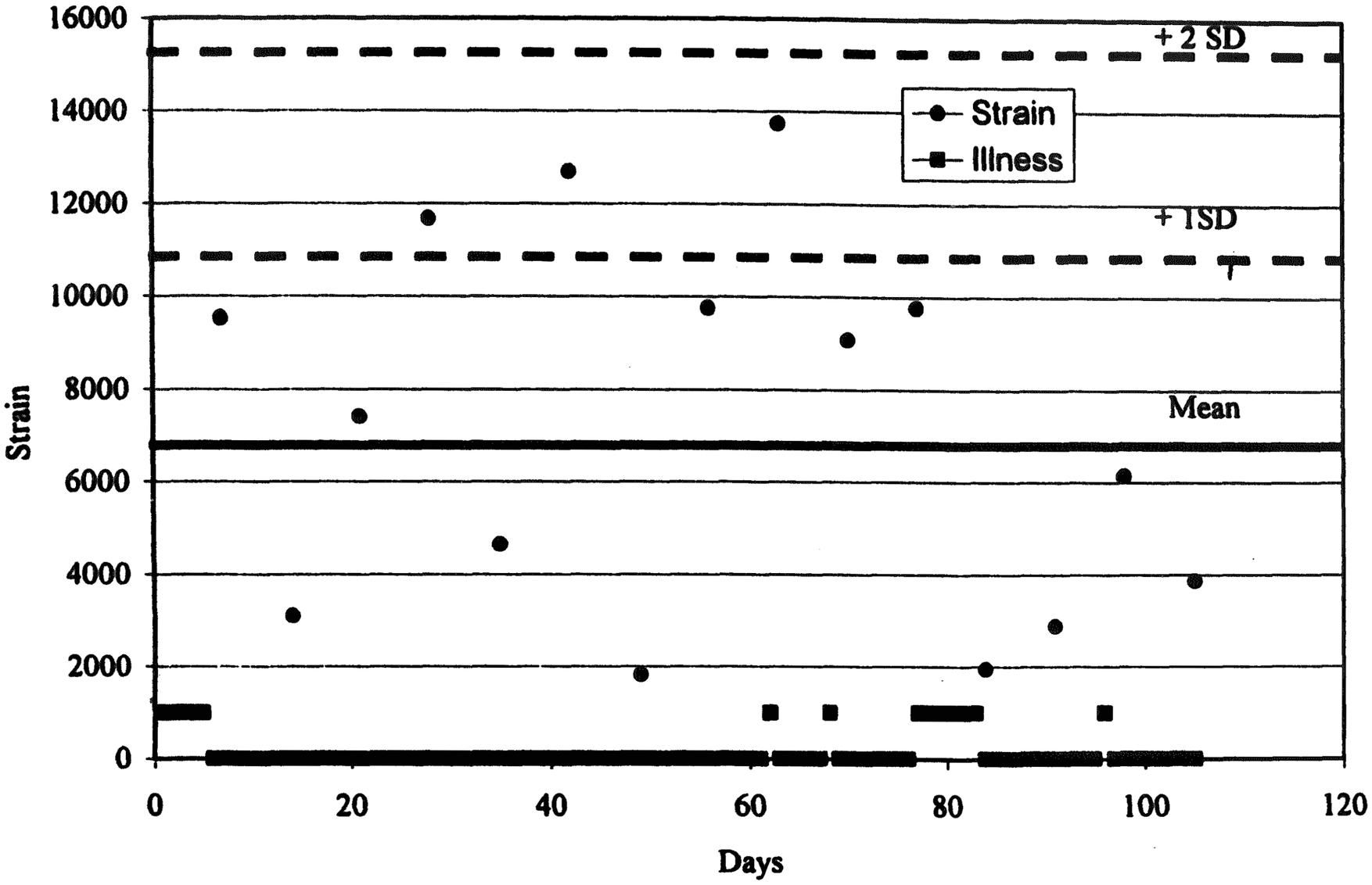

Figure 8. Weekly strain and illness for subject 3

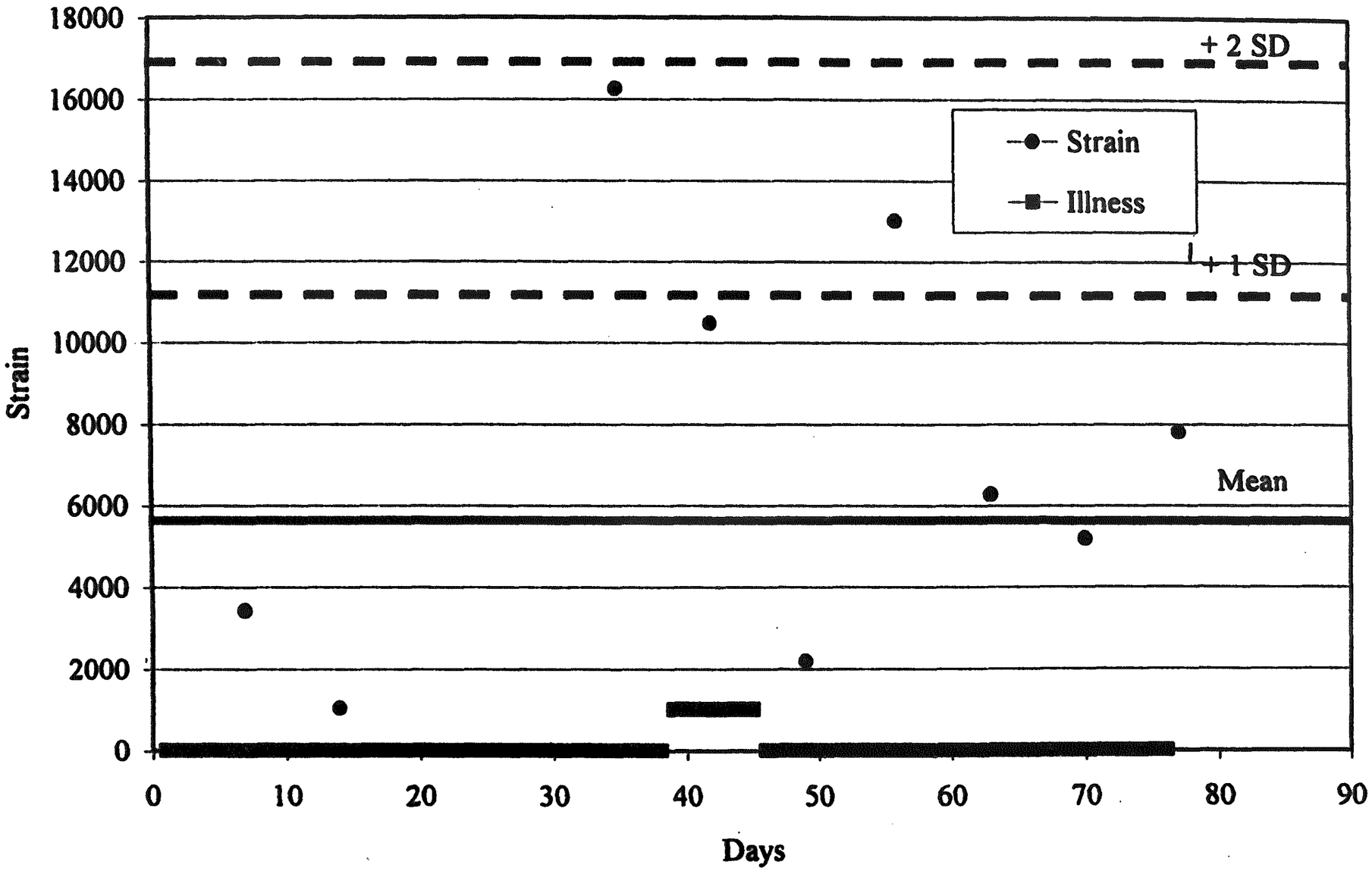

Figure 9. Weekly strain and illness for subject 4

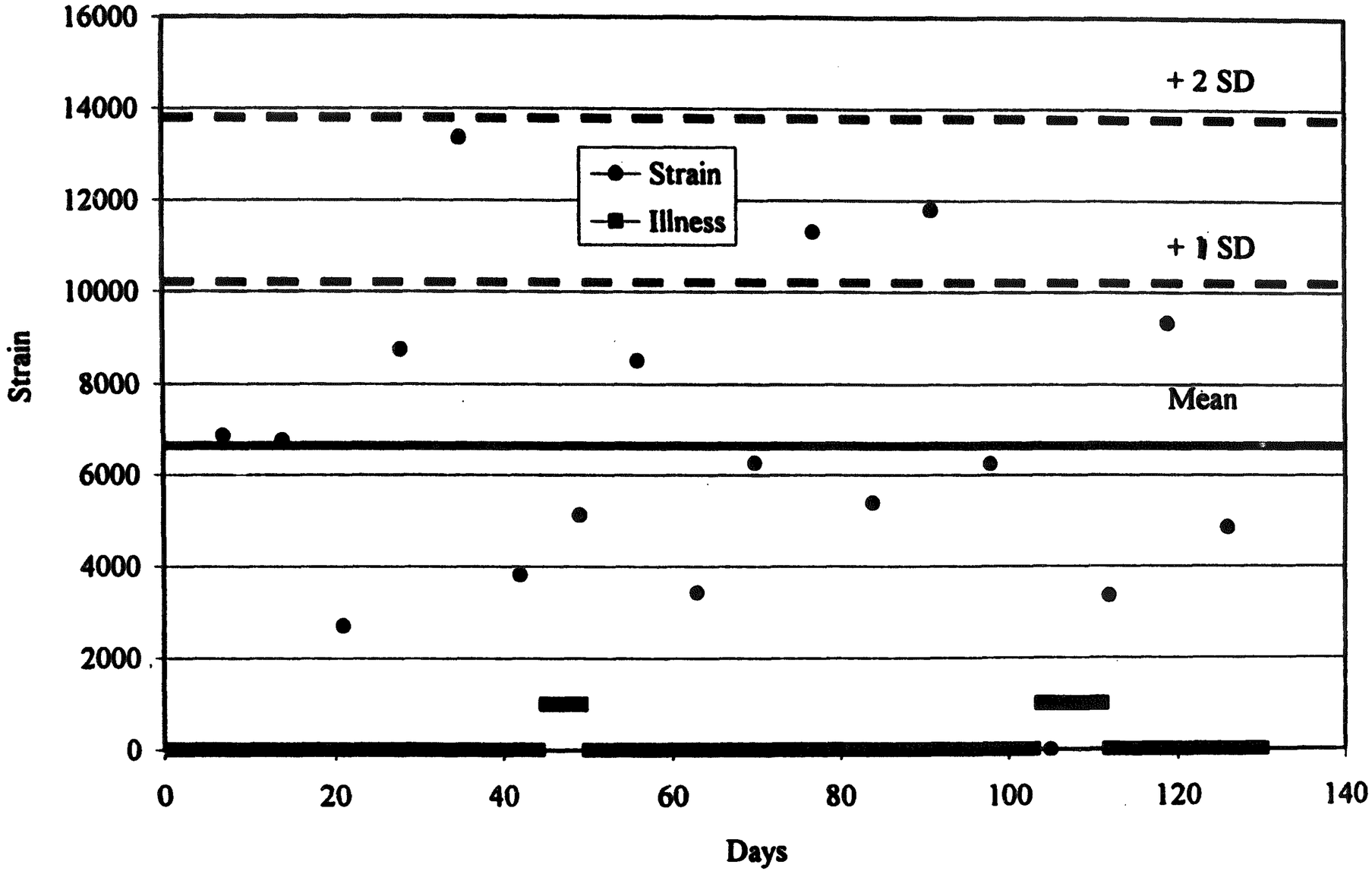

Figure 10. Weekly strain and illness for subject 5

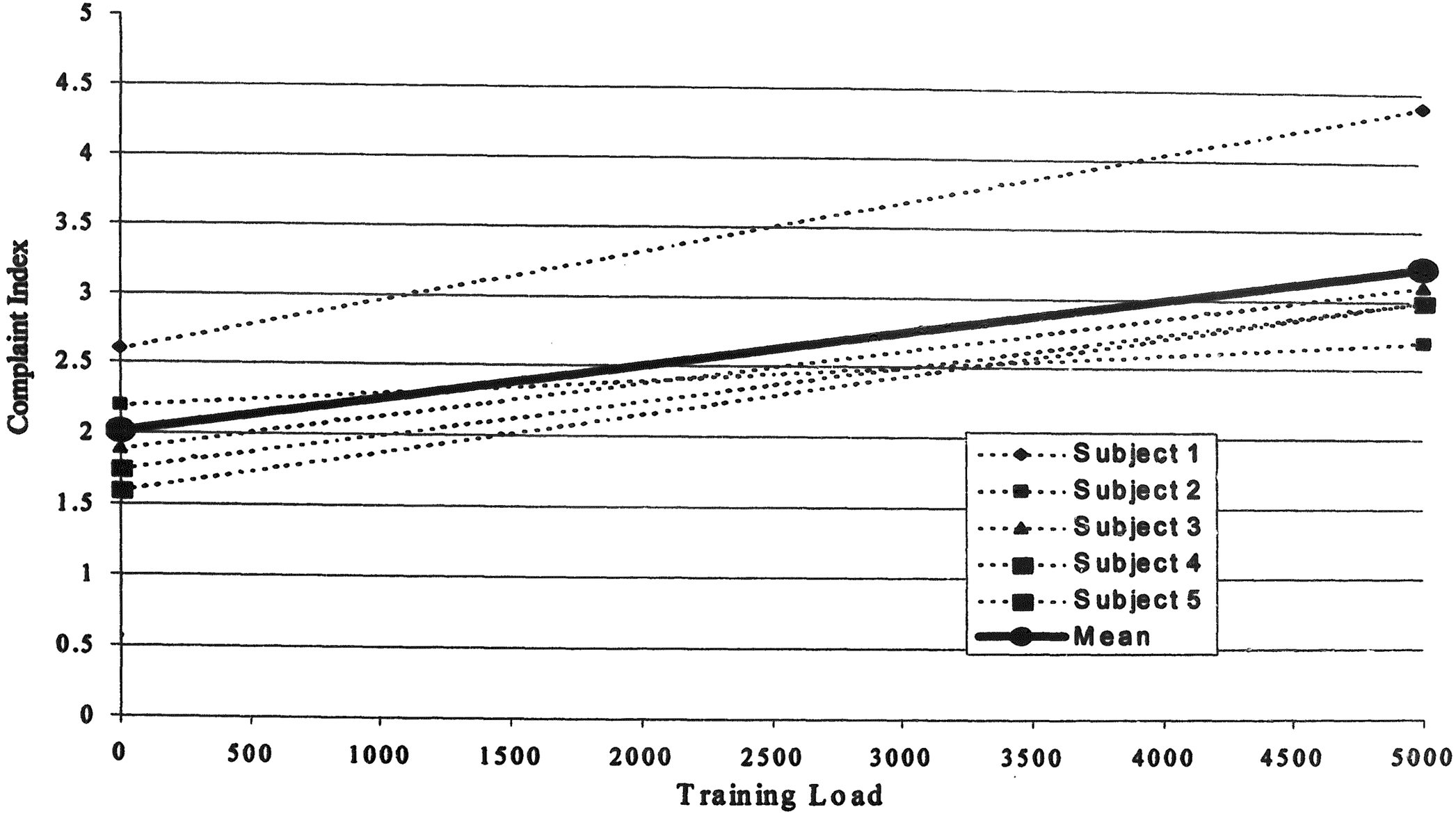

Figure 11. Group complaint index versus training load

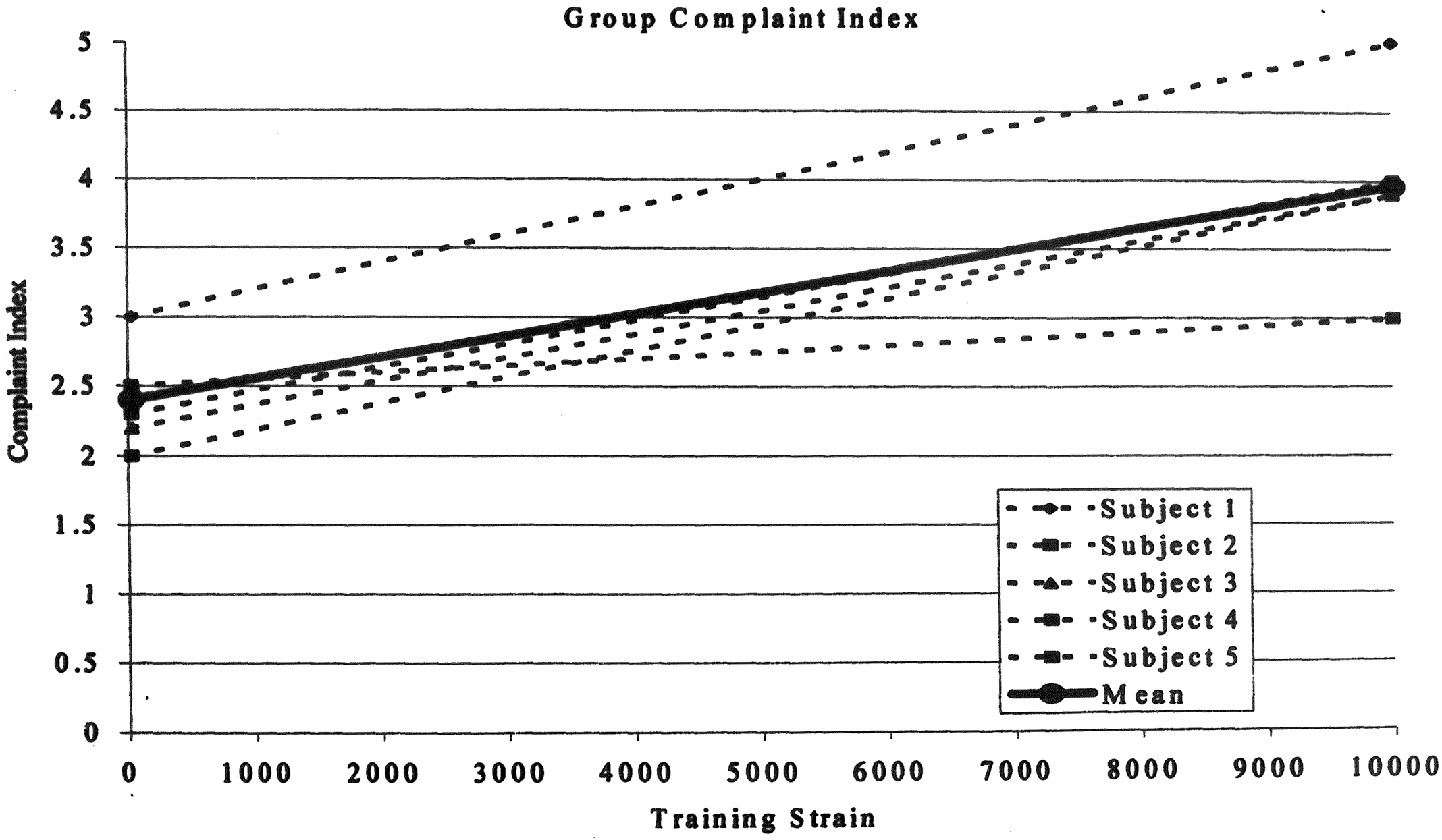

Figure 12. Group complaint index vs. training strain

DISCUSSION

Illness and injury are undesired outcomes that are relatively frequent throughout the career of an elite athlete. The primary purpose of this study was to make preliminary observations to determine if there was a relationship among indices of training and the incidence of illness and injury in elite speedskaters. The athletes recorded their training using a method that integrates the overall exercise session RPE and the duration of the training session. Measures of muscle aches and pains along with the athletes' state of mental well-being were also reported. Illnesses were noted and correlated with indices of training load and training strain. Weekly training load and weekly training strain were also compared to the weekly complaint index, calculated as the product of muscle aches and pains and mental well-being.

Throughout the seven-month study, there was a low overall incidence of illness and injury. The correspondence between spikes in the indices of training and subsequent illness was noted. Only a moderate relationship between training indices and the incidence of illness was found. Although many of the elite athletes reported various muscle aches and pains along with a lower state of mental well-being, only a moderate to weak relationship was observed between illnesses/injuries and the complaint index. Furthermore, there was no evidence of a threshold behavior.

Previous work using similar means of monitoring training in athletes has suggested that heavy, monotonous training may increase the likelihood of developing overtraining syndrome (4,5,6). Foster (4) found that a high percentage of

illnesses (84%) could be explained by individual athletes exceeding individually identifiable training thresholds. This suggested that simple methods for monitoring the characteristics of training may allow athletes to achieve the goals of training while minimizing the likelihood of developing OTS. It is highly probable that because of previous work with this population, the structure of the training program devised by the coaches included sufficient rest to prevent OTS despite high training loads.

Currently there is no universally accepted global hypothesis of OTS. In an attempt to integrate all of the present hypotheses into one, Smith (11) proposed the cytokine hypothesis of overtraining. This hypothesis suggests that repetitive micro-trauma to the muscular, skeletal, and/or joint systems is frequently the initiator of OTS. As an athlete continues to train and compete, adaptive microtrauma occurs in the muscles. Rest days and recovery are essential for this damage to heal. Often times, an athlete continues to train and receives insufficient rest and recovery. This may result in local acute inflammatory responses which may lead to chronic inflammation and initiate a cluster of behaviors associated with OTS.

In this study, many athletes reported various muscle aches and pains along with a lower state of well-being, but no definable OTS was observed. Difficulty in acquiring data and the small sample size may explain why no OTS was observed. A lack of cooperation from the athletes in returning data may have confounded the outcome. It was a challenge to obtain continuous data from each athlete throughout the study. Often periods of two to four weeks would pass with no data being returned. Many options were tried in an attempt to receive the completed data. Athletes had the choice of

communicating through e-mail, the postal service, and the telephone. In addition, due to training and competition, many of the athletes were traveling quite frequently, often outside the U.S. This may have caused problems with returning data promptly and may explain why some athletes discontinued returning data all together.

Although these elite speedskaters did not reach a training threshold that would induce OTS, previous work (4) has shown a relationship between indices of training and the incidence of illness. Many of the behaviors associated with OTS may be linked to the inflammatory response induced by excessive musculoskeletal stress. Although no OTS was evident, the results of this study demonstrate a potential approach to testing the cytokine model of OTS. This study failed experimentally possibly as a result of difficulty in receiving data. It is also highly probable that because of previous work with this population that the structure of the training program devised by the coaches may have prevented long-term inflammatory responses that are proposed progenitors of OTS. If the problems with data collection can be solved, then repeating this study may provide more meaningful results.

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APPENDIX A

FIGURES 1-10

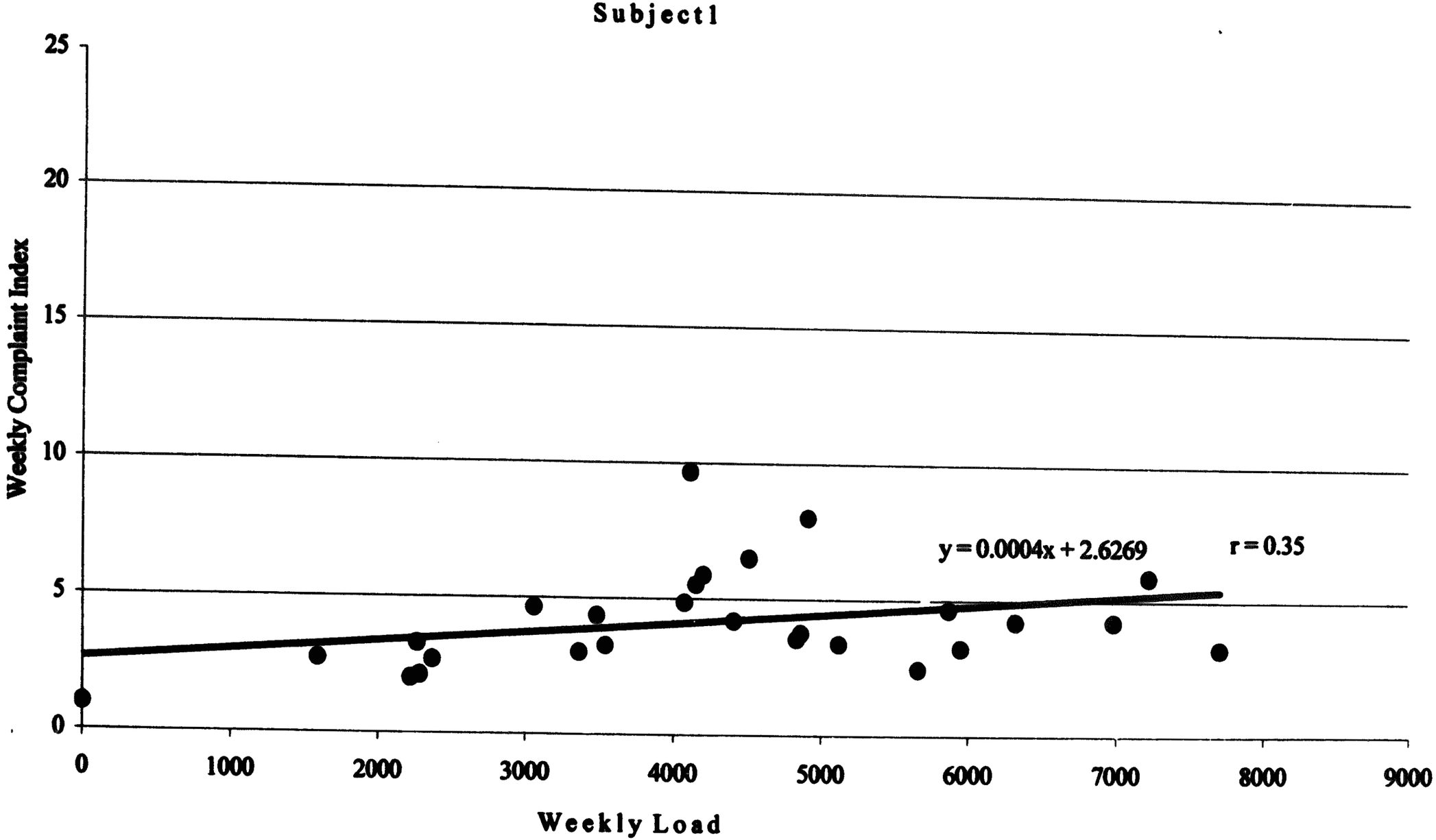

Figure A-1. Weekly complaint index versus weekly load

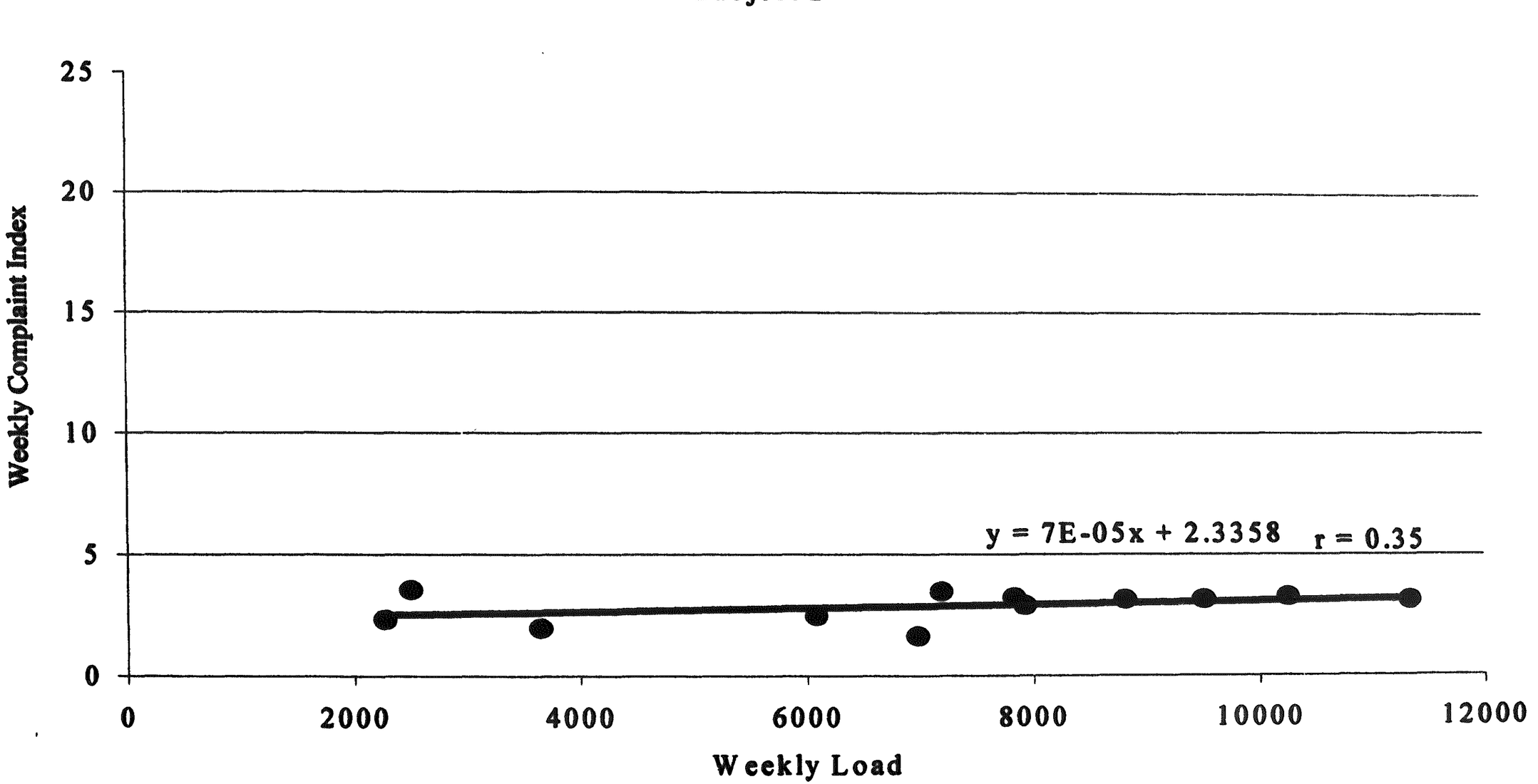

Figure A-2. Weekly complaint index versus weekly load

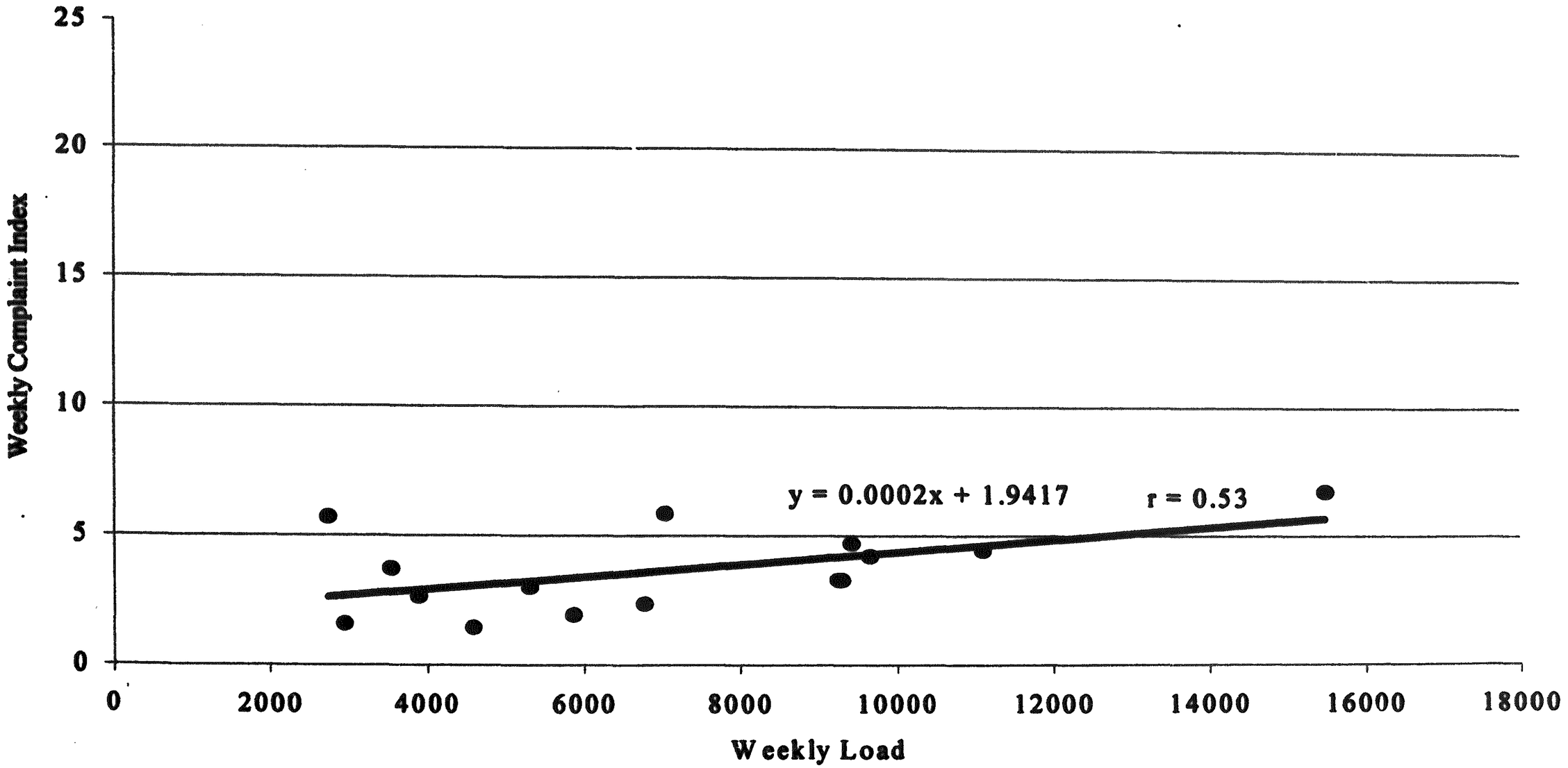

Figure A-3. Weekly complaint index versus weekly load

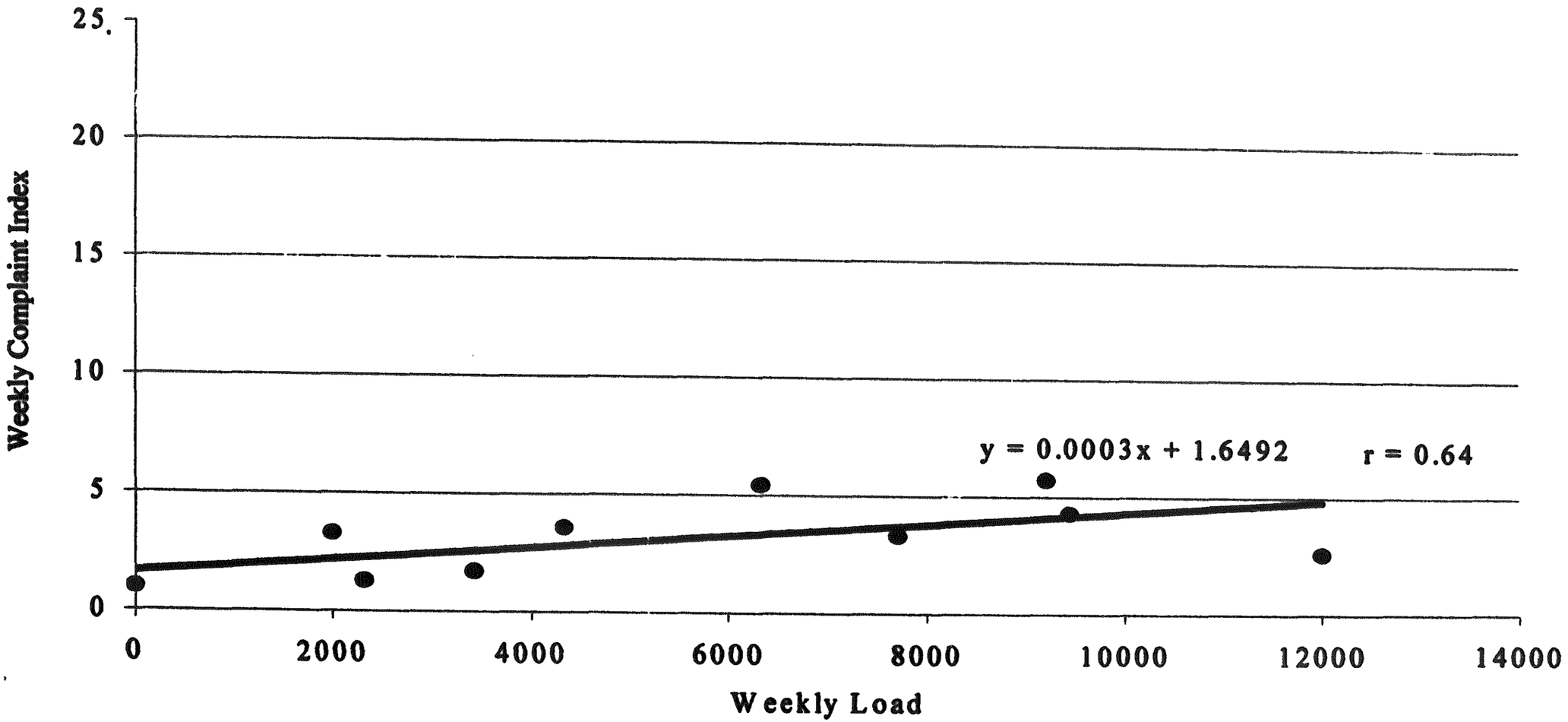

Figure A-4. Weekly complaint index versus weekly load

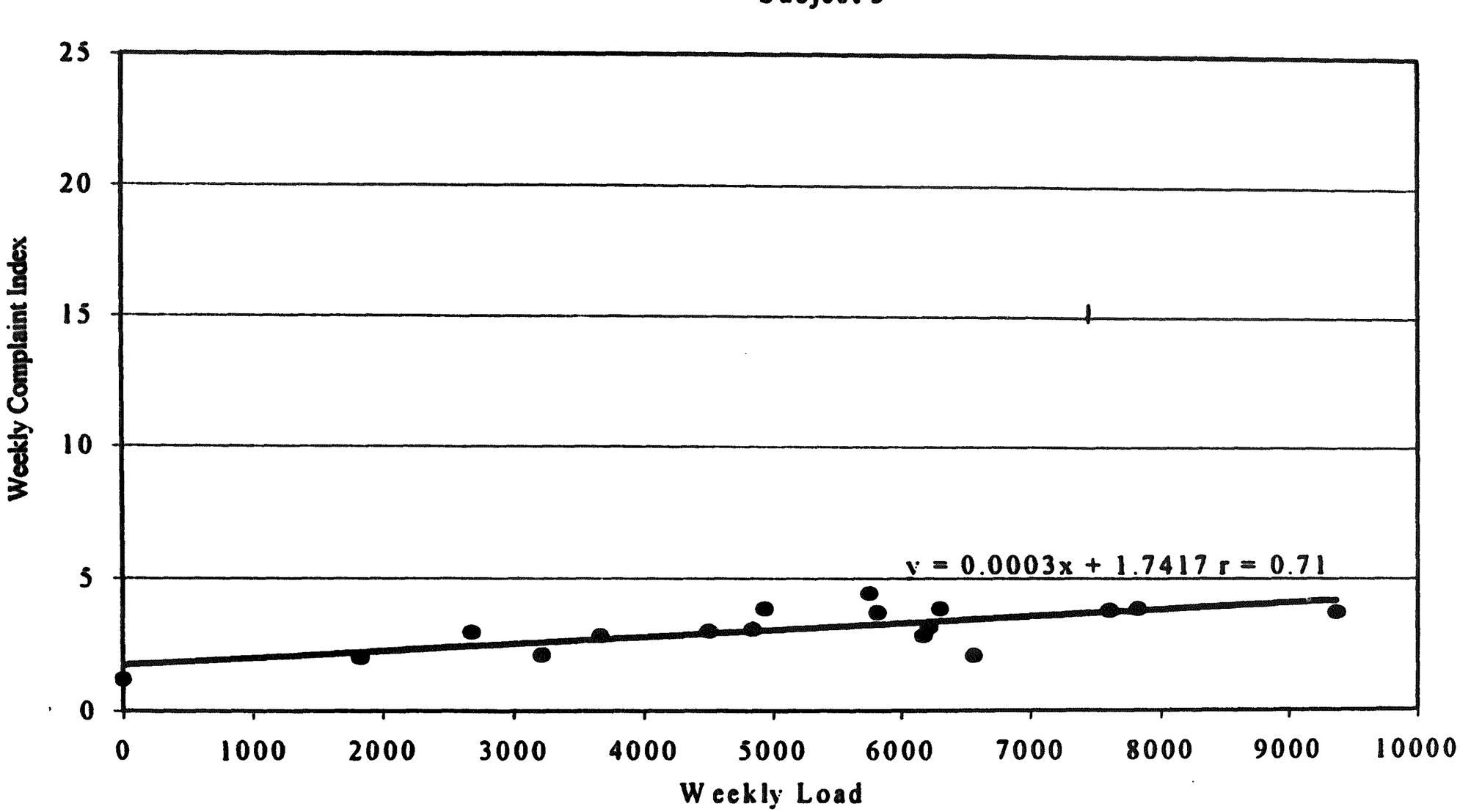

Figure A-5. Weekly complaint index versus weekly load

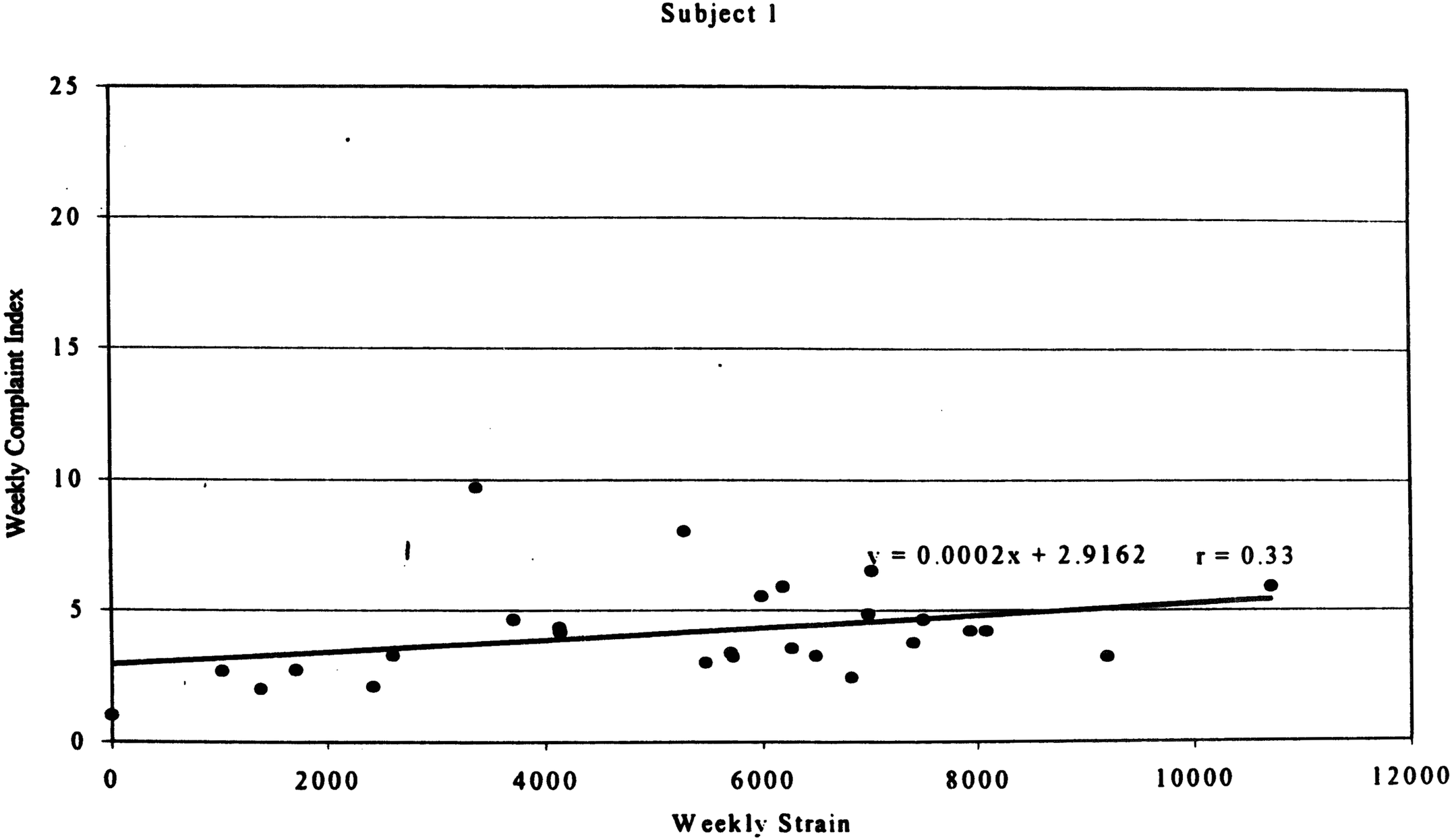

Figure A-6. Weekly complaint index versus weekly strain

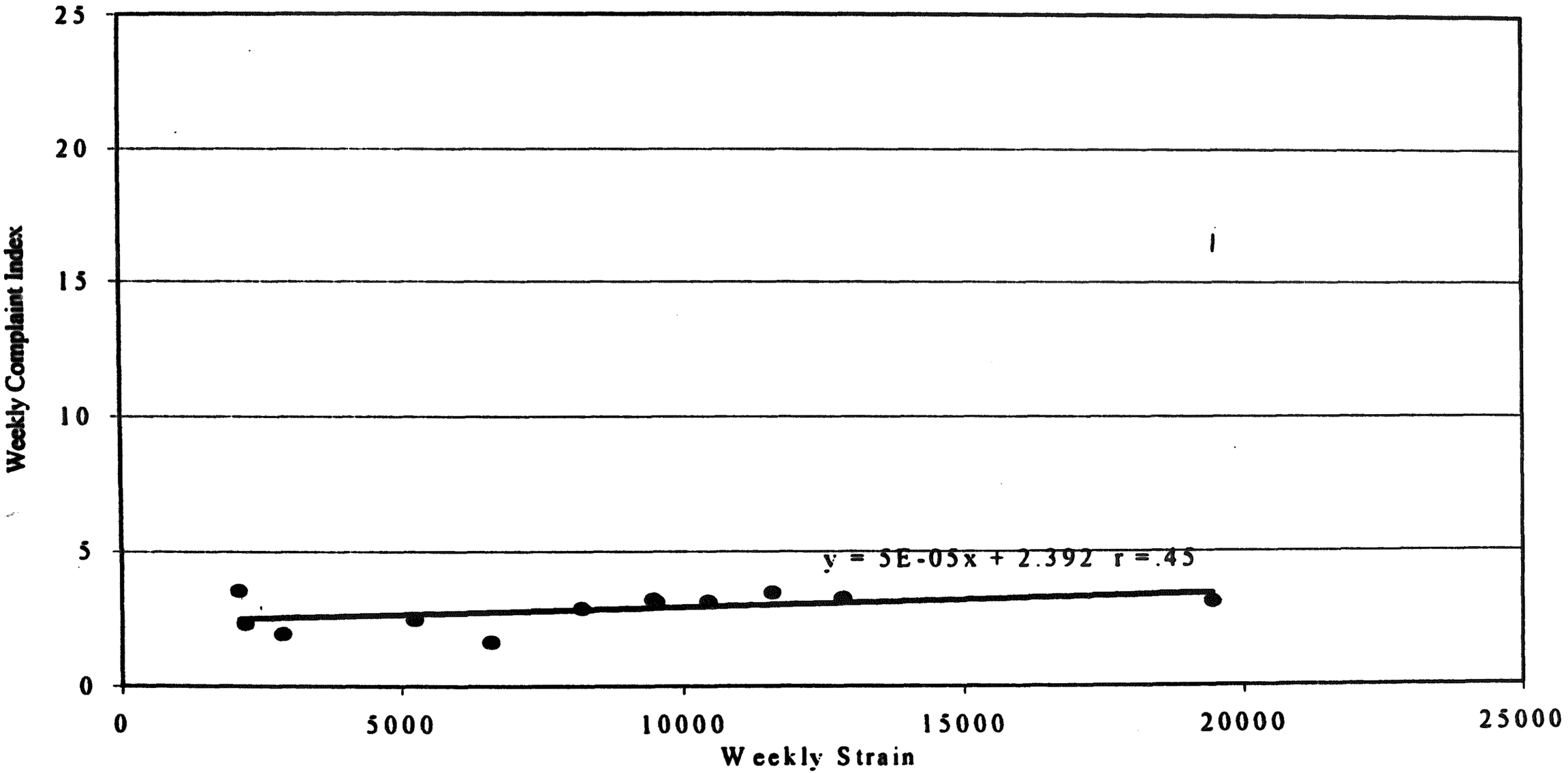

Figure A-7. Weekly complaint index versus weekly strain

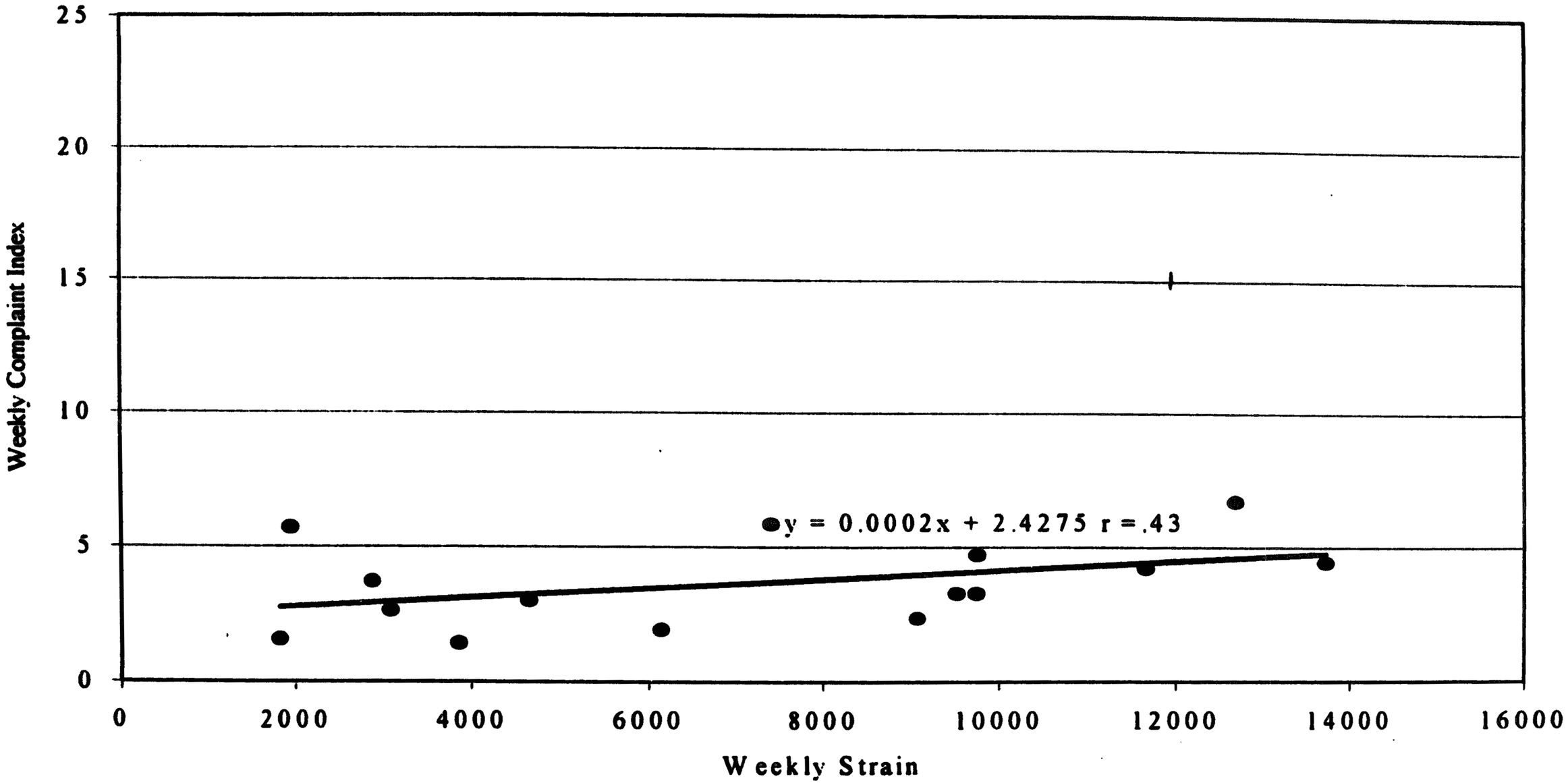

Figure A-8. Weekly complaint index versus weekly strain

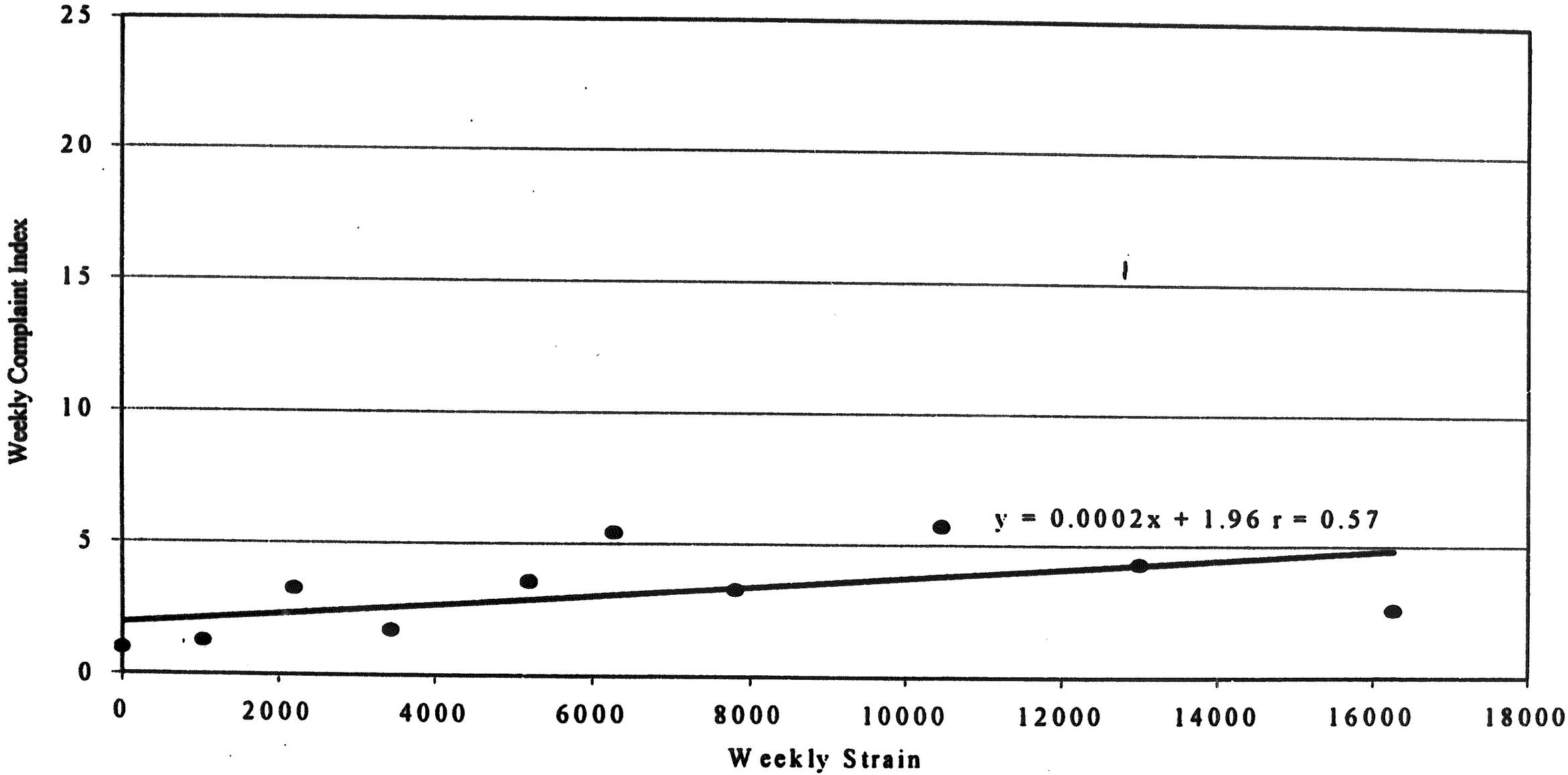

Figure A-9. Weekly complaint index versus weekly strain

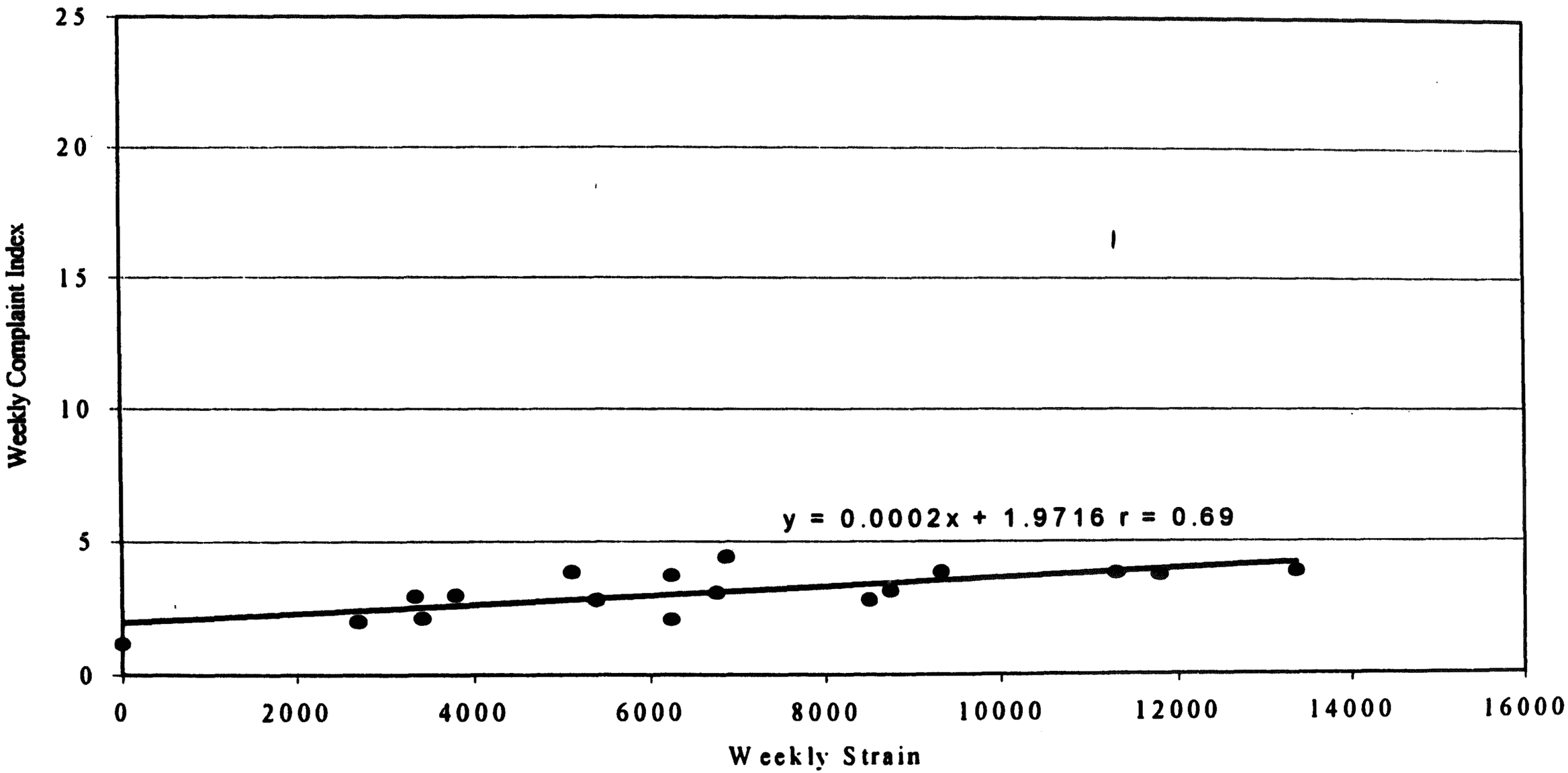

Figure A-10. Weekly complaint index versus weekly strain

APPENDIX B

INFORMED CONSENT

Informed Consent For:

Relationship among Indices of Training and the Incidence of Illnesses and Injuries

I, _____ give my informed consent to participate in this study designed to monitor the training of athletes. This study is being conducted to acquire information pertaining to the training programs experienced by athletes and how well this matches to the training program designed by the coach. I consent to the publication or presentation of results as long as identity is not released.

I have been informed that the only risks associated with this study are within the scope of training including fatigue, muscle soreness and injuries. There are no foreseeable risks involved with the recording of training data.

I have been informed that by completing the training log-questionnaire I will be provided with information that could eliminate some common mistakes in training and this information will allow my coach to design a better training program for me.

I have been informed that I will be required to complete a daily training log-questionnaire, which could take 10 minutes to fill out. The training log-questionnaire involves reporting on intensity and duration, along with questions regarding illness, injury, muscular aches and pains, and state of mental well-being. Data collection will begin July 2000 and continue through March 2001.

I have been informed that the training logs will be in the investigator's possession at all times. Discussion may occur between the investigator and the research advisor in order to configure the data for adequate completion of the project.

I have been informed that any questions I have in the participation of this study I can contact Amy Cimbalnik, Graduate Student in the department of Exercise and Sport Science, UW-La Crosse or Dr. Carl Foster, Faculty Research Advisor in the ESS department at UW-La Crosse. Questions regarding the protection of human subjects may be addressed to Dr. Garth Tymeson, Chair, UW-La Crosse Institutional Review Board (608) 785-8155.

I have been informed that my participation is voluntary and I may withdraw from this study at any time without penalty.

Participant's Signature: _____ Date: _____

Parental Signature: _____ Date: _____
(If less than 18 years of age)

Investigator: _____ Date: _____

APPENDIX C

REVIEW OF RELATED LITERATURE

REVIEW OF RELATED LITERATURE

Introduction

The life focus of an elite athlete is not only competition but also, very importantly, training for competition. It is believed that with increased training levels, athletic performance will improve. When the athlete demands more than his/her body can tolerate, performance often begins to deteriorate. When a deterioration in performance occurs in association with an intensive training schedule, a phenomenon known as overtraining syndrome occurs (OTS) (32).

Overtraining can be classified as short-term and long-term overtraining (22). Short-term overtraining is represented by a decrease in performance capacity and training fatigue lasting a few days up to two weeks. Long-term overtraining is characterized by a disturbance of mood state, a reduction in maximum performance capacity, and muscle soreness lasting weeks up to months (22). Athletes that suffer from OTS are also often predisposed to injury and illness.

It has been suggested that by monitoring the characteristics of training, the athlete should be able to achieve the goals of training while minimizing undesired training outcomes such as injury, illness, and deterioration in performance (15). In the past it has been difficult to do this due to a lack of an adequate method to quantify training load. Bannister et al. (3) developed the training impulse (TRIMP) concept of calculating training, based on integrating the intensity and duration of each day's training. The method has been modified by others including Busso et al. (9), Mujika et al. (28),

Foster (15), and Foster et al. (16,17). Foster (15) and Foster et al. (16) have used a simple modification of the rating of perceived exertion (RPE) scale to represent the global intensity of the entire training session by multiplying the duration of the training session by the RPE to derive the training load of that session. Training monotony, training strain, and negative adaptations to training may also be derived. Training monotony can be determined by dividing the daily mean load by the standard deviation over a week. Training strain can be calculated by multiplying the weekly training load by the monotony. Foster (15) found that negative adaptations to training were mostly related to training strain. Monitoring of training loads may be the way to optimize athletic performance by allowing the coach or athlete to visualize the relatively positive (load) and negative (strain) aspects of the training program.

The following provides a review of literature pertaining to: 1) the development and expansion of general strategies for the monitoring of training; 2) hypotheses related to the development of overtraining syndrome; 3) the relationship between illness and OTS; and 4) the relationship between injuries and OTS.

General Strategies for the Monitoring of Training

Aerobics Point System

In an attempt to understand the training response, it would be appropriate if there was a method that integrated the effects of intensity, duration, and frequency into a single number representative of the "input"of a training session. In return, this "input" might result in a particular "output" or change in performance. In 1968, Cooper (11) developed the "aerobics" point system in an attempt to reach this goal. Aerobic exercises were

quantified by means of a point system. The "aerobics" point system was based on absolute intensity and duration of several different physical activities. This estimate of the absolute exercise intensity was multiplied by duration to reach a single number or "points" which represented the "input" from the training session. The "aerobics points" system was a very popular approach to monitoring exercise due to its simplicity and was a central factor in the emergence of wide public interest in fitness beginning in the late 1960s. However, since relative training intensity dominantly influences training responses, the use of absolute training intensities limited the usefulness of the "aerobics points" system of monitoring training (14).

<u>TRIMPS System</u>

In the early 1990s, Morton et al. (27) and Fitz-Clarke et al. (13) developed the concept of the training impulse (TRIMP). The TRIMP was seen as an effective way to integrate the intensity (represented by average % heart rate reserve) and duration of training. This concept was paired with an understanding of directionally opposite effects on fitness and fatigue created by any particular TRIMP. It was recognized that a TRIMP contributes to both fitness and fatigue, and performance is related to the difference between these two quantities (13). Performance can be determined by summing the interaction between fitness and fatigue. Although the TRIMP concept is very useful for expressing the training load undertaken by an athlete, it does have some limitations. The intensity component of the TRIMP approach is dependent on heart rate recordings. Given the availability of electronic HR monitors, recording and evaluation of data are not difficult. However, if an athlete forgets to wear the monitor or if there is a malfunction of

the monitor, no data will exist for that training session. A second limitation is that during very high intensity exercise, HR fails to represent the total input of the exercise load.

The TRIMP concept has been extended by Busso et al. (10) and Mujika et al. (28). Replacing the average percentage of HR reserve, intensity was represented by a rating system based on percentages of maximal possible performances. Mujika et al. (28) studied the effects of training on performance and assessed the response to tapering. Fitness indicators were estimated from the positive influences of training (e.g., Load) while fatigue indicators were estimated from negative influences of training (e.g., fitness-fatigue). A reduction in negative influences resulted in an improvement in performance during taper. No improvement was seen in the positive influences during taper. Additionally, positive influences did not change significantly during the taper (28). This showed that the TRIMP concept is a valuable method to describe the interaction of training on performance (28).

Busso et al. (10) also expanded the TRIMP concept by studying the effects of training on performance using time-varying parameters. Variations over time allowed for changes in responses to training to be explained. Busso et al. (9) found that a systems model composed of fitness and fatigue could account for performance changes observed during training. Additionally, a time-varying model proved to be a valuable tool for providing a way of studying adaptations to training (10).

Rating of Perceived Exertion

The TRIMPS concept was further extended by Foster et al. (17) who developed a method of monitoring training based on using the category ratio version of Borg's Rating

of Perceived Exertion scale (4). By replacing the average percentage of HR reserve, the modification of the Borg RPE scale represented the training intensity. The session RPE, a single number that represents the overall intensity of a training session, is collected from the athlete after each training session. The session RPE is then multiplied by the duration of the session which results in the training load. This training load can then be summated over a week to derive a single number that represents the TRIMP that the athlete accumulated over the week. It has been shown that the session RPE corresponds to the average percent heart rate reserve, to the HR-blood lactate relationship, and to the summated HR score (18). Foster et al. (16) used this method of monitoring training to relate training load changes to performance. They noted that across a wide range of training loads that a 10-fold increase in training level was associated with a 10% improvement in cycling time trial performance.

It was also suggested that total training load and a lack of training variability (monotony) is associated with the development of illness and/or injury seen in OTS (15). Foster and colleagues (17) created a method to quantitatively measure the training "monotony" with the use of the training load calculation. The training monotony is calculated by taking the daily average load and dividing it by the standard deviation for the week. In addition to high training monotony, high training loads have been linked to negative adaptations to training (15). Therefore, Foster suggested that the product of training load and training monotony would determine the training strain. It was found that the training strain accounted for a high percentage of illnesses in individual athletes.

The simple expansion of the RPE method of monitoring characteristics of training allows an athlete to achieve goals while decreasing the chances of negative outcomes.

The RPE method for monitoring training developed by Foster (15) has also been used and refined by Busschaert and colleagues (8). Busschaert et al. (8) conducted a study that involved using a training log that included physical, physiological, and psychological parameters. The purpose of the study was to create a quantification of training with the use of the training log. Busschaert et al. (8) refined the RPE method of monitoring of training by adding muscle soreness and mental well-being into the formula for global daily training activity. It was found that the training log created a tool to predict overtraining.

Overtraining

Athletes train excessively and at high intensities in an attempt to optimize athletic performance. Many believe that with increased training levels and practice frequencies, athletic performance will increase. There comes a time though, when the athlete demands more than the body can deliver. During this time, the athlete's training includes high volume and high intensity with insufficient recovery time. It has been argued that this type of training predisposes the athlete to muscular, skeletal, and/or joint trauma (32). An increased volume or training load combined with insufficient recovery often results in a decline in performance. This decline in performance is considered the universal criterion associated with overtraining (32).

Lehmann et al. (22) define overtraining as an imbalance between training and recovery, exercise and exercise capacity, stress and stress tolerance. Other signs and

symptoms that may be evident before the decline in performance include muscle aches and pains, illness, fatigue, and depression (32). There are many symptoms that have been associated with overtraining. Fry et al. (20) categorized these symptoms into groups which include psychological processing, physiological performance, immunological, and biochemical parameters. Some of the symptoms include emotional instability, loss of appetite, muscle damage, headaches, increased susceptibility to illnesses, colds, and allergies, and hypothalamic dysfunction. It is obvious that overtraining cannot be defined by one symptom. Rather, several symptoms in various combinations accompany overtraining. It is this cluster of symptoms coupled with the absence of any clear pathophysiologic antecedent that is referred to as overtraining syndrome.

Presently, there is no single accepted global hypothesis for the genesis of OTS. Several hypotheses have been proposed to explain the cause of OTS. Areas of focus have included the role of the hypothalamus, sympathetic and parasympathetic imbalances, catabolic and anabolic imbalances, blood levels of the amino acids glutamine and tryptophan, glycogen depletion, and a lack of day-to-day variation in training.

Lehmann et al. (21) studied the role of the hypothalamus in relationship to OTS (21). Lehmann et al. (21) proposed that neuroendocrine dysfunction may be a contributing factor to OTS. The hypothalamus plays a major role in neuroendocrine function by regulating blood levels of stress hormones such as cortisol, epinephrine, and norepinephrine (32). In addition, it also regulates the gonadal hormones, testosterone and estradiol. When an athlete is training appropriately, the hypothalamic-pituitary axis is stabilized. On the other hand, when an athlete undergoes extreme stress from intense

training and competition the hypothalamic-pituitary axis may become altered resulting in changes in the concentrations of blood stress hormones and gonadal hormones. Reduced and abnormal release of cortisol, adrenocorticotropic hormone, growth hormone, and prolactin have been reported in overtrained runners after insulin induced hypoglycemia (19). Therefore, changes in circulating concentrations of both stress and gonadal hormones have been associated with OTS (20).

Further support for the hypothalamus-pituitary dysfunction is development of amenorrhea in female athletes. Ovarian function becomes impaired due to a decrease in pituitary hormone secretion. This further results in an abnormal hypothalamic control of gonadal hormones (20).

Sympathetic and parasympathetic imbalances have been associated with the genesis of OTS (20). Lehmann and Foster (19) found that a decrease in intrinsic sympathetic activity resulted in a 40-70% decrease in nocturnal urinary catecholamine secretion in overtrained runners and soccer players. Catecholamines function to effect powerful physiological responses (6). A decrease in catecholamines will not allow an athlete to respond or adapt to training. In many ways, this is a type of physiologic beta blockade.

Catabolic and anabolic imbalances may have a role in OTS. After sustained, heavy exercise a catabolic phase occurs. This catabolic phase represents a decrease in tolerance to exercise. It is during this phase that biochemical and hormonal changes occur. Following the catabolic phase, an anabolic phase occurs. It is during this phase that the body increases its adaptation to stress. The ultimate result of this increase in

adaptation to stress is an increase in performance levels. When training levels become too intense or too frequent, an imbalance between the catabolic and anabolic phases may occur resulting in a decreased tolerance to exercise. Changes in circulating hormone concentrations have been shown to affect the rate of recovery after exercise (20).

The hormones testosterone and cortisol play a critical role in the catabolic and anabolic balance. Aldercreutz and colleagues (1) have shown that the reduction of cortisol production and elevation of testosterone is of prime importance for anabolic processes. Intense exercise leads to increased cortisol and decreased testosterone secretion (20). This results in an unfavorable testosterone/cortisol balance, which causes catabolic effects. This ratio has been associated with the catabolic state that is reported in overtraining (20).

The development of OTS may be attributable to an amino acid imbalance. Investigators (29) have specifically focused on the reduction of circulating levels of the amino acids tryptophan (TRY) and glutamine. These researchers have hypothesized that the imbalance of branched chain amino acids (BCAA) may be associated with central fatigue of OTS. Newsholme et al. (29) noted an increased uptake of BCAA by muscle tissue during severe sustained exercise. It is thought that this may lead to an increase in TRY by the brain. Once in the brain, TRY is converted into serotonin (32). Researchers (29) believe that increases in serotonin result in mood and behavioral changes that discourage further exercise. These mood and behavioral changes are often associated with OTS.

Researchers (29) have also proposed that a reduction in blood glutamine is responsible for the impaired immune response and increase in infection rate that is often evident in OTS. Glutamine is an amino acid found in plasma that plays an important role in metabolism (32). It is also required for the synthesis of new DNA and RNA during proliferation of lymphocytes and for synthesis and repair in macrophages (25). Therefore, a decrease in circulatory levels of glutamine may cause a decline in the function of the immune system (29). Long duration/intense training may cause a decrease in blood levels of glutamine. Foster and Lehman (19) found a decrease in glutamine in overtrained runners. Another study (32) involving overtrained athletes reported a reduction in glutamine thus making a reduction in glutamine a good indicator of OTS.

Muscle glycogen depletion may contribute to OTS (12). Costill et al. (12) studied the effects of doubling the training intensity on male collegiate swimmers over a 10-day period. Four out of the twelve subjects showed signs of OTS. These athletes had difficulty completing the training load, significantly reduced muscle glycogen stores, consumed fewer calories, and failed to maintain the carbohydrate intake requirement. Therefore, Costill et al. (12) suggested that chronic muscle glycogen depletion causes fatigue which results in a decrease in performance. Additionally, low muscle glycogen stores can reduce the levels of BCAA which can result in fatigue. Although these conditions are often seen in OTS, the glycogen hypothesis of overtraining has not been supported (33). Snyder (33) increased the training load of competitive cyclists for a period of two weeks. During this time, the cyclists also increased carbohydrate intake to

ensure sufficient carbohydrate intake. They found that all of the subjects met criteria for short-term overtraining and all subjects maintained normal glycogen levels. It was concluded that some other mechanism must be responsible for the genesis of OTS.

Foster and Lehman (19) proposed that the monotony theory of overtraining, a lack of day to day variation in training, could initiate OTS. It has been suggested that the daily sameness of hard training makes the athlete more prone to injury by putting stress on the musculoskeletal system (32). Bruin and colleagues (7), using racehorses as a model, have shown that this constancy of the training load may influence the genesis of OTS. The horses tolerated increases in training load as long as the training during the recovery days was light. If the training on regeneration days became too intense, the horses developed OTS. This suggests that training monotony along with total training load can create a stress that can result in OTS (19).

As stated earlier, no all encompassing hypothesis for OTS exists. In an attempt to integrate all of the present hypotheses into one, Smith (32) proposed the cytokine hypothesis of overtraining. The cytokine hypothesis of overtraining suggests that repetitive trauma to the muscular, skeletal, and/or joint system is frequently the initiator of OTS (32). Training and competing results in adaptive microtrauma (AMT) to muscle, bones, and connective tissue. Appropriate training programs that include rest days and allow time for recovery are needed in order for these injuries to heal. Therefore, it has been suggested that joint structures involved in high volume repetitions would initiate AMT (32). Previous research suggests AMT results in a mild inflammatory response to promote healing. The inflammatory healing process promotes muscle, bone, and tissue

adaptations which results in greater physical capabilities. Musculoskeletal trauma produced from subacute exercise results in release of local inflammatory factors known as cytokines. These cytokines, soluble hormone-like proteins, allow for acute inflammatory responses which in turn promote healing (32). Local acute inflammation becomes chronic with training imbalances. When chronic inflammation occurs, the cytokines activate monocytes which produce more proinflammatory cytokines. This results in systemic inflammation. The cytokine hypothesis of overtraining proposes that systemic inflammation causes several of the signs and symptoms associated with OTS.

Systemic inflammation may be the primary cause of the mood and behavioral changes that are often seen in overtrained athletes. Products of the immune system that are external to the central nervous system communicate with the brain. More specifically, cytokines appear to be the messenger molecules that convey information from the periphery to the CNS (32). Healing behaviors referred to as "sickness", "vegetative", or "recuperative" are activated in response to the message that is delivered to the central nervous system (32). The result of these behaviors includes reduced appetite, weight loss, depression, fear, and sleep disturbances (32). These results are all associated with OTS.

Hypothalamic functions that appear altered in OTS may be related to the cytokine hypothesis of OTS. According to the cytokine hypothesis, systemic inflammation could account for decreased testosterone levels and increased cortisol levels in overtrained athletes. Proinflammatory cytokines activate the hypothalamic-pituitary-adrenal axis during systemic inflammation. Activation of the hypothalamic-pituitary adrenal axis

results in the release of corticotropin releasing hormone (CRH) which stimulates the release of pituitary adrenocorticotropin releasing hormone (ACTH). ACTH then stimulates the release of cortisol into the circulation (6). Therefore, systemic inflammation could account for the elevated cortisol levels in OTS. Cytokines may also be responsible for the decrease in testosterone. Lutenizing-hormone releasing hormone (LHRH) is released from the hypothalamus. LHRH controls the release of the pituitary gonadal hormone LH which induces the release of testosterone and estradiol. Cytokines inhibit the release of LHRH which can result in a decrease in testosterone.

The amino acid imbalances of tryptophan and glutamine, imbalances associated with OTS, may also be understood in relation to the cytokine hypothesis. In athletes suffering from OTS, investigators (29) found reduced circulating levels of TRY. It is believed that a greater uptake of TRY by the brain occurs during OTS. This uptake of TRY causes an increase in brain levels of serotonin which results in mood and behavioral changes (29). The uptake of TRY into the brain is dependent on many factors. One of these factors is the free and bound plasma concentration of TRY (32). Albumin acts as the carrier of TRY and influences the rate of influx into the brain. During systemic inflammation, serum albumin concentrations are reduced. Therefore, the availability of TRY to the CNS is also reduced.

The reduction in blood levels of glutamine may also be related to the cytokine hypothesis of OTS. Foster and Lehman (19) found low levels of glutamine in athletes suffering from overtraining. Glutamine is one of the primary amino acids released from

muscle and is essential for gluconeogenesis and maintenance of blood glucose levels. It is also essential for lymphocyte proliferation and macrophage activity (25). Therefore, there is an increased need for glutamine when trauma occurs. A reduction in food intake is usually associated with trauma (32). To achieve appropriate blood glucose levels for the brain and other organs, gluconegensis is up-regulated. The trauma may be responsible for the decrease in blood glutamine which is associated with impaired immune responses and increased infections that are seen in overtrained athletes.

The muscle glycogen depletion that is sometimes associated with OTS may be caused by large volumes of training, systemic inflammation, and pro-inflammatory cytokines. Systemic inflammation with elevated pro-inflammatory cytokines may be the result of excessive stress. Elevated pro-inflammatory cytokines trigger a behavioral mood pattern. It has been suggested that reduced muscle glycogen levels in OTS may be the result of reduced food intake caused by cytokine-induced anorexia (32). Cytokines directly influence the hunger center of the hypothalamus by suppressing the appetite. A suppressed appetite results in a reduction in caloric intake. Transportation of glucose into the muscle may also be inhibited by muscle injury. Thus, muscle injury could also contribute to reduced muscle glycogen levels.

Overtraining occurs when the body undergoes excessive stress without adequate rest and recovery. A variety of hypotheses have been proposed to account for OTS, but presently no all encompassing hypothesis exists. Smith (32) suggests that many of the characteristics and symptoms of OTS could emerge from the presence of an injury. Systemic inflammation attempts to integrate previous research into one hypothesis.

Illness

The immune system responds to increased physical activity. It is thought that regular participation in moderate exercise enhances the immune system. Conversely, it has been shown that sustained intense exercise causes immunosuppression (5,24,31). It is also agreed that stress of competition may make athletes more prone to infectious illnesses (5). Elite athletes engage in intensive training at least once a day, have the psychological stress of major competition, and often are overtrained. This puts them at an increased risk of infection, primarily upper respiratory tract infection (URTI) (24).

Currently there are three hypotheses that have been proposed to explain the relationship between regular exercise and susceptibility to infection. A J-shaped model describing the relationship between exercise and URTI has been proposed by Niemann and Cannarella (30). This model suggests that moderate exercise training results in a lower risk of URTI when compared to the sedentary population whereas excessive amounts of high-intensity exercise results in an above average risk of URTI.

Pederson et al. (31) have proposed an "open window" hypothesis. According to this model, the "open window" is the time period after intensive exercise during which the athlete is at increased risk of infection. Whether or not this "open window" in the immune system occurs is dependent on the intensity and duration of exercise and also on whether muscle damage occurred (24). Moderate exercise results in stimulation of immune function during and for a short period following exercise. Intensive exercise results in an initial stimulation of immune function but is soon followed by a longer lasting suppression (24). It is during this period of immune suppression that the athlete is

at increased risk of infection. Elite athletes may spend a large amount of time within this period of immune suppression due to daily intensive training. It is hypothesized that if the athlete does not allow the immune system to recover severe immunosuppression could occur.

Lastly, a neuroendocrine model has been proposed to explain the immune response in regards to varying exercise intensities (24). It is proposed that during exercise, there is a release of immunodulatory hormones that result in either immunostimulation or immunosuppression, depending upon the intensity of the exercise. Moderate exercise causes the release of immunostimulatory hormones such as growth hormone, endorphins, and cytokines. Immunosuppressive hormones such as cortisol and catecholamines are released once intensity levels of exercise exceed a critical threshold. This model could explain the relationship between exercise intensity and the incidence of URTI.

Intensive exercise training may deplete glutamine and Vitamin C, factors that are necessary for proper function of the immune system. Overtrained athletes often have a reduction in glutamine and Vitamin C levels which suggests that the increased incidence of illness in these athletes may be associated with OTS. Smith (32) suggests that systemic inflammation may be the cause of the compromised immune system in overtrained athletes. A surgical example is used to explain the relationship between systemic inflammation and immunosuppression. After surgery/injury, inflammation occurs in an attempt to mobilize immune responses (32). Anti-inflammatory factors such as cortisol are also released to counteract the pro-inflammatory effects. Prolonged,

intense, counteraction results in immunosuppression. Smith (32) suggests that by the time the athlete realizes he/she has OTS, the athlete has most likely been exposed to pro-inflammatory cytokines and anti-inflammatory factors for an extended period of time, resulting in immunosuppression.

It is well known that overtraining and stress increase the susceptibility to infections. When training levels are not decreased, the severity of the disease increases. Both bacterial and viral infections may occur as a result of overtraining. It has been suggested that elite and endurance athletes are more susceptible to URTI than the general population during periods of excessive training (5). Vitamin C supplementation may reduce the incidence of URTI (31). Brenner et al. (5) have also recommended that athletes avoid excessive conditioning and allow for sufficient recovery time.

Injuries

Elite athletes tend to pursue heavy training in an attempt to optimize athletic performance. These athletes are often predisposed to negative consequences such as OTS and orthopedic injuries. There are two classifications of injuries that often occur: acute and chronic (2). Acute injuries are defined as those injuries precipitated by a single, sudden, violent trauma such as a sprain. Chronic injuries, also known as overuse injuries, are defined as injuries that result from repeated microtrauma to the tissue (23).

Overuse injuries are quite common in elite athletes due to high volume/intensity training with insufficient rest. It has been proposed that the volume of vigorous physical training may be an etiologic factor for exercise-related injuries (2). Almeidea and colleagues (2) found that weekly injury rates were positively correlated with the duration (hours) of

vigorous physical training. It was also suggested that abrupt increases in training volume may further contribute to injury risk.

Whether or not injuries induce OTS or are the result of OTS, they still hinder performance. Therefore, there is great interest in determining if there is a way to predict when an athlete is most susceptible to injury. Some athletes are more prone to injury than others. Internal or external risk factors can contribute to the occurrence of an athletic injury. Internal risk factors include biomechanics, fatigue, conditioning, flexibility, and somatotype of the athlete. External risk factors include weather, field conditions, training, and rules (26).

Prevention programs need to be implemented in order to avoid injuries. Monitoring the characteristics of training may be the key to minimizing undesired training outcomes.

Summary

In conclusion, due to high volume and high intensity training, elite athletes are often predisposed to injuries, illnesses, and OTS. Although no global hypothesis of OTS exists, the cytokine hypothesis of overtraining attempts to integrate previous research into one encompassing idea. In the past there was a lack of an adequate method to quantitate training load. Recent developments have created a method to monitor training. Monitoring characteristics of training allows an athlete to achieve goals while decreasing the chances of negative outcomes.

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