A Dynamic Model of Etiology in Sport Injury: The Recursive Nature of Risk and Causation

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Abstract: The purpose of this manuscript is to outline a new model representing a dynamic approach that incorporates the consequences of repeated participation in sport, both with and without injury. This model builds on the previous work, while emphasizing the fact that adaptations occur within the context of sport (both in the presence and absence of injury) that alter risk and affect etiology in a dynamic, recursive fashion. Regardless of the type of injury, it is often preceded by a chain of shifting circumstances that, when they come together, constitute sufficient cause to result in an injury. If we are to truly understand the etiology of injury and target appropriate prevention strategies, we must look beyond the initial set of risk factors that are thought to precede an injury and take into consideration how those risk factors may have changed through preceding cycles of participation, whether associated with prior injury or not. This model considers the implications of repeated exposure, whether such exposure produces adaptation, maladaptation, injury or complete/incomplete recovery from injury. When feasible, future studies on sport injury prevention should adopt a methodology and analysis strategy that takes the cyclic nature of changing risk factors into account to create a dynamic, recursive picture of etiology.

Key Words: Injury, etiology, cause, multifactorial

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INTRODUCTION

Injury reduction, control, and prevention are important goals for clinicians, researchers, athletes, and the active population. We should seek to remove barriers not only to continuing competitive participation but also to maintaining an active, healthy lifestyle. Given that an understanding of causes and risks are prerequisites for injury prevention, several models have been developed to understand the interplay of different factors along the path to injury.

Injuries occur when energy is transferred to the body in amounts or at rates that exceed the threshold for human tissue damage.[1] In sport injuries, we are usually referring to mechanical energy transfer. These conceptual definitions usually give way to operationalization of injuries that meet certain time-loss from activity or medical treatment criteria. Indeed, a recent consensus statement on injury definitions and data collection procedures in football (soccer) suggested that injuries are: "Any physical complaint sustained by a player that result from a football match or football training, irrespective of the need for medical attention or time-loss from football activities."[2]

In 1992, van Mechelen put forth a model of prevention based on injury surveillance, identification of risk factors, and implementation of prevention strategies.[3] In 1994, our group published a multifactorial model of causation[4] based on a modification of work in infectious disease.[5] Our multifactorial model (Figure 1) attempted to account for the interaction of multiple risk factors, both internal (intrinsic) and external (extrinsic).[4,6,7] It highlighted the importance of examining intrinsic predisposing factors as well as those extrinsic factors that interact to make an athlete susceptible to injury, before an injury-inciting event occurs.

Bahr and Krosshaug elaborated on the characteristics of the inciting event as a component of the causal pathway.[8] Specifically, these investigators discussed the importance of the playing situation and player/opponent behavior in addition to the global and detailed biomechanical description as elements of the inciting event.

Our experience over the past decade has led us to conclude that there are limitations to the approach that has been taken to date. Implicit in these models, is a linear paradigm. That is, events follow each other sequentially from a beginning point to an end point. This paradigm follows the approach used in classical cohort studies, where individuals who are exposed or not exposed to some (risk) factor are followed forward in time to measure outcome of disease (injury). Often in these types of studies, a specific, finite end point, such as occurrence of cancer or death, is measured.

However, the nature injury in sport is different. First, exposure is a combination of both possessing a risk factor and then participating (to a greater or lesser degree) with the risk factor. An individual may be exposed to the same or different risk factors repeatedly through multiple participations. Injuries may or may not occur under similar conditions. In most cases, the occurrence of injury does not permanently remove an individual from participation and, therefore, may not represent a finite end point.

This being the case, a linear approach that contains a start point and an end point does not reflect the true nature of injury in sport. This fact has been previously recognized by

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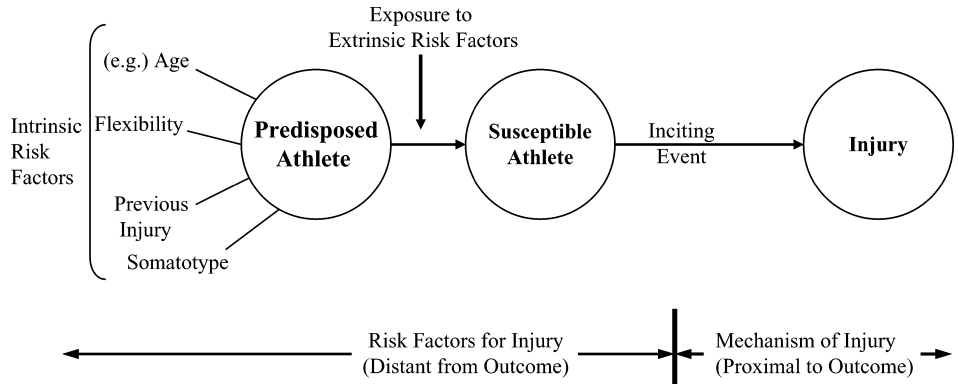

FIGURE 1. Previous multifactorial model of athletic injury etiology.[4]

Gissane et al, who noted that a linear model does not account for what happens after injury.[9] These authors developed a cyclical model for the investigation of contact sports to account for the return of healthy/fit players to sport. It was recognized that athletes could return to a lower level of sport, but their approach did not permit these players to return to the cycle of exposure.[9] What is not emphasized in prior models is that there may also be recurrent changes in susceptibility to injury in the course of sports participation without injury and that these exposures can produce adaptation and continually change risk.

The purpose of this manuscript is to outline a new model representing a dynamic approach that incorporates the consequences of repeated participation in sport, both with and without injury.

A DYNAMIC, RECURSIVE MODEL OF SPORT INJURY

In a real life sporting environment, a participant's risks are dynamic and can change frequently. Moreover, one exposure to a potential inciting event can alter an athlete's intrinsic risk factors and change their predisposition to injury. The athlete can then be exposed to the same or different extrinsic risk factors and have a different susceptibility. This paints a recursive picture where an athlete can enter a given athletic event cyclically with a differing set of risk factors (even though most other elements of the athlete and playing environment may remain constant).

The proposed model (Figure 2) is recursive in that one exposure can alter risk factors and allow the athlete to cycle through the model repeatedly. Aside from the possibility of retirement from sport, this model can be seen to operate independent of outcome.

Intrinsic Risk Factors: Predisposed Athlete

Each athlete has their own particular set of intrinsic factors or risks (eg, bone strength, neuromuscular control, age, previous injury history, etc.). A risk factor may be minimized as the athlete participates and adapts to the environment or to potentially injurious situations without sustaining injury. For example, exposure to body contact in collision sports may produce injury, but it may also result in adaptation and strengthening. If intrinsic strength improves, the athlete may be less predisposed to injury. In this example, exposure to extrinsic factors and other events (which might otherwise incite an injury) could actually have the effect of reducing intrinsic risk, thereby lowering overall injury risk.

The opposite may also hold true. If repeated body contact produces asymptomatic microtrauma and lowers strength or reduces neuromuscular control, the athlete may be more predisposed to injury. Then, the exposure to the same extrinsic (risk) factors and same mechanism or event would result in the athlete being injured. In this case, we refer to the event as a mechanism of injury.

Extrinsic Factors: Susceptible Athlete

The same logic may apply to changes in extrinsic risk factors. For example, the nature of events while participating might lead the athlete to adopt a more (or less) protective piece of equipment. We can also think of "behavioral" effects of the environment such as reaction to other athletes, game conditions, officiating decisions, the spectator environment, and the level of importance attached to a particular game. Also, reaction to rule changes or equipment may result in risk modification, changing susceptibility to injury. This concept of risk compensation has been the subject of two recent editorials by Hagel and Meeuwisse[10] and McIntosh.[11]

The proposed model (Figure 2) also assumes that any risks may interact with any other risks to produce joint interaction effects. As such, intrinsic factors may interact with extrinsic factors to produce a joint interaction. Essentially, the "susceptible athlete" phase is where the intrinsic and extrinsic risks and the interactions between all of the risks accumulate.

Injury Decision

The next stage is the occurrence of (potentially) injurious events. There are several possibilities considered in this model.

Injury

The original model only considered the possibility that an inciting event occurs to produce injury. The important consideration here is that when an inciting event results in an injury outcome, we consider the combination of preceding factors as part of the cause.

Recovery

Injury in sport rarely represents the same finite end point as in other areas of epidemiology (ie, motor vehicle fatalities or

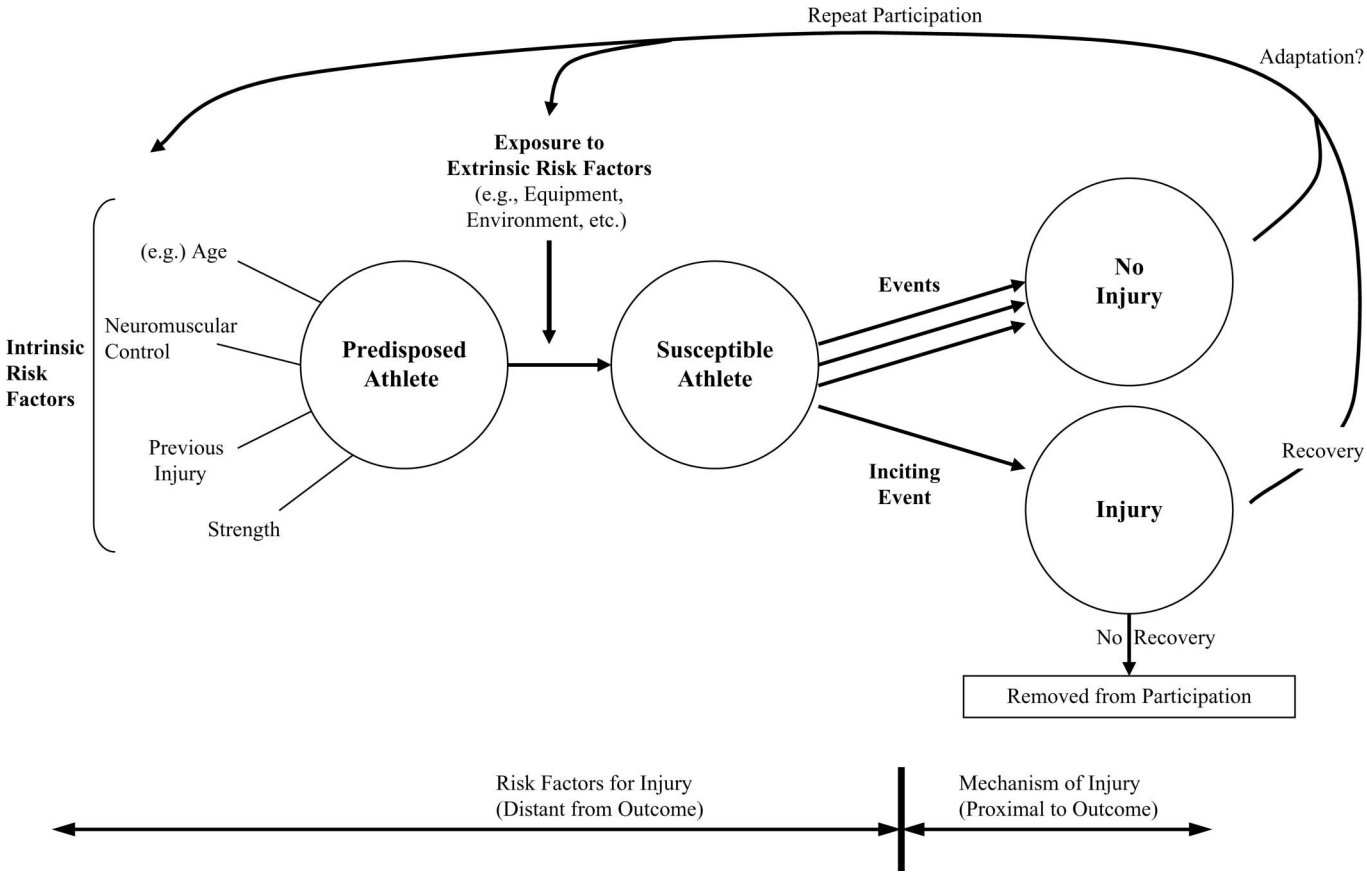

FIGURE 2. A dynamic, recursive model of etiology in sport injury.

cancer mortality). If an injury does occur, withdrawal from further exposure may be the result; more often, recovery will facilitate reentry into sport participation (after medical attention or at a later date), potentially with a new set of intrinsic risk factors. In this respect, their predisposed status will now be different. Recovery and reentry into participation may also result in an athlete changing their equipment or other extrinsic risk factors. In this respect, their susceptible status will also be different.

Removal

Unfortunately, not all athletes return to sport after injury. Removal from participation would then represent a finite end point in terms of further risk or injury. This may occur due to inadequate recovery from injury, or a choice to no longer accept risk. Most injuries are sport-specific in nature. Although some athletes may change sport after injury, they should be considered "removed" from the further potential for exposure or injury in the sport being studied.

No Injury

The other possibility that has not received a great deal of attention to date is that the athlete may be exposed to potentially injurious situations but not sustain injury. In fact, in most circumstances, participation does not result in an injury. Rather, adaptations or maladaptations may occur to tissues, equipment, etc. that modify intrinsic and/or extrinsic risk factors on the basis of the interaction of the event attributes and preexisting risks. The same characteristics outlined by Bahr and Krosshaug as components of an inciting event (ie, playing situation, player/opponent behavior, global and detailed biomechanical description)[8] could be considered as important aspects of non-injury events. The potential effects of these events may be immediate, or they could be latent. With this scenario, we consider the changing factors as risks, but in the absence of injury, we do not consider them causes.

Entry into the Cycle

Due to the cyclic nature of the model, the athlete can enter the injury chain at any point. For example, there might be an event (eg, heavy training producing fatigue) causing adaptation that does not result in time-loss from participation and does not require medical attention. Therefore, it would not meet the definition of injury in most studies. In Figure 2, this would be considered "events" producing no injury.

However, an intrinsic risk factor (eg, neuromuscular control) could be altered on the basis of fatigue. Secondly, this alteration could interact with another preexisting risk factor (eg, a previous partial medial collateral ligament tear with residual laxity). The next day, the athlete might play sport again in the presence of the same extrinsic risk factors as previously (eg, a high friction surface) and perform a maneuver that they have done many times (eg, plant/decelerate/twist).

However, under the conditions of altered intrinsic risk factors, the same maneuver then becomes an inciting event in which the same motion (eg, the plant/decelerate/twist) becomes the mechanism of injury for an injury (eg, an acute anterior cruciate ligament tear).

In one respect, this situation could be considered linear in that it began with one new and one old intrinsic risk factor. However, it could also be considered to begin with the heavy training load or subclinical event of fatigue.

IMPLICATIONS OF THE DYNAMIC, RECURSIVE MODEL

The concepts of combining risk factors have been described in different language by Rothman as "necessary" and "sufficient" causes, and the combination of factors as "causal pies."[12] The application of these causal concepts has also been described previously in reference to sport injury.[6] However, the dynamic, recursive nature of repeated exposure with varying risk factors before the occurrence of injury has not received sufficient attention in the context of sport. This nature has important considerations for injury prevention and how we approach both research design and analysis.

Implications for Study Design and Data Analysis

To date, the linear approach taken in much of the design and analysis of sport injury studies may have been quite appropriate, particularly if risk factors are stable over time, and a multifactorial approach is taken. However, it is also plausible that past studies have found no associations because the assumption of stable risk factors was not valid. If this is the case, we may need to reexamine these risk factors using study designs and analysis techniques that account for a dynamic picture of risk that changes over time.

Measurement of this dynamic concept is not simple. For example, in the past, researchers might have measured how preseason strength predicts injury risk (during a season). However, this type of design assumes that strength remains constant up to the point of injury. If we take the dynamic-recursive approach, one could theorize that it is the pattern of change in strength that influences injury risk more so than the absolute value. If this were the case, one would need to measure strength at some regular interval to model how changes in strength influence injury risk.

Other areas of epidemiology, such as the study of pharmacological outcomes, face some of the same issues in dealing with recurrent events and cumulative exposure. This experience can guide the design and analysis strategies for sport injury. Miettinen and Caro[13] have discussed the issue of excess risk of adverse drug reactions where etiologic problems can essentially be simplified to an assessment of exposed versus not exposed for the outcome of interest with reference to etiologically relevant time.

Miettinen noted that the key is to look back from the time of injury to an etiologically relevant time period (eg, the last session of play before the injury occurred or the cumulative effect of playing on artificial turf over a number of sessions) and compare those who are and those who are not exposed to the risk factor of interest during that time period, but who are effectively the same regarding all other risk factors of interest.[14] This concept incorporates whatever adaptations take place for the athlete, because the comparison of interest should be among those who have experienced similar adaptations (to control confounding) but who have and have not experienced the exposure. In fact, "membership" in these study populations can be considered dynamic or open from one exposure to the next.[14] This analytic approach is design-independent from an etiologic point of view in that it applies to both case-control and cohort studies (both retrospective and prospective studies).

A cohort of athletes followed from the start of the season will arguably have a changing risk factor profile. Consideration needs to be given to capturing and using the risk factor data, including exposure, during an etiologically relevant time period, looking back from the time of injury, rather than forward from the beginning of the season. Changing exposure and adaptation present analytic challenges if viewed through a prospective lens but are simplified if a retrospective outlook is adopted.

It should be emphasized that there is a difference between the design of the study (data collection) and the analysis approach. In general, a prospective study design is considered superior because the collection of data tends to be a more structured and monitored part of the research study execution. While cohort studies typically use an outcome of relative risk, it is also possible to use more retrospective analysis techniques on studies that have used prospective data collection.

If changes in multiple risk factors are to be assessed, then a multivariate analysis technique must be employed. In simplest terms, we can view the analysis of injury events in relation to a particular exposure as a simple contingency table if we dichotomize outcome (ie, 1 if the athlete sustains injury; 0 otherwise) and exposure (ie, 1 if exposed in etiologically relevant time; 0 if not). The odds ratio provides a measure of the association between the risk factor and outcome. With only one or a few potential confounders considered, we can use stratified analysis techniques as adjustment factors for our exposure-disease relation.[15] However, if we must control for more than a few confounding variables, we would most likely need to use multivariate models, as the number of strata could become unwieldy. For dichotomous outcomes, logistic regression may be used to estimate the exposure-outcome relation in the presence of covariates.[16] With this approach, the problem of a changing covariate profile (ie, time-dependent covariates) is not an issue, as these changing covariates would simply need to be operationalized within the etiologically relevant time period. That is, athletes who have and do not have the outcome of interest, but who are the "same" in all other ways, including the timing and nature of changing covariates, would be compared in order to estimate the exposure-disease relationship. An alternative is to use a Poisson regression approach to estimate rates of injury per (for example) 1000 athlete exposures or 1000 player hours. Cox proportional hazards regression is another approach for the estimation of injury rates dealing with time to event data (eg, injury). A more complete description of the differences between these methods is beyond the scope of this article, but

can be found in Callas et al.[17] Further, generalized estimating equations[18,19] and cluster analysis[20] can be used to account for correlated outcomes (eg, variation in injuries may be less within individuals/teams than between individuals/teams).

The presence or absence of risk factors (and confounders) can only be considered if they are measured.[6] The timing and nature of this measurement becomes particularly important in a dynamic, recursive model. The analysis will only be limited by the degree to which precise documentation of exposure and covariates (including any temporal changes in the level of these) can be captured.[13]

Given that the exposure histories can differ widely on a number of different parameters, the design and analysis must reflect the correct variables. The variable itself may change, or the level of the same variable may be altered. If a baseline covariate is expected to change over time (ie, strength, previous injury) and a follow-up measurement is done, then the covariate of interest considered in the analysis may not be the baseline measure or a single pre-injury (follow-up) measure, but rather the change over time between baseline and follow-up (eg, strength difference or change). In terms of measurement, it may not be that we need to measure different variables, but that we need to measure current variables differently (ie, repeatedly) to account for changes over time. In terms of analysis, it is important to develop different variables to account for the cyclic (and variable) nature of exposure to risk factors. The important point here is that exposure to a risk factor in sport cannot be seen as a static event, since the exposure is repeated under changing conditions. Therefore, both the design of the study and analysis strategy must accommodate changing risks.

PREVENTION

One can imagine an almost infinite number of scenarios based on the combination of intrinsic and extrinsic risk factors and the number of events, cycles, and time that passes before an injury actually occurs. However, in order to prevent injury, we must identify, target and attempt to ameliorate the effects of modifiable risk factors through the introduction of appropriate and timely injury prevention strategies. The optimal opportunity to intervene could be at a number of different stages of this model or at a certain number of cycles through the model (ie, at some optimal level of participation or exposure). Regardless of which point in the cycle or at which iteration though the cycle, the goal should remain the same: to intervene before an injury occurs (primary prevention). This may be accomplished through modification of both intrinsic and extrinsic risk factors. Safety rules, protective gear, and changes in the playing environment may be altered on a group basis.

However, the optimal intervention approach may be individual in nature. To maximize their effectiveness, targeted group prevention strategies may require individual customization on the basis of individual levels and variability of risk factors over time (and possibly, their interaction). Clearly, maximizing the effectiveness of any prevention strategy will also be dependent on the ability to accurately capture changes in levels of any risk factor through appropriate methods of measurement.

CONCLUSION

This model builds on the previous work by Meeuwisse,[4,6] Gissane,[9] and Bahr,[8] while emphasizing the fact that adaptations occur within the context of sport (both in the presence and absence of injury) that alter risk and affect etiology in a dynamic, recursive fashion.

Regardless of the type of injury, (eg, acute, traumatic, or gradual onset, overuse, recurrent), it is often preceded by a chain of shifting circumstances that, when they come together, constitute sufficient cause to result in an injury. If we are to truly understand the etiology of injury and target appropriate prevention strategies, we must look beyond the initial set of risk factors that are thought to precede an injury and take into consideration how those risk factors may have changed through preceding cycles of participation, whether associated with prior injury or not.

When feasible, future studies on sport injury prevention should adopt a methodology and analysis strategy that takes the cyclic nature of changing risk factors into account to create a dynamic, recursive picture of etiology.

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