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REVIEW ARTICLE

# The Impact of Triathlon Training and Racing on Athletes' General Health

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**Abstract** Although the sport of triathlon provides an opportunity to research the effect of multi-disciplinary exercise on health across the lifespan, much remains to be done. The literature has failed to consistently or adequately report subject age group, sex, ability level, and/or event-distance specialization. The demands of training and racing are relatively unquantified. Multiple definitions and reporting methods for injury and illness have been implemented. In general, risk factors for maladaptation have not been well-described. The data thus far collected indicate that the sport of triathlon is relatively safe for the well-prepared, well-supplied athlete. Most injuries 'causing cessation or reduction of training or seeking of medical aid' are not serious. However, as the extent to which they recur may be high and is undocumented, injury outcome is unclear. The sudden death rate for competition is 1.5 (0.9–2.5) [mostly swim-related] occurrences for every 100,000 participations. The sudden death rate is unknown for training, although stroke risk may be increased, in the long-term, in genetically susceptible athletes. During heavy training and up to 5 days post-competition, host protection against pathogens may also be compromised. The incidence of illness seems low, but its outcome is unclear. More prospective investigation of the immunological, oxidative stress-related and cardiovascular effects of triathlon training and competition is warranted. Training diaries may prove to be a promising method of monitoring negative adaptation and its potential risk factors. More longitudinal, medical-tent-based studies of the aetiology and treatment demands of race-related injury and illness are needed.

**Key Points**

The sport of triathlon appears to be relatively safe for the majority of well-trained, well-prepared athletes.

The demands of triathlon training and racing, and their influence on injury and illness, are not well-described.

More prospective investigation of the health-related effects of triathlon participation, with a view to producing better training and racing guidelines, is warranted.

## 1 Introduction

The sport of triathlon involves a sequential swim, cycle and run over a variety of distances and formats [1]. At any given life-stage, the triathlete is likely to be focusing his or her training on preparation for the shorter-distance sprint or Olympic-distance races, or for longer-distance half-Ironman to Ironman events. Athletes in the 35–39 years and 40–44 years age groups form the majority of participants [2].

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Non-elite athletes who compete against other athletes within the same 5-year age range (hereafter referred to as 'age-groupers'), and particularly those who are less experienced [3], are less likely to be coached than elite athletes. According to a study by the USA Triathlon organization, although only 26 % of athletes did not 'want or need a coach', 47 % did not have a precise training plan [4]. The sport of triathlon has been shown not to be 'the sum of its component sports' (because the neuromuscular adaptations to cycling training, for example, interfere with those elicited by running [5, 6]). Little research that can help the triathlete train in an optimal, sport-specific, manner has been published, however. The training that is involved in preparation for competition for the various triathlon event formats and distances [7, 8] has been insufficiently quantified [9]. Few detailed longitudinal investigations [10–12] of how changes in training factors may be reflected by changes in injury and illness status are available. The risk profile of the athlete as he or she goes into competition, and the extent to which this is mirrored by race-related problems, has not been investigated. Although training diaries have been cited as a crucial diagnostic aid in the management of 'tired' triathletes [10] and are reportedly the triathletes' most commonly used method of feedback on training efficacy [3, 12], minimal examination of the extent to which such logs may be used to minimize maladaptation has occurred.

This article reviews the literature regarding triathlon training and racing loads and their effects on the immune system, oxidative stress and cardiovascular status. The extent of and putative risk factors for illness and injury in able-bodied athletes participating in road-based triathlons are described. We report how the development of specific illnesses or injuries may be influenced by the environmental conditions and/or cross-training that is involved [1]. The triathlon-specific research that has thus far been conducted into potential indicators of maladaptation is discussed. Issues that will have to be addressed if the results of future studies are to lead to practical improvements in training and racing practice are highlighted.

## 2 Triathlon Training

Only one calculation of mean weekly training duration data from the literature for each discipline, comparing Olympic-distance and Ironman-distance specialists, has been published [9]. These mean values broadly agree with retrospective data that were obtained 10 years earlier for age-groupers [13, 14]. Weekly training volumes for world-ranked elite triathletes have not been well-documented but are clearly higher [15]. No examination of the extent to which training practice has changed over time has been published. However, several differences between sex, ability and event-distance groups that were noted in 1993 (Table 1) may still hold. Olympic-distance athletes may spend less overall time per week than Ironman athletes doing longer, low intensity, 'long run' ($p < 0.05$ for both sexes) and 'long bike' sessions ($p < 0.05$, for females only). The length of such individual sessions is likely less for Olympic-distance than for Ironman-distance athletes ($p < 0.05$). Superior Olympic-distance athletes also do more speed work cycle and fewer long-run sessions per week (both $p < 0.05$), and inferior Olympic-distance athletes do more back-to-back cycle–run transition training than Ironman athletes ($p < 0.05$) [12].

In addition, nor are many detailed prospective longitudinal training studies [8, 12, 16] available. Neal et al. [16] analyzed the training-intensity distribution of ten recreational-level athletes (mean ± standard deviation [SD] age 43 ± 3 years) over the 6 months leading up to an Ironman race. Three training periods (January–February, March–April, and May–June) and 4 testing weeks, were involved. The athletes spent (mean ± SD) 69 ± 9, 25 ± 8, and 6 ± 2 % of the total training time for the three training periods combined doing low-, mid- and high-intensity exercise, respectively.

Prospective data for ten Olympic-distance athletes who finished within the top 50 at their non-drafting national championships 21 weeks later, in 1994, have also been reported [12]. The athletes were members of a national squad but given that their data pre-date the inception of the drafting rule for elite racing, the increased professionalism of the sport since it gained Olympic status, and that they were focusing on domestic races rather than on the international circuit, they are only likely to be representative of well-trained age-groupers. Approximately 25, 56 and 19 % of training time was spent swimming, cycling and running, respectively. Nearly 70 % of training time in each discipline was spent below racing intensity. The changes in training volume and intensity that occurred in the squad which included the latter athletes are illustrated in Figs. 1 and 2. It is important to note that the relative proportion of training time that was spent at higher intensity levels and the overall weekly rate of overall change in training stress became increasingly greater as the athletes progressed towards the competitive period.

Only conference abstracts exist to support the premise that elite athletes [17] with a current world ranking also spend approximately 70 % of their exercise time below racing intensity. Little is known about the training of such athletes other than it can vary widely, even between athletes with the same coach [8], that international travel may be involved, and that altitude training is widely practiced in the lead-up to competition.

**Table 1** Selected potential intrinsic and extrinsic factors for maladaptation that have been found to vary with sex, distance specialization and ability in triathletes (reproduced from Vleck [12], with permission)[a]

| Variable | | Ability | | | | Event distance | | | | | Sex | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | E OD M vs. SE OD M | E OD M vs. NE OD M | SE OD M vs. NE OD M | E OD F vs. SE OD F | OD vs. IM | E OD M vs. E IM M | SE OD M vs. E IM M | E OD F vs. E IM F | SE OD F vs. E IM F | M vs. F squad | E IM vs. E IM F | E OD M vs. E OD F | SE OD M vs. SE OD F |
| Competitive experience (years) | Swim | – | – | – | * | – | * | * | ** | – | – | – | – | – |
| | Cycle | *** | – | – | – | – | – | – | – | – | – | – | – | – |
| | Run | *** | – | – | – | – | – | – | * | – | – | – | – | – |
| | Triathlon | *** | – | – | – | – | – | *** | – | – | – | – | – | – |
| Psychological state | Sad or depressed | – | – | – | – | – | – | – | – | – | – | – | – | * |
| | Stressed | – | – | – | ** | – | – | – | – | – | ** | – | – | ** |
| | Tense/ anxious | – | – | – | – | – | – | – | – | – | – | – | – | * |
| | Worried | – | – | – | – | – | – | – | – | – | ** | – | – | – |
| | Restless sleep | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | Cannot cope | – | – | – | * | – | – | – | – | – | ** | – | – | *** |
| | Need to get away | – | – | – | – | – | – | – | – | – | – | – | – | ** |
| | Mood disturbance | – | – | – | * | – | – | – | – | – | – | – | – | ** |
| Level reached in cycling | | – | – | – | – | – | – | – | – | – | – | * | – | – |
| Best distance | | – | – | – | – | – | *** | – | – | – | – | – | – | – |
| Orthopaedic problems | | – | – | – | – | – | * | – | – | – | – | – | – | – |
| Weekly training time (h) | Running | ** | – | – | – | – | – | – | – | – | – | – | – | – |
| | Long runs | *** | – | – | * | – | – | – | ** | – | – | – | – | – |
| | Overall | – | – | – | – | – | – | – | * | – | – | – | – | * |
| Weekly training distance (km) | Overall | *** | – | – | – | *** | – | * | – | – | – | – | – | *** |
| | Swimming | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | Cycling | *** | – | – | – | – | – | – | – | – | – | – | – | – |
| | Running | *** | – | – | – | *** | – | – | – | – | ** | – | – | – |
| Number of sessions per week | Swimming, cycling and running | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | Swimming (overall) | – | – | – | – | – | – | – | – | – | ** | – | – | – |
| | Cycling (overall) | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | Running (overall) | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | Speed work bike | – | – | – | – | * | – | – | – | – | – | * | – | – |
| | Hill repetition cycle sessions | – | – | – | – | – | – | – | – | – | ** | – | – | – |

**Table 1** continued

| Variable | | Ability | | | | Event distance | | | | | Sex | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | E OD M vs. SE OD M | E OD M vs. NE OD M | SE OD M vs. NE OD M | E OD F vs. SE OD F | OD vs. IM | E OD M vs. E IM M | SE OD M vs. E IM M | E OD F vs. E IM F | SE OD F vs. E IM F | M vs. F squad | E IM vs. E IM F | E OD M vs. E OD F | SE OD M vs. SE OD F |
| | Back-to-back cycle run training | * | – | – | – | – | – | – | – | – | – | – | – | – |
| | Hill repetition run sessions | – | – | – | – | – | – | – | – | – | ** | – | – | – |
| | Long runs | – | – | – | – | * | – | – | – | – | – | – | – | – |
| | Other types of run session | – | – | – | – | – | – | – | – | – | ** | – | – | – |
| Length of each session | Each long cycle | – | – | – | – | * | – | – | – | – | – | – | – | – |
| | Each long run | – | – | – | – | * | – | – | – | – | – | – | – | – |
| Warm up/ warm down | Pre-swim | – | – | – | – | ** | – | – | – | – | – | – | – | ** |
| | Post-swim | – | – | – | – | – | – | – | – | – | ** | – | – | – |
| | Pre-cycle | – | – | – | – | – | * | – | – | – | – | * | – | – |
| | Post-cycle | – | – | – | – | * | * | – | – | – | – | – | – | – |
| Stretching | Pre-swim | – | – | – | – | * | – | – | – | – | – | – | – | * |
| | Post-cycle | – | – | – | – | – | * | – | – | * | – | – | * | * |
| | Pre-run warm-up | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Technique analysis | Swim | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | Run | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | Transition | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Train with single-sport athletes | Swim | – | – | – | – | – | – | – | – | – | – | – | – | – |
| | Cycle | – | – | – | * | *** | * | – | – | * | – | – | – | – |
| | Run | – | – | – | – | – | – | – | – | – | – | – | – | – |
| Type of coach | Cycle | – | – | – | – | – | – | – | – | – | *** | * | – | – |
| | Run | – | – | – | – | – | – | – | – | – | *** | * | – | – |
| Periodised training[b] | | *** | – | – | – | – | – | – | – | – | – | – | – | – |

– indicates no information, *E* 1994 elite (most likely corresponding to higher ability, well-trained recreational athletes of today), *IM* Ironman distance (i.e. 3.8-km swim, 180-km cycle, 42.2-km run), *F* female, *M* male, *NE* non-elite (recreational) athletes, *OD* Olympic distance (i.e. 1.5-km swim, 40-km cycle, 10-km run), *SE* 1994 sub-elite (most likely corresponding to good, well-trained, recreational athletes of today

* $p < 0.05$, ** $p < 0.02$, *** $p < 0.01$ from the group marked with the same symbol and in the same row of the table

[a] No differences were observed between the various groups in the use of clipless pedals, use of different types of cycle handlebars or gearshift systems

[b] For the entire year as opposed to from race to race

## 3 Triathlon Competition

The length of the competitive season, and the number and type of competitions that it involves, may differ markedly both between elite athletes and age-groupers [12], and with event-distance specialization. The relative intensity at which competition is performed has been insufficiently quantified, but also differs [18–25] (Table 2). The extent to which it does so is unclear given that most studies have used different physiological

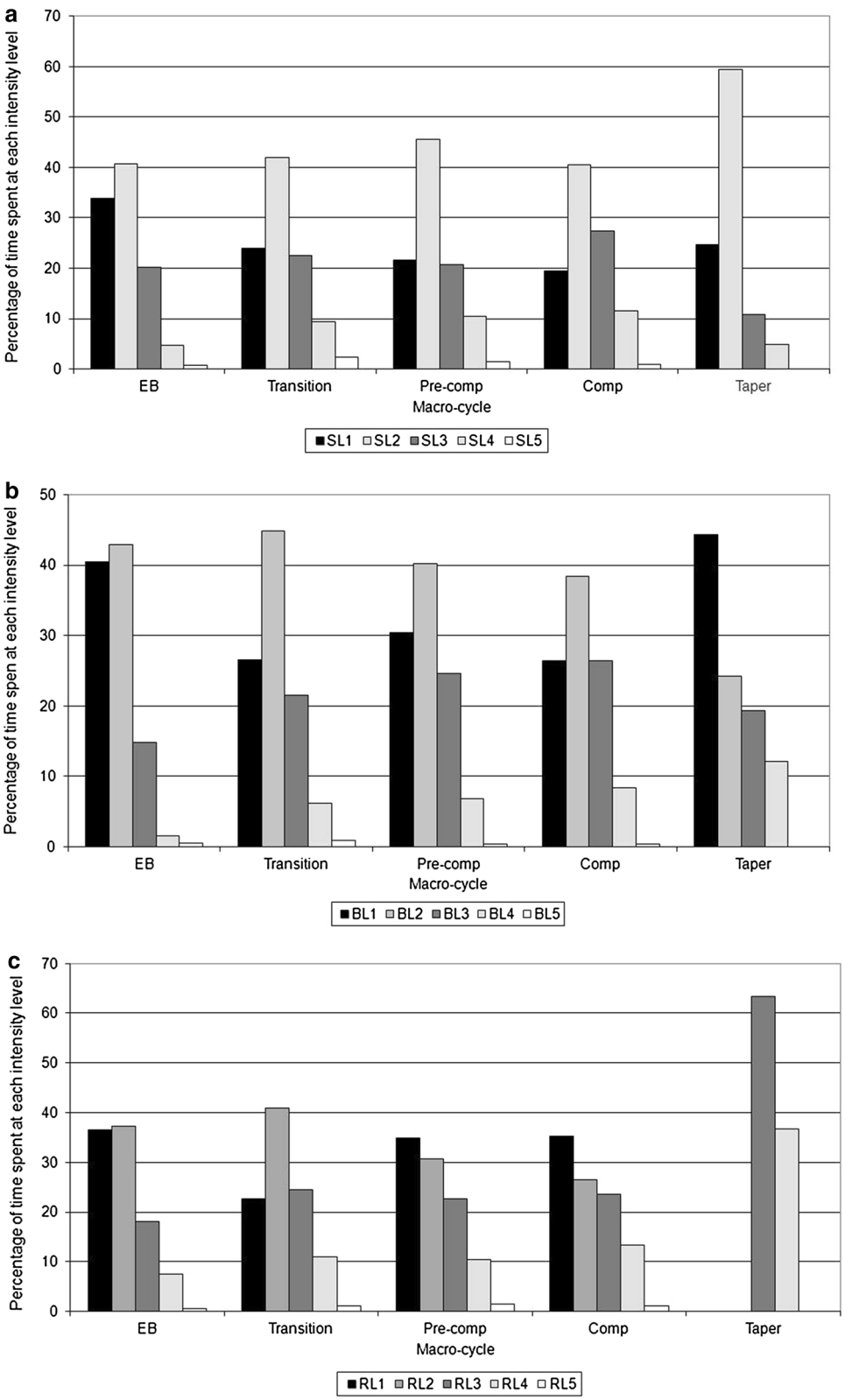


**Fig. 1** Changes in distribution of training intensity of Olympic-distance triathletes over a two-peak competitive season: (**a**) swim, (**b**) bike, (**c**) run (reproduced from Vleck [12], with permission.) *EB* endurance base, *Pre-comp* pre-competition, *Comp* competition, *S* swim, *B* bike, *R* run, *L* intensity level (rated as 1–5, with 1 being the lowest intensity)

markers for competition intensity. Few studies [18, 19, 26] have obtained data relating to the physiological and other demands of triathlon swimming. This is despite potentially hazardous interactions between environmental temperature, water temperature, currents, marine life, other athletes, exercise intensity and duration, as well as

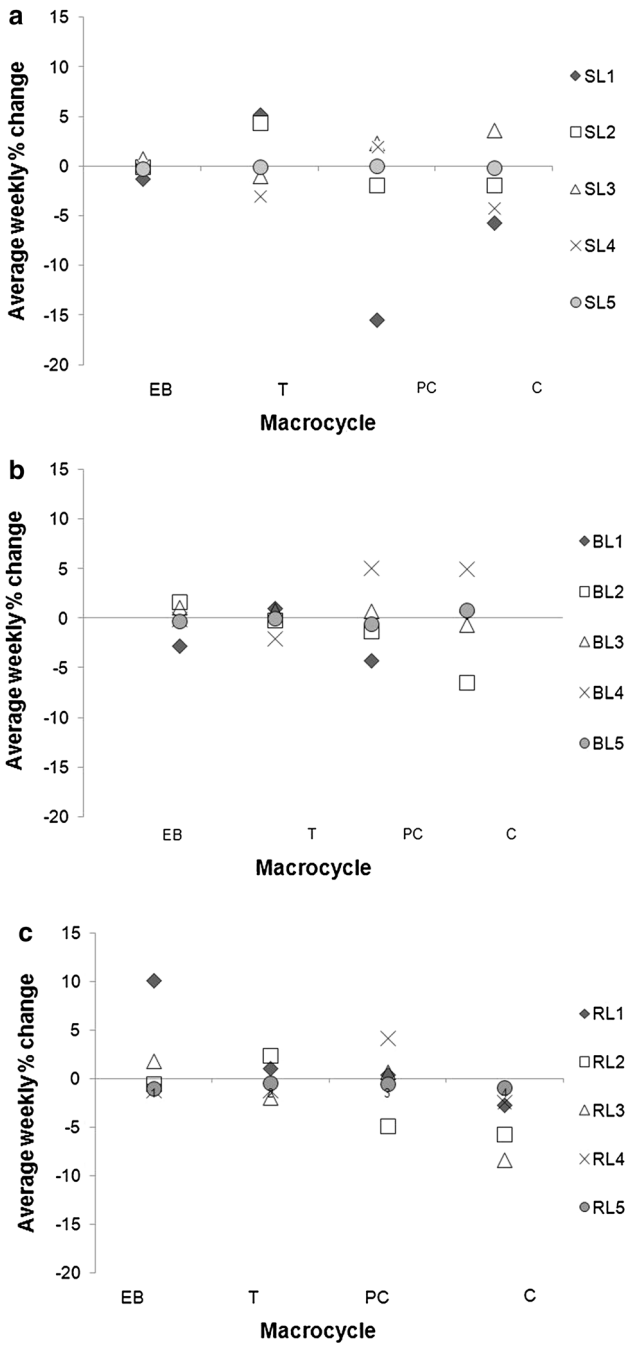


**Fig. 2** Changes in weekly rates of total training stress (arbitrary units) across consecutive macro-cycles of a two-peak competitive season in Olympic-distance triathletes: (**a**) swim, (**b**) bike, (**c**) run (reproduced from Vleck [12], with permission). *EB* endurance base, *T* transition, *PC* pre-competition, *C* competition, *S* swim, *B* bike, *R* run, *L* intensity level (rated as 1–5 with 1 being the lowest intensity)

'feed-forward' fatigue effects from one discipline to the next [27].

As the intensity and duration of competition changes, so may the thermal stress that is experienced by the athlete. Hypoglycaemia, dehydration [28], changes in blood electrolyte concentration and muscle damage [29] may all occur. The relative extent to which they occur in short-distance races is unknown. Muscle damage [30, 31] seems to be the most significant of these issues in half-Ironman-distance events [29]. The extent to which the triathlete may be at risk for hypo/hyperthermia and other heat-related illness in sprint distance events is related to environmental temperatures, humidity and degree of prior heat acclimatization [32]. Water temperatures at International Triathlon Union-sanctioned events start at 13 °C (for 1,500 m) or 14 °C (for 3,000–4,000 m) [33]. The upper allowable limits are 20–24 °C depending on athlete ability and race distance/format. They may be adjusted down according to water–air temperature differences and the weather. Maximum allowable time spent in the water also varies with event distance and athlete ability group. Total body water turnover with Ironman competition can be around 16 L or 1.33 $L.h^{-1}$ [25]. Dehydration is usually estimated via measurements of body mass loss. With Ironman competition, this may be 3–8 % of the pre-start value (i.e. almost double that of half-Ironman [23, 29]) in males [25, 34, 35]. It was not reported to be significant in female age-groupers [36]. Body weight may also increase with competition in athletes with exercise-associated hyponatremia [34, 37–42]. Both hyponatraemia—which is rare in races lasting less than 4 h, but common in those lasting over 8 h [39], and heat illness [32] are discussed elsewhere [35, 37, 40, 41, 43–46]. However, normally (but not always [25]) plasma volume decreases with short-distance competition [47], and is either maintained or increased (by 8.1–10.8 %) after Ironman competition [48–50].

## 4 Immune, Oxidative and Cardiovascular Responses to Triathlon Training and Competition

Although the demands of training and competition are not well-described, it has been suggested [51] that triathletes do 'extreme amounts of exercise'. Some empirical as well as epidemiological data suggest that such excess may be associated with DNA modulation, increased risk of cardiovascular or pulmonary events [52–58], and/or impaired immune status. Cumulative oxidative stress [54], increased oxidation of plasma lipoproteins and a subsequent potential contribution to atherosclerosis may potentially offset the positive effects of endurance training. Indeed, it has been postulated that U- and S-shaped relationships between exercise (load) and health exist in age-groupers [51] and elite athletes, respectively [59].

### 4.1 The Immune Response

Longitudinal studies of the response of white blood cell (WBC) counts or other immune system markers to triathlon

training are scarce. According to a 10-year retrospective study of Australian Institute of Sport (i.e. elite) athletes [60] who presented without illness, triathletes had lower resting total WBC and neutrophil counts than athletes from other sports (Table 3) [61–88]. The authors concluded that this probably reflected a training-induced adaptive anti-inflammatory response operating within broader homeostatic limits rather than any underlying pathology. They also found that the aerobic component of the sports that they surveyed exhibited a large positive correlation with monocyte counts in males ($r = 0.51$) and a moderate positive correlation in females ($r = 0.34$). Their group probably involved mostly or all Olympic-distance specialists. Rietjens et al. [65] also observed many elite (probably Olympic distance) triathletes to exhibit haematological values near or below the lower limit of the normal range. However, a 4-year prospective study of Spanish elite triathletes [71] showed WBC counts to lie within the normal limits within both the pre-competitive and competitive periods. However, 16 % of the triathletes in the Australian Institute of Sport study displayed neutropenia and 5 % displayed monocytopenia, respectively. This observation (which was supported by Philip and Bermon [89]) is of clinical interest. Neutropenic individuals are generally more susceptible to bacterial infection, such as might occur after inadequate treatment of a seemingly trivial skin abrasion. The reason for neutropenia, in particular, is unclear. It may be due to exercise-induced neutrophil apoptosis and consequent lower neutrophil lifespan. When the running section of normal triathlon training is intensified [90] (as illustrated in Figs. 1 and 2), infection risk (as measured by symptoms of upper respiratory tract infection [URTI] and increased congestion) may rise. Whether this means that short- and long-distance specialists, who likely differ in the proportion of their training that is spent at higher intensities, may differ in immune status is unknown. A 6-month prospective study of competitive-level athletes preparing for Ironman competition [62] demonstrated accumulation of differentiated and transition T cells, at the expense of naïve T cells. This accumulation could compromise host protection to novel pathogens during periods of heavy training [63] (especially when the athlete is at altitude [91, 92] and/or during excessive international travel [93]). Certainly, Southern Hemisphere athletes were reported to have a lower infection risk in their 'off-season' [64]. The opposite has also been reported, however [94].

Competition has been reported not to pose any acute health risks to healthy athletes who come well-prepared and well-supplied [95], but immune suppression can occur within the post-race recovery period (electronic supplementary material [ESM] Table S1) [24, 25, 29–31, 35, 36, 40, 44, 47, 48, 50, 53, 55–58, 67, 69, 70, 95–155]. The observed decreases in WBC, for example, are unlikely to be wholly explainable by plasma volume expansion as the magnitude of cell count differences is larger than the typical race-related change in plasma volume. The suggestion [104] that completing an Olympic-distance triathlon may decrease the level of immunoglobulin A (IgA)-mediated

**Table 2** Physiological demands of (actual or simulated) triathlon competition (values expressed as mean ± SD)[a]

| Study | Distance | Percentage of maximal oxygen uptake[b] | | Percentage of maximal heart rate | | | Percentage of maximal aerobic speed/maximal aerobic power/peak running speed | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Cycle | Run | Swim | Cycle | Run | Swim | Cycle | Run |
| Taylor et al. [18] | Simulated sprint (lab)[c] | 82.1 ± 6.0 | 89.7 ± 4.9 | – | 89.6 ± 3.5 | 91.9 ± 1.9 | – | 68.2 ± 7.2 | 87.5 ± 3.0 |
| Binnie et al. [19] | Simulated sprint (lab)[c] | – | – | – | – | – | – | – | – |
| Gonzalez-Haro et al. [20] | Simulated OD swim-cycle (lab) | 82.8 | – | – | 92 | – | 98 ± 2 | 77 ± 10 | – |
| Bernard et al. [21] | OD (field)[d] | – | – | – | 91 ± 4 | – | – | 60 ± 8 | – |
| Le Meur et al. [22] | OD (field)[d] | – | – | – | 92 ± 3<br>F 92 ± 2 | – | – | 63.4 ± 6.5<br>F 61 ± 7.5 | – |
| Gillum et al. [23] | ½ IM[e] | 68 | 70 | – | – | – | – | – | – |
| Laursen et al. [24] | IM[e] | – | – | 80 | – | – | – | – | – |

– indicates no information, *F* female, *½ IM* half-Ironman (i.e. 1.9-km swim, 90-km cycle, 21-km run), *IM* Ironman distance (i.e. 3.8-km swim, 180-km cycle, 42.2-km run), *lab* laboratory, *OD* Olympic distance (1.5-km swim, 40-km cycle, 10-km run), *SD* standard deviation

[a] All values in the table refer to males unless otherwise specified

[b] No swim-related data are available

[c] In both cases, the cycle section involved a 500 kJ (approximately 20 km) task

[d] Draft-legal (i.e. in which slip-streaming behind another cycle(s) is allowed within the cycle section)

[e] Non-drafting

**Table 3** Immunological, oxidative and cardiovascular responses to triathlon training

| Study | Athlete level | Marker type | Marker | Measure | Result |
|---|---|---|---|---|---|
| Diaz et al. [61] | 17 elite | White blood cell count | – | Season start, pre-competition, start and end of race period for four consecutive seasons | Non-significant effect of period, season or season period. Neutropenia in 8, monocytopenia in 9, and lymphopenia in 1 at some point |
| Horn et al. [60] | 48 healthy rested elites | | – | Overnight 'at rest' sample. Comparison across multiple sports. | Neutropenia ($<2 \times 10^9$/L) in 16 %, monocytopenia ($<0.2 \times 10^9$) in 5 % |
| Cosgrove et al. [62] | 10 recreational IM | Changes in peripheral differentiated and senescent T cells | – | 27, 21, 15, 9 and 3 weeks (June) prior to and 2 weeks post-race | 1 % ↑ of differentiated (KLRG1+/CD57−) CD8+ T cells and 'transitional' (CD45RA+/CD45RO+) CD4+ and CD8+ T cells with training. Two weeks post-race: differentiated CD8+ T cells at T0 level, ↑ senescent CD4+ T cells, ↓ naïve (CD45RA+/CD45RO) cells |
| Pool et al. [63] | 13 M tri, 8 M recreationally active controls | Immune function | Endotoxin induced IL-6 release in whole blood cultures | 24 h post-exercise | [Tri-plasma IL-6] and in vitro [basal IL-6] and [endotoxin activated IL-6] > that of controls. Post-endotoxin: [newly induced IL-6] in tri < in controls |
| Broadbent [64] | 15 IM, 12 UT controls | Haematology, CD4(+) lymphocyte transferrin receptor (CD71) expression, CD4(+) intracellular iron and URTI | – | Every 4 weeks for 1 year | Tri < control values for Hb (10 months), MCHC (9 months), platelet (11 months) and CD4(+)CD71(+) (1 month). Tri < controls for CD4(+)CD71(+) [3 months]; Fe(3+) [1 month]. Less URTI in tri |
| Rietjens et al. [65] | 7 M, 4 F elite | Haematology | Hb, haematocrit, erythrocyte count, mean corpuscular Hb content, mean corpuscular volume and plasma ferritin | 102 samples over 3 years | Erythrocyte count ↓ in race compared with training season. Hematological values < lower limit of normal range in off-, training- and race-season in 46, 55 and 72 %, respectively |
| Gouarne et al. [66] | 9 UT, 10 tri | Hormonal parameters | Salivary cortisol response to waking, overnight urinary cortisol, cortisone and catecholamine excretion | 10-month season | Overnight urinary cortisone excretion for tri > UT |
| Knez et al. [67] | 16 ½IM, 29 M age-matched healthy controls | Oxidative stress and antioxidant status | [MDA]; GPX, CAT and superoxide dismutase activity | – | ½IM resting GPX > controls. IM resting plasma [MDA] < controls, IM GPX and CAT > controls |
| Medina et al. [68] | 5 F, 10 M | Oxidative stress markers and prostaglandin metabolites | – | Pattern of isoprostane and prostaglandin metabolites in urine post-training | ↓ [Tetranor-PGEM and 11beta-PGF(2alpha)] and [IsoP 8-iso-PGF(2alpha)]. ↑ (vascular $PGI_2$ metabolite). Variation possibly linked to training |
| Banfi et al. [69] | 7 elite, 5 controls | Growth factors and chemokines | VEGF, EGF, MCP-1, IL-8 | T0: 1-day pre-race season start | Tri EGF and IL-8 > control EGF |
| | | | | T1: 30-min post-tri | Tri VEGF, EGF, MCP-1 and IL-8 > control VEGF, EGF, and MCP-1 |
| Konig et al. [70] | 42 M | Homocysteine levels | Plasma [total Hcy], [vitamin B(12)], and [folic acid] | Pre- and post 30 days training, pre- and post-sprint tri | No change in Hcy post-training. [Folate] > in high-training group post-training |

**Table 3** continued

| Study | Athlete level | Marker type | Marker | Measure | Result |
|---|---|---|---|---|---|
| Diaz et al. [71] | 5 elite M | Overtraining parameters 5 weeks up to major race vs. values at season onset | Total testosterone, CK, urea, total cortisol | Wednesday and Thursday of 1-week microcycles with high loads on Monday, Tuesday, Friday and Saturday | Urea and CK over 4/5 loading weeks > T0 values |
| Spence et al. [72] | 32 elite, 31 AG tri and cyclists, 20 UT controls | Respiratory health | URTI | Nasopharyngeal and throat swabs for subjects with two or more URTI symptoms over 5 months | 37 URTI episodes in 28 subjects. Infectious agents seen in 11 (2 control, 3 AG and 6 elite). Incidence rate ratios for illness in controls and elites > AG |
| Knopfli et al. [73] | 7 elite | | $FEV_1$ extrapolation of decrease in $FEV_1$ to BH limit | 8-min track run at intensities equal to anaerobic threshold. Tests at 4.4 ± 2.8 °C, −8.8 ± 2.4 °C and 3.6 ± 1.5 °C, and humidity of 52 ± 16, 83 ± 13 and 93 ± 2 % | BH ↑ within 2 years. Three athletes with BH. After extrapolation of the decrease in $FEV_1$, it was determined that 21–57 % of athletes had newly developed BH per year |
| Claessens et al. [74–77] | 52 tri, 22 controls | Structural and functional cardiac adaptations | Ventricular premature beat incidence | Number of VPB within last 2 min of maximal exercise tests on treadmill and bidirectional two-dimensional echo-doppler exam for five consecutive beats | Tri > controls for VPB and late passive diastolic filling period amplitude of excursion of the interventricular septal endocardium at the end of the LV diastole just after atrial contraction values. Tri < controls for (P top-onset systolic septal contraction) interval and P top-LV posterior wall systolic contraction interval. Tri had more incomplete right bundle block. Tri: concentric and eccentric hypertrophy and evidence of supernormal diastolic LV function. Tri max diastolic LV and RV internal diameter, diastolic interventricular septum thickness and diastolic LV posterior wall thickness > controls. It was not always the best tri who had the most significant structural cardiac adaptations |
| Douglas et al. [78, 161] | 26 tri, 17 controls | | M-mode LV echograms and doppler recordings of LV inflow velocity | – | Tri > controls for LV wall thickness, relative wall thickness, LV mass and doppler-derived ratio of early-to-late LV inflow velocities. No difference in resting systolic function, diastolic LV fractional shortening or end systolic stress |
| Knez et al. [79] | 44 tri, 44 active controls | | Brachial BP, central haemodynamics (↑ aortic BP, wave reflection, augmentation index, ejection duration, timing of reflected wave | – | No significant difference for augmentation index, timing or reflected wave, brachial or central pulse pressure. Tri > controls for sub-endocardial perfusion capacity, sub-endocardial perfusion and ejection duration |

**Table 3** continued

| Study | Athlete level | Marker type | Marker | Measure | Result |
|---|---|---|---|---|---|
| Scharf et al. [80] | 26 elite M, 27 non-athletic M controls | | Indexed LV and RV myocardial mass, end-diastolic and end-systolic volumes, stroke volume, ejection fraction, and cardiac index at rest; ventricular remodelling index and maximum LA volume | – | Combination of eccentric and concentric remodeling with regulative ↑ of atrial and ventricular chambers. Tri atrial and ventricular volume and mass indexes > controls. Tri LV and RV end-diastolic volumes > normal range in 25/26) Findings different from other types of elite |
| Platen et al. [81] | 18 tri, 69 UT/ trained student controls | Bone health | BMD | Athletes vs. controls, screening questionnaire | Lumbar spine, femoral neck, trochanter major and intertrochanteric BMD < trained controls. Femoral neck and Ward's triangle values > UT |
| Shellock et al. [82] | 20 M, 9 F | | Knee cartilage abnormalities | – | Abnormal MRI findings no greater than age-related changes for other athletic populations and UT |
| Smith and Rutherford [83] | 8 tri, 13 UT | | Regional bone density | – | No difference in spine and total BMD between tri and controls. Serum testosterone < in tri |
| McClanahan et al. [314] | 9 M, 12 F | | Total body, arms and leg BMD | Just before and immediately after 24-week competitive season | No adverse changes in BMD |
| Muhlbauer et al. [84] | 9 tri, 9 inactive controls | | Knee joint cartilage thickness | Via nuclear MRI | No significant difference between groups in patella, femoral trochlea, lateral femoral condyle, medial femoral condyle, medial and lateral tibial plateau cartilage thickness |
| Maimoun et al. [85] | 7 M | Bone metabolism, bone turnover; sexual, calciotropic and somatotropic hormones | Total and regional BMD, bone-specific alkaline phosphatase, osteocalcin, and urinary type I collagen C-telopeptide | Start of training and 32 weeks later | ↑ BMD for lumbar spine and skull but not total body or proximal femur, ↑ 1alpha,25-dihydroxyvitamin D3, insulin-like growth factor-1 and bioavailability of insulin-like growth factor-1 index. ↓ Bone-specific alkaline phosphatase. No change in parathyroid hormone, [testosterone], [insulin-like growth factor-binding protein-3] and [cortisol] |
| Newsham-West et al. [86] | 8 M and 7 F sub-elite, 17–23 years | Tibial morphology | Medial, anterior and lateral cortex thickness. Oedema/ stress fracture on nuclear MRI | Comparison of stress fracture and non-stress fracture groups | Significantly different medial cortex thickness between groups. Those with oedema within the cancellous bone or a stress fracture on MRI took time off within 2 years due to stress fracture |

**Table 3** continued

| Study | Athlete level | Marker type | Marker | Measure | Result |
|---|---|---|---|---|---|
| Lucia et al. [87] | 9 Elite | Reproductive health | Percentage body fat, hormonal profile (resting levels of follicle-stimulating hormone, luteinizing hormone, total and free testosterone, and cortisol), and seminograms | Three times within season (winter, competitive, and rest period) | Triathlon training does not adversely affect hypothalamic-pituitary-testis axis |
| Vaamonde et al. [88] | 45 including tri | | Sperm parameters (volume, liquefaction time, pH, viscosity, sperm count, motility, and morphology) | – | Morphology reaching clinical relevance for tri. Parameters tended to ↓ as training ↑ |

– indicates no information, ↓ indicates decrease, ↑ indicates increase, *[]* concentration, *AG* age-groupers, i.e. non-elite athletes who compete against other athletes within the same 5-year age range, *BH* bronchial hypereactivity, *BMD* bone mineral density, *BP* blood pressure, *CAT* catalase, *CK* creatine kinase, *EGF* extracellular growth factor, *F* female, $FEV_1$ forced expiratory volume in 1 s, *GPX* glutathione peroxidase, *Hb* haemoglobin, *Hcy* haemocyanin, *IsoP* isoproterenol, *IL* interleukin, *IM* Ironman (i.e. 3.8-km swim, 180-km cycle, 42.2-km run), *½IM* half-Ironman (i.e. 1.9-km swim, 90-km cycle, 21-km run), *LA* left atrial, *LV* left ventricular, *M* male, *MCHC* mean corpuscular hemoglobin content, *MCP-1* monocyte chemoatractant protein-1, *MRI* magnetic resonance imaging, *PGEM* prostaglandin E2 metabolite, *PGF* prostaglandin F, $PGI_2$ prostaglandin I2, *RV* right ventricle, *T0* baseline, *tri* triathlete, *URTI* upper respiratory tract infection, *UT* untrained, *VEGF* vascular endothelial growth factor, *VPB* ventricular premature beats

immune protection at the mucosal surface has been supported by data obtained over repeated short-distance races [108]. As triathletes may be exposed to waterborne microorganisms during the swim discipline, such a decreased IgA-mediated immunity may increase the risk of post-race URTI [156, 157]. Neutrophil death [107] has been seen immediately after half-Ironman-distance competition in males. Significant alterations in oxidative stress and immunological markers have also been recorded 20 min after Ironman-distance competition [113].

Nonetheless, such immune system alterations, as well as the muscle damage and metabolic changes that are induced by Olympic-distance competition, decline rapidly [103, 109]. Five days after Ironman competition, all the oxidative stress markers that were assayed by Neubauer et al. [55–58] and Wagner et al. [122]—the changes in which may have partly been due to muscle damage [123]—had returned to baseline levels [129]. The extent to which any postulated 'infection window' may exist or persist once the athlete has finished competing appears to be affected by the existence of positive adaptive mechanisms. Such mechanisms, which may include upregulation of repair mechanisms and increased activity of the endogenous antioxidative system, are likely to be highly related to the individual's training and performance status.

### 4.2 Oxidative Stress

It is possible that significant differences in the magnitude of oxidative stress markers [68] may be obtained when poorly trained vs. well-trained athletes, athletes with lower vs. higher antioxidant status, or even different periods of the training year [158] are compared. Even minor differences in training status among the same athletes can result in different alterations in markers of lipid peroxidation [55–58]. Data obtained from half-Ironman- and Ironman-distance athletes, as well as controls [67], also suggest the existence of a dose-response relationship between oxidative enzyme adaptation and the response to ultra-endurance exercise. Although it is unclear exactly how triathlon training or race duration, intensity and/or frequency may affect the propensity for DNA damage [122], better training levels may enhance protection against oxidative stress [112, 159].

### 4.3 Cardiovascular Responses

The other effects of triathlon training and/or competition with potential health-related repercussions include platelet and coagulation activation [64, 68, 119, 130, 138, 159] and other cardiovascular system-related changes [74–78, 80, 132, 135, 143, 145, 150–152, 160–163]. Platelet activation (which may increase the risk of thromboembolytic events) and markedly increased plasmin formation may occur during competitions lasting over 2 h [130, 138, 164]. Both appear to be triggered by run-induced mechanical stress on thrombocytes and/or inflammation [130]. However, Olympic-distance triathlon was found to have no significant negative effects on either left ventricular function or myocardial tissue in adult males [151]; nor was Olympic-distance competition found to affect blood B-natriuretic peptide concentration—a marker of cardiac failure—in regularly-trained triathletes [149]. Elevated levels of troponin and B-type natriuretic peptide were noted 45 min after both half-Ironman- and Ironman-distance races, and both markers correlated with decreased right ventricular ejection fractions [136, 144]. Although the levels of these indicators of myocardial injury were back to normal within

1 week, Ironman competition was reported [141] to often result in persistently raised cardiac troponin T (cTnT) levels (agreeing with Rifai et al. [140]). This increase in CTnT was associated with echocardiographic evidence of abnormal left ventricular function. Therefore, abnormal left ventricular function [144] may increase with race distance [135, 143]. Although such abnormal left ventricular function generally disappears within 24 h [135], it may be linked to the occurrence of pulmonary oedema [165–167].

However, even when short-term right ventricular recovery appears complete, long-term training and competition may lead to myocardial fibrosis and remodeling in a small, genetically susceptible, percentage of athletes [74–77, 168]. This theoretically might provide a foundation for atrial and ventricular arrhythmias and increase cardiovascular risk, particularly in older athletes. La Gerche et al. [144] found increased right ventricular remodeling in well-trained endurance athletes with a longer competitive history. Their results suggest a cumulative effect of repetitive ultra-endurance exercise on right ventricular change and possibly myocardial fibrosis. The long-term sequelae of the structural or other alterations that occur to the adult triathlete heart with training and competition [74–77] warrant further investigation. The long-term consequences of the transient functional abnormalities that have also been observed post-triathlon in children [134] are also unknown. More ventricular premature beats at the end of a maximal exercise test have been noted in well-trained adult triathletes than in controls [75]. However, it was not the triathletes with the best competition results who had the most characteristics of eccentric and concentric left ventricular hypertrophy; nor did the athletes who exhibited the greatest training volumes exhibit the most extensive heart adaptations. Nonetheless, the triathlete who displays the first indications of evolution to a pathological hypertrophic and dilated cardiac myopathy, i.e. ventricular premature beats and other specific electrocardiographic and echocardiographic findings, is a candidate for ‘sudden cardiac death’ [75]. Acute changes in baseline hemodynamics and autonomic regulation (characterized by a decrease in stroke index, blood pressure, total peripheral resistance index, baroreceptor sensitivity, vagal modulation of the sinus node, and increased heart rate, cardiac index, and sympathetically-mediated vasomotor tone) that occur with competition may also make Ironman-distance athletes vulnerable to orthostatic challenge post-race [145, 169].

### 4.4 Other Responses with Potential Health Consequences

The other responses to triathlon training and racing that have potential health consequences include changes in bone mineral density. One study involving adolescent females [170] concluded that the generalised anatomical distribution of triathlon training load does not significantly enhance total bone mineral density. Junior males, on the other hand, exhibited lower bone mineral density than athletes from other sports [81]. They had significantly elevated levels in most femoral regions, but exhibited no differences from untrained controls at L2 and L3 of the lumbar spine. The authors concluded that training regimes with high volume but low intensities do not, or only slightly, induce osteogenic effects, while a variable training protocol with short-lived but high-intensity forces will have the highest positive stimulatory effects on bone formation. The implications for fracture risk (e.g. in the Wards triangle, as a result of cycle falls) are unknown. Thinner anterior tibiae and the presence of oedema on magnetic resonance imaging (MRI) appears to be a precursor to stress fracture development, however [86]. In Ironman triathletes, the spectrum of abnormal MRI findings of the knee and shoulder was no greater than age-related changes previously reported for other athletic populations and non-athletes [82, 171]. Little else is known regarding the extent to which the susceptibility to skeletal problems of triathletes [81, 83, 170, 172, 173] is affected by training-induced modulation of circulating hormone levels [85, 87] and/or relative energy deficiency in sport [174]. Some triathletes exhibit disordered eating [175, 176] and may suffer from anorexia nervosa [177], bulimia nervosa [178], or other nutritional disorders [172, 173, 179], all of which may influence susceptibility to injury and/or illness.

## 5 Illness

Our knowledge of the degree to which the immunological/oxidative stress of training and racing is reflected by the occurrence of illness is limited. Only six groups [10–12, 64, 71, 94] have prospectively investigated triathlon illness. Vleck collected 25.1 ± 5.6 weeks (mean ± SD) of Olympic-distance national squad athlete daily training diary data in 1994. The eight athletes concerned trained 8:10 ± 2:06 hh:mm (mean ± SD) per week, and raced (mean ± SD) 20.3 ± 10.9 times. They rated (mean ± SD) 6.4 ± 3.4 of such events as ‘best performances’. Training and injuries were recorded. The athletes also logged the occurrence of each one of the highest cited symptoms within each of Fry et al.’s 12 classes of putative overtraining symptoms [180–182]. These symptoms [12] were interpreted as symptoms of ‘illness’. The athletes logged 247 such separate incidents. Delayed-onset muscle soreness (DOMS) was the most commonly reported symptom, followed by ‘heavy legs’, loss of appetite and then virus-related symptoms. Such symptoms coincided with self-

diagnosed performance decrement on 15 % of occasions. Performance [135] also declined on 34.7 and 21.5 % of the occasions that DOMS and headaches were reported, respectively. It declined on less than 7 % each of the times that the athlete recorded heavy legs, a sore throat, gastric problems, or reported viral infection. In 66.7 % of DOMS cases and 76.9 % of cases of heavy legs, the performance decrement could have been due to another illness-related symptom, or even to an injury. Interestingly, the athletes never reported a drop in performance on the same day that they reported a 'stuffy nose', 'loss of appetite', 'chest cold', 'head cold', 'sleeplessness' or 'nausea'. The athletes neither explicitly stated the criteria that they used to decide whether a drop in performance had occurred or not, nor how training was interrupted or modified because of illness.

Andersen et al. [94] implemented a slightly different illness definition, with Ironman athletes. They defined illness as any health problem that was not related to the musculoskeletal system (e.g. respiratory tract infections, influenza or gastrointestinal infections, and not DOMS). Over 26 weeks, 156 cases affecting 104 athletes (i.e. 60 %) were reported, equating to 5.3 illnesses per 1,000 athlete days. Nine percent of cases did not lead to any time loss, 34 % led to 1–3 days off, 36 % led to 4–7 days off, 19 % led to 8–28 days off and 3 % led to more than 28 days off.

Medical diagnosis of illness can itself be problematic [183]. It is certainly unclear to what extent upper respiratory symptoms in triathletes may be due to infection or to other non-infectious inflammatory symptoms that mimic a URTI [183]. Of 25 cases of URTI symptoms that were reported for 63 triathletes and cyclists [72], 28 % each were due to rhinovirus and influenzae (A and B), 16 % to parainfluenzae, 8 % each to *Streptococcus pneumoniae* and coronavirus, and 4 % each to Epstein–Barr virus reactivation and metapneumovirus. Four percent of URTI symptoms were unaccounted for and could have been due to local drying out of the mucosal surfaces and increased exposure to airborne pathogens [184], to bronchial hyperreactivity (the rate of development of which has been reported to be 195–286 faster in elite athletes than is normal for asthma development [72, 73, 185–187]) or to muscle damage-induced migration of inflammatory cytokines [183]. The incidence of URTI in both the triathletes and the untrained controls who were assessed by a yearlong study [64] was lower than the international average of two per year.

Thus, the extent to which the immune changes that occur as a result of the stress of triathlon training and/or racing alter overall disease susceptibility [156, 157] is not usually likely to be major, but is unclear. However, the conditions that are involved in open-water swimming may increase the risk for specific conditions [187] such as *Acanthamoeba keratitis* [188], and for uncommon diseases such as schistosomiasis [189, 190] and leptospirosis. Leptospirosis has been incurred by triathletes training [191] and competing [192–197] in contaminated surface water. Crucially, the affected athletes were only diagnosed as having been infected after awareness of a leptospirosis outbreak [198] was independently established for the race locality. The clinical presentation of leptospirosis varies and may present similar symptoms to common febrile illnesses. Thus, there is also a potential problem in triathletes of illness being misdiagnosed [199, 200]. The fact that an inappropriate management strategy (with potentially negative repercussions for rehabilitation time) may then be implemented was recently highlighted [199]. However, the extent to which such issues occur is unknown. At present, the overall outcomes of triathlete illness in terms of economic cost, training time loss and/or even performance decrement [201] are unquantified. Only (potentially) indirect evidence of the extent to which illness may lead to changes in training load exists [12, 202]. The national squad triathletes who were examined by Vleck in 1994 [12, 202] logged lower average weekly training durations than were expected of top-level athletes of that time [9]. Unfortunately, as illness and injury can overlap, it can prove difficult to ascertain the real outcome of either in isolation [12].

## 6 Injury

Injury 'causing cessation of training for at least one day, reduction of training, or seeking of medical aid' has been reported to affect 29 % [13] to 91 % [202, 203] of adult triathletes at any one time (ESM Table S2) [12, 14, 32, 39, 42, 94, 204–226, 228–232, 238]. The wide range of reported values is likely due to a failure to standardize methodology or surveillance between studies as the International Olympic Committee (IOC) guidelines recommend [233]. The other methodological difficulties with the triathlon injury literature have been reviewed [234–237] and are not repeated here. Only one retrospective study has compared the prevalence of training-related injury between different sex, ability-level and event-distance specialization groups, using the same definition and reporting methods in each case [12, 13], with no difference being found. No one has yet conducted a similar comparative study across all the triathlon age groups. Nor does the proportion of athletes who report for medical aid at sprint distance events [32] appear to be influenced by age, sex or competitive experience. Whether the same consistently applies to all the other triathlon distances and formats [238] is unknown.

Obtaining meaningful injury incidence values for triathletes is a challenging task. This is partly because of

difficulties in quantifying and weighting overall training stress across (at least) swimming, cycling, running and weight-training [239]. The typical presentation and characteristics of overuse injuries also makes them difficult to record in epidemiological studies when time-loss definitions are used [240]. No sudden death rates for training exist and there is no long-term international registry system for this within races. The sudden death rate for USA Triathlon-sanctioned events over 2006–2008, involving 959,214 participants, was estimated by Harris et al. [241] at 1.5 (0.9–2.5) deaths per 100,000 participations, with an average age at death of (mean ± SD) 42.8 ± 10.1 years. It was (but not significantly) greater in males and in races with more participants. When data from 2003 to 2011 (for triathlon, duathlon, aquathlon, and off-road triathlon events) were examined [242], an approximate figure of one death per 76,000 participants per year was obtained. The absolute fatality rate increased with participation rates. Most were rated as sudden cardiac death events, yielding a higher rate than reported for half marathons and marathons between 2000 and 2010 [243] (i.e. 0.28 and 0.52 per 100,000, respectively). According to Harris et al. [241], sudden death during swimming accounted for 1.4 (0.8–2.3) deaths per 100,000 participations per year. The equivalent values for triathlon cycling and running were 0.1 (0.01–0.07) and 0.0 (0.0–0.3). Slightly, but not significantly, higher death rates were recorded for the races with short (<750 m) or longer (>1,500 m) swims than for those with 750–1,500 m swims. It is not known why.

Self-assessed overuse injury incidence rates of 0.74–76.7 per 100 athletes, and of 10.0–23.8 per 1,000 training and racing hours, respectively (depending on the month of the year), have been obtained prospectively for small ($n$ = 11–43) samples of Olympic-distance triathletes [12]. Values of 1.39 and 18.45 incidences per 1,000 training and racing hours over various distances, respectively, have also been obtained [232]. The injuries were not confirmed by medical diagnosis. A total of 20.1 presentations for medical assistance per 1,000 h of sprint-distance, Olympic-distance and fun-distance (i.e. 0.15–0.3 km swim, 7–10 km cycle, 1–3 km run) competition has been recorded [238]. Although few directly comparable data exist, injury rates are usually thought to be higher within competition [94, 221, 222, 232]. The incidence of (traumatic) crowding-, hydration- and/or heat-related injuries in particular is also thought to be higher (ESM Table S3) [39, 203, 206–209, 212–214, 222, 226, 229, 230, 238], although no training-related studies appear to have assessed these issues. The lack of detail of assessment that has been involved in most larger-scale studies also makes it difficult to assess how widespread the problems that have only or mostly been reported by case studies (e.g. ESM Table S4) [41, 54, 166, 177, 188, 191, 196, 200, 244–274, 321–324], and that may to some extent be 'triathlon specific', actually are.

No prospective intergroup (age, sex, ability or event distance) comparisons of injury incidence rates exist for the endurance base, pre-competitive and competitive periods. Only one study [230] has investigated the effect of race distance and athlete ability level on the temporal occurrence of race injuries—a topic with clear implications for the depth and timing of provision of medical support. Wind speed, humidity, and dry-bulb temperatures in the study in question varied widely, but the extent to which this was over each race or between events is unclear. Injury (defined in this case as a presentation for medical assistance) affected 10.8 % of half-Ironman- and 37.7 % of Ironman-distance age-group starters, respectively. Previously, it was reported to affect 15–25 % of elite Ironman-distance competitors [275, 276]. Most athletes took 5–9 h to finish. A total of 72.2 % of half-Ironman injuries occurred between hours 6 and 7, during which time medical personnel needed to be prepared for 78 presentations for assistance per 1,000 race starters. No equivalent rates exist for shorter-distance events. The proportion of injuries that were severe was higher during the Ironman event than for the half-Ironman, and was calculated to be (mean ± 95 % confidence interval) 38.2 ± 6.0 % of those receiving treatment at any given time. Treatment duration increased with finishing time. The highest proportion of severe injuries occurred in the half-Ironman athletes who took longer to finish, or the Ironman athletes who were faster, than the rest of their cohort.

Contusions, abrasions/grazes and blisters are the most commonly reported short-distance race injuries [238]. At half-Ironman events, dehydration (50.8 %) and muscle cramps (36.1 %) are the primary medical diagnoses. Both have been reported in almost equal proportions (38.9 vs. 37.7 %) at an Ironman-distance event [230]. The percentage of so-called race injuries that are actually existing, training-related injuries that have been exacerbated by competition is unknown. Injury outcome after a race has finished (e.g. death from complications arising from chest infection) is also not described (ESM Table S5) [12–14, 32, 206, 208, 211, 212, 214–218, 220, 222, 226]. Gradual-onset overuse injuries are the most commonly reported training injuries. They have been reported to occur in approximately three times as many athletes as do acute injuries [209, 215, 232, 277] (ESM Table S6) [12, 14, 94, 203, 206, 209–211, 213, 214, 216, 217, 221, 222, 225, 226, 232, 237, 238]. The true value may be higher given the fact that retrospective recall is generally poorer for overuse injuries than for traumatic injuries [232].

Most athletes rate their training-related injuries as 'minor' to 'moderate' (i.e. incurring up to 21 days off) when a time-loss definition is used. However, according to Finch

"it is often the medically less severe injuries that are considered to be more severe by the athlete, although they do not require medical treatment, as they have the potential to severely limit an athlete's performance" [278]. Many injured triathletes may continue training [12, 217, 226, 277]. Running, cycling and swimming training is modified in 17–21, 26.2–75 and 42–78 % of injury cases [202], respectively, and injury recurrence is probably a major issue [202, 279].

We highlighted the fact that the influence of certain injury risk factors may differ with sex, format and event-distance specialization (Table 1 and Sect. 2) [12, 280]. Minimal examination into which putative risk factors are most highly linked to injury in each group has taken place (Table 4 and ESM Tables S4 and S5) [12–14, 32, 41, 54, 166, 177, 188, 191, 196, 200, 203, 206–222, 224–226, 232, 238, 244–274, 281, 315, 317, 318, 321–324]. Although various potential (and even triathlon cross-training-specific) mechanisms of injury have been speculated upon [217, 236, 237, 282–284], they are largely unverifiable. For example, drowning was the reported cause of death for the swim fatalities recorded by Harris et al. [241], but drowning lacks the accurate methods of risk exposure that are needed to establish aetiology [285]. The actual cause could be something else (e.g. autonomic conflict [286, 287], deterioration in performance [288] in cold water, swim-induced pulmonary oedema [249], or hyperthermia). It is noteworthy that all the swim deaths occurred in open water, raising the question as to whether there is something about mass participation competition that is significant [286]. Periodic health screening (such as the IOC Periodic Health Evaluation [289]) is not routinely implemented in the sport of triathlon to screen for risk factors for sudden cardiac death [290]. With only one abstract on the topic published thus far, the extent to which triathletes enter races with pre-existing medical conditions is unknown. Importantly, of the sudden deaths reported by Harris et al. [241], seven of nine athletes were found on autopsy to have had cardiovascular abnormalities. Six had mild left ventricular hypertrophy. Two years later, the USA triathlon fatality incidents study [242] concluded, despite incomplete access to relevant medical data, that most non-traumatic deaths were likely due to sudden cardiac death. However, injuries are usually attributed to "a result of failure to adjust pace within safe limits for specific environmental conditions" [209, 237], or to "inadequate implementation of (race) safety precautions"[247].

## 7 Training and Performance Status Indicators

It has been said that "a fine line exists between the level of training that is required for optimal performance, and that which induces problems" [291]. Laboratory-based (physiological, immunological, haematological, cardiorespiratory and biochemical) testing may therefore sometimes be conducted to ascertain the individual's health status. Only some markers have been shown to be related to triathlon performance and thereby possess criterion validity (Table 5) [10–12, 29, 30, 65, 68, 90, 145, 223, 292–305]. Whether they are sensitive enough to detect a drop in performance before it becomes competitively meaningful [306] is unclear. To date, peak power output and blood pressure variability appear to be the only variables that are correlated with triathlon performance that have been used [300] in prospective investigations of the links between training and health in triathletes [10–12, 16, 304]. Peak power output appeared not sensitive enough to detect the early signs of overreaching in well-trained males [300]. Whether it may react later to more extended exhaustive training is unknown.

In any case, by the time an underlying problem has been confirmed in the laboratory, it may be too late. Ideally the individual's distress markers should be monitored far more regularly, in conjunction with his/her training, and on an ongoing basis. Indeed, it has been observed that as regards heart rate variability (HRV) related data [305], for example, attempting to diagnose the athletes' physical status from records obtained on a single isolated day may be a somewhat meaningless exercise. Weekly averages and rolling averages of RR-interval (the interval from the peak of one QRS complex to another on an electrocardiogram)-related values and the coefficient of variation of HRV, on the other hand, were shown to differ between an athlete who developed non-functional overreaching and a control. These results complemented data obtained in swimmers—in which a shift in autonomic balance towards sympathetic predominance 1 week earlier was linked to increased risk for URTI and muscular problems [307].

Although HRV holds promise as an indicator of maladaptation, as do baroreflex sensitivity and blood pressure variability [145], monitoring it may only prove realistic for some. Dolan et al. [3] reported that only 20.9 % of triathletes used a heart rate monitor. In contrast, 45.5 % kept a training diary [12]. Training diary compliance is therefore likely to be higher. The question arises as to whether the right things are being monitored in the diary, as well as how the data are being analyzed. Scores on questionnaires such as the Daily Analysis of Life Demands for Athletes (DALDA) [90], the Recovery-Stress Questionnaire for Athletes (RESTQ-Sport) [303], the Perceived Stress Scale (PSS), the Training Distress Scale (TDS), the Athlete Burnout Questionnaire (ABQ) and the Multi-component Training Distress Scale (MTDS) [10, 11], as well as on a combination of shortened versions of the Profile of Mood States (such as the Brunel Mood Scale [BRUMS] and the

**Table 4** Risk factors for injury that have been directly assessed in the triathlon literature (modified and updated from Vleck [202], with permission)

| Possible risk factor | Injury variable | Significant relationship (at the 95 % confidence level or higher) observed between risk factor and injury variable | |
|---|---|---|---|
| | | Yes | No |
| Sex | Overuse injury occurrence | Vleck [12] (Retros: anatomical location) | Collins et al. [211], Villavicencio et al. [226] (BP), Williams et al. [210], Manninen and Kallinen [216], Egermann et al. [222] (LB), Zwingenberger et al. [232], Korkia et al. [214], Burns et al. [221], Gosling et al. [238] |
| | Number of injuries | – | Vleck [12] (Retros: OD, IM of E, SE or rec level) |
| Age | Injury occurrence | Egermann et al. [222], Gosling et al. [238] | Collins et al. [211], Zwingenberger et al. [232] |
| Height | Injury occurrence | – | Vleck and Garbutt [14], Vleck [12] (Retros: OD F and IM of both sexes), Korkia et al. [214] |
| Body mass index | Injury occurrence | – | Collins et al. [211], Vleck and Garbutt [14], Korkia et al. [214], Villavicencio et al. [226], Vleck [12] (Retros: OD F and IM of both sexes) |
| COL5A1 CC1 genotype | Exercise-associated muscle cramping | O'Connell et al. [315] | – |
| Foot type, orthopaedic problems | Injury occurrence | Burns et al. [225] | Vleck [12] (Retros: F), Vleck and Garbutt [14] |
| Orthopaedic problems | Overuse injury incidence | – | Vleck and Garbutt [14] |
| Previous injury | Injury incidence | Korkia et al. [214]***, O' Toole et al. [203], Burns et al. [221], Migliorini [213], Villavicencio et al. [226] (BP, NP) | Manninen and Kallinen [216] (lower limb, LB) |
| Achilles tendon, hamstring, knee and lower-back injury | Calf injury occurrence | Vleck and Garbutt [14] | – |
| Diet | Injury occurrence | – | Vleck and Garbutt [14][a] |
| Use of NSAIDs | Hyponatremia | Wharam et al. [281] | – |
| Restless sleeper, restless sleep, health worries | Overuse injury incidence | – | Vleck and Garbutt [14] |
| Psychological state/total mood disturbance (basic analysis)/ daily or weekly hassles | Overuse injury incidence | Fawkner et al. [219] (daily hassles)[b] | Vleck and Garbutt [14] |
| Position on cycle/degree of trunk flexion on cycle/use of aerobars | Overuse injury incidence | – | Vleck and Garbutt [14], Manninen and Kallinen [216] (LB) |
| Cycle gear ratio/crank length | Cycle injury | – | Massimino et al. [209], Vleck and Garbutt [14] |
| Use of and type of clipless pedals | Overuse injury incidence | – | Vleck and Garbutt [14] |
| Cycle cadence | Overuse injury incidence | – | Massimino et al. [209], Vleck and Garbutt [14] |
| Cycling cadence trained at | Overuse injury incidence[e] | – | Massimino et al. [209], Vleck and Garbutt [14] |
| Faulty running shoe construction | Plantar fasciitis | Wilk et al. [220][c] | – |
| Training in other sports | Overuse injury incidence | Collins et al. [211]* | Manninen and Kallinen [216] |
| Sporting background | Injury occurrence | Williams et al. [210] (B) | Vleck and Garbutt [14], Korkia et al. [214] |
| Initial sporting background | Overuse injury incidence[d] | Williams et al. [210] (B) | Collins et al. [211] |
| Level reached in single sport | Injury incidence | – | Vleck [12] (Retros) |

**Table 4** continued

| Possible risk factor | Injury variable | Significant relationship (at the 95 % confidence level or higher) observed between risk factor and injury variable | |
|---|---|---|---|
| | | Yes | No |
| Years of competitive experience | Injury occurrence | Burns et al. [221] (R), Williams et al. [210] (T, $r = 0.17$***) | Vleck [12] (Retros: S, B, R, elite OD M), Villavicencio et al. [226] (NP) |
| | Injury incidence | Korkia et al. [214], Williams et al. [210], Egermann et al. [222], Villavicencio et al. [226] (NP) | Vleck and Garbutt [14] |
| Years of competitive swimming or cycling experience | | | Vleck [12] (Retros: E, SE or age-group OD M or F) |
| Years of competitive running experience | Number of running injuries | Vleck [12] (Retros: IM M, $r = 0.59$**) | Vleck [12] (Retros: OD M) |
| Number of triathlons participated in/years of triathlon experience | Low BP or neck pain | Villavicencio et al. [226] (NP), Korkia et al. [214] | Collins et al. [211] |
| Athletic status | Overuse injury incidence | – | Collins et al. [211], Villavicencio et al. [226] (BP) |
| Athlete ability level | Injury incidence | Shaw et al. [224], Egermann et al. [222] | Korkia et al. [214], Vleck and Garbutt [14] (for anatomical location) |
| Performance level | Injury incidence | Egermann et al. [222] (muscle tendon injury) | Zwingenberger et al. [232] (top 50 or bottom 50 %) |
| Personal best time | Injury to specific anatomical site | – | Vleck and Garbutt [14] (OD: T, S, B, R) |
| Main competitive distance | Injury occurrence | Williams et al. [210] (F) | Korkia et al. [214], Williams et al. [210] (M) |
| Race distance trained for | Overuse injury incidence[d] | (For anatomical location) Vleck [13] (for IM vs. OD) | Vleck 2010 [12] (Retros, F) |
| Race distance done | Medical assistance | Gosling et al. [238] (B and R) | Gosling et al. [238] (S) |
| Race distance (IM vs. triple IM) | Hyponatremia prevalence | Rust et al. [317] | – |
| Degree and specificity of coaching and feedback | Overuse injury incidence[d] | – | Collins et al. [211], Vleck and Garbutt [14] (not detailed), Egermann et al. [222] (yes/no), Zwingenberger et al. [232] (yes/no) |
| Back-to-back cycle run transition training (yes/no) | Overuse injury incidence[d] | – | Vleck and Garbutt [14] |
| Presence of medical care (yes/no) | Injury | – | Egermann et al. [222] |
| Presenting for medical aid in race (yes/no) | Injury incidence | Gosling et al. [32] | – |
| Stretching practice/flexibility | Injury incidence[d] | Negative, Massimino et al. [209] (Ank, Ach [before bike and after swim])[e] | Ireland and Micheli [206], O'Toole et al. [203], Manninen and Kallinen [216] |
| Warm-up/cool-down practice | Number of injuries | Burns et al. [221] | Ireland and Micheli [206], Vleck and Garbutt [14], Korkia et al. [214] |
| Warm-down/stretching after training | Overuse injury incidence[d] | – | Vleck and Garbutt [14] |
| Cool-down practiced (yes/no) | Number of injuries | – | Korkia et al. [214] |
| Altered blood flow | Gastrointestinal symptoms | – | Wright et al. [318] |
| Number of races per season/participation in competition/time spent competing | Overuse injury incidence[d] | Zwingenberger et al. [232] | Villavicencio et al. [226] (CP) |

**Table 4** continued

| Possible risk factor | Injury variable | Significant relationship (at the 95 % confidence level or higher) observed between risk factor and injury variable | |
|---|---|---|---|
| | | Yes | No |
| Training time | Injury incidence[d] | Egermann et al. [222] (T), Shaw et al. [224] (T, B, R) | Villavicencio et al. [226] (CP), Ireland and Micheli [206] (SBR); Korkia et al. [214] (SBR), Shaw et al. [224] (S), Murphy [207], Zwingenberger et al. [232] (<10 h or ≥10 h), Manninen and Kallinen [216] (B time and LB) |
| Time spent cycling | Number of cycling injuries | Vleck and Garbutt [14] ($r = 0.28$***)[f] | – |
| Time spent running | Number of running injuries | Vleck [12] (Retros: [E and SE OD and IM] F, $r = 0.63$**) | – |
| | Number of cycling injuries | Vleck and Garbutt [14] (Retros, $r = 0.26$***) | – |
| Time spent running | Occurrence of Achilles tendon injuries | Vleck [12] (Retros: OD M, $r = 0.44$***) | – |
| Time spent doing long runs | Number of running injuries | Vleck [12] (Retros: SE M, $r = -0.76$***, E OD F, $r = 0.90$*) | Vleck [12] (Retros: SE F) |
| Amount or percentage of training time spent in each discipline | Injury incidence[d] | – | Ireland and Micheli [206] |
| Average time doing intervals, hard, moderate, easy and hill training in all disciplines combined | Injury incidence[d] | – | Korkia et al. [214] |
| Total time spent doing speed cycle work during a race week without taper | Number of injuries | Vleck [12] (Retros, $r$ = 0.29–0.60) * to *** depending on group | – |
| | Lower-back injury prevalence | Vleck [12] (Retros: OD M, $r = 0.52$***) | – |
| Percentage of (cycle) training spent doing interval work | Number of overuse injuries | Vleck [12] (B) (SE OD M, $r = 0.92$***) | O'Toole et al. [203] (B, R) |
| Percentage of time spent or number of sessions spent doing cycle hill repetitions | Number of injuries | Vleck [12] (Retros OD M, $r = -0.44$* and $-0.39$* | Massimino et al. [209], Vleck [12] (Retros IM M), O'Toole et al. [203] (B, R) |
| Time out of seat during training sessions | Number of injuries | – | Massimino et al. [209] |
| Percentage of time spent or number of sessions spent doing run hill repetitions | Number of injuries | Vleck [12] (Retros, M SE OD and E OD M) | – |
| Increased percentage of time spent doing quality or track run work | Number of injuries | Vleck [12] (Retros, $r = 0.66$ for M and 0.91 for F***) | Massimimo et al. [209] (R) |
| Training distance | Number of (cycling) injuries | – | Ireland and Micheli [207], Collins et al. [211], Egermann et al. [222] |
| | Number of run overuse injuries | Vleck [12] (Retros: [E, SE and NE] M, $r = 0.23$*) | – |
| | Injury incidence[d] | Burns et al. [221] | Massimino et al. [209] (KI), Korkia et al. [214], O'Toole et al. [203] (S, B, R), Manninen and Kallinen [216] (LB) |
| Swimming distance | Number of run injuries | Vleck and Garbutt [14] ($r = 0.34$**) | – |
| Overdistance swim work, fartlek, hypoxic, kick, pull in swim | Incidence of swimming injuries | – | Massimino et al. [209] |

**Table 4** continued

| Possible risk factor | Injury variable | Significant relationship (at the 95 % confidence level or higher) observed between risk factor and injury variable | |
|---|---|---|---|
| | | Yes | No |
| Weekly cycling distance | Number of injuries | Williams et al. [211] ($r = 0.14$**) | – |
| | Number of run injuries | Vleck and Garbutt [14] ($r = 0.25$*) | – |
| Cycling overdistance, pace, cadence | Number of cycling injuries | – | Massimino et al. [209] |
| Increased cycle overdistance work | Number of cycling injuries | – | Massimino et al. [209] |
| Distance covered during run hill repetitions | Occurence of Achilles tendon injuries | Vleck [12] (Retros: OD M, $r = 0.92$***) | – |
| Higher pre-season running mileage | Number of injuries | Burns et al. [221] | – |
| Mileage for week before event | KI incidence[d] | – | Massimino et al. [209] |
| Number of triathlon workouts per week | Injury incidence[d] | Vleck and Garbutt [14] (RI, $r = 0.25$*) | Korkia et al. [214] |
| Number of 'other' (than speed, long or hill repetition) cycle sessions per week | Number of overuse injuries | Vleck[12] (Retros: OD M, $r = 0.35$*) | Vleck [12] (Retros: IM M) |
| Number of other types of cycle session, increased percentage of time sent doing cycle interval work | Number of injuries | Vleck [12] (Retros, $r = 0.92$ for both)* | – |
| Number of run sessions per week | Number of run injuries | Vleck and Garbutt [14] ($r = 0.23$*) | – |
| Number of run speed sessions | Injury incidence[d] | Vleck [12] (Retros, $r = 0.56$ for IM)* | – |
| Number of hill repetition run sessions per week | Number of overuse injuries | Vleck [12] (SE OD M, $r = 0.92$***) | – |
| Number of other (i.e. not speed, hill repetition or long) run sessions | Injury incidence[d] | Vleck [12] (Retros, $r = 0.63$ for OD F*) | – |
| Long-run session time | Injury incidence[d] | Vleck [12] (Retros, $r = 0.86$ for SE OD F*) | – |
| | Number of running injuries | Vleck [12] (Retros: SE OD M, $r = 0.76$**) | Vleck [12] (Retros: SE OD F, $r = 0.76$**) |
| Duration of speed run sessions | – | Vleck [12] (Retros: IM M) | Vleck [12] (Retros: IM F) |
| Training sequence | Injury/KI incidence[d] | – | Massimino et al. [209], Korkia et al. [214] |
| Strength training (yes/no) | Overuse injury incidence[d] | – | Korkia et al. [214], Collins et al. [211], Manninen and Kallinen [216] (LB) |
| Combined intensity work for all three disciplines | Injury incidence[d] | – | Korkia et al. [214] |
| Pace/intensity (not in detail) | Injury incidence[d] | Vleck [12] (Retros, cycle work)*, Massimino [206] (Ank, Ach) | Massimino et al. [209] (K), Korkia et al. [214], O'Toole et al. [203] |
| Increase in training load | Injury incidence[d] | Vleck [12] (Pros) | Korkia et al. [214] (Pros) |
| Cycled faster | Foot, ankle, Achilles tendon injury | – | Massimino et al. [209] |
| Increased other (i.e. not long, hill repetition or speed) cycle training | Foot, ankle, Achilles tendon injury | Vleck [12] (Retros, $r = 0.35$)* | – |

**Table 4** continued

| Possible risk factor | Injury variable | Significant relationship (at the 95 % confidence level or higher) observed between risk factor and injury variable | |
|---|---|---|---|
| | | Yes | No |
| Weighted combined cycle and run training in intensity levels 3–5 of 5 (with level 5 being the highest intensity) | Injury incidence[d] | Vleck [12] (Pros)* | – |

– indicates no information, *Ach* Achilles tendon, *Ank* ankle, *B* bicycling, *BP* back pain, *CP* cervical pain, *E* 1994 elite (probably most similar to very-well-trained recreational athletes), *F* female, *IM* Ironman (i.e. 3.8-km swim, 180-km cycle, 42.2-km run), *K* knee, *KI* knee injury, *LB* lower back, *M* males, *NE* non-elite, *NP* neck pain, *NSAIDs* non-steroidal anti-inflammatory drugs, *OD* Olympic distance (i.e.1.5-km swim, 40-km cycle, 10-km run), *Pros* prospective study, *R* running, *Rec* recreational, *Retros* retrospective study, *RI* running injuries, *S* wimming, *SE* 1994 sub-elite (probably most similar to good age-groupers, i.e. athletes competing within their 5-year age-group band, of today), *SBR* swim, bike and run, *T* triathlon

* $p < 0.05$, ** $p < 0.02$, *** $p < 0.001$

[a] Very limited data. Potential links between diet/disordered eating/occurrence of female athlete triad and triathlon injury have not yet been investigated

[b] Value correlation coefficient not given because it was calculated for a three-sport sample

[c] Previous lower-limb pain was not linked to the onset of lower-back pain

[d] Unless a prospective study, most incidence data actually refer to incidence proportions

[e] Lumbar pain linked with prior foot, ankle or knee injury

[f] Some indication of a sex, age, event distance and or athlete ability/experience effect seen in this study

Profile of Mood States for Children [POMS-C]) [10, 12, 300] and various signs and symptoms of illness and injury [12], assess mood disturbance, perceived stress and training or other distress symptoms to various degrees. They may all potentially be incorporated into such logs.

Main et al. [11] found, using linear mixed modeling, that both various combinations of training factors and psychological stressors (as monitored on a weekly basis via the PSS, BRUMS, TDS and ABQ) were linked with signs and symptoms of both illness and injury in age-group triathletes. The number of training sessions and the number of completed run sessions per week, as well as perceived programme difficulty (see Tables 1 and 4), had significant effects on signs and symptoms of URTI, injuries or minor aches and pains, although less so than did individual athlete scores on the PSS [308]. We note that the TDS itself (which was developed from the list of distress symptoms that Fry et al. [309] identified from interviews with fit individuals who were exposed to repeated intense training) was later validated against performance in a laboratory time-to-fatigue trial. TDS responses were also compared across a high-intensity training group and a control group of triathletes, and decreases in running performance in the training but not the control group were reflected by the athletes' TDS scores [310]. However, neither actual nor self-assessed performance was assessed in the study by Main et al. [11].

Certainly, potential indicators of the fitness fatigue response, or of performance (as indeed may training-related risk factors for injury and illness), are likely to function better if they have been tailored to the individual athlete. Vleck [12] retrospectively calculated individual specific peak performance norms for various indicators on Fry et al.'s (longer) 1991 list [180, 181] of potential overtraining symptoms, for each of eight national squad triathletes. The fact that these norms were only obtainable over an average of six 'best performance' occasions rather than the recommended eight [311], even though the study lasted approximately 6 months, underlines the difficulties in producing such norms. The extent that the weekly values for each distress indicator diverged from the individual athlete's peak performance norm were then modelled together with composite training load scores and self-reports of performance decrement, using binary logistic regression. The combination of the heavy legs and DOMS scores for the same week, the composite appetite score for the previous week, the POMS-C confusion factor score for both 2 and 3 weeks before, and the POMS-C anger factor score for the previous week increased the predictive power of the model for performance decrement. New overuse injury had previously been shown to be associated with an increase in combined weighted cycle and run training at higher intensity levels 2 weeks prior to onset. Interestingly, prediction was not improved by incorporation of any derived training:stress recovery variables for each of the athletes into the model. This may have been due to the difficulty in producing valid, individual-specific indices that account for relative rather than absolute changes over time in the training stress to which each athlete is exposed.

**Table 5** Selected studies that have related physiological, cardiovascular, immunological, neuromuscular, endocrinological and/or psychobiological markers to triathlon performance, non-functional overreaching, burnout or overtraining

| Study | Group | Design | Markers | Result |
|---|---|---|---|---|
| de Milander et al. [292] | 468 IM M, 200 M controls | Genotype comparison of fastest, middle and slowest IM finishers, and controls | IL-6 −174 G/C, 5-HTT 40 base-pair insertion–deletion, 30 base-pair variable number of tandem repeat MAO-A gene polymorphisms | No direct associations between IL-6 −174 G/C, 5-HTT 44 base-pair insertion–deletion, and MAO-A 30 base-pair variable number of tandem repeat gene polymorphisms and endurance perf, although central governor theory implies IL and serotonin levels play a role in endurance capacity |
| Van Schuylenbergh et al. [293] | 10 | Cycle- and run-graded maximal exercise test, two to three 30-min constant-load tests in swimming, cycling and running to establish their maximal lactate steady state. Sprint race 2-weeks post | HR, power output or running/swimming speed and [BLA] at regular intervals. Oxygen uptake | Stepwise multiple regression analysis run speed and swim speed at maximal lactate steady state, and [BLA] at run maximal lactate steady state, best prediction of perf |
| Hue [294] | 8 elite M | Stepwise multiple regression of links between OD draft legal time and variables within a laboratory 30-min cycle, 20-min run trial | [BLA] | Predicted triathlon time (s) = 1.128 (distance covered during run of cycle-run time-trial [m]) + 38.8 ([BLA] at end of cycle in cycle run time-trial) + 13,338 |
| Laursen et al. [295] | 21 | Correlation between IM perf, HR and HR at various laboratory-based cycle or run thresholds | $VO_2$peak, $VT_1$ HR, $VT_2$ HR, HR deflection point | Mean HR during cycle and run of IM related to ($r = 0.76$** and 0.66***), and not different from, $VT_1$. Difference between race cycle HR; and HR at $VT_1$ related to run time ($r = 0.61$***) and overall race time ($r = 0.45$*) |
| Schabort et al. [296] | 5M, 5F elite | Correlation of laboratory test variables 4 days post OD race with maximal swimming test results over 25 and 400 m, bike peak power output, bike $VO_2$peak, run $V$max, run $VO_2$peak | Cycle PPO, cycle $VO_2$peak, run Vmax, run $VO_2$peak, 25- and 400-m swim time. Steady state $VO_2$, HR and [BLA] during cycle and run laboratory tests | Five most significant predictors of triathlon perf were [BLA] at 4 W $kg^{-1}$, run [BLA] at 15 kph, run Vmax, and cycle $VO_2$peak. Stepwise multiple regression analysis: race time (s) = −129 (peak treadmill velocity [kph]) + 122 ([BLA] at 4 W $kg^{-1}$) + 9,456 |
| Millet and Bentley [297] | 7 M juniors, 6 F juniors, 9 senior M, 9 senior F | Correlation between laboratory (submaximal treadmill run 1, maximal then submaximal cycle, submaximal treadmill run 2) variables and OD perf | Run 1 EC, cycle PPO, cycle $VO_2$max, cycle VT, cycle EC, run 2 EC | Overall triathlon time (min) correlated with cycle $V0_2$max ($r = -0.80$***) and cycle PPO in watts ($r = -0.85$***) |
| Millet et al. [298] | 15 elite M | As above | – | Swimming time correlated with $W$(peak) ($r = -0.76$*) and economy ($r = -0.89$***) in the short-distance athletes. Cycle time in triathlon correlated with $W$(peak) ($r = -0.83$*) in long-distance athletes |

**Table 5** continued

| Study | Group | Design | Markers | Result |
|---|---|---|---|---|
| Miura et al. [299] | 17M | Correlation between OD perf and simulated laboratory triathlon (30-min swim, 75-min cycle, 45-min run, all at 60 % $VO_2$max) | $VO_2$peak and EC in each discipline | OD triathlon (total time) correlated with swim $VO_2$max ($r = -0.621$***), cycle $VO_2$max ($r = -0.873$***), run $VO_2$max ($r = -0.891$***), swim EC ($r = 0.208$, not significant), cycle EC ($r = 0.601$***) and run EC ($r = 0.769$***). Correlation between swim time and swim $VO_2$max ($r = -0.648$***), cycle time and cycle $VO_2$max ($r = -0.819$***), between run time and run $VO_2$max ($r = -0.726$***), between swim time and swim EC ($r = 0.550$*), between cycle time and cycle EC ($r = 0.613$***), and between run time and run EC ($r = 0.548$*) |
| Rietjens et al. [300] | 7 M | Correlation tested pre and post 2-week period of training load (i.e. 200 % prior volume and 115 % prior intensity) | Maximal incremental cycle ergometer test with continuous ventilatory measurements and [BLA] values, time trial, basal blood parameter tests (red and white blood cell profile), growth hormone, insulin-like growth factor 1, adrenocorticotropic hormone, [cortisol], neuroendocrine stress test [short insulin tolerance test, combined anterior pituitary test and exercise], a shortened POMS, RPE and cognitive reaction time test | ↑Training period resulted in ↑ training load, training monotony and training strain. RPE during training ↑, total mood score ↑. Reaction times ↓. No changes in exercise-induced plasma hormone values, nor short insulin tolerance test values. During the combined anterior pituitary test only cortisol ↓ after intensified training. Hb ↓, Hct, red blood cell count and mean corpuscular volume tended to ↓. No effect on physical performance (incremental test or time trial), maximal blood lactate, maximal heart rate and white blood cell profile. The most sensitive parameters for detecting overreaching are reaction time performance, RPE and the shortened POMS |
| Robson-Ansley et al. [90] | 8 M | 4 weeks training, including 3 successive days of intensified run interval training in weeks 2 and 3. Saliva and blood sampling 1 × week$^{-1}$ | Leukocyte counts; neutrophil function; plasma IL-6; CK activity; and cortisol. Signs and symptoms of stress | Plasma IL-6 and CK activity ↑ after intense training. Neutrophil function ↓ but total leukocyte and neutrophil counts, plasma cortisol and salivary immunoglobulin A unchanged. ↑ Symptoms of stress despite no change in sources of stress during training |

**Table 5** continued

| Study | Group | Design | Markers | Result |
|---|---|---|---|---|
| Seedhouse et al. [301] | 8 | Day 1: 10-km swim, 165-km cycle; day 2: 261-km cycle; day 3: 85-km run. Baseline HR, MAP and pulmonary function 2 days pre-race. HR and MAP <30 min prior to race start and 10 min post. Pulmonary function immediately post-race | HR, MAP and pulmonary function | Lower baseline resting HR correlated with faster race times. ↓ $FEV_1$ and peak expiratory flow over race correlated with perf. HR and MAP had strongest association with total race time prediction (54 and 19 % of total). When ↓ in pulmonary function included, peak expiratory flow associated with 87 % of total race time prediction |
| Gratze et al. [145] | 27 M | Multivariate regression analysis of beat-to-beat hemodynamic and autonomic parameters for supine rest and active standing pre, 1 h post and up to 1 week post IM | HR, SBP, DBP, TPRI | 0.05–0.17 Hz band of diastolic blood pressure variability before competition and weekly net exercise training, but not the other hemodynamic and autonomic parameters, related to perf time |
| Balthazar et al. [302] | 8 M professional | Correlation between salivary data on competition day, 7 days post, and short tri perf | Cortisol, testosterone | Early morning cortisol, not testosterone/cortisol ratio, correlated with perf |
| Del Coso et al. [29] | 25 well-trained M | Correlation between jump height and leg muscle power for countermovement jump pre and post ½IM with muscle damage | CK, myoglobin | Leg-muscle fatigue correlated with blood markers of muscle damage |
| Margaritis et al. [30] | 12 racing, 5 not | Correlation between serum enzyme activity and markers of muscle damage, from 2 days prior to 4 days post LD competition | Maximum voluntary contraction, DOMS, and total serum CK, CK myoglobin isoenzyme, LDH, aspartate aminotransferase and alanine aminotransferase activities | Extent of and recovery from muscle damage cannot be evaluated by magnitude of changes in serum enzyme activities. Muscle enzyme release cannot be used to predict magnitude of muscle function impairment caused by muscle damage |
| Medina et al. [68] | 10 M, 5 F | Pattern of iso-prostanes and prostaglandin metabolites in urine after triathlon training | – | Variation in 6-keto PGF(1 alpha) after exercise is linked to their precursor prostaglandin: a useful marker of vasodilation and inhibition of platelet aggregation |
| Sharwood et al. [223] | 258 IM | Establish relationships between body weight changes and serum sodium concentration during and after IM, and post-race fluid status, rectal temperature, including the incidence of hyponatremia. Weighed at registration, immediately pre-race, immediately post-race, and 12 h later. Blood samples at registration and immediately post-race. Rectal temperatures measured post-race. BP and [serum sodium] at registration and immediately post-race. Rectal temperatures and medical exam post-race | – | Percentage change in body weight linearly related to post-race serum sodium concentrations but unrelated to post-race rectal temperature or running perf in the marathon. No evidence that more severe levels of weight loss or dehydration related to either body temperatures or ↓ perf. Large changes in body weight not associated with prevalence of medical complications or rectal temperatures but associated with serum sodium concentrations |

**Table 5** continued

| Study | Group | Design | Markers | Result |
|---|---|---|---|---|
| Millet et al. [304] | 4 elite | Effects of training load (calculated from exercise HR) on anxiety and perceived fatigue, over 40 weeks | Anxiety and perceived fatigue self-reported 2 × week$^{-1}$ | Relationship ($r = 0.32$***) between training loads and anxiety identified using a two-component model: a first, negative (i.e. anxiety decreased), short-term (tau (1) = 23 days) function, and a second, positive, long-term (tau (2) = 59 days) function. Relationship between training loads and perceived fatigue ($r = 0.30$***), with one negative function (tau (1) = 4 days) |
| Barnett et al. [303] | 1 elite F | Retrospective examination, via dynamic linear models and mediating variable analysis, of case study data of association of training load with SE | SE (perf.) via RESTQ-Sport (2 × week$^{-1}$ for 137 days); fatigue/'lack of energy', 'being in shape' psychosocial states | Concurrent and lagged training loads positively associated with perf-related SE |
| Main et al. [11] | 20 M, 10 F well-trained | Linear mixed modelling of 45 weeks of training and SAS data | Training factors, SAS | SAS associated with ↑ in training factors*. Greatest impact on SAS by psychological stressors***. Common overtraining symptoms affected by ↑ training and psychological stressors*. Mood disturbance not affected by training factors* but by ↑ in psychological stressors***. Each of the three athlete burnout subscales affected by psychological*** stressors and training factors* |
| Main and Landers [10] | 1 M | Visual inspection of retrospective case study of weekly ABQ and MTDS, obtained for 45 weeks from season start, in conjunction with semi-structured interviews and consultation with sports doctor | ABQ factor 1 (reduced sense of accomplishment), ABQ factor 2 (sport devaluation), ABQ factor 3 (emotional and physical exhaustion); MTDS factors 1–6 (depression, vigour, physical signs and symptoms, sleep disturbances, perceived stress, and fatigue, respectively) | Athlete burnout and overtraining syndrome may develop simultaneously and be confused with each other |
| Plews et al. [305] | 1 M, 1 F elite | Linear regression of daily HRV data obtained over 77 days for one athlete who became NFOR and one who did not | 7-day rolling average of the log-transformed square root of the mean sum of the squared differences between R-R intervals, coefficient of variation of HRV (CV of the aforementioned variable) | 7-day rolling average of the log-transformed square root of the mean sum of the squared differences between R-R intervals ↓ towards race day in NFOR athlete, remaining stable in control. In the NFOR athlete, coefficient of variation of HRV revealed large linear ↓ towards NFOR (i.e. linear regression of HRV variables vs. day number towards NFOR, while these variables remained stable for the control) |

**Table 5** continued

| Study | Group | Design | Markers | Result |
|---|---|---|---|---|
| Vleck [12] | 8 | Prospective longitudinal training diary-based study over 26 weeks | POMS-C vigour, confusion, depression, tension and anger factor scores; difference between individual normative peak performance values and composite weekly values for resting morning HR, gastric disturbance, DOMS and heavy legs; performance decrement and injury | Probability of self-assessed performance decrement = 1/[1 − (exp (−2.68)*exp (0.75 heavy legs-DOMs score)*exp (0.298 number of standard deviations outside the peak performance composite appetite score that the score in other weeks of the analysis accounted for)* exp (0.595 POMS-C confusion score 2 weeks prior)*exp (0.383 POMS-C confusion score 2/3 weeks prior)* exp (0.528 POMS-C anger score 1-week prior)] |
| Rietjens et al. [65] | 7 M, 4 F elite | 102 blood samples over 3 years | Hb, Hct, RBC, mean corpuscular Hb, mean corpuscular volume, mean corpuscular Hb content and plasma ferritin. The data were pooled and divided into three periods; off-season, training season and race season | Only RBC ↓ during race season compared with training season. Hematological values below lower limit of normal range in 46, 55, and 72 % of athletes during the off-, training- and race seasons, respectively. Hb and ferritin values most frequently < normal range. Training at 2,600 m for 3 weeks showed Hb, Hct and mean corpuscular volume |

– indicates no information, ↑ indicates increased, ↓ indicates decreased, *ABQ* Athlete Burnout Questionnaire, *[BLA]* blood lactate concentration, *BP* blood pressure, *CK* creatine kinase, *CV* coefficient of variation, *DOMS* delayed-onset muscle soreness, *DBP* diastolic blood pressure, *EC* economy, *F* female, $FEV_1$ forced expiratory volume in 1 s, *½IM* half-Ironman (i.e. 1.9-km swim, 90-km cycle, 21-km run), *Hb* haemoglobin, *Hct* haematocrit, *HTT* hydroxytryptamine transporter, *HR* heart rate, *HRV* heart rate variability, *IL* interleukin, *IM* Ironman, *LD* long distance, *LDH* lactate dehydrogenase, *M* male, *MAO-A* monoamine oxidase A, *MAP* maximal aerobic power, *MTDS* Multi-component Training Distress Scale, *NFOR* non-functionally over-reached, *OD* Olympic distance (i.e. 1.5-km swim, 40-km cycle, 10-km run), *perf* performance, *PGF* prostaglandin, *POMS* Profile of Mood States, *POMS-C* Profile of Mood States-C, *PPO* peak power output, *RBC* red blood cell count, *RESTQ–Sport* Recovery Stress Questionnaire for Athletes, *RPE* rating of perceived exertion, *R-R interval* the interval between the peak of one QRS complex to another on an electrocardiogram, *SAS* signs and symptoms of injury, *SBP* systolic blood pressure, *SE* self-efficacy, *TPRI* total peripheral resistance index, *Vmax* peak speed, $VO_2peak$ peak oxygen uptake, $VO_2$ oxygen uptake, $VO_2max$ maximal oxygen uptake, *VT* ventilatory threshold, $VT_1$ first ventilatory threshold, $VT_2$ second ventilatory threshold, *W(peak)* peak power output

* $p < 0.05$, ** $p < 0.02$, *** $p <$ at least 0.01

Despite its problems (which may have been partially due to improper parameters being used to indicate training strain and performance), the Banister 'fitness-fatigue' model has also been used [304], in this case to examine the effect of training on self-perceived fatigue and anxiety. The study is noteworthy because self-report measures arguably exhibit reliable dose-response relationships with training load. Self-assessment of fatigue also circumvents the problem of obtaining sufficient race or time-trial performance data for the modelling process. Both measures were found to hold potential for the early detection of training-related problems.

Feelings of fatigue, in addition to loss of performance, can have a major impact on the self-efficacy of the athlete. Dynamic systems modelling of performance-related self-efficacy, in conjunction with mediation analyses of 'being in shape' and 'fatigue/lack of energy', has also therefore been used to track longitudinal training adaptation in two separate elite females [303, 312]. In this case, the 'norm' on each of the scales of the RESTQ-Sport values that was required for the model was obtained from questioning the athlete rather than from long-term data collection. Positive effects of training on self-efficacy, which were partly explained by feelings of 'being in shape' and suppressed by feelings of 'fatigue/lack of energy', were observed. Promisingly, modification of the relationship between lagged training load and 'fatigue/lack of energy' was seen and was particularly pronounced in the temporal proximity of an injury. Although no attempt to actually predict injury or illness was made, the dynamic systems approach may hold especial promise as a potential method of modelling the relationships between training and illness in triathletes

**Table 6** Selected limitations of the health-related triathlon literature and recommendations as to how they might be addressed

| Issue | Consensus to develop and implement | Key studies to undertake |
|---|---|---|
| Quantification of the levels of risk/training stress to which triathletes expose themselves[a,b] | Universal systems of categorising subjects' level of athletic ability and event distance specialization, for the purpose of research | How training in each of the individual triathlon disciplines, and weight training, should be weighted for the purpose of calculating total summed training load |
| Effect of training on injury, immune, oxidative and cardiovascular status | Agreement on the key issues and markers to monitor on a longitudinal prospective basis | Comparison against age-matched healthy controls |
| Investigation of possible links between oxidative and/or immunological status and illness incidence | Definition of illness | The extent to which this is influenced by transference between disciplines |
| Determination of the risks of competition[a,b,c] | Universal reporting methods for race injuries and illnesses [319, 320] (including logs for their associated medical care requirements such as staff specialisation and treatment duration), to be implemented across national and international governing bodies[d,e,f] | Follow-up of sudden death incidents by retrospective questioning of next-of-kin for autopsy reports/pre-existing medical conditions of the athletes in question[f,g] |
| | | Extent to which risk of heat illness is influenced by competition length, equipment restrictions, and/or environmental conditions (such as water temperature) |
| | | The extent to which injury risk and treatment duration changes with competition length and environmental conditions |
| Comparison of the outcomes of triathlon training and competition with those of untrained healthy controls[a,c] | – | Incidence and short-term outcome of illness in triathletes |
| | | Extent to which injury recurs |
| | | Long-term sequelae of the structural and other changes to the heart that occur with triathlon training and competition |
| | | Extent of sudden cardiac death in training as well as in competition |

– indicates no information

[a] As modified by age, ability level and/or event-distance specialization

[b] As modified by competition duration, course topography, equipment restrictions and/or environmental conditions (such as water temperature)

[c] Including for how long any such effect lasts

[d] Must include a definition of recurring injury, to be used in prospective studies

[e] Must include details of the conditions under which (and, as far as possible, how) the injury occurred. This is particularly important for research into the possible aetiology of swim-related deaths

[f] Perhaps incorporating a health and performance risk grading system similar to that of Dijkstra et al. [313]

[g] As per Kim et al. [243]

because it can avoid the possible problems with athletes' or coaches' (as opposed to researchers') reliance on mainly visual analyses [10] of graphical profiles. Visual analysis is easily done and facilitates athlete–coach discussions. It is therefore 'friendly'. However, visual analysis may not account for the effects of factors that mask the true relationship between explanatory and outcome variables, or for auto-correlation between successive observations. It can neither quantify dose-response relationships between training/racing and signs and symptoms of illness/injury, nor their temporal variation. This complicates the design of an appropriate programme of intervention. It also means that much work still remains to be done in this field before clear guidelines as to what the athlete should do and what he/she should monitor, if health and performance are to be maximised, can be arrived at.

## 8 Conclusion

Neither the stress to which triathletes subject themselves nor what this means for their wellbeing has been comprehensively evaluated. Little scientific data are available to aid triathletes, most of whom are older age-groupers, balance the multi-discipline training that is required in their sport. Any negative effects of racing on immunological, oxidative, cardiovascular and humoral parameters appear, for the majority of athletes, to be transient and non-severe.

For most athletes, injury and illness incurred whilst training also appears to be of minor or moderate severity. However, injury recurrence rates have not been investigated and the long-term effects on health of triathlon training and racing are relatively unknown. For both to be fully elucidated, issues such as the development of a consensus statement on the definition and reporting of both (first time and recurring) injury and illness, and the development of an international registry for sudden death incidents, need to be addressed (Table 6) [243, 313].

Some clues exist as to whether the degree of influence of specific risk factors for maladaptation may differ with different athlete attributes such as sex, age group and event-distance specialization. Both injury and infection risk may be greater within periods of higher intensity work. They may also be greater at specific points within competition (e.g. when fatigue is setting in). These clues should be followed up by (possibly training diary-based) longitudinal prospective studies. Such studies would allow more comprehensive evaluation of the risk factors for, and warning signs of, any negative outcomes of training and racing stress. Better management strategies may then be developed for any negative health issues that may arise as a result of triathlon training and racing.

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