tigated individual mediators or effectors, which limits the interpretation of effector function in the tissues. Furthermore, cytokines often have crosstalk or cumulative effect and insight in the group effect of cytokines and chemokines would provide more accurate information about the net effect.

The scoring systems ought to be used to define the appropriate therapy. Damage control surgery and damage control orthopedics are currently used strategies to limit the incidence of organ failure after trauma [76,77]. Timing of surgery is essential in this damage control approach and recent literature provides a timeframe for planning interventions [78,79]. This timeframe, which is based on database analysis, is not fully complementary with the activation status of the innate immune system. According to the measurements of neutrophils (oxidative burst and L-selectin) hyper-inflammation is at its maximum 6 hours after trauma, whereas according to the damage control timeframe hyper-inflammation is present between day 2–4 [20,26]. Despite this problem in defining the timeframe, solutions are sought to prevent the excessive inflammation. A recent therapy that became available, hemoglobin based oxygen carriers as alternative for packed red blood cells, show promising results in limiting the inflammatory response [28]. The start of hypo-inflammation is less well defined and more individual determined, which makes therapy more difficult.

Conclusion

Several studies have shown a relationship between the severity of trauma and the resulting immune response [75]. The injury to the host can be expressed in scoring systems and these have become important prognostic tools to calculate the risk based on clinical signs and symptoms in combination with inflammatory parameters [68]. It is likely that a threshold needs to be reached before clinical symptoms become evident. The loss of barrier integrity of different organs seems to play a major role in the development of complications in both the pro-inflammatory period and the anti-inflammatory period. Studies which focus on the interaction between host and innate immunity are to be performed to resolve the post-traumatic complications resulting in organ failure. Immunomonitoring with interpretation of group effects of cytokines or analysis of effector cells in interaction with tissue may lead to more intensive immunomonitoring and the adjustment of therapeutic and supportive strategies for the optimalization of care for trauma-patients.

Abbreviations

ARDS: Acute respiratory distress syndrome

CARS: Compensatory anti-inflammatory response syndrome

CRP: C-Reactive protein

GM-CSF: Granulocyte macrophage colony stimulating factor

HMGB-1: High mobility group box 1

ICAM-1: Intercellular adhesion molecule 1

IL-n: Interleukin-n

ISS: Injury Severity Score

MAC-1: Macrophage 1

MARS: Mixed antagonist response syndrome

MHC-II: Major histocompatibility complex II

MIF: Macrophage migration inhibitory factor

MODS: Multiple organ dysfunction syndrome

MOF: Multiple organ failure

ROS: Radical oxygen species

sICAM: Soluble ICAM

SIRS: Systemic inflammatory response syndrome

TGF: Tumor growth factor

TNF-α: Tumor necrosis factor α

Competing interests

The author(s) declare that they have no competing interests.

Authors' contributions

FH participated in the design of the review and drafted the manuscript.

LK revised the manuscript critically on the content of effector processes till the final version was reached.

GR revised the manuscript critically on the mediator processes till the final version was reached.

LL participated in the design of the review and revised the manuscript till the final version was reached.

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