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Letter to the Editor

Re: Comment on "Incidence of risk factors for bloodstream infections in patients with major burns receiving intensive care: A retrospective single-center cohort study"

Dear Editor,

We completely agree that the overuse of broad-spectrum antibiotics can cause selection of resistant bacterial populations and is not recommended [1,2].

In the retrospective study by Fochtmann-Frana et al. 27% of all patients did not receive any antibiotic treatment [3]. In the recent past and the present in our university institution we do not routinely use broad-spectrum antibiotics. The initiation of broad-spectrum antibiotic was always an individual decision. An empiric broad-spectrum antibiotic treatment was only initiated upon clinical signs and symptoms of serious infections such as sepsis, pneumonia, upper or complicated urinary tract, intra-abdominal infection, or deep wound infection in severely burned patients requiring intensive care. It is and was a standard that before starting antibiotic therapy at least 2 blood cultures and any other appropriate samples were taken, and if a causative organism was identified the antibiotic therapy was de-escalated.

It is evident that an increase of blood stream infections caused by multidrug resistant (MDR) bacteria from none in 2003 to up to 8% of the cases in 2014 was observed [3]. However, we do not agree that this was only caused 'by the liberal prescription of piperacillin/tazobactam since 2007' in our institution. In the present study, 4 out of 10 (40%) patients suffering from BSIs caused by MDR bacteria were transferred from countries with a higher prevalence of MDR [3,4]. These patients were often pre-treated with broad spectrum antibiotics, had often spent days in the other facility/hospital without surgical treatment and were already colonized with MDR when admitted to our institution [3,5]. It was previously suggested that due to late wound closure and increased usage of broad-spectrum antibiotics, further replacement by MDR bacteria can occur [6].

Nevertheless, we completely agree that careful consideration should always be given as to whether the antibiotic therapy is necessary or not. To treat or not to treat should always be an individual decision. In our institution we reevaluate daily the necessity of the antibiotic therapy in addition to a regular consultation of infectious disease specialists to discuss the current antimicrobial therapies of complicated cases.

Conflict of interest statement

There are no financial or other relationships, which may lead to a conflict of interest.

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Letter to the Editor

Frailty and more: Age-related outcome factors in burns

Dear Editor,

Age is one of the most important outcome predictors in burns [1]. It is commonly accepted that chronological age does not necessarily reflect the biological age of patients. Accordingly, Ward et al. could show in their recently published paper 'Frailty: an independent predictor of burns mortality following in-patient admission' that implementation of a frailty index independently predicts in-hospital and one-year mortality for burns of any size [2]. Moreover, combining the Modified Baux Score and the Frailty Score could even further improve mortality prediction. We completely agree with the authors' conclusion that routine integration of a frailty index on cases involving elderly patients, would definitely improve clinical decision-making in stratifying patients for optimal burn care.

However, we believe that evaluating frailty is just one approach to incorporate biological age in the assessment for patient stratification. Elderly burn patients may present without frailty; however, their skin may display advanced age-related alterations, which can also influence patient performance. Intrinsic ageing produces thinner, atrophic skin [3] impacting burn depth and burn wound progression. Moreover, numerous extrinsic variables, most importantly UV radiation, smoking and pollution, play a role in accelerating skin ageing [4]. In contrast to intrinsically aged skin, skin subjected to extrinsic ageing (mainly UV exposure) presents as hypertrophic, dull, rough and with deep wrinkles [3]. In addition to these visible changes, age-induced alterations (summarised in Table 1) in cellular compartments, extracellular matrix components, the immune system as well as the inflammatory state have been described [5,6].

Although wound healing is not intrinsically impaired in the aged population, alterations have been observed at basically any stage of this process (reviewed in Ref. [5,6]). Considering intrinsic and extrinsic factors for ageing, we assume that biological skin age is highly variable within a population of approximately the same chronological age and may vary even within a single individual.

In order to reliably link biological skin age to patient outcome after burn trauma, easily quantifiable biomarkers would be highly desirable. So far a number of different biomarkers have been suggested to assess biological (skin) age including assessment of shortening of the telomeres, the hormonal status, general inflammatory markers or apoptosis markers [3]. So far, very few have been investigated that directly link biological age to patient response to burn injury. Driven by their observation that elderly patients are more likely to develop full-thickness injuries from partial-thickness thermal burns Farinas et al. set out earlier this year to determine local inflammatory responses in young and aged patients [7]. Apart from changes in macrophage numbers and activation state, they could also identify two signalling molecules (CCL5/RANTES, and EGF) that significantly differed between the two groups analysed. These results suggest it would be worthwhile to further investigate the local wound (immune) environment in order to improve and personalise care for burn patients [7].

As Ward et al. [2] concluded in their recent paper, it would be certainly interesting to expand frailty assessment to patients less than 65 years of age, *i.e.* the young elderly (60–65), and to patients at risk of early senescence. Furthermore, we propose a more elaborated stratification system by including not only the frailty aspect, but also biological skin age. We are aware that further research is much-needed to

Table 1 – Summary of alterations in aged skin (modified from Ref. [5,6]).

	Alteration upon age
Keratinocyte proliferation	↓
Keratinocyte migration time	↑
Fibroblast proliferation	↓
Melanocyte count	↓
Collagen	↓
Macrophage count	↓
Langerhans-cell count	↓
Inflammation	↑
T-cell infiltration	↓
Microvascularisation	↓