

## Professor Stefan D. Anker, MD, PhD, FESC

### BIOGRAPHY



Professor of (Tissue) Homeostasis in Cardiology & Metabolism, Charité Berlin, Germany

**Stefan D. Anker** is Professor of (Tissue) Homeostasis in Cardiology & Metabolism (W3) at Charité Berlin (from June 2017). Dr. Anker studied medicine at Charité Berlin and completed his clinical training in Germany and the UK. He obtained his M.D. from Charité Medical School, Berlin, Germany (1993), and his Ph.D. (1998) at National Heart & Lung Institute of Imperial College London. He was Professor of Cardiology & Cachexia Research (W2) at Charité (2002-14), and Professor of Innovative Clinical Trials (W3) in Göttingen (2014-17).

Dr. Anker has authored more than 750 original papers, reviews, and editorials that are well cited (total citations: >49,000, h-index: 107, papers with  $\geq 200$  citations: 59; source: Scopus, 4th July, 2017). For his work Dr. Anker has won several prizes, and obtained a number of fellowships and grants, including 2 from NIH (WARCEF trial), 2 EU-FP7 & 2 IMI/Horizon2020 grants. He was co-ordinator for "SICA-HF" study (EU-FP7).

Dr. Anker is Vice President of the European Society of Cardiology (ESC, 2016-18), serving on the ESC board since 2012. Dr. Anker serves in the board of the Heart Failure Association (HFA) of the ESC since 2006; he was HFA President (2012-14), and currently chairs the HFA committee on regulatory affairs. He is founding Editor-in-Chief of the first open access heart failure journal ESC Heart Failure. Dr. Anker serves on the editorial boards of 4 scientific journals (including *European Heart Journal* and *European Journal of Heart Failure*), and he worked in several ESC Guideline task forces.

Dr. Anker is the founding president of the International Society on Sarcopenia, Cachexia and Wasting Disorders (SCWD – see [www.cachexia.org](http://www.cachexia.org)). Dr. Anker is founding Editor-in-Chief of the *Journal of Cachexia, Sarcopenia and Muscle* (JCSM, 2016-IF: 9.70, see [www.jcsm.info](http://www.jcsm.info)).

Dr. Anker was and is member of >30 international clinical trial steering committees, chairing or co-chairing several currently (FAIR-HF2, RESHAPE-HF2, EMPEROR-HFpEF, Fair-HFpEF) as well as in the past (incl. FAIR-HF, TIM-HF, AUGMENT-HF, IMPULSE-HF, and BACH). He served in a number of DMC's (chairing 4) and end-point committees (chairing 4).

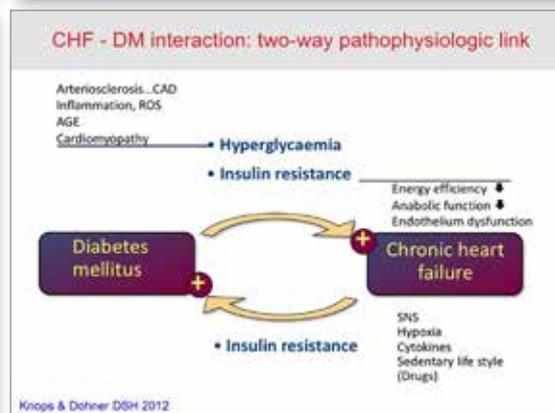
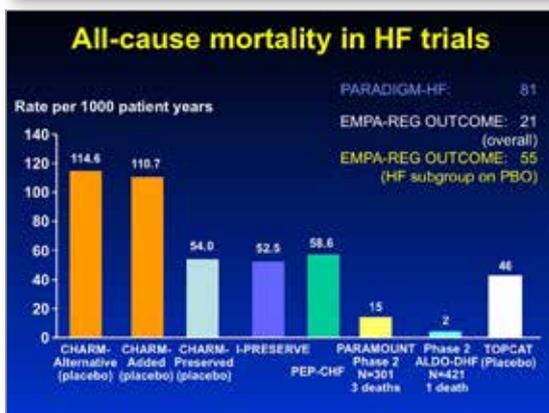
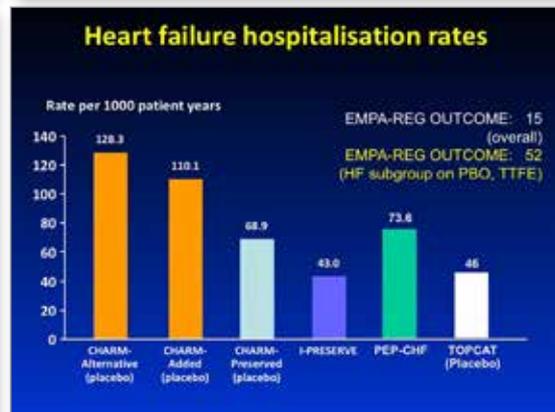
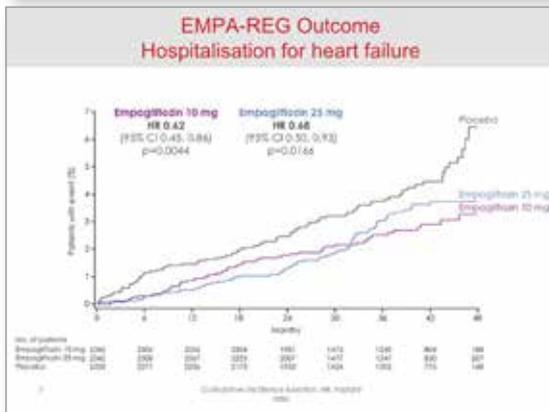
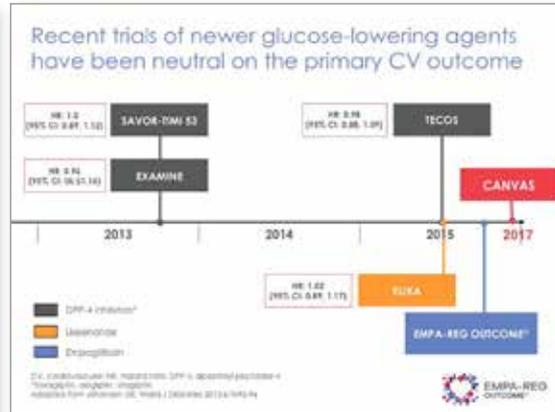
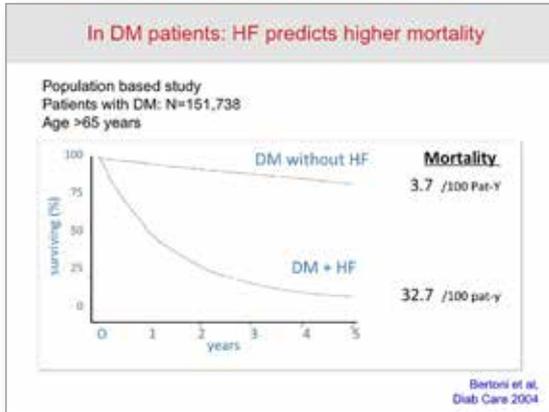
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## ABSTRACT

### **Heart failure & Diabetes: Time for a more unified approach**

Diabetes and HF frequently coexist in the same patient, while diabetes per se is a relevant risk factor for the development of HF over time. In registries of HF, the prevalence of diabetes ranges from 13% to 47%, with the actual number probably being right in the middle, (i.e., ~30%). Because the prevalence values of both HF and diabetes are steadily increasing in Western countries, an exponential growth in the number of diabetic HF patients is expected in the years to come. Diabetic HF patients and nondiabetic ones are clinically different and hence require different therapeutic approaches.

Glycemic exposure is not the only important factor in the pathophysiological interaction between HF and diabetes. Treatment also plays a major role in this regard. The list of cardiovascular considerations in the treatment of diabetes is long, but the recent addition of empagliflozin could be an interesting exception. The EMPA-REG OUTCOME trial showed a surprisingly high relative risk reduction of 35% in the rate of hospitalization for HF (as well as of all-cause and cardiovascular mortality), which likely also means that fewer cases of new-onset HF developed over time. Importantly, the general results of the EMPA-REG OUTCOME trial also held true in the subgroup of 706 patients who had HF at baseline. The positive effects of empagliflozin on these events might have been due to a combination of effects, including an antidiabetic and insulin-sensitizing impact, as well as some diuretic and neuroendocrine inhibitory effects and possibly also vasodilation. If the results can be confirmed, then we may have to rethink our therapeutic approaches to diabetes in patients with HF.



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