**Response to Reviewers: Modelling the effects of antibiotic usage in livestock on human salmonellosis.**

**Reviewer Point 1.1:**The paper relies heavily on the model, but I found that the study did not align the work well with One Health or human health. Some additional data on the actual risks of salmonellosis from eating products from these animals is not explained and some additional context is needed.

We thank the reviewer for their comment. We have sought to clarify the wording throughout the manuscript, that antibiotic curtailment interventions in livestock have an impact on *human* salmonellosis, and therefore has a clear one health link. Examples can be found in the *Results* and the *Discussion* sections (lines 240-241 and 348-349).

“*Curtailment of antibiotic usage (τ → 0 g/PCU) in the fattening pigs case studies resulted in the largest increase in the daily incidence of human salmonellosis with a 1.11-fold…*”

“*…were found to effectively mitigate increases in the daily incidence of human salmonellosis following curtailment of antibiotic usage in livestock.*”

We also agree with the reviewer regarding the “actual risks of salmonellosis from eating products” not being discussed. We now also mention that this was a limitation to the study, and that future studies aiming to more accurately describe the farm-to-fork pathway could utilise dose-response modelling to simulate infection probability upon exposure, as seen in risk assessment literature (lines 416-420).

“*For example, transmission of Salmonella spp. from animals-to-humans was simplified using a single parameter in this study. Future models could utilise non-linear microbial load dose-response models to more accurately quantify infection risk upon human exposure to Salmonella spp. on food products [1]*”

**Reviewer Point 1.2:** The authors state that Listeria and VTEC would have different dynamics (L393). I did not think this section was explained well.

We thank the reviewer for their comment. We have now rewritten this paragraph to make it clear that *Listeria* spp. and VTEC are commonly found in the human intestinal flora as commensal organisms. This is in comparison to the transient infection in humans and clear food animal origin of *Salmonella* spp. Therefore, the impact of foodborne transmission and subsequent interventions to target this pathway on the infection probability of *Listeria* spp. and VTEC will likely be different than for *Salmonella* spp. (lines 392-399).

“*It is also likely that there will be a less clear link between improvements in farm-to-fork hygiene and the incidence of opportunistic infections of commensal pathogens, such as Listeria spp. and E.coli (i.e. VTEC). This contrasts with Salmonella spp. modelled in this study, which has clear food animal origins and predominantly results in self-limited colonisation and infection in humans [2]. Factors such as the extent of host-immunosuppression, microbial community interactions and nosocomial transmission may play a larger role than the animal-to-human transmission pathway in determining the extent of Listeria spp. and E.coli infection in humans [3, 4].*”

**Reviewer Point 1.3:** Could the authors add a paragraph listing limitations of the study?

We thank the reviewer for their comment, and we have now added an additional paragraph detailing further limitations of the mathematical model (lines 415-428). We also note that previous paragraphs in the *Discussion* highlight several limitations (data for model fitting, applicability to other pathogens, etc.). However, in these paragraphs we also detail how our results are robust to these limitations.

“*The compartmental model structure chosen in this study was simplified for model tractability and certain phenomena were implicitly assumed or modelled. For example, transmission of Salmonella spp. from animals-to-humans was simplified using a single parameter in this study. Future models could use non-linear microbial load dose-response models to more accurately quantify infection risk upon human exposure to Salmonella spp. on food products [1]. Future models could also explicitly model mechanisms driving strain coexistence, such as within-host competition, with this mechanism known to impact AMR dynamics following the implementation of interventions [5]. The impact of country level adherence to antibiotic curtailment interventions on human and livestock AMR could also be of interest, to explore the impact of population structure on intervention efficacy. Finally, the relationship between livestock antibiotic usage and resistance was assumed to be linear in this study, with this also being assumed in related literature [5, 6]. Exploring the functional form of this relationship may also provide useful insight into the range of potential scenarios following curtailment interventions at the one health interface.*”

**References**

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2. Centers for Disease Control and Prevention. *Salmonella in the Caribbean - 2013: Infection with Salmonella*. Atlanta: Centers for Disease Control and Prevention. (2014). Available from: <https://www.cdc.gov/training/SIC_CaseStudy/Infection_Salmonella_ptversion.pdf>.

3. L Poirel, Madec J-Y, Lupo A, Schink A-K, Kieffer N, Nordmann P, et al. Antimicrobial resistance in Escherichia coli. *Microbiology Spectrum*. (2018). 6(4):6.4. 14.

4. S Becattini, Littmann ER, Carter RA, Kim SG, Morjaria SM, Ling L, et al. Commensal microbes provide first line defense against Listeria monocytogenes infection. *Journal of Experimental Medicine*. (2017). 214(7):1973-89.

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6. S Rahman, Hollis A. The effect of antibiotic usage on resistance in humans and food-producing animals: a longitudinal, One Health analysis using European data. *Frontiers in Public Health*. (2023). 11:1170426.