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Research article

Landscape biodiversity correlates with respiratory health in Australia

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ABSTRACT

Megatrends of urbanisation and reducing contact with natural environments may pose a largely unappreciated risk to human health, particularly in children, through declining normal (healthy) immunomodulatory environmental exposures. On the other hand, building knowledge of connections between environments, biodiversity and human health may offer new integrated ways of addressing global challenges of rising population health costs and declining biodiversity. In this study we are motivated to build insight and provide context and priority for emerging research into potential protective (e.g. immunomodulatory) environmental exposures. We use respiratory health as a test case to explore whether some types and qualities of environment may be more beneficial than others, and how such exposures may compare to known respiratory health influences, via a cross-sectional ecological epidemiology study for the continent of Australia. Using Lasso penalized regression (to interpret key predictors from many candidate variables) and 10-fold cross-validation modelling (to indicate reproducibility and uncertainty), within different socio-geographic settings, our results show surrogate measures of landscape biodiversity correlate with respiratory health, and rank amongst known predictors. A range of possible drivers for this relationship are discussed. Perhaps most novel and interesting of these is the possibility of protective immunomodulatory influence from microbial diversity (suggested by the understudied 'biodiversity hypothesis') and other bioactive agents associated with biodiverse environments. If beneficial influences can be demonstrated from biodiverse environments on immunomodulation and human health, there may be potential to design new cost-effective nature-based health intervention programs to reduce the risk of immune-related disease at a population level. Our approach and findings are also likely to have use in the evaluation of environment and health associations elsewhere.

1. Introduction

There is growing awareness of the numerous mechanisms and co-benefits that link natural and biodiverse environments with human health. It is important to understand such connections given the global challenges of escalating population health costs and declining biodiversity (WHO and SCBD, 2015). As reviewed elsewhere (Craig et al., 2016; Keniger et al., 2013; Myers et al., 2013; Sandifer et al., 2015; WHO and SCBD, 2015), there are broad and interacting mechanisms of human health impact from environmental change, many of which are well-studied. However, some potentially important environmental influences on human health remain understudied due to their multidisciplinary nature. A key example is described by the biodiversity hypothesis (von Hertzen et al., 2011), and related microbial 'old friends' mechanism (Rook, 2013), as recognized by the World Health Organization (WHO and SCBD, 2015) and World Allergy Organization (Haahtela et al., 2013), which highlights that environmental microbiota (or communities of microorganisms from the surrounding environment) overlap and interact with human commensal microbiota, contribute to human microbial diversity and may provide important beneficial immunomodulatory roles. As discussed later, there are potential links between different environments, their

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microbiotas and other possible bioactive agents (e.g. volatile organic compounds, VOCs; air ions), and via direct and aerobiological exposures, possible immunomodulatory effects and human health influences (e.g. see Liddicoat et al. (2016), their Fig. 1).

The possibility of populations receiving some level of inadvertent ambient beneficial or adverse immunomodulatory influence associated with different types and qualities of environment, highlights a potentially important gap in our awareness of possible links between environments and human health. Megatrends of urbanisation and reducing contact with natural environments may pose a largely unappreciated risk to human health through declining normal (healthy) immunomodulatory environmental exposures. Children may be particularly impacted by inadequate exposures during the critical early period of immune system development (Wopereis et al., 2014) where immunoregulatory commensal microbiota can be acquired through random environmental encounters (Artis, 2008). For example, children who grow up in environments with diverse microbial exposures such as traditional farms, are less prone to developing asthma and atopy (allergic sensitization) (Ege et al., 2011; Stein et al., 2016). Beneficial immunomodulatory environmental exposures are also suggested into adulthood (Douwes et al., 2007; Rottem et al., 2015; von Hertzen and Haahtela, 2006). Moreover, a lack of appropriate environmental exposures and deficient immune training and regulation may also impact other areas of immune-related human health including susceptibility to infectious disease (as below).

Respiratory health provides a conspicuous test case to explore environment-human health associations that have plausible microbiota-mediated linkages, because breathing offers a primary mode of exposure for people interacting passively with the environment. Throughout life, millions of litres of air move through the human respiratory tract, which provides one of the first points of contact with environmental contaminants and bioaerosols (airborne microbiota and bioactive agents). Airway epithelial tissues provide a protective arsenal of physical barriers, niche-occupying commensal microbiota, antimicrobial compounds, and receptors ready to orchestrate immune responses (Parker and Prince, 2011; Whitsett and Alenghat, 2014). Environmental microbiota can interact directly with respiratory mucosal immune receptors or via ecological interactions with host commensal microbiota. Similar interactions can also occur in the gut, influencing immune- and health-status, after environmental microbiota deposit in the airways and are transported by cilia to be swallowed (Rook, 2013). Importantly, dysregulation of the airway epithelial innate immune system can be associated with compromised immunity and chronic inflammation (Parker and Prince, 2011). Immune dysfunction may involve adverse feedbacks that reinforce imbalance (or dysbiosis) of host microbiota (Haahtela et al., 2013), which in turn may favour pathogenic microbes and increase susceptibility to infectious disease. As discussed later, there is often not a clear distinction between infectious and non-infectious respiratory disease outcomes, for example, where one type of disease (e.g. cold or influenza) can exacerbate symptoms of another (e.g. asthma). This means there is potential for environments and their microbiota to impact immune status, which in turn may have potential broad underlying (and population-level) influence on multiple infectious and non-infectious respiratory diseases.

If particular macro- and landscape-scale features of the environment (e.g. types of vegetation, soil, land use, and their diversity) can be associated with human health benefits (and ultimately supported by new knowledge of underlying causal mechanisms), it may be possible in the future to design new cost-effective, landscape and urban green space interventions with concurrent benefits for public health and biodiversity conservation. Informing such outcomes would require a large body of multidisciplinary research. The work presented here represents an early step.

Our motivation for this study is to build insight and provide context and priority for further research into these types of potential beneficial environmental exposures, through building on existing, inexpensive data. Given the possible abovementioned links between environments, immune development or dysfunction, and infectious and non-infectious respiratory disease, we examine available aggregated respiratory health outcome data in a cross-sectional ecological epidemiology study spanning the continent of Australia. Our aim is to test whether some types and qualities of environment may be more beneficial than others, and how such exposures may compare to known respiratory health influences. We appreciate that using aggregated health response data represents a limitation in terms of loss of specificity to link environmental influence with any particular disease. On the other hand, this approach may offer greater sensitivity to detect possible broad environmental influence on multiple respiratory disease outcomes.

Due to many unknowns (e.g. possible agents, behaviourally- and temporally-mediated exposures, requisite exposures, immunomodulatory and other possible physiological pathways), the use of environmental proxies is warranted as a pragmatic investigation tool (Liddicoat et al., 2016). Proxies allow us to consider a variety of (including possible beneficial immunomodulatory) environmental influences on human health. We might expect to see correlative signals between health outcomes and environmental exposures that are consistent with sources of microbial diversity, such as biodiverse environments, diversity in land use, and soils high in clay and/or organic matter content (Liddicoat et al., 2016; Rook, 2013; von Hertzen and Haahtela, 2006).

We use a data-intensive approach suited to this emerging area of scientific inquiry where it is important to gain an early understanding of key relationships among many variables, and note that models will improve iteratively over time (Elliott et al., 2016). Our modelling approach reflects the highly faceted nature of environments and is adept at handling large numbers of (included potentially correlated) candidate predictors. To guide future work, we provide clear interpretation and ranking of previously unaccounted environmental influences among important predictors of our respiratory health data.

2. Methods

We use an array of specially-prepared environmental covariates to estimate environmental exposures, each allowing for a potential surrounding zone of influence (section 2.2). To cater for geographically-variable environmental and multimodal social predictors, we stratify our analysis into three different socio-geographic groups spanning the Australian continent (section 2.3). We develop an automated screening algorithm to filter out extraneous variables and assess selected transformations of candidate predictors to help optimize linear relationships in subsequent modelling (section 2.4). We use least absolute shrinkage and selection operator (or Lasso) penalized regression (a contemporary machine-learning algorithm (Tibshirani, 1996)) to interpret key predictors from large numbers of candidate variables, and purpose-built 10-fold cross-validation (CV) modelling to indicate reproducibility and uncertainty in our results (section 2.5). This approach puts the onus on variables to compete and display strength and consistency of predictive value.

2.1. Public health and contextual data

We use merged 2011/12 and 2012/13 Social Health Atlas of Australia (PHIDU, 2015, 2016) data for respiratory disease public