Urbanisation of floodplain ecosystems: Weight-of-evidence and network meta-analysis elucidate multiple stressor pathways

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HIGHLIGHTS

- Complexity of multiple stressors can preclude holistic understanding of ecosystems.
- We applied a framework to quantify complex impacts on urban floodplain ecosystems.
- Complexity was quantified via network meta-analysis and weight-of-evidence approaches.
- Study revealed evidence gaps of pressure and stressor impacts on ecological receptors.
- Networks illustrated novel disturbance pathways for hypothesis generation.

GRAPHICAL ABSTRACT
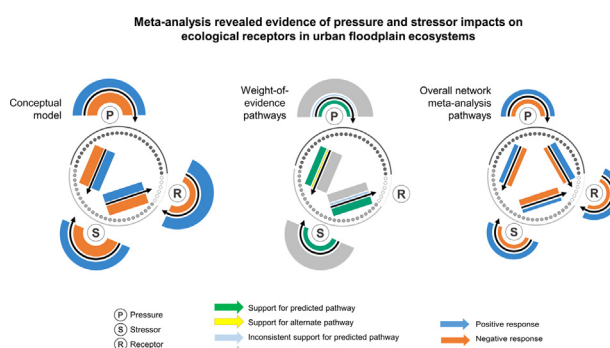
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ABSTRACT

Freshwater floodplains are dynamic, diverse ecosystems that represent important transition zones between terrestrial, riparian, subsurface and aquatic habitats. Given their historic importance in human development, floodplains have been exposed to a variety of pressures, which in combination have been instrumental in driving changes within these ecosystems. Here, we present an evidence-based framework to explore direct and indirect effects of pressures and stressors on floodplain ecosystems and test this structure within the urban landscape. Evidence was obtained from peer-reviewed scientific literature, focusing on effects of key pressures and stressors on receptors, including species composition (e.g., species presence-absence, diversity) and ecosystem function (e.g., biomass, decomposition). The strength of direct and indirect effects of individual and multiple stressors on biological receptors was quantified using two separate analyses: an evidence-weighted analysis and a quantitative network meta-analysis using data extracted from 131 studies. Results demonstrate the power of adopting a systematic framework to advance quantitative assessment of floodplain ecosystems affected by multiple stressors. While direct pathways were generally stronger and provided the core network skeleton, there were many more significant indirect pathways indicating evidence gaps in our mechanistic understanding of these processes. Indeed, the importance of indirect pathways (e.g. increase in impervious surface → increase in the accumulation rate of sediment nutrients) suggests that embracing complexity in network meta-analysis is a necessary step in revealing a more complete snapshot of the network. Results from the weight-of-evidence approach generally mirrored the direct pathway structure and demonstrated the strength of incorporating study quality

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alongside data sufficiency. Networks illustrated novel disturbance pathways (e.g., decrease in habitat structure → decrease in structure and function of aquatic and riparian assemblages) that can be used for hypothesis generation for future scientific enquiries. Our results highlight the broader applicability of adopting the proposed framework for assessing complex environments, such as floodplains.

1. Introduction

Urbanisation pressures currently threaten freshwater ecosystems, and these pressures are likely to strengthen as 68% of the global population is projected to live in urban areas by 2050 (Faulkner, 2004; United Nations, 2018). Through construction of impervious surfaces, urbanised areas alter water dynamics (McGrane, 2016), sediment and nutrient loads (Sonneman et al., 2001; Faulkner, 2004; Lee et al., 2006), carbon fluxes (Sickman et al., 2007), light regimes (Bergeron and Strachan, 2012), water temperature and chemistry (Oke, 2004; Zhang et al., 2015), and water pollution levels (Grimm et al., 2008). Combined, these stressors can lead to changes in surface water hydrology (Faulkner, 2004; Lee et al., 2006; O'Driscoll et al., 2010), declining groundwater levels (Rose and Peters, 2001; Mount et al., 2001), and decreasing microclimate regulation and function (Zhang et al., 2015, 2016). These stressors cause degradation of biodiversity, species richness, and abundance of at-risk plants and animals (Nehlsen et al., 1991; May et al., 1997; Sonneman et al., 2001; Faulkner, 2004; Morgan and Cushman, 2005), altering microbial, diatom, and macroinvertebrate assemblages (Sonneman et al., 2001), and ultimately impacting ecosystem function, species interactions and the provision of ecosystem services.

As human settlement began in floodplains, it is not surprising that floodplains and their associated wetlands remain the habitats most threatened by urbanisation, particularly in coastal areas (Reinelt et al., 1998; Sonneman et al., 2001; Faulkner, 2004; Kentula et al., 2004; Lee et al., 2006; Patenaude et al., 2015; Zhang et al., 2016). Globally, these key habitats are experiencing accelerated loss (Davidson, 2014; Hu et al., 2017) with factors such as eutrophication, water abstraction, contamination, climate change, and invasion of exotic species, contributing to the loss of ecosystem functioning. More troubling are the issues surrounding changes to the hydrological regimes of these systems, with the practices of filling, dyking, draining, and reclamation of floodplain ecosystems (Brinson and Malvárez, 2002) all linked to urbanisation. These pressures are especially pronounced in developing and transition economies (Pauchard et al., 2006) where wetlands are often viewed as marginal lands suitable for natural resource exploitation and reclamation to support growing populations (Bosma et al., 2017; Greenland-Smith et al., 2016; Turyahabwe et al., 2017) rather than as critical providers of essential ecosystem services.

Floodplain wetlands are of critical global importance due to their unique nature as highly productive and biodiverse habitats that provide valuable ecosystem services, such as flood protection, food security, and societal well-being (Tockner and Stanford, 2002). These wetlands are intrinsically linked to terrestrial ecosystems, surface and groundwater, soils, and riparian zones, with interactions among these ecosystems serving to maintain their health and resilience (e.g., Brunke and Gonser, 1997; Arthington et al., 2010; Vander Vorste et al., 2016a, 2016b). In terms of biodiversity, floodplain wetlands support high habitat diversity and a disproportionately large number of biota compared to their global extent (Knopf and Samson, 1994; Kelsey and West, 1998; Tockner and Stanford, 2002; Flinn et al., 2008). Despite warnings from the science community of their critical endangerment (e.g., the Cuiabá Wetlands Declaration; Society of Wetland Scientists, 2008), pressures on these ecosystems and their preservation are often downplayed or simply ignored by regulators and developers (Ripple et al., 2017; Finlayson et al., 2011).

As societal use of these ecosystems has evolved, the associated primary drivers of ecosystems change (i.e., urbanisation, agriculture) have degraded biodiversity and ecosystem functioning (Brinson and Malvárez, 2002). While there is consensus that intense urbanisation pressures will have negative effects on biodiversity, the effects of moderate urbanisation are less clear, or at least less discernible (McKinney, 2008). The paradox that species richness may indeed increase with low-to-moderate urbanisation (McKinney, 2008) could be attributed to replacement of local species by non-native species, spatial heterogeneity, disturbance (i.e., the intermediate disturbance hypothesis; Connell, 1978), and scale. Of all major drivers of freshwater ecosystem change, urbanisation 'hides in plain sight', because most of the evidence for these patterns comes from highly focused, localised studies that fail to consider the large suite of potential pressures associated with urbanisation, which drive multiple stressor impacts. Here, we argue that by embracing their complex nature, we can gain greater insights into the interlinked pressures present in these environments and how local ecosystems respond, thus improving our ability to manage urbanisation pressures more effectively.

Given the complexity of how the adjacent components of floodplains (e.g., wetland, groundwater, river, and terrestrial ecosystems) interact and transfer energy and materials, they are perhaps better understood as 'meta-ecosystems' (Loreau et al., 2003). Understanding multiple stressor impacts on such spatially and temporally complex systems remains a key challenge for ecologists (Dafforn et al., 2016). The DPSIR framework (Driver-Pressure-State-Impact-Response model of intervention; Smeets and Weterings, 1999; further adapted by Baird et al., 2016) offers a structure to quantify pathways between components to improve our understanding of highly complex systems and to assess multi-stressor responses of ecosystems.

Here, we review existing literature on the effects of urbanisation on floodplain wetlands. Specifically, we apply an explicitly multi-stressor approach within an overarching DPSIR-compatible framework (Van den Brink et al., 2019, this volume), supporting science-to-policy linkages through the use of formal, quantitative meta-analysis approaches. These analyses are used to identify the pressures and pathways by which urbanisation impacts floodplain wetlands and the biodiversity and ecological responses that arise. Additionally, we explore how this approach can support quantification of indirect pathways, elucidating hidden linkages among pressures, stressors, and drivers. We illustrate the power of quantitative meta-analytical approaches and the importance of considering pressures from a multiple stressor context. Our results provide resource managers with quantifiable evidence to support management and restoration efforts and highlight knowledge gaps to inform future research needs.

2. Methods

2.1. Meta-analysis framework

The proposed framework consists of two parts: an environmental filter and a transmitting function to allow effects to propagate to higher levels of biological organisation (Van den Brink et al., 2019, this volume). Applying the framework consists of eight steps designed to identify pressures, stressors, their interactions and biotic groups sensitive to the stressors (Van den Brink et al., 2019, this volume). These are used to identify potential responses of biotic and functional groups and form an

ecological model to predict the interaction of multiple stressors within an ecosystem. Data are then used to predict the resultant impact of multiple stressors on the ecosystem studied. We applied the proposed framework within floodplain wetlands and identified several drivers of multiple stressor impacts on the ecosystem (e.g. agriculture, forestry, industry, water management, climate change). However, we chose to focus on one of the most pressing societal drivers influencing river floodplains today and throughout history: urbanisation. We defined urbanisation as the driver of primary *pressures* and secondary *stressors* on the floodplain ecosystem, and denoted *receptors* as the ecological endpoints within the framework that are often measured as part of routine monitoring.

A conceptual, iterative model to describe DPSIR pathways was developed during the initial expert workshop. We predicted directional changes between pairwise pressure-stressor and stressor-receptor nodes that would cause quantifiable directional responses based on our mechanistic understanding of these pairwise pathways (Fig. 1). The conceptual model was analysed using two literature-based methods to quantitatively assess the impacts of urbanisation on freshwater floodplains: 1) a weight-of-evidence pathway analysis, called Eco Evidence (Norris et al., 2012), and 2) a structured network meta-analysis (Schwarzer et al., 2015). The Eco Evidence approach allowed us to assess the mechanisms behind predicted pathways among pressures, stressors, and receptors from our conceptual model via a structured weight-of-evidence approach using an adapted causal criteria analysis that allows us to strengthen confidence in these pairwise pathways using available

evidence (Norris et al., 2012). The network meta-analysis, which is increasingly applied to complex clinical studies in human medicine (Schwarzer et al., 2015), allowed our analysis to move beyond identification of direct pathways towards quantifying the strength of direct and indirect pathways of both individual and multiple pressures and stressors on biotic receptors (Neupane et al., 2014).

### 2.2. Standardised literature review

Given the highly complex pathways among pressures, stressors, and receptors in response to the driver of urbanisation in floodplain ecosystems, we used an a priori literature extraction focusing on six core pressure themes identified as being important to ecosystem integrity during workshop discussions: sediments, nutrients, surface flow, contaminants, groundwater, and water temperature. Applying strict standardised operational criteria to minimise bias among extractions (Singh, 2017), literature searches were completed using Google Scholar, with the same standardised Boolean terms at the start of each search [(urbanisation OR urbanization) AND (floodplain OR "flood plain")]. Each search was then tailored to the component of interest, for example ("water temperature" OR "stream temperature). These standardised searches yielded an average of 18,460 results (SD = 7308.9) and papers for the first 250 hits by relevance of each search combination were downloaded ($n = 1500$ peer-reviewed papers total). Preliminary paper screening involved a structured assessment of the quality, relevance, and information content of each study (e.g. availability of raw data, graphical data that could be extracted, full statistical output). These screening measures resulted in 131 studies supporting single (e.g. pressure (P) → stressor (S), pressure → receptor (R) or stressor → receptor) and multiple (e.g. P → S → R) pathways in single and multiple stressor environments across the six broad pressure themes. Data and study information were extracted, including pressure theme, location, study type, collection and analysis methods, and summary results. Additionally, individual and multiple pathways among the core pressures, stressors, and receptors were identified using simple linkage statements and supported via statistical results. For the quantified network meta-analysis, data were further summarised to support pathways including means, sample sizes, and standard deviations of pathway responses for both control and impact groups.

2.3. Identified nodes

Standardised nodes were identified from the terms extracted during the literature review (Table 1). Terms were merged to maximise the number of evidence items within a node without compromising the complexity of the pathway network. Nodes were classified as pressures ($n = 27$) (e.g. impervious surfaces, erosion, channel form), stressors ($n = 25$) (e.g. riparian/floodplain cover, large woody debris, soil organic matter), and receptors ($n = 6$) (e.g. function or composition, aquatic or riparian).

2.4. Weight-of-evidence analysis

We developed a simple conceptual model to explore associations among the 58 standardised nodes using the EcoEvidence approach (Fig. 1; Norris et al., 2012). We predicted that directional changes across the pressure, stressor, and receptor nodes would cause quantifiable directional responses, and individual evidence items were paired with each pathway. Only evidence with statistical significance ($\alpha = 0.05$) was included in the final weight-of-evidence analysis because the weight-of-evidence approach is to find evidence to support individual node-to-node pathways rather than to assess strength of pathways. Where statistically significant pathways were observed, support from the evidence was assessed according to its study design, where higher weights were assigned to studies with stronger designs (e.g. greater replication). For each pathway, the weight of each paired evidence item was summed for those supporting the pathway and for those refuting the pathway. The two summed values were compared to a
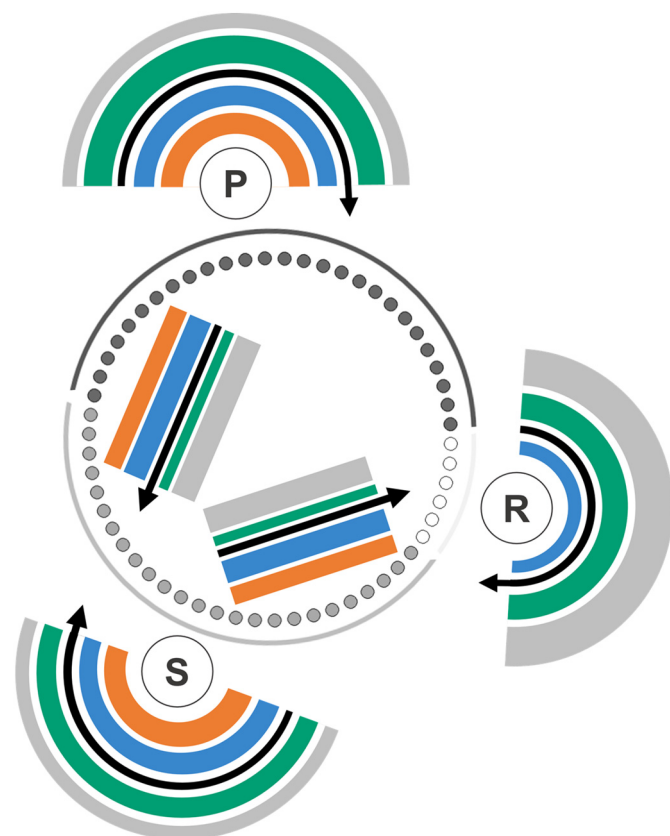
Fig. 1. Conceptual model of the effects of increasing urbanisation on pressures, stressors and biological receptors in freshwater floodplain habitats. Predicted pathways were developed from our existing mechanistic understanding of directional responses from pressures (P) to stressors and stressors (S) to receptors (R). The lines represent the relative number of predicted pathways between nodes. The colour of the line represents the directionality from the primary node to the response node: orange = increase-increase; blue = increase-decrease; green = decrease-increase; and grey = decrease-decrease. The circles represent the individual nodes used in the analyses (Table 1). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Table 1

Standard codes and terms for nodes used in the network meta-analysis. P indicates pressure, S indicates stressor and R indicates receptor. Nodes represented in the weight-of-evidence analysis are indicated by •. Mean (SD) standardised mean difference treatment effect presented for direct, indirect and overall pairwise pathways. Number of statistically significant ($p < 0.05$) pathways for each node are presented.

Node ID	Term	Direct pathways	Indirect pathways	Overall pathways
• P01	Impervious surface	2.415 (1.810)	2.226 (1.470)	2.176 (1.451)
		$p < 0.05 = 24$	$p < 0.05 = 44$	$p < 0.05 = 50$
• P02	Flow: Magnitude	0.083 (1.328)	0.396 (0.952)	0.445 (0.979)
		$p < 0.05 = 7$	$p < 0.05 = 21$	$p < 0.05 = 21$
• P03	Erosion	0.047 (0.609)	0.937 (1.061)	0.922 (1.058)
		$p < 0.05 = 2$	$p < 0.05 = 26$	$p < 0.05 = 28$
P04	Sediment flux	−0.215 (0.053)	0.216 (0.965)	0.216 (0.965)
		$p < 0.05 = 0$	$p < 0.05 = 16$	$p < 0.05 = 16$
• P05	Channel size	1.581 (1.368)	0.703 (1.008)	0.746 (1.027)
		$p < 0.05 = 2$	$p < 0.05 = 19$	$p < 0.05 = 22$
• P06	Channel form	−0.236 (0.505)	−0.372 (1.056)	−0.376 (1.047)
		$p < 0.05 = 1$	$p < 0.05 = 29$	$p < 0.05 = 30$
• P07	Flow: variability (flashiness)	0.985 (1.327)	0.186 (0.967)	0.191 (0.965)
		$p < 0.05 = 3$	$p < 0.05 = 16$	$p < 0.05 = 16$
• P08	Proximity to urban area	−1.286 (0.410)	−0.343 (1.053)	−0.325 (1.041)
		$p < 0.05 = 2$	$p < 0.05 = 25$	$p < 0.05 = 25$
• P09	Deposition	−0.110 (0.593)	2.472 (2.200)	2.509 (2.181)
		$p < 0.05 = 2$	$p < 0.05 = 51$	$p < 0.05 = 53$
• P10	Coarse substrate	−0.147 (0.352)	−0.341 (1.119)	−0.337 (1.109)
		$p < 0.05 = 0$	$p < 0.05 = 30$	$p < 0.05 = 31$
P11	Sediment depth	−0.439 (0.459)	−0.823 (1.405)	−0.827 (1.398)
		$p < 0.05 = 1$	$p < 0.05 = 41$	$p < 0.05 = 42$
• P12	Slope	−0.714 (1.363)	−1.125 (1.649)	−1.144 (1.645)
		$p < 0.05 = 4$	$p < 0.05 = 45$	$p < 0.05 = 45$
• P13	Baseflow (surface)	−0.178 (0.821)	0.402 (1.023)	0.415 (1.031)
		$p < 0.05 = 2$	$p < 0.05 = 14$	$p < 0.05 = 15$
• P14	Floodplain wetted area	−0.135 (0.543)	1.051 (1.453)	1.038 (1.454)
		$p < 0.05 = 1$	$p < 0.05 = 41$	$p < 0.05 = 43$
P15	Velocity	0.368 (0.437)	−0.417 (1.221)	−0.423 (1.220)
		$p < 0.05 = 2$	$p < 0.05 = 29$	$p < 0.05 = 30$
• P16	Turbidity	0.466 (0.176)	0.186 (0.977)	0.172 (0.968)
		$p < 0.05 = 0$	$p < 0.05 = 14$	$p < 0.05 = 14$
• P17	Fine substrate	0.439 (0.448)	0.118 (0.966)	0.117 (0.966)
		$p < 0.05 = 0$	$p < 0.05 = 13$	$p < 0.05 = 13$
P18	Sedimentation rate	0.544 (0.361)	0.831 (1.494)	0.832 (1.493)
		$p < 0.05 = 1$	$p < 0.05 = 37$	$p < 0.05 = 38$
• P19	Groundwater levels	−5.286 (3.373)	−0.132 (1.121)	−0.134 (1.121)
		$p < 0.05 = 1$	$p < 0.05 = 23$	$p < 0.05 = 23$
P20	Habitat structure	1.142 (1.112)	−0.777 (2.036)	−0.759 (2.040)
		$p < 0.05 = 4$	$p < 0.05 = 45$	$p < 0.05 = 46$
• P21	Substrate size	0.891 (1.210)	−1.395 (3.294)	−1.375 (3.293)
		$p < 0.05 = 4$	$p < 0.05 = 53$	$p < 0.05 = 54$
• P22	Fragmentation	2.598 (0.773)	−0.042 (0.982)	−0.026 (0.986)
		$p < 0.05 = 2$	$p < 0.05 = 10$	$p < 0.05 = 11$
• P23	Flow: duration/frequency of events	−0.203 (0.198)	0.052 (1.061)	0.052 (1.054)
		$p < 0.05 = 0$	$p < 0.05 = 9$	$p < 0.05 = 10$
• P24	Sinuosity	0.431 (0.148)	−0.065 (0.972)	−0.067 (0.967)
		$p < 0.05 = 0$	$p < 0.05 = 11$	$p < 0.05 = 12$
• P25	Groundwater recharge/exchange	−4.296 (0.944)	−0.415 (1.649)	−0.367 (1.530)
		$p < 0.05 = 2$	$p < 0.05 = 24$	$p < 0.05 = 25$
P26	Riparian area	6.371 (1.077)	−0.478 (1.670)	−0.406 (1.683)
		$p < 0.05 = 0$	$p < 0.05 = 0$	$p < 0.05 = 0$
• P27	Channel migration	0.870 (0.166)	−0.138 (1.046)	−0.139 (1.048)
		$p < 0.05 = 0$	$p < 0.05 = 7$	$p < 0.05 = 7$
• S01	Nutrients (N, P, C) (short-term)	−1.306 (3.474)	−0.246 (1.216)	−0.155 (0.996)
		$p < 0.05 = 6$	$p < 0.05 = 24$	$p < 0.05 = 22$
• S02	Riparian/floodplain cover	−0.023 (1.134)	−0.180 (1.013)	−0.167 (0.964)
		$p < 0.05 = 7$	$p < 0.05 = 21$	$p < 0.05 = 20$
• S03	Conductivity	1.559 (1.289)	−0.163 (2.107)	−0.167 (2.107)
		$p < 0.05 = 10$	$p < 0.05 = 50$	$p < 0.05 = 51$
• S04	pH	−0.157 (1.078)	−0.075 (1.020)	−0.091 (1.020)
		$p < 0.05 = 5$	$p < 0.05 = 21$	$p < 0.05 = 21$
• S05	Surface water non-essential toxic substances	−0.465 (0.593)	−0.108 (1.021)	−0.086 (1.005)
		$p < 0.05 = 2$	$p < 0.05 = 20$	$p < 0.05 = 21$
• S06	Sediment non-essential toxic substances	0.595 (0.611)	−0.082 (1.021)	−0.094 (1.021)
		$p < 0.05 = 2$	$p < 0.05 = 21$	$p < 0.05 = 21$
• S07	Water temperature: Magnitude (summer)	−0.527 (0.746)	−0.246 (1.453)	−0.243 (1.449)
		$p < 0.05 = 4$	$p < 0.05 = 39$	$p < 0.05 = 42$
• S08	Large woody debris	0.131 (1.731)	0.275 (1.347)	0.263 (1.361)
		$p < 0.05 = 11$	$p < 0.05 = 39$	$p < 0.05 = 39$
S09	Sediment nutrients (N, P, C)	0.732 (0.746)	−0.180 (1.102)	−0.186 (1.110)
		$p < 0.05 = 5$	$p < 0.05 = 19$	$p < 0.05 = 19$
S10	Water temperature: Magnitude (winter)	−0.430 (0.288)	−0.125 (1.035)	−0.114 (1.025)

Table 1 (continued)

Node ID	Term	Direct pathways	Indirect pathways	Overall pathways
• S11	Sediment organic matter	$p < 0.05 = 1$ −0.641 (0.623)	$p < 0.05 = 20$ −0.167 (1.058)	$p < 0.05 = 21$ −0.149 (1.057)
S12	Sediment non-essential toxic substances (storm)	$p < 0.05 = 2$ −0.087 (0.017)	$p < 0.05 = 20$ 0.008 (0.970)	$p < 0.05 = 21$ 0.009 (0.970)
S13	Surface water non-essential toxic substances (storm)	$p < 0.05 = 0$ −0.189 (0.024)	$p < 0.05 = 13$ −0.057 (0.990)	$p < 0.05 = 13$ −0.057 (0.990)
• S14	Organic matter	$p < 0.05 = 17$ 1.101 (0.584)	$p < 0.05 = 15$ 0.114 (0.979)	$p < 0.05 = 17$ 0.112 (0.971)
S15	Soil non-essential toxic substances	$p < 0.05 = 1$ 0.383 (0.154)	$p < 0.05 = 15$ −0.214 (1.100)	$p < 0.05 = 16$ −0.236 (1.076)
• S16	Sediment nutrient accumulation rate (N, P, C)	$p < 0.05 = 0$ 2.192 (0.427)	$p < 0.05 = 17$ −0.377 (1.252)	$p < 0.05 = 17$ −0.395 (1.198)
• S17	Oxygen	$p < 0.05 = 1$ 0.400 (0.572)	$p < 0.05 = 21$ 0.129 (0.960)	$p < 0.05 = 22$ 0.130 (0.966)
S18	Groundwater non-essential toxic substances	$p < 0.05 = 2$ 0.630 (0.460)	$p < 0.05 = 15$ −0.217 (1.072)	$p < 0.05 = 15$ −0.226 (1.063)
S19	Nutrients (N, P, C) (long-term)	$p < 0.05 = 1$ 2.703 (0.251)	$p < 0.05 = 19$ 1.415 (1.769)	$p < 0.05 = 18$ 1.438 (1.778)
S20	Soil nutrients (N, P, C)	$p < 0.05 = 0$ 0.108 (0.215)	$p < 0.05 = 1$ −0.105 (0.986)	$p < 0.05 = 1$ −0.109 (0.985)
S21	Riparian/floodplain diversity	$p < 0.05 = 0$ −1.053 (0.764)	$p < 0.05 = 13$ 0.157 (0.965)	$p < 0.05 = 14$ 0.136 (0.969)
S22	Soil organic matter	$p < 0.05 = 2$ −1.046 (0.794)	$p < 0.05 = 12$ 0.895 (1.233)	$p < 0.05 = 12$ 0.862 (1.222)
S23	PAR	$p < 0.05 = 1$ 0.209 (0.078)	$p < 0.05 = 25$ −0.079 (0.971)	$p < 0.05 = 26$ −0.076 (0.971)
• S24	Evapotranspiration	$p < 0.05 = 0$ 4.000 (0.523)	$p < 0.05 = 13$ 1.971 (1.888)	$p < 0.05 = 13$ 1.971 (1.880)
• S25	Habitat quality	$p < 0.05 = 2$ 3.617 (0.335)	$p < 0.05 = 42$ 1.021 (1.296)	$p < 0.05 = 43$ 1.066 (1.327)
• R01	Function (aquatic)	$p < 0.05 = 1$ −0.145 (1.275)	$p < 0.05 = 8$ −0.026 (0.939)	$p < 0.05 = 9$ −0.047 (0.970)
• R02	Composition (aquatic)	$p < 0.05 = 13$ −0.489 (0.973)	$p < 0.05 = 24$ −0.379 (0.993)	$p < 0.05 = 27$ −0.398 (0.974)
• R03	Composition (riparian)	$p < 0.05 = 9$ 1.428 (1.603)	$p < 0.05 = 20$ −0.310 (1.004)	$p < 0.05 = 22$ −0.305 (0.967)
• R04	Function (riparian)	$p < 0.05 = 2$ 0.835 (0.077)	$p < 0.05 = 19$ −1.338 (1.168)	$p < 0.05 = 19$ −1.300 (1.175)
• R05	Fecal coliform	$p < 0.05 = 0$ −2.007 (0.333)	$p < 0.05 = 4$ −2.553 (1.684)	$p < 0.05 = 4$ −2.554 (1.681)
R06	Chlorophyll a	$p < 0.05 = 1$ 0.651 (0.060)	$p < 0.05 = 46$ 0.764 (1.038)	$p < 0.05 = 47$ 0.762 (1.038)
		$p < 0.05 = 0$	$p < 0.05 = 5$	$p < 0.05 = 5$

standardised threshold value (Norris et al., 2012): support for predicted pathway (≥20 points in support, <20 points against); support for alternate pathway (<20 in support, ≥20 against); inconsistent evidence (≥20 in support, ≥20 against); and insufficient evidence (<20 in favour, <20 against).

2.5. Quantitative meta-analysis

Network meta-analysis expands pairwise meta-analysis to combine direct and indirect evidence for pathways across multiple nodes (Schwarzer et al., 2015). The analysis combines individual lines of evidence between nodes, allowing for single (P → P, P → S, S → S, P → R or S → R) and multiple (P → S → R) pathways (Fig. 1). Data were extracted for studies where comparisons were made between reference (non-urbanised) and impact (urbanised) samples and a frequentist network meta-analysis model was developed. The standardised effect size using Hedge's g was calculated for each pathway because it represents an estimate of the standardised mean difference that is not biased by small sample sizes (Gurevitch and Hedges, 2001). The network meta-analysis was completed using a random-effects model that assumes the observed estimates of treatment effect vary across studies because of both measured differences in addition to sampling variability: the included evidence items were assumed to be a random sample from all possible studies that met the inclusion criteria for the review (Riley et al., 2011). This is particularly important for an ecological study where the different nodes have a wide range of natural variation in

their values. Finally, potential publication bias was assessed via funnel plots and the associated Egger's test via the *meta* package (version 4.9-2, Schwarzer, 2018). The network meta-analysis was completed using the *netmeta* package (version 0.9-8, Rücker et al., 2018) within the R Environment (version 3.5.1, R Core Team, 2018).

3. Results

3.1. Pressure, stressor and receptor nodes

From the initial 1500 articles, only 8.7% met the standardised criteria for inclusion but included floodplains from all major continents (Fig. 2). Data were extracted from 131 studies for the analysis (Supplementary Information 1), where the majority (~76%) were associated with multiple node pathways; 34.4% of the studies were associated with both the quantitative and weight-of-evidence analyses, 60.3% were only used for the weight-of-evidence analysis, and 5.3% were used only for the quantitative network meta-analysis (Fig. 2). From the included studies, we extracted 58 standardised nodes revealing 689 statistically significant overall pathways from the network meta-analysis and 79 assessed pathways from the weight-of-evidence approach.

3.2. Weight-of-evidence approach

Pathways represented 21 pressure, 14 stressor, and five receptor nodes (Table 1). Evidence was available to assess directionality and

support for 79 pairwise predicted pathways (P → P, P → S, P → R, S → S, and S → R) (Fig. 3). There was insufficient evidence to reach conclusions for the majority (54 of 79) of the predicted pathways, as they were only represented by one or two lines of evidence. Of the remaining 25 pathways, we found support for 20 hypotheses, but found support for only one alternate pathway (Fig. 3). Finally, inconsistent evidence was found for four hypotheses, but these patterns were trending towards support for each predicted pathway (Fig. 3).

Strong variation was observed in the number of pathways from different nodes, with the majority of nodes linked with few lines of evidence (Fig. 3). Inconsistent pathways had greater than average lines of evidence (average = 19, SD = 11.1 for inconsistent pathways; average = 5, SD = 7.2 for all pathways). Indeed, only nine nodes were associated with >10 lines of evidence in the analysis, reflecting the strength of pathways associated with those nodes. Not surprisingly, heavily-supported nodes were associated with strongly supported hypotheses, with an average ratio of 0.93 (SD = 0.07) for support:alternate evidence weight for those pathways.
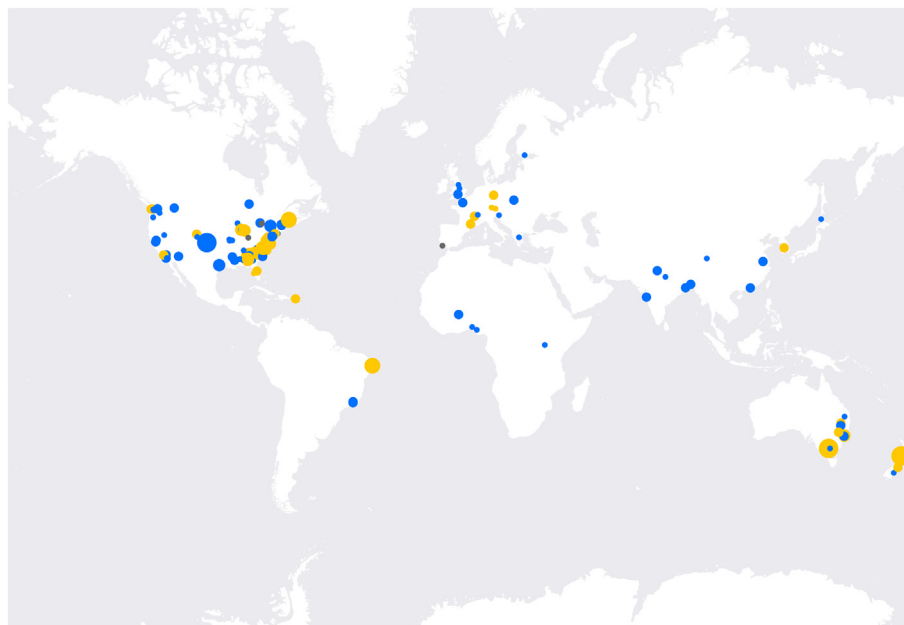
3.3. Quantitative network meta-analysis

From the 52 studies used in the network meta-analysis, 14 were from pairwise node studies while the remaining 38 represented multiple node studies. These pairwise and multiple node studies resulted in 936 pairwise comparisons, where pressure nodes represented 56.5% of the primary nodes and 16.3% as secondary nodes (Fig. 4a). The 25 stressor nodes were associated with both primary and secondary nodes in pathways (42.4% as primary, 46.4% as secondary) (Fig. 4a). The six receptor nodes were primarily associated with secondary nodes in each pathway (10.8% as primary, 37.3% as secondary) (Fig. 4a). Between-node pathways (e.g. P → S, P → R, S → R) were observed more frequently than within-node pathways (e.g. P → P, S → S, R → R), 66.6% of pathways versus 33.4% of pathways, respectively. Pairwise pathway representation from the evidence varied with nodes (average Hedge's standardised treatment effect = 0.320, SD = 3.96), where the majority of pairwise pathways were supported by evidence but the number of evidence items was generally low (average number of lines of evidence = 2.84, SD = 3.50).

A random-effects network meta-analysis model was developed from 936 pairwise evidence items, resulting in a mixed treatment comparison with some closed loops within the network. The random-effects network meta-analysis model yielded a generalised heterogeneity statistic, Q_{total}, of 3114.53 (df = 879, $p < 0.05$) where Q_{within} was 2041.57 (df = 637, $p < 0.05$) and $Q_{between}$ was 1072.96 (df = 242, $p < 0.05$). As Q_{total} exceeds df, the additional evidence strengthens the case that the excess dispersion is not zero and that the true effects vary. The within-design heterogeneity was further explored, where 292 designs from the 769 lines of evidence were included in the analysis. Within the 94 designs represented by more than one study, 49 demonstrated less heterogeneity between the contributing studies than was expected by chance ($p > 0.05$). The random-effects model had a τ^2 value of 1.272, reflecting the true heterogeneity of the model, and an I^2 value of 71.8%, reflecting the proportion of observed dispersion that was due to this heterogeneity. The model was assessed for the potential impacts of publication bias on the results. First, data were examined via funnel plots, which suggested some asymmetry around the mean effect size. Egger's regression test and Spearman rank corrections were calculated to quantify the potential bias. Results of the Spearman rank correlation tests indicated a low correlation between standardised effect size and standard error ($n = 936$, $r_s = 0.20$, $p < 0.05$) and a significant relationship between effect size and pooled sample size (n = 936, $r_s = -0.14$, $p < 0.05$). Similarly, Egger's regression test was statistically significant for both comparisons: between the standardised effect size and standard error ($t = 2.62$, df = 934, $p < 0.05$) and between the standardised effect size and the pooled sample size ($t = -4.57$, df = 934, $p < 0.05$), which may indicate bias towards larger effect sizes. Funnel plot assessments and statistical tests indicated significant asymmetry in the dataset, but graphical comparisons of regression lines showed that they were not significantly different. Indeed, the results did not necessarily indicate significant publication bias in our data, as data asymmetry may also be attributable to true heterogeneity in the dataset, reflecting the diverse node inputs (i.e. six different pressure themes and multiple stressors and receptors) and indicated by the high Q_{total} from the original model (Nakagawa and Santos, 2012).

The sensitivity of the network meta-analysis to effect size outliers was assessed using Rosenthal's method to estimate a fail-safe number. The fail-safe number, which is the number of non-significant or missing observations that would be needed to change the significance of our findings (Rosenthal, 1979), was calculated using the *fsn* function within the *metafor* package (version 2.0.0, Viechtbauer, 2017). The fail-safe

Fig. 2. Location of studies used in the analyses. Blue circles represent studies only used in the weight-of-evidence analysis, grey circles represent studies only used in the quantitative network meta-analysis, while yellow circles represent those in both analyses. Points are scaled by the number of lines of evidence extracted from the studies. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)
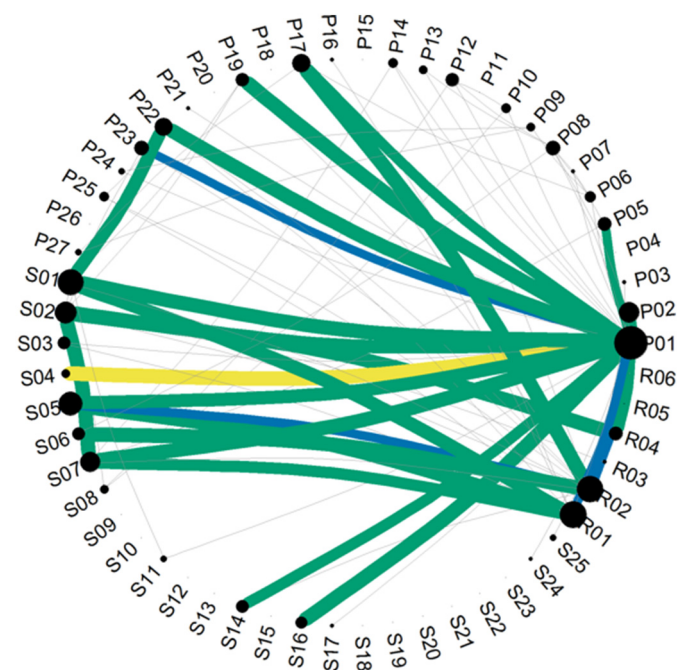
Fig. 3. Quantified pairwise pathways between nodes from the weight-of-evidence analysis (see Table 1 for code descriptions). Colour of pathways represents level of support for predicted pathways: Green pathways = support for predicted pathway; yellow pathways = support for alternate pathway; dark blue pathways = inconsistent evidence where there is a large amount of evidence both for and against the predicted pathway; and grey pathways = insufficient evidence where there is not enough evidence to assess the predicted pathway. Node size is relative to the number of lines of evidence supporting that node. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

number was estimated as 1598 (> 936, the number of input pathways) using a target alpha level of 0.05 as compared with the observed significance of 0.034, suggesting that the results are reliable despite the observed asymmetry (Rosenthal, 1979). A secondary trim and fill analysis was completed using the *metafor* package (version 2.0.0, Viechtbauer, 2010) to quantify the impact of missing observations on the meta-analysis (Nakagawa and Santos, 2012). The trim and fill analysis identified 64 pathways as potential outliers needed to restore symmetry, but the corrected random-effects model with 64 added pathways performed poorly ($p > 0.05$) with a τ^2 value of 2.2471 and an I^2 value of 81.9%.

We observed 79 statistically significant direct pairwise pathways ($p < 0.05$; Fig. 4b). The directionality of the supported pathways was generally positive (60.8% vs. 39.2%) and generally associated with P → P, P → S or S → S pathways (Fig. 4b). Overall, there were few significant pathways linking pressures or stressors to the ecological receptors (12.7% of the significant direct pathways) and directionality was generally both positive and negative. However, the supported overall direct pathways generally had a high standardised treatment effect (average = 4.22, SD = 4.30), but this did not often reflect those pairwise pathways with high evidence weight. For example, P01 (impervious surface) → S01 (short-term N, P, C nutrients) had the greatest amount of evidence, but the standardised treatment effect for this pathway was not statistically significant ($p > 0.05$), likely reflecting the mixed directionality for this pathway. This does not mean that there is no effect, but instead that the directionality of the pathway may change depending on the study and likely quantifying different pathways. The indirect pairwise pathways were heavily supported in the analysis, with 668 statistically significant ($p < 0.05$) pathways observed (Fig. 4c). On average, the standardised treatment effect for these significant pathways was weaker than the direct pathways (average = 2.43, SD = 1.21) (Fig. 4c). Pathways were generally within-node (e.g. P → P or S → S) or between specific

node types (i.e. P → S). Receptor nodes were only associated with 9.7% of the overall pathways (but 17.2% of the significant overall pathways), and they were generally negative (70.6% of the significant overall pathways) in their directionality (Fig. 4d). The strongest indirect pathways were heavily associated with pressure nodes, particularly P09 (deposition) and P21 (substrate size), which accounted for over half of the strongest indirect pathways (Fig. 4c). Finally, for the overall pairwise standardised treatment effect, 649 (69.3%) statistically significant pathways ($p < 0.05$) were observed, with the strongest pathways generally mirroring those from the indirect pathways (Fig. 4d). Again, the overall standardised treatment effect was weaker than the direct standardised treatment effect (average = 2.39, SD = 1.20), and pathways were generally associated with P → P, P → S or S → S pathways (81.9% of the overall pathways) (Fig. 4d).

4. Discussion

We illustrated the potential for the wider application of a mechanism-based, data-driven DPSIR framework that can directly integrate available data with published literature to quantify directly measured pathways and indirectly inferred pathways via both weight-of-evidence and quantitative network meta-analysis. Using standardised data extraction methods, the two analytical approaches were complementary and supported the formal assessment of urbanisation impacts in floodplain ecosystems by testing our predicted direct pathways and identifying key indirect pathways that warrant additional exploration. Specifically, these approaches allowed us to 1) develop a mechanistic understanding of how urbanisation has impacted freshwater and associated riparian systems, 2) highlight knowledge gaps in our mechanistic understanding of these impacts, and 3) make hypotheses about how previously unknown or poorly-studied pathways of urbanisation impacts are expected to affect these ecosystems and their functional attributes.

The 77 significant direct pathways in the network meta-analysis generally mirrored the supported pathways from the weight-of-evidence approach. The significant direct pathways were dominated by physical properties and processes (e.g., flow (magnitude), slope, erosion) as pressures on a subset of stressors and receptors (e.g., short-term nutrients (N, P, C), LWD, aquatic composition and function). These direct results highlight the benefit of both the network meta-analysis and the weight-of-evidence approaches, as they strengthen support by combining information for these pathways, thereby informing the DPSIR framework structure.

The structure of the overall treatment effects from the network meta-analysis was heavily influenced by indirect pathways, which were not explicitly measured in studies within this analysis. Indirect pathways help to identify knowledge and data gaps, and also provide a framework for developing new pathways that can be further tested provided they are biologically meaningful; for example, a decrease in floodplain wetted area → decrease in LWD could be driven by a loss of riparian area and a loss of LWD sources. The indirect pathways were sometimes limited by available data and node information; the indirect pathway showing a decrease in substrate size → decrease in aquatic composition, for instance, bypassed the stressor component of the pathway. However, indirect pathways overall did provide context and a mechanistic understanding for complex and unexpected patterns observed in the literature. For example, while only 5 studies (3.8%) from our corpus considered receptor-receptor linkages, and none (0 of 4) of the direct pathways covered by those studies were significant, 5 indirect pathways were significant ($n = 14$ total); indirect pathways like these could provide support for receptor-receptor feedback loops, which are difficult to study but may be common in natural systems. A recent meta-analysis of multiple stressors in freshwater systems revealed that the net effects, across all stressor pairs and response metrics, were consistently antagonistic or additive (Jackson et al., 2016); however, while this synthesis summarised multiple stressor effects in freshwater systems, it was unable to reveal indirect pathways, and, therefore,
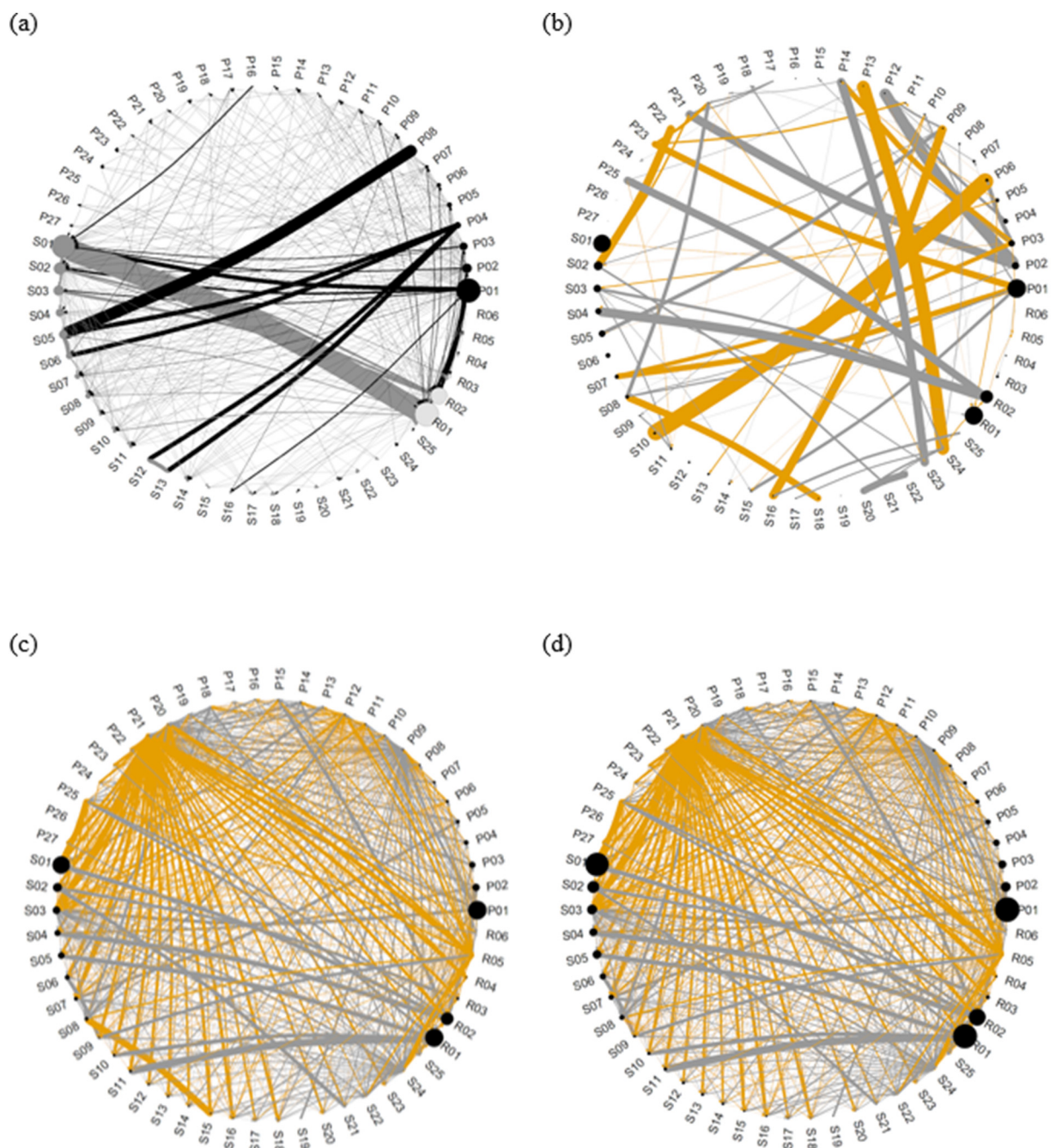
Fig. 4. Quantified pathways between nodes (see Table 1 for code description). The size of individual nodes represents the number of lines of evidence supporting that node. (a) Thickness of lines represents the log-transformed weights from random effects model for pairwise node comparisons. The colour of the lines and nodes represents the pathway node source category: pressures are black, stressors are mid-grey and receptors are light grey; (b) Thickness of lines represents standardised treatment effect between two nodes for direct pathways. The colour of the lines represents the direction of the pathway: positive pathways are grey and negative pathways are orange; (c) Thickness of lines represents the standardised treatment effect between two nodes for indirect pathways. Line colour as for (b); (d) Thickness of lines represents the overall standardised treatment effect between two nodes. Line colour as for (b). For figures (b) - (d), only statistically significant (p < 0.05) pathways are shown and node size as for (a). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

provide a mechanistic understanding of multiple stressor effects. A network meta-analysis approach, like the one we have illustrated here, could further improve our understanding of freshwater systems, illustrating hidden mechanisms of the observed patterns and providing hypothesised pathways for future study.

4.1. Response complexity of biodiversity and ecosystem function to urbanisation

Previous assessments of the impacts of urbanisation have elucidated how urbanisation is associated with loss of vegetation (habitat) and the simplification of vegetative structure (McKinney, 2008) and remaining habitat (Marzluff and Ewing, 2001). Such factors are known to decrease the biodiversity of animals whose diversity correlates with vegetative complexity and plant species richness, such as birds and aquatic fauna (Savard et al., 2000). Urbanisation is also strongly associated with increases in contaminants (e.g. heavy metals, pesticides, pharmaceuticals, nitrogen, sewage) and sedimentation within water systems (Mekonnen and Hoekstra, 2015; Schuler and Relyea, 2018; Strokal et al., 2018). Such contaminants have been linked to reduced biodiversity in both stream (e.g., fish and macroinvertebrates) (Beasley and Kneale, 2002; Muñoz et al., 2016; Bere et al., 2016) and groundwater (e.g. stygofauna)

systems (Boulton et al., 2003; Gibert et al., 2009). Lastly, urbanisation has been strongly linked to changes in hydrological patterns; such changes can profoundly alter habitat availability and diversity, impacting species composition, productivity, and ecosystem functioning (Junk et al., 2006). Most of these patterns were also observed and quantified via both direct and indirect pathways in our meta-analysis, but our approach allowed us to place these results in the context of the wider framework and identify novel hypotheses from the network.

The mixed responses, in terms of significance, magnitude, and path direction, documented in our network meta-analysis are surprising given what we know about how urbanisation is expected to affect biodiversity and ecosystem function. The general lack of strong directionality across a majority of nodes in our analysis may reflect the homogenising driver of urbanisation (McKinney, 2008; Alvey, 2006). This homogenisation effect can have a complicated and divisive influence on local biodiversity due to complex ecological responses and tolerances to single and multiple stressors and pressures (McKinney, 2008).

Spatial and temporal heterogeneity could also reflect apparent contradictions between predicted pathways and those quantified within our analyses. Within shorter periods, species invasions within urban landscapes can actively outpace species loss, particularly for smaller or less mobile organisms that can capitalise on heterogeneous habitat patches (McKinney, 2002, 2006; Tait et al., 2005). Initial increases in alpha richness reflect the greater spatial diversity in habitat patches, which is generally caused by changes to habitat structure and increased invasion at the broader scale, leading to increases in beta diversity (Niemelä, 1999; Aronson et al., 2014). However, these changes can be highly scale dependent (Hogsden and Hutchinson, 2004); for example, urbanisation yielded net increases in channel size via flow changes and erosional processes (supported via weight-of-evidence analysis) but at the broader floodplain scale, channel area was reduced via increased impervious surfaces and loss of riparian zones (supported by both network meta-analysis and weight-of-evidence approach). Four pathways were classified as inconsistent within our weight-of-evidence approach, likely reflecting the highly variable responses of the studies. For example, we predicted that increases in surface water non-essential toxic substances would result in a net decrease in aquatic composition. While there was a large amount of support for this predicted pathway, the direction of the response was mixed, suggesting that the inconsistencies may reflect the broad nature of the categories (i.e., different non-essential toxic substances have different toxicity impacts).

A final explanation for the variability in the responses and deviation from some of our predicted pathways could be due to methodological decisions made during our analyses. First, variation in direct pathways between nodes may also occur because they incorporate signals of unquantified patterns (Harris and Heathwaite, 2005; Heathwaite, 2010). This may be reflected in our network meta-analysis, where many more indirect compared to direct pathways were identified (668 significant indirect pathways versus 79 significant direct pathways). This is not surprising given that the internal pathways in our study were complex and often driven by variation along a spatio-temporal gradient. Indeed, impacts of pressures and stressors acting within an urban landscape are highly varied across both space and time, leading to long-term, cumulative effects. This high heterogeneity is reflected in the 58 nodes within the analyses, which themselves often consist of multiple terms from single and multiple pressure or stressor studies. Second, the variation in standardised treatment effects may also reflect the fact that the weight-of-evidence approach used only studies with significant pathways, while the network meta-analysis considered all possible pathways. The strength of between-node pathways was driven by both study design (in the weight-of-evidence approach) and consistency (in both analyses), where quantitative pathways with inconsistent directionality were associated with an absence of statistically significant pathways between respective nodes, regardless of the size of the standardised treatment effect.

Consequently, real biological pathways governed by many factors of small effect or by many indirect mechanisms would be overlooked by the weight-of-evidence approach. Indeed, it was surprising that additional pathways were not supported via either the weight-of-evidence approach or the direct treatment effects from the network meta-analysis despite the large amount of evidence within both analyses. This may be due to a lack of suitable data for our analyses, with the majority of data being local and having a subject-specific focus, thereby precluding a broader understanding of linkages among pressures, stressors, and receptors. This is particularly evident given the stronger support for both direct and indirect pathways associated with pressures and stressors, but not with receptors, which are generally biological endpoints measured during biomonitoring. Third, the varying support for individual pathways may reflect the mixed directionality of receptor responses, given the broad ecological categories (e.g., "aquatic function" or "aquatic composition").

Collectively, these mechanisms could explain the mixed biological responses of composition and function in our meta-analysis. The six receptor variables associated with aquatic or riparian function were not statistically significant and did not demonstrate a strong mean standardised effect size for either their direct or indirect pathways. However, aquatic and riparian composition were generally stronger in their pathway strengths and statistical significance, although these were still weak in comparison with P → P or P → S pathways. Urbanisation severity varied across the studies in our meta-analysis, such that studies looking at moderate urban gradients sometimes showed positive biotic responses, while those with extreme gradients showed negative responses; thus, mixed responses could have led to net weak or neutral effects or variation in the direction of responses among nodes. This interpretation would be supported by the intermediate disturbance hypothesis (Connell, 1978). In the context of urbanisation, this hypothesis predicts that moderate levels of human disturbance will promote the coexistence of many types of species, including early successional native species as well as introduced species (Zerbe et al., 2003). This hypothesis has been supported in several urban studies, where species richness increases at intermediate levels of urbanisation (Blair and Launer, 1997; Germaine and Wakeling, 2001; Marzluff, 2001, 2005; Leveau and Leveau, 2005). Additionally, spatial heterogeneity can lead to a specific case of the intermediate disturbance hypothesis, called the disturbance heterogeneity model (Porter et al., 2001), where the intermediate state is defined spatially as opposed to temporally, and reviews of urban ecology demonstrate how the spatial diversity of urban habitats promotes higher beta diversity (spatial turnover) in some taxonomic groups (Rebele, 1994; Niemelä, 1999). This mechanism is particularly true for plants, and to a lesser degree invertebrates, which have relatively small geographic ranges (Gaston et al., 1998), but vertebrates are less likely to show increased species richness in urban habitats because of the large spatial scale required to maintain viable population sizes. The mixed nature of our biotic responses (i.e., grouping all organism responses together), therefore, could also explain the limited or weak biotic responses to pressures and stressors in our meta-analysis.

4.2. Information gaps, limitations of multiple-stressor studies, and areas in need of research focus

A high-level outcome of our weight-of-evidence approach and network meta-analysis is that we were able to quantify and visualise gaps in the literature related to multiple stressors in urbanised systems. While there was abundant, statistically significant evidence supporting P → P, P → S, and S → S pathways, there was much less direct evidence supporting ecological receptor nodes (i.e., composition and function) (Fig. 3, Fig. 4a). Ecological receptors, which are often the measured endpoints for routine monitoring, were only examined 18% of the studies in our analysis. When studies did look at the influence of urbanisation on ecological receptors, they often assessed only the impact of percent impervious surfaces or single pressure-receptor or stressor-receptor

pathways. This underscores the importance of studies reporting complete pressure-stressor-receptor linkages, allowing for the assessment of mechanisms underlying responses by which urbanisation influences biodiversity and ecosystem function. Among the pathways including ecological receptors in our corpus of literature (38.0% of pathways for the network meta-analysis), most looked at biodiversity and community structure (35.1%) or ecosystem function (50.0%), or linked pressures, stressors, or biodiversity patterns to ecosystem function, especially in a multiple stressor studies. However, the ecological receptors were pooled in our study to maximise the strength of pathway evidence, and so in the future it will be important to examine the multiple stressor context for specific functions, evaluating them from an ecosystem services perspective (sensu Allan et al., 2013; Acuña and Garcia, 2019).

Despite the few multi-arm studies that include ecological receptors in the literature, our use of network meta-analysis enabled quantification of not only observed, direct links among pressures, stressors, and receptors, but also indirect links, enabling a more holistic multiple-stressors perspective. These indirect pathways elucidated between-habitat pathways, for example between surface and sub-surface processes, which are difficult to measure. Specifically, assessment of indirect pathways enabled quantification of path significance and path strength of linkages that have not been documented in the literature. This allows us to glean information on which nodes and pathways missing from the literature are likely to be important, providing a framework for hypothesis generation for future studies and research.

Observations about information gaps identified in our study generally corroborate and extend what we know from other meta-analyses and reviews on urbanisation. While many case studies document how urbanisation is associated with different aspects of biodiversity, we do not have a strong consensus on the commonalities and context dependence of these impacts across urban systems, especially when it comes to understanding the mechanisms by which urbanisation impacts biodiversity in ecological systems (Roy et al., 2016). Additionally, while many studies of urban-rural gradients describe the spatial effects of urbanisation on species-richness by examining changes along an intensity gradient (McKinney, 2002; Marzluff, 2001; Chace and Walsh, 2006), temporal studies in urban areas are rare (McKinney, 2008). Accurate, detailed urban inventories of biota are unavailable for long periods, likely because of funding limitations, changing environmental values, and changing political administrations, which affect the perpetuity of most long-term monitoring programs. Information on urbanisation is particularly scarce in developing countries, which face limited funding, a dearth of qualified and dedicated professionals, and a lack of planning for information systems associated with urban development projects (Madon and Sahay, 2018). The lack of scientific research and ecological resource management programs in developing countries facing rapid urbanisation (Glaeser and Henderson, 2017; Madon and Sahay, 2018; Roy et al., 2016; Fig. 2) underscores the importance of aligning scientific priorities with stakeholder interests in lower-income urban areas, because 1) impoverished people are disproportionately affected by urbanisation, especially at high levels of urbanisation (Stephens et al., 1997; Moore et al., 2003; Liddle, 2017), 2) scientific study in these regions can benefit from a wider global coverage of the impacts of urbanisation and uncover novel disturbance mechanisms (Roy et al., 2016), and 3) increased scientific attention and funding for research in these areas can increase engagement of local communities and enhance environmental management (Allen, 2003; Roy et al., 2016).

4.3. Importance of multiple stressor approaches to understand ecosystem complexity in a changing world

Our study highlights the value of assessing multiple stressors in ecosystem management, an approach that has been strongly advocated (Tockner et al., 2010; Baird et al., 2016; Dafforn et al., 2016; Griffen et al., 2016), but is rarely quantified (Van den Brink et al., 2016). It also reveals the deeper complexity of floodplain ecosystems, and the interactions among pressures, stressors and receptors influencing their response to urbanisation. By taking a multiple stressor approach we can reveal the complex pathways by which individual stressors interact to impact ecosystem functioning, especially in context of anthropogenic environmental change. Adopting an analytical approach that considers multiple stressors is particularly important from a management standpoint, since stressors will interact, impacting ecosystems: additive, non-additive, antagonistic, and synergistic effects have all been documented in freshwater systems (Folt et al., 1999; Coors and De Meester, 2008; Townsend et al., 2008; Ormerod et al., 2010; Piggott et al., 2015; Jackson et al., 2016). Despite this knowledge, most studies fail to embrace this complexity, measuring only a few of the many stressors, often in controlled, unnatural settings such as laboratories or field mesocosms. The oversimplification of these reductionist studies can lead to erroneous conclusions about the impact of stressors on ecological receptors, as interactive, indirect, or unmeasured stressors are often important, especially in highly impacted systems.

Our DPSIR-based multi-stressor methodology moves beyond "faith-based" approaches (sensu Hilbourn, 2006), supporting quantitative, evidence-based decision making, while avoiding the confirmation bias of expert panels (Baird et al., 2015). We deliberately chose floodplains as the focus for this paper, not only because they are among the most threatened of freshwater ecosystems, but because they exemplify the type of system which should be managed explicitly within a multiple-stressor framework. In doing so, we have given examples of neglected or hidden pathways of effect, providing quantitative evidence of the need to consider these pathways not only when observing and assessing the state of floodplains, but also to support their 'wise use' (sensu Finlayson et al., 2005) for science-informed decision-making. Because it allows adaptation to changing conditions and goals, the framework provides a flexible yet structured system to hypothesise and quantify pathways within a highly complex, multiple-node environment where the monitored ecosystem component (usually the receptor) is affected by multiple pressures and stressors occurring across different spatio-temporal scales. As we are faced with unprecedented, accelerating anthropogenic environmental change (Steffen et al., 2015, 2018), it is no longer acceptable to ignore the complexity of multiple-stressor systems. We now have the tools to tackle this complexity, and we must do so, acknowledging that while the resulting outcomes may be imperfect, they are still vital to inform sustainable development practices in the human-dominated ecosystems of the Anthropocene.

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