

Sources and Mitigation of Nationwide Parasiticide Water Pollution

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Abstract

Despite being banned for outdoor agricultural use since 2018, the neonicotinoid insecticide imidacloprid continues to be detected in English freshwaters at potentially harmful concentrations. This study investigates the contribution of domestic pet ectoparasiticides, e.g., treatments for flea, tick, and mite infestations, as putative sources of imidacloprid. For the first time, high frequency daily and year-round measurement of untreated influent into a major wastewater treatment plant (WWTP) in the UK is used to characterise imidacloprid sourced from the highly urbanised catchment it serves. In addition, all available samples from WWTP effluent and Environment Agency (EA) monitoring sites from running waters (e.g., rivers and streams) across England in the years leading up to and after the ban are used to quantify concentrations in receiving waters at a national scale. These data quantify the year-round presence of imidacloprid in untreated and treated wastewater and in waterways across England since the ban for the first time. More than a third of EA samples in rivers and streams are shown to have exceeded *lowest predicted no-effect concentration* (PNEC) for adverse biological effects. Imidacloprid in untreated wastewater, and at many sites downstream of WWTPs, exceeded chronic and acute toxicity thresholds year-round. We demonstrate that if a monitoring site has a WWTP upstream, it is > 3 times more likely to be contaminated with imidacloprid. Bayesian regression modelling shows that concentrations were likely to be higher if a WWTP had a higher pet population in its catchment. These results indicate that parasiticide use has been the primary source of pollution through down-the-drain emissions since the ban. We explored hypothesised mitigation strategies and suggest several options to substantially reduce contamination without need for extensive changes to wastewater treatment practices. These findings highlight the need to revise consumer advice, product marketing, authorisation and monitoring procedures for ectoparasiticial products to ensure that their environmental risks are adequately assessed and managed.

Keywords

Pesticide, Imidacloprid, Wastewater, Banned, England, Rivers, Waterways

The sources and transport pathways of many chemical contaminants in waterways, in particular pesticides, are poorly understood. Establishing contamination of running waters is especially important because of their crucial role in supporting biodiversity as well as human, pet and environmental health, and because they can increase the spatial range and impacts of pollutants across space and time. We report multiple novel approaches and findings that may have major consequences for this 'One Health' issue¹. Here, we establish the first catchment to national-scale case study of the highly toxic neonicotinoid insecticide imidacloprid in UK waterways. We

demonstrate how sources and pathways of the chemical can be identified and used to inform potential mitigation strategies by considering hydrological connections and measured concentrations in the catchment. For the first time we draw together and critically evaluate water industry, government agency and academic data for a highly potent chemical toxin in consistent use within a large proportion of UK households.

Contamination by neonicotinoid insecticides has been reported at toxic concentrations in many waterways globally, and historically this has been largely attributed to agricultural use and subsequent run-off into streams and rivers²⁻⁹. Their potential for adverse effects on the environment led to ban for outdoor agricultural use in such settings in the UK and EU since 2018^{8,10-15}. There was a marked decrease in the use of imidacloprid as a plant protection product (PPP) in the UK, from ~43,000 kg /year in 2005 to negligible usage by 2019 (see Methods and Supporting Information)¹⁶. As part of Retained EU Law, the UK government is committed to maintaining restrictions on neonicotinoid pesticides¹⁷ and at present, there are no authorised PPPs containing imidacloprid⁷. Industry has reported that since the agricultural ban, imidacloprid concentrations in UK surface waters show a “consistent decline”¹⁶. Having market-wide sales, usage, hydrological and wastewater monitoring data, and more robust statistical design, would improve the currently limited assessment of contamination.

Despite the cessation of routine outdoor agricultural use, imidacloprid continues to be detected in UK freshwaters, often at concentrations known to have ecotoxicological effects^{8,18-20} (Fig. 1). Notably, it has been observed at potentially harmful concentrations in urban catchments, where agricultural activity is minimal or absent^{19,20}. A recent risk assessment of 164 contaminants of emerging concern (CECs) in waterways across Greater London from 2019 to 2021 identified imidacloprid as the highest-risk compound, with a risk quotient (RQ) of 19.6, substantially exceeding the *high environmental risk threshold* (RQ = 10), calculated by Egli et al.²¹. Recent investigation of 140 freshwater samples in UK rivers across southern Wales from 2021 to 2023 indicated widespread risk to invertebrate life from imidacloprid⁹. Results from that study are compared to our analysis of ca. 1890 samples across English freshwaters (see Supporting Information).

Increasingly, attention has turned to the potential contribution of domestic pet treatments as the source of year-round and pervasive environmental contamination by imidacloprid^{9,22,23}. They were introduced into veterinary medicinal products (VMPs) in 1997 and appear to have had broadly consistent use in the UK in the last decade (2014 to 2024): this usage has been estimated at 3618±620 kg/year^{16,23}, equivalent to ~1.6±0.3 million doses per year if the mean monthly dose is 190 mg per animal and a dose is administered every month^{16,23} (see Supporting Information). Preston-Allen et al.²⁴ indicate that pet product sales of imidacloprid increased from ~1000 kg/year in 2009, to >3000 kg/year in 2014, and > 2500 kg/year in 2019. Their data and the compilation from Brown et al.¹⁵ suggest broadly consistent combined PPP usage and VMP sales in those pre- and post-ban years.

Despite the strong seasonality in infestation rates²⁵, and against the British Veterinary Association's own guidelines, it has become common industry and veterinary practice to encourage pet owners to administer flea and tick treatments routinely and prophylactically year-round²⁰. Following their application (e.g., via collars and spot-on solutions), the active ingredients in VMPs that contain imidacloprid spread across the skin and fur of treated animals to exert their intended effects. Several pathways for entry into sewage systems, watercourses and water bodies have been identified, including wash-off from bathing, swimming, and rainfall, as well as following excretion in urine and faeces after absorption through the skin^{19,26,27}. Pathways involving shed hair or dander carrying residues into household drains through pet-bathing and owners washing their hands or pet beddings after anti-flea/tick treatment, which can reach waterways via discharge of WWTPs, have also been identified^{9,28,29}. Secondary transfer, such as between treated and

107 untreated pets, further amplifies dispersal, particularly in urban areas with high pet density³⁰. These
108 potential emission routes suggest that VMPs could be a significant source of current imidacloprid
109 contamination in UK freshwaters via their presence in municipal wastewater. Concern around the
110 use of pet parasite medication containing imidacloprid and other pesticides (e.g., fipronil) has led to
111 the recent (2026) UK Parliament House of Lords Pet Parasite Medication Inquiry³¹. A timeline of
112 key events relevant to UK waste and surface water contamination with imidacloprid is shown in Fig
113 S1.

114

115 **Imidacloprid in English municipal wastewater**

116 Urban wastewater contamination has been suggested as a major point source of imidacloprid in
117 UK fresh waters^{32,33}, but our understanding of the sources of most chemicals, including
118 imidacloprid, is limited by the spatio-temporal resolution and extent of previous work. Nevertheless,
119 it is increasingly clear that WWTPs act as conduits, moving contaminants they receive from their
120 catchments into receiving waterbodies via treated effluent^{28,34}. Perkins et al.²⁰, for instance,
121 analysed monitoring data collected between 2016 and 2018 from 20 river sites in England and
122 found that imidacloprid concentrations were significantly higher at sites immediately downstream of
123 wastewater discharge points compared to those located > 2 km from the nearest upstream
124 WWTPs. Those results align with the conclusions of another more focused study³⁵, which identified
125 a sub-catchment containing a major WWTP as the dominant source of imidacloprid along a
126 tributary of the River Thames. We seek to understand whether use of VMPs is reflected in
127 imidacloprid concentrations in wastewater at catchment-to-national scales. To do this, we
128 produced and investigated a new inventory of intensive year-round sampling of influent at a major
129 urban English WWTP and combined it with extensive national-scale environmental monitoring data
130 from surface waters from across England (see Methods and Supporting Information).

131

132 **Transport of imidacloprid in English waterways**

133 The complexity of UK waterways, which often receive inputs from multiple wastewater
134 discharges³⁶, underscore the need to consider entire watersheds and their hydrological
135 connectivity when attempting to relate measured concentrations to sources upstream. However,
136 nationwide spatial hydrological mapping and statistical interrogation has not been attempted to
137 date for imidacloprid nor for most contaminants. Recent studies have pointed out that, in addition
138 to WWTPs, combined sewer overflow (CSO) discharges, misconnected sewers and dog swimming
139 can be important, and in some cases dominant, sources of a number of contaminants, including
140 imidacloprid^{9,37–39}. These studies emphasise the need to develop new methods that can
141 incorporate multiple inputs and the transport of chemicals through fluvial networks when assessing
142 their environmental impacts downstream³⁶. In this study we establish how digitally mapped
143 drainage networks can be used to relate imidacloprid concentrations measured at EA monitoring
144 sites on running freshwater (e.g., rivers and streams) to wastewater sources upstream efficiently
145 with a reproducible and computationally efficient methodology that could also be applied to future
146 monitoring and high-resolution multi-chemical datasets across multiple scales, from, local point
147 sources within a catchment to full national coverage.

148

149 Our approach to assessing the role of down-the-drain wastewater in contamination of waterways
150 downstream is structured to test two hypotheses. The first is that sites that have WWTPs located
151 upstream are more likely to have detectable concentrations of imidacloprid than sites receiving no
152 known treated effluent. The second hypothesis is that imidacloprid concentrations are positively
153 correlated with the size of the pet population in the catchments of upstream WWTPs. We use the
154 *population equivalent* measure at individual WWTPs as a proxy for pet population size in their
155 catchments because, as far as we are aware, the size of pet populations in WWTP catchments is
156 not currently monitored (see Methods). More broadly, we explore how sources of contaminants
157 (e.g., domestic pet parasitocides) can be identified at a catchment-to-national (i.e., England) scale
158 using environmental concentration data collected as part of routine regulatory efforts.

159

Addressing these hypotheses required a detailed, extensive, and exhaustive *de novo* assembly of chemical and hydrological data. Concentration data from 2014 to 2023 were used to establish temporal patterns before and after the ban. We enhanced the foundational chemical data by positioning geolocated EA monitoring sites within new digital hydrological networks generated for each relevant catchment (see Methods). Locating the monitoring sites on the digital drainage network was aided by contextual biomonitoring, hydrological, and chemical information across the UK, which is amongst the most extensive in the world making it an ideal case study^{9,20,22,29,40}. For the first time, watersheds for each EA monitoring site on running water across England were mapped and used to identify upstream WWTPs and CSOs (see Methods and Data Availability Statement). The mapping explicitly accounts for the hydrological connectivity between sample sites and upstream sources. Our approach contrasts with more traditional coarse grid square approaches that do not incorporate the hydrological reality on the ground^{16,19,20,22} and are therefore of limited use in linking contaminants to their sources.

Results

We found year-round contamination above PNEC nationwide in influent and effluent wastewater and at nearly all of the EA monitoring sites, and our population proxy for pet density predicted contamination for concentrations spanning almost four orders of magnitude (Fig. 1-7).

Imidacloprid in English Running Freshwaters

Despite some notable gaps in the data, for instance there is negligible overlap between the CIP3 and EA data in space and time, and there was limited EA sampling during the COVID-19 pandemic in 2020 (Fig. 2a), we still found year-round contamination both before and several years after the ban. Fig. 1 and Tables S1-S2 highlight some of the finer-grained geographical distribution in sampling effort and detection.

Between 2019 and 2023, imidacloprid was detected at least once at 78 of the 110 (71%) monitoring sites (Fig. 1a), and in 49.6% of individual samples. Of the 85 running water sites analysed with mappable upstream catchments, 28 (33%) had no measurable imidacloprid contamination. It was detected frequently at concentrations above the limit of detection (LOD) in the Thames, Anglian, and South East River Basin Districts (RBDs). In contrast, rivers in the Solway Tweed and Northumbria RBDs had very few samples recorded ($n < 50$), and those that exist had low measured concentrations (see Fig. 1b & Tables S1-S2). With 'non-detect' values set to 0 (see Methods and Supporting Information), the overall mean and median imidacloprid concentrations were 0.013 $\mu\text{g/L}$ and 0 $\mu\text{g/L}$, respectively. The mean and median values for all positive detects were 0.026 $\mu\text{g/L}$ and 0.012 $\mu\text{g/L}$, respectively. Fig. 1b compares maximum imidacloprid concentration recorded at each monitoring site with lowest predicted no-effect concentration (PNEC; 0.0068 $\mu\text{g/L}$ ⁴¹), and chronic (0.035 $\mu\text{g/L}$) and acute (0.2 $\mu\text{g/L}$) toxicity thresholds estimated by Morrissey et al.⁴². This toxicity benchmark was exceeded in 35% of all running water samples collected during the post-ban period.

Imidacloprid was detected in 52 to 74% of all river monitoring samples in each year before the ban (2014 to 2018) and in 36 to 59% of the samples in each year after the ban (2019 to 2023; Fig. 2a). Mean annual concentrations were all greater than PNEC and all years, except 2014, had samples that exceeded the 0.035 $\mu\text{g/L}$ chronic toxic threshold (Fig. 2b). Samples with imidacloprid concentrations equal to or exceeding the acute toxic threshold (0.2 $\mu\text{g/L}$) were collected in 2015, 2018, 2019, 2021 and 2022 (Tables S3-S4).

Monthly distributions of the fluvial samples reveal some subtle differences in the seasonal dynamics of imidacloprid concentrations pre- and post-ban (Fig. 3-5). For instance, calculated mean concentrations were above lowest PNEC throughout the year in the pre-ban years, with highest concentrations measured from April to October, during the main agricultural growing season. The post-ban period had mean measured concentrations that exceeded PNEC consistently throughout the year, although there was also some evidence of an uptick between July and October, which could be associated with treatment during the peak infestation period for ectoparasites²⁵.

217 At the most granular scale, Fig. 4 shows all measured imidacloprid concentrations at the individual
218 sites with the highest sampling effort. Even among these most highly resolved temporal data there
219 is considerable variability across seasonal to interannual time scales and no clear post-ban linear
220 declining trend was apparent.

221

222 ***Links between imidacloprid in running fresh water and upstream WWTPs***

223 Eighteen of the 85 mapped watersheds did not have upstream WWTPs (Fig. 6). After the ban in
224 2018, imidacloprid was detected at 44% of the monitoring sites with no upstream WWTPs and at
225 73% of the sites with upstream WWTPs (Table S5). In support of our first hypothesis, logistic
226 regression revealed a significant association between the presence of WWTP and the detection of
227 imidacloprid ($p = 0.0256$). The WWTP exposure Odds Ratio, OR = 3.40 (95% Confidence Interval:
228 1.17–10.3): i.e., imidacloprid detection was > 3 times more likely when a WWTP was present
229 upstream. These results emphasise the importance of non-agricultural sources, particularly in
230 urban areas.

231

232 Twenty-eight sites had no measurable imidacloprid, and of those, 10 had no reported upstream
233 WWTP. The remainder (which had at least one reported upstream WWTP) were only sampled
234 once or twice over five years, which is <1 % of the sampling effort at the two most intensively
235 sampled sites. Among the eight sites that have detected imidacloprid but without an upstream
236 WWTP (Table S5), six have upstream CSOs and two of these are within 1 km. This could
237 potentially explain the detection of imidacloprid at those sites (see Supporting Information for
238 further analysis). In addition, dogs swimming and sewer misconnections may account for some
239 imidacloprid contamination, as recently suggested by Egli et al.²¹ for ponds in England and Hadley
240 et al.⁹ for rivers in southern Wales.

241 In addition to our extensive national-scale spatial data, near continuous temporal daily sampling of
242 influent wastewater from October 2021 to January 2023 at one of the UK's largest WWTPs has
243 established that year-round influx of imidacloprid to sewer networks was remarkably consistent:
244 0.083 ± 0.047 µg/L (Methods & Supporting Information). All measured concentrations in that dataset
245 exceeded the PNEC (0.0068 µg/L) and 92% of them exceeded the chronic toxicity limit of 0.035
246 µg/L. The results emphasise the 'pseudo-persistence' and year-round down-the-drain sources of
247 imidacloprid at ecotoxicologically-relevant concentrations, and only subtle seasonal changes in
248 concentrations, irrespective of peak tick-and-flea infestation periods (see Supporting Information).

249

250 ***Predicating contamination from proxy population data***

251 In support of our second hypothesis, positive relationships were observed between imidacloprid
252 concentration at the EA monitoring sites and the proxy for pet population in upstream watersheds
253 (see Methods; Fig. 7). Fitting all measured concentrations at each of the 84 sites with a mappable
254 upstream catchment yielded a predictive model of the form $i = p^m/c$, where i is estimated
255 imidacloprid concentration and, p , is *population equivalent density* (see Methods and Supporting
256 Information). m and c are coefficients with respective values of 0.56 and 1122 estimated by our
257 Bayesian hurdle model fit to all data. Models fit to the mean or median concentrations at each site,
258 with values less than LOD assumed to be zero, $\frac{1}{2}$ LOD or LOD, yield similar results (see
259 Supporting Information); a rule of thumb is $i \approx p^{1/2}/750$.

260

261

262 **Discussion**

263 Our new approach is the first to combine multiple lines of chemical, hydrological, and geospatial
264 evidence from industry, government and academia to characterise contamination of a widespread
265 toxin from catchment to national scales. These data were then modelled with a novel application of
266 Bayesian statistics to predict contamination spanning almost four orders of magnitude from a proxy
267 of pet population size across England. Across a large proportion of sites and sampling occasions
268 contamination was well above PNEC and there was a strong urban signature clearly linked to
269 WWTPs. The environmental half-life of imidacloprid, its widespread contamination years after the

270 agricultural ban, particularly in urban areas, and the strong predictive relationship we have
271 uncovered in combination suggest pet parasiticides as the major source, rather than legacy
272 agricultural pollution.

273 The findings support the growing consensus that upstream wastewater has been a major source of
274 year-round imidacloprid contamination of English waterways since its usage in outdoor agricultural
275 settings was severely curtailed from 2016 and banned outright from 2018^{9,21,43}. Analysis of samples
276 from untreated and treated wastewater, and from running freshwater monitoring sites shows that
277 imidacloprid concentrations were above detection limits year-round across England, even many
278 years after the ban. We note that imidacloprid is reported to have a half-life of a few days to
279 months in soils and potentially orders of magnitude less in water exposed to UV light⁴². More than
280 a third of the individual samples at the freshwater monitoring sites had concentrations that
281 exceeded PNEC, often by a considerable margin. We currently do not have both deep and broad
282 sampling of contamination nationwide. Intensively sampled influent wastewater from one of the
283 UK's largest urban WWTPs shows pseudo-persistent year-round contamination. These
284 complimentary data add depth to the spatial overview from EA data that is pointing to broad, year-
285 round, contamination. Although a single dose is a tiny amount in absolute terms, these chemicals
286 exceed PNEC in less than 0.01 µg/L, which when multiplied by the millions of cats and dogs in the
287 UK, many of which are receiving multiple doses per year, clearly has the potential to scale into a
288 national and particularly urban contamination issue.

289 Imidacloprid contamination of nine rivers in Wales has recently been related to WWTP outfalls and
290 sewer misconnections⁹. That study was based on 140 samples and contamination reached up to
291 0.076 µg/L. Our collation of EA data, which includes 1890 samples, is compared to the Welsh data
292 in Supporting Information (Fig S6). In addition, we incorporate a further 423 daily composite
293 samples from WWTP influent and CIP data into our study: taken together the data across England
294 show contamination is far higher in terms of both medians and peaks (>0.2 µg/L). The obvious
295 year-round consistency of imidacloprid occurrence could be explained by year-round prophylactic
296 treatment of pets with VMPs, combined with some additional remedial treatment in summer
297 months.

298 We found that measured EA concentrations can be related to sources upstream, and that
299 considering drainage networks brings the picture into much sharper focus. Upstream WWTPs were
300 associated with elevated downstream imidacloprid concentrations, supporting our hypothesis that
301 contamination concentrations are positively correlated with the pet population. Here, we assumed
302 a positive correlation between *population equivalent density* values (see Methods) and pet
303 ownership, and by extension, between pet population and imidacloprid-containing veterinary
304 medicine usage. The use of other proxies, for instance, the number of households recorded in UK
305 Census 2021 data, or mapped pet populations and treatment, e.g., from prescribing or VMP sales
306 data, could also be explored in future work. While the use of proxies can provide a practical means
307 to estimate VMP inputs, more direct information about pet ownership rates and treatment practices
308 across regions would be helpful for further and deeper analyses (e.g., with citizen science and
309 industry engagement⁴⁴) but such data are not currently available (see Supporting Information).

310 It is also important to note that linking imidacloprid concentrations to only WWTP effluent could
311 underestimate additional contributions from other sources, such as CSOs, sewer misconnections
312 and direct disposal throughout the catchment^{9,35}. Correlating CSO discharge event logs with EA
313 site monitoring timestamps could help explain episodic imidacloprid spikes observed in receiving
314 waters. Further, an alternative to relying on inference from WWTP *population equivalent* estimates
315 or other proxies, actual measured imidacloprid concentrations in wastewater effluents, if they were
316 to become available in the future, would provide more direct evidence of chemical release.
317 However, data are currently unavailable at the scales of both spatial and temporal coverage and
318 resolution required. The most relevant data available is the UK Water Industry Research's

Chemical Investigation Programme (CIP), which provides imidacloprid effluent load data for a selection of WWTPs in the post-ban period⁴⁵. Unfortunately, all but one monitored WWTP fall outside the 85 mappable watersheds upstream of EA monitoring sites on running water, preventing deeper formal analysis (see Supporting Information for an extended analysis of CIP and downstream EA data).²¹

Based on our new insights, we suggest several actions for improving the current evidence base, the regulatory environment and usage of imidacloprid to reduce potential contamination risks. First, regular water quality monitoring will provide a step-change in capabilities to identify and quantify imidacloprid in waterways. We also recommend higher-frequency sampling across seasons, at least within a subset of more intensively surveyed model systems, combined with more extensive spatial coverage across systems. Doing so will provide information that can then be used to develop forward and inverse models to precisely apportion imidacloprid to sources and predict future contamination risks, including under different management or regulatory scenarios³⁵. The risk-based approaches currently taken by regulatory agencies differ from those based on public perception of local waterways. A corollary is that incorporating citizen science may provide significant added value to the mapping of imidacloprid concentrations. Two recent large-scale citizen-science based fully quantitative water surveys perhaps indicate a way forward to mapping imidacloprid concentrations at scale. These surveys, undertaken in 2024 and 2025, coordinated by Earthwatch and our teams highlighted the risk of neonicotinoids^{46,47}, with imidacloprid having the highest risk of all the *chemicals of emerging concern* tested, including several other pesticides monitored. Evaluating the usefulness of citizen science data as an adjunct, or alternative, to regulatory chemical monitoring at scale would be a useful next step.

Potential mitigation scenarios and future solutions

We assess how hypothetical mitigation strategies could impact imidacloprid concentrations in rivers throughout the year and at critical times during the life cycle of many freshwater organisms exposed to this parasiticide. First, switching from year-round monthly dosing to a single dose in a single month during the peak infestation period could in theory reduce inputs by about 12-fold. We estimate that if the samples collected from 2019 to 2024 at the 85 EA monitoring sites with mappable upstream catchments had only exceeded PNEC in one of the peak infestation months, the total number of samples exceeding PNEC year-round would have been reduced by 85 to 91%. See Methods and Supporting Information for extended results including for LOD, chronic and acute limits. Second, moving to a model in which only cats, which have been reported to be ca. 3 times more likely to be seen by vets with a flea infestation than dogs²⁵, are treated prophylactically during the peak-infestation months of July to October is estimated to reduce imidacloprid load to 21% of its current value (see Supporting Information). Moving to a model in which parasiticides are only available via prescription based on diagnosed clinical need (e.g. an observed infestation) could reduce imidacloprid load further still and push even more sites below the critical lowest PNEC threshold. This approach would mirror a proposed change in practice in Switzerland currently under consultation, which could provide a useful future Before-After-Control-Impact case study⁴⁸.

The Supporting Information document describes the methodologies used to assess hypothetical mitigation strategies in greater detail and their caveats. We emphasise here that development of effective mitigation strategies would massively benefit from improvements to the public reporting of dosing and the assessment of the prevalence of fleas and ticks in pet populations. Linking such developments to improvements to the spatial and temporal coverage of sampling of imidacloprid in the environment could dramatically improve understanding of its distribution year-round and its impact on the environment. Nonetheless, it is clear at this stage that dosing only in peak infestation periods and based on clinically diagnosed need could provide temporal refugia for riverine insects in particular, as this is when generations overlap and recruitment peaks, potentially providing greater population resilience than might be the case for reductions in use during winter months when entire populations of many sensitive taxa are present solely in the aquatic larval phase.

The framework we developed here could be applied more broadly to monitor chemicals of concern in general. This includes other parasiticides that are being used at scale in the UK and elsewhere (e.g. fipronil), those that have a relatively small but growing proportion of market value (e.g.

fluralaner), and those that will form the next generation. A first important step to improving our understanding of these chemicals in the environment is to develop a more robust, coherent and consistent monitoring scheme that can use more diverse sources, maps onto the hydrological reality on the group and is scalable, as we have done here. Beyond addressing these current limitations around data, monitoring and modelling, and our specific focus on imidacloprid, there are wider systemic issues with the environmental assessment and regulation of pet parasiticides in general that need to be addressed. Currently, pet parasiticides do not require a Phase II risk assessment, based on the assumption of minimal environmental exposure⁴⁹: our data show that this is clearly not the case and so we advocate for more robust and realistic field testing in particular. This approach could then help guide more ethical and less damaging usage whilst avoiding “regrettable substitution” from simply replacing one contaminant with another that may subsequently turn out to be even more toxic.

Conclusions

Identifying sources and drivers of pesticides is crucial for establishing effective strategies to reduce contamination of waterways. We demonstrate how sources of the neonicotinoid ectoparasiticide imidacloprid, prevalent in English waterways despite being banned for outdoor agricultural use since 2018, can be identified. We make use of 1,890 samples collected from 110 running freshwater sites across England by the UK’s Environment Agency between 2019 and 2023 to demonstrate that the presence of upstream wastewater treatment plants increased the likelihood of detecting imidacloprid more than three-fold. The results indicate that treated effluent is a significant source of imidacloprid. The positive correlation between WWTP-derived *population equivalent density* and mean imidacloprid concentrations supports the hypothesis that domestic pet ectoparasiticides are the primary source of this contaminant through down-the-drain emissions. With current pet populations and preventive treatment frequencies, current regulatory assumptions of negligible environmental exposure from companion animal treatments appear to no longer be reliable. Future research is needed to characterise the spatial and temporal dynamics of imidacloprid occurrence and distribution in freshwater and the source-to-sink trajectories of ectoparasiticides in waterways. Doing so would massively benefit from enhanced water quality monitoring, including routine sampling across seasons and broader spatial coverage. Such data would provide information to support the development of methodologies that can accurately and precisely apportion contaminants to their sources. Addressing these issues would provide the toolkit needed to establish sources of contaminants, including but not restricted to pesticides, and their fate in waterways with global applicability.

Methods

Imidacloprid concentrations in influent wastewater

We measured imidacloprid concentrations in 423 24-hour composite samples of influent wastewater collected at a major UK WWTP from October 2021 to January 2023. The WWTP served a population equivalent (*p.e.*) of >1 million. The assembled data represents the most temporally resolved and continuous measurement of imidacloprid in influent wastewater from a single, heavily urbanised catchment globally to date. An extended explanation of the sampling and analytical methods is given in Supporting Information.

Imidacloprid concentrations at English running water sites

A subset of the EA’s semi-quantitative, single-point calibration, water quality screening data, generated using liquid chromatography mass spectrometry (LC-MS), was used to investigate imidacloprid concentrations (see Code and Data Availability Statement for details). Positive detections are defined as measured concentrations equal to or greater than the reported lower limit of detection (LOD = 0.001 µg/L for imidacloprid). Concentrations less than LOD (‘non-detects’) are often not reported by the EA. Following Lapworth et al.⁵⁰, we developed an inventory of ‘non-detects’, which assumes that samples in the EA dataset with no reported imidacloprid measurements had concentrations below LOD (as per reported EA protocols; see Environment

432 Agency⁵¹.

433 The resultant chemical dataset contains 1890 samples collected between 2019 to 2023 at 110
434 sites located on fresh running water ways in England⁵¹ (Fig. 1). Numbers of samples available for
435 each site varied considerably, from 1 to >170 over five years. Eight sites were monitored
436 intensively, each with more than 140 individual samples collected. In contrast, 56 sites (51%) were
437 sampled only once. These data complement 140 samples from 62 sites in southern Wales
438 investigated by Hadley et al.⁹ (see Supporting Information).

439
440 A variety of approaches exist to assess the impact of censored data (e.g., data with concentrations
441 below LOD) on calculated statistical moments⁵² (e.g., mean and variance). Given that the
442 distribution of imidacloprid concentrations below (and above) the LOD is poorly understood, we
443 test the impact of three simple substitution- and one removal-based approaches, assessing the
444 impact of assuming extreme values and widely used assumptions. We note that Perkins et al.²⁰
445 show that calculated statistics (e.g. p-values) tend to be insensitive to the precise value of LOD
446 chosen. First, following Perkins et al.²⁰, we substituted 'non-detect' entries in the database of
447 samples for 1/2 LOD. Second, we assessed the impact on results if non-detect values are set to
448 the LOD, and third, if they are set to zero. Finally, we assessed the impact of removing non-detect
449 values from the dataset. The resulting estimates and statistical inferences from this study are
450 included in the Supporting Information. Given the similarity of results for the substitution-based
451 approaches and lack of a rationale for removing samples from the dataset, our subsequent
452 analysis sets 'non-detect' entries to zero. We acknowledge that other statistical approaches could
453 be fruitful, especially as more becomes known about the distribution of imidacloprid concentrations
454 in surface waters.

455 456 **Mapping Watersheds and WWTPs**

457 Watersheds upstream of river sites sampled post-ban were mapped using standard flow routing
458 techniques implemented with the hydrology toolset in ArcGIS Pro 3.2 as follows⁵³. First, sinks (i.e.
459 cells with no surrounding lower cells) in the UKCEH Integrated Hydrological Digital Terrain Model
460 (IHDTM), which has a cell size of 50 × 50 m, were filled using the iterative Spatial Analyst fill tool⁵⁴.
461 Flow directions were then calculated using the D8 algorithm⁵⁵. Flow accumulation downstream was
462 then computed, and the results were used to establish a stream raster by assuming overland flow
463 for cells. The raster was generated for cells with upstream area > 4 km², which is a conservative
464 estimate for the initiation of overland flow for UK rivers. Stream Order was assigned using the
465 Strahler method⁵⁶. Flow distances were calculated from sample sites to the boundary of their
466 upstream catchment. Finally, monitoring sites were placed on the vectorised stream network using
467 automated snapping tools in ArcGIS; additional manual adjustment was made to ensure that all
468 sites were associated with the correct drainage pathway following visual inspection and use of
469 contextual information (EA Water Quality Archive, aerial imagery and googlestreetview; see
470 Supporting Information for details and examples). Watershed boundaries were then mapped using
471 the ArcGIS watershed tool and validated using the Catchment Data Explorer⁵⁷ and IHDTM
472 cumulative catchment area data⁵⁴. Twenty-five running water sites had equivocal positions in the
473 digital network even after the high-resolution snapping because flow routing was locally complex,
474 e.g., due to low-gradient industrial landscaping or highly braided marshland—those sites were
475 therefore excluded from further analyses. In total, watersheds for 85 river sites were produced,
476 with areas of 1.6 to 9,958 km², and a median of 386 km² (Fig. 6).

477 English WWTP locations upstream of sample sites and information about the size of the population
478 served by the plants were obtained from the Department for Environment, Food and Rural Affairs⁵⁸
479 (Defra; see Data & Code Availability statement). Only plants serving a *p.e.*, greater than 2000 were
480 reported, in accordance with the Urban Waste Water Treatment (England and Wales) Regulations
481 1994. These data were filtered to include only WWTPs that reportedly discharge into freshwater
482 bodies, the rest of the dataset is excluded from further analysis in this study. Reported discharge
483 locations for WWTPs were associated with the respective digital watersheds.

Pet population and land use upstream of monitoring sites

As far as we are aware, direct information (e.g., census data) about residential pet populations in the catchments served by UK WWTPs is not publicly available. In this study *p.e.* data provided by Defra is used as a proxy for pet populations in WWTP catchments. This approach has the benefit of making use of relevant data provided by the regulatory body for WWTPs across England. Following guidance from the EA, *p.e.* served by a WWTP was defined as the *aggGenerated* values provided by Defra in their Wastewater Treatment in England dataset. This value is the number of 'population equivalent, *p.e.*' (= *P*) values as defined in the European Union Urban Waste Water Treatment Directive 91/271/EEC Article 6.2⁵⁸, which states "1 population equivalent" or '(1 *p.e.*)' means the organic biodegradable load per day, having a five-day biochemical oxygen demand (BOD5) of 60 g of oxygen per day". It is "calculated on the basis of the maximum average weekly load entering the treatment plant during the year, excluding unusual situations such as those due to heavy rain", and includes domestic wastewater or a mixture of domestic wastewater with industrial wastewater and/or run-off rain water.

Population equivalent density, *D*, for each monitoring site was calculated using the *p.e.* values from Defra, and calculated watershed area, *A*,

$$D = \frac{\sum_{i=1}^n P_i}{A}$$

where *n* is the number of WWTPs in the watershed upstream of the monitoring site, and *P_i* is the *p.e.* value for each of the upstream WWTPs. Some Welsh WWTPs discharge into rivers that flow into England. Consequently, locations of Welsh Water (Dwr Cymru) WWTPs were obtained from Southwest Environmental Limited⁵⁹, however exact agglomeration loads were not available and therefore could not be incorporated into our calculations. Mean and median human population densities served by WWTPs in each catchment were 423 and 104 persons/km², respectively (N = 84). Site 50022, which has the majority of its watershed in Wales, was excluded in the population density analysis due to the lack of Welsh WWTP *population served* data. See Hanley et al.⁸ for study of Welsh rivers and a comparison of their measurements and the data used in this study in Supporting Information. The highest WWTP-serving population density was in the watershed of monitoring site 59010550, on River Tame near Birmingham. It is located 1.8 km downstream of Minworth STW, one of the largest sewage treatment plants in the UK serving ca. 1.75 million people.

Land use information (10-m pixel resolution) was obtained from the Centre for Ecology and Hydrology⁶⁰ and reclassified into six aggregate categories: arable, built-up and gardens, grassland, mountain-heath-bog, water and woodland. The locations of mapped CSOs were obtained from Stream⁶¹ and Welsh Water⁵⁹ (Fig. 6).

Likelihood of finding imidacloprid downstream of WWTP

To establish the likelihood of detecting imidacloprid in waterways that have upstream WWTPs, EA monitoring sites were first grouped into those with and without upstream WWTPs. The percentage of detections of imidacloprid and the corresponding 95% Wilson confidence intervals (CIs) were calculated for each group. Logistic regression was used to determine whether the presence of upstream WWTPs significantly affects the likelihood of detecting imidacloprid at monitoring sites. The odds ratio (OR) of detections occurring in the presence of upstream WWTPs vs. them not occurring was then calculated to quantify the strength and direction of this association (Table S5).

Annual and monthly changes in imidacloprid concentrations

Generalised additive models (GAMs) were used to generate smoothly varying functions to represent annual and monthly changes in imidacloprid concentrations (see Fig. 2–3). Locally estimated scatterplot smoothing (LOESS) was used to smooth pre- and post-ban monthly concentrations, and the standard error was calculated (see Fig. 5).

Correlations between imidacloprid concentrations and pet populations upstream

To investigate the role treatment of companion animals might play in determining imidacloprid concentrations in English waterways since 2019, estimates of *population equivalent density* upstream were compared to mean imidacloprid concentrations in log-log space. All analyses were conducted in R v4.2.1⁶². We modelled imidacloprid concentrations across the samples in two ways: (1) a simpler analysis working with averages of concentrations across sites, and (2) a more detailed analysis that accounts for repeated measurements through time. These models were conducted using *brms*⁶¹ (see Supporting Information). We elected to use a Bayesian framework in order to propagate uncertainties in sampling and observed variation through into our estimates of imidacloprid concentrations, and so best inform policy as to the confidence and magnitude of the observed effects. Model coefficients are shown in the main text across both model formulations to emphasise the robustness of our findings to model formulation and structure.

(1) Working with mean imidacloprid concentrations across all measurements at each site, and $\log_{10}$ -transformed population equivalents, we fit a Bayesian hurdle model. This hurdle model allows us to separately estimate the probability that a site's imidacloprid concentration is above the detectable threshold (through a logistic, logit-link) and then, given a site has a detectable level of imidacloprid, the measured concentration (assuming a lognormal distribution of the chemical, as we observed here). Because the *population equivalent density* data were log-normally distributed but with some sites where *population equivalent density* = 0, rather than excluding those sites or adding a constant to all the sites, we chose to $\log_{10}$ -transform all population values greater than 0 and add a dummy-variable with 0s where *population equivalent density* was 0, and 1 where it is >0 (inspired by Little⁶⁴). These models were fit with 4 chains each with 2000 iterations (1000 were discarded as burn-in); all $\hat{r} = 1.00$ and ESS > 2000 with no obvious signs of non-convergence or divergence warnings. Model results were qualitatively identical to Spearman-rank correlations and models of sample medians. Full model outputs are provided in Supporting Information.

(2) The sampling of each site varied through time, with some sites being well-sampled (two sites were sampled 168 times) but the majority visited only once (40 visited once, 12 twice, of 84 sites). Those sites that were well-sampled were visited at varying, uneven intervals. To account for this non-even sampling through time, we extended our hurdle model to account for both variation in sampling across sites, as well as the temporal sequence of sampling. To ensure our results were conservative, we accounted for this variation in three ways: (a) the modelling of imidacloprid detection (through a hierarchically drawn term for each site in the logit-component of the model), (b) the modelling of imidacloprid concentration (through another hierarchically drawn term for each site in the lognormal-component of the model), and (c) the overall error structure of the model (adding a conditional autoregressive term estimated for each site). This approach was chosen because it is so conservative with respect to our study question: it provides three separate null options for the capturing of variance across sites other than *population effective density* units. The use of Bayesian hierarchical modelling is central to this approach, as they permit robust estimation of such models [reviewed in Gelman et al.⁶⁵]. These models were fit with 4 chains each with 4000 iterations (2000 discarded as burn-in); all $\hat{r} = 1.00$ and ESS > 3000 with no obvious signs of non-convergence or divergence warnings. Model results were qualitatively identical to Spearman-rank correlations and models of sample medians. Full model outputs are provided in Supporting Information.

Chemical concentrations are most often presented, and most readily interpreted, when presented in $\log_{10}$ units, but lognormal (hurdle) models are most readily fit using the natural logarithm (to the base e). We therefore fit models #1 and #2 above using a natural logarithm (ln) for their lognormal components, and converted our coefficients to $\log_{10}$ by dividing by $\ln(10)$, since, for any variable 'x': $\ln(x) = \ln(10) \times \log_{10}(x)$.

Quantifying impact of hypothesised mitigation strategies

We quantify the reduction in the number of water samples that exceeded toxic limits and total imidacloprid load from hypothesised mitigation strategies that centre on reducing dosing of cats and dogs. The Supporting Information document describes the methodologies and assumptions used to calculate the reductions and associated caveats, which mostly arise because of the lack of data available to quantify current dosing rates and infestation prevalence.

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Data and Code Availability

Environment Agency water quality monitoring data GC-MS and LC-MS semi-quantitative screening data can be assessed at <https://www.data.gov.uk/dataset/0c63b33e-0e34-45bb-a779-16a8c3a4b3f7/water-quality-monito>. The NORMAN ecotoxicology database can be assessed at <https://www.norman-network.com/nds/ecotox/lowestPnecslIndex.php>. Wastewater treatment in England data can be assessed at <https://www.data.gov.uk/dataset/d7e2c57b-110a-462b-97a0-9833e7d26cc2/wastewater-treatment-in-england>. Welsh Water wastewater location data was extracted from https://www.southwest-environmental.co.uk/further%20info/in%20depth/sewage_works_maps/welsh_water_sewage_treatment_works_location_maps.html. IHDTM elevation and IHDTM cumulative catchment area data can be assessed at <https://www.ceh.ac.uk/data/integrated-hydrological-digital-terrain-model>. Countries boundaries data is available from <https://geoportal.statistics.gov.uk/maps/82f271c23d514958a352001629fa4075>. Major towns and cities boundaries data is available from <https://geoportal.statistics.gov.uk/maps/980da620a0264647bd679642f96b42c1>. Catchment boundary data was extracted from Environment Agency's catchment data explorer at <https://environment.data.gov.uk/catchment-planning>. Land cover map data is available from <https://catalogue.ceh.ac.uk/documents/017313c6-954b-4343-8784-3d61aa6e44da>. Combined sewer overflow data for major UK water companies can be accessed at <https://corporate.dwrcymru.com/en/community/environment/event-duration-mon> and <https://www.streamwaterdata.co.uk/pages/storm-overflows-data>. UKWIR Chemical Monitoring Programme data is available at <https://ukwir.org/water-chemicals-investigation-programme>.

The watershed delineation workflow using ArcGIS Pro and the R code used for data processing and data analysis in this study are both documented and available from <https://github.com/JiayiLiu354/Imidacloprid>.

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Figures

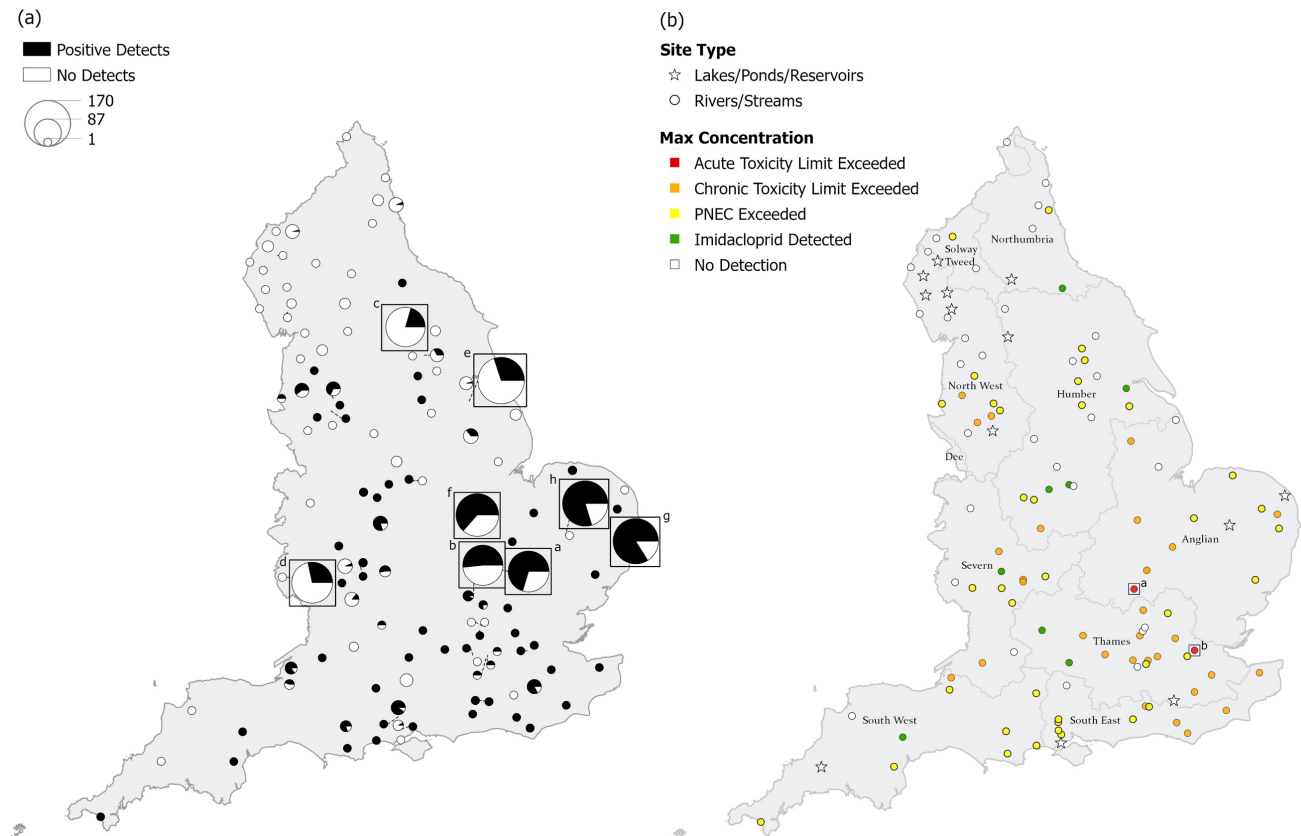


Fig. 1: Imidacloprid in English waterways. (a) Detections of imidacloprid in English waterways after the ban for outdoor agricultural use: 2019 to 2023. Pie charts show number of samples in which imidacloprid was detected or not detected at Environment Agency (EA) waterway monitoring sites. Time series for annotated sites are shown in Fig. 4: a = 12M08, b = 19M07, c = 49100488, d = 50050, e = ANCN5, f = NENE550W, g = WAV120, h = WEN250, where site codes are those used by the EA. (b) Maximum recorded imidacloprid concentration at EA monitoring sites post-ban: 2019 to 2023. Limit of detection (LOD) = 0.001 µg/L; PNEC = 0.0068 µg/L; chronic and acute toxicity limits are 0.035 µg/L and 0.2 µg/L, respectively. Annotated polygons = River Basin Districts. Circles labeled a and b are the monitoring sites (16M08 and MD02) with the highest maximum concentrations (see Supporting Information for details on standing waters, which are not part of this study).

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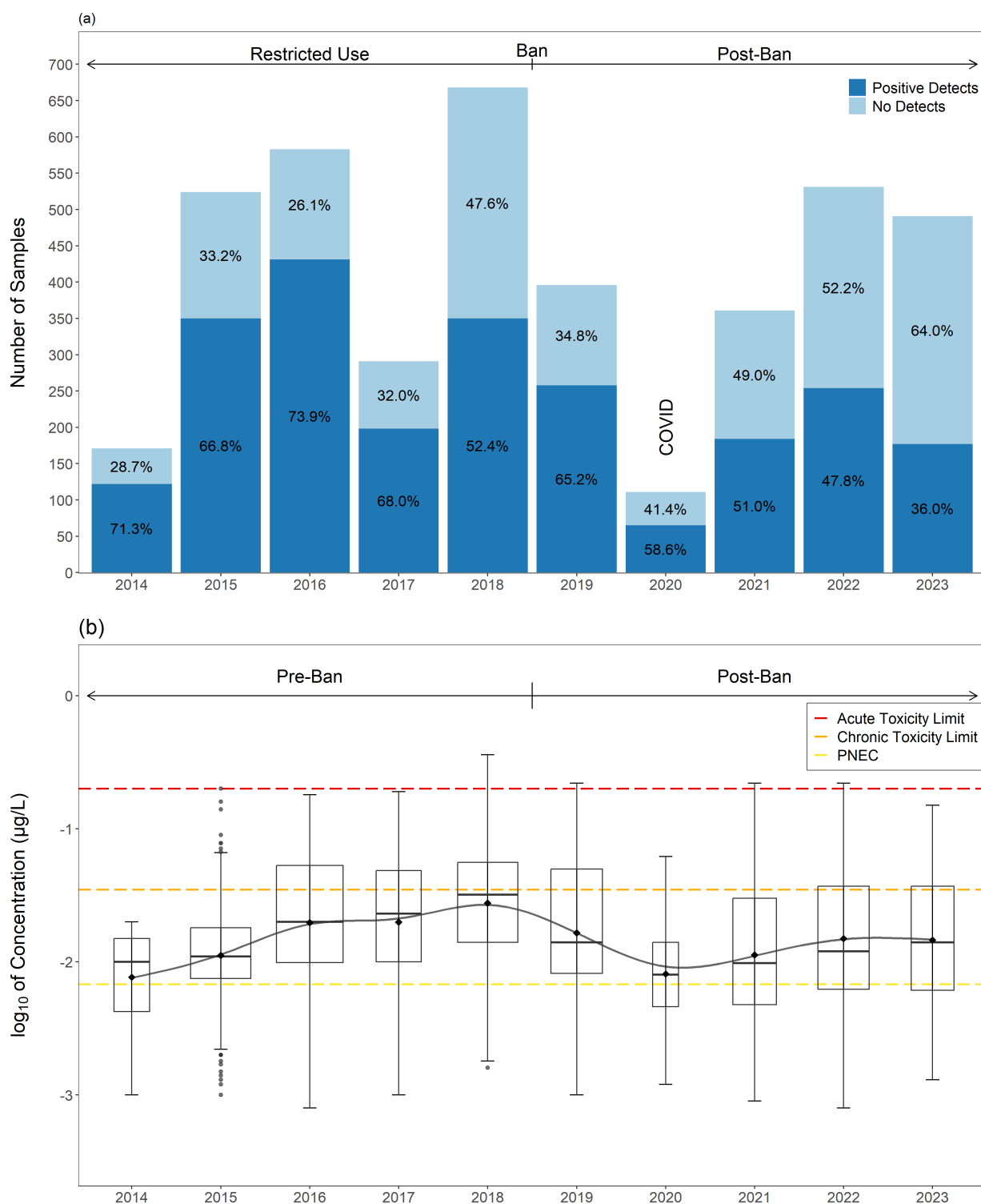


Fig. 2: Annual measurements of imidacloprid concentrations. (a) Count of Environment Agency samples at all sites shown in Fig. 1a from 2014 to 2023, separated by detections and no detections of imidacloprid. Note pre- and post-ban periods. (b) Log-transformed imidacloprid concentrations above LOD from 2014 to 2023. Horizontal black lines = median values; boxes = inter-quartile range (IQR); whiskers = $1.5 \times \text{IQR}$; circles = mean values (within IQR) and outliers. Non-detects were removed prior to log-transformation. Yellow dashed line = PNEC ($0.0068 \mu\text{g/L}$). Chronic and acute toxicity limits = $0.035 \mu\text{g/L}$ and $0.2 \mu\text{g/L}$, respectively.

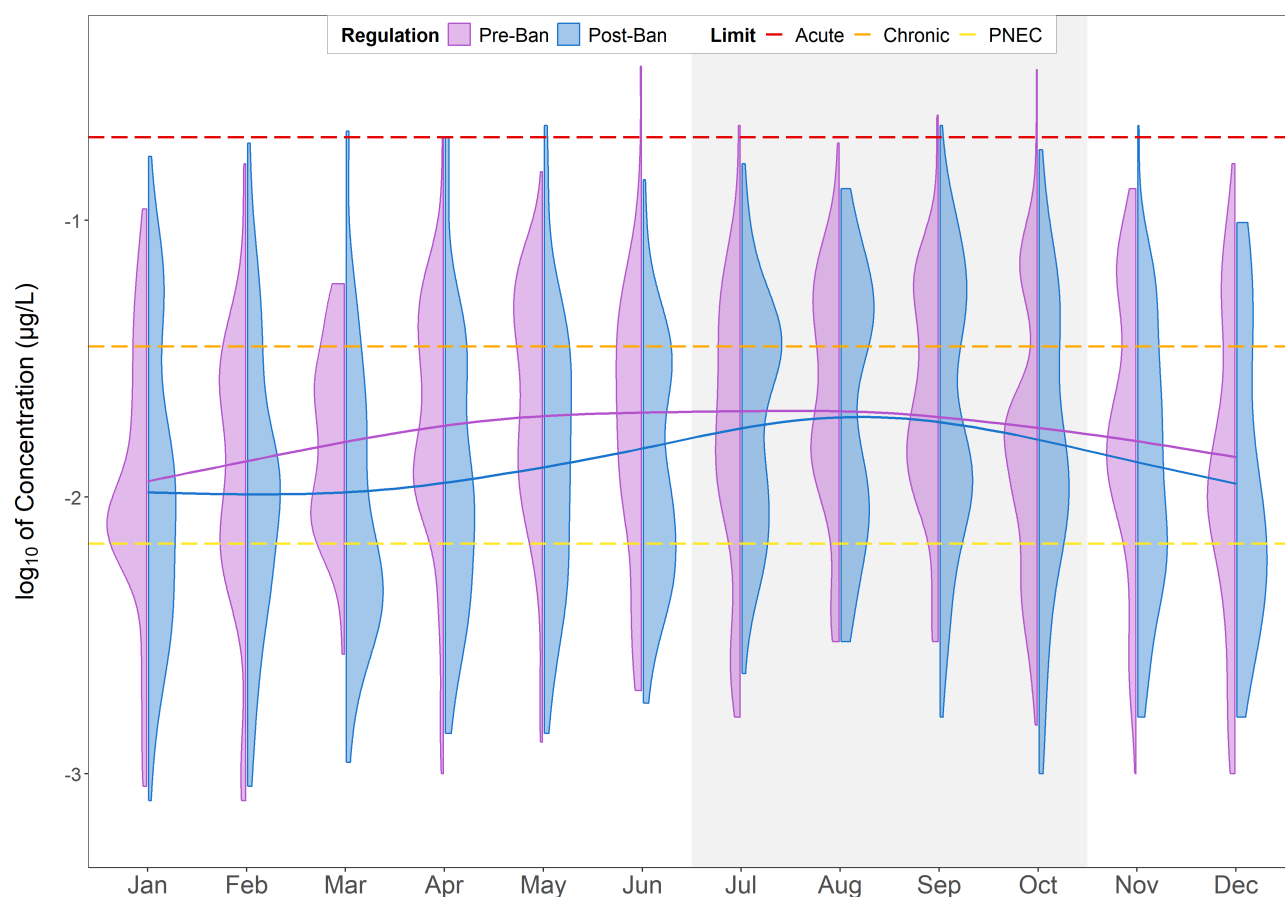


Fig. 3: Distributions of monthly log-transformed concentrations above LOD, comparing pre- (purple) and post- (blue) ban samples. N = number of samples per month; note: samples with concentrations below LOD (0.001 µg/L) were removed prior to log-transformation. Yellow dashed line = PNEC (0.0068 µg/L); chronic and acute toxicity limits = 0.035 µg/L and 0.2 µg/L, respectively. Shaded background = high flea infestation period. Solid purple and blue curves = generalized additive models (GAM) for pre- and post-ban samples.

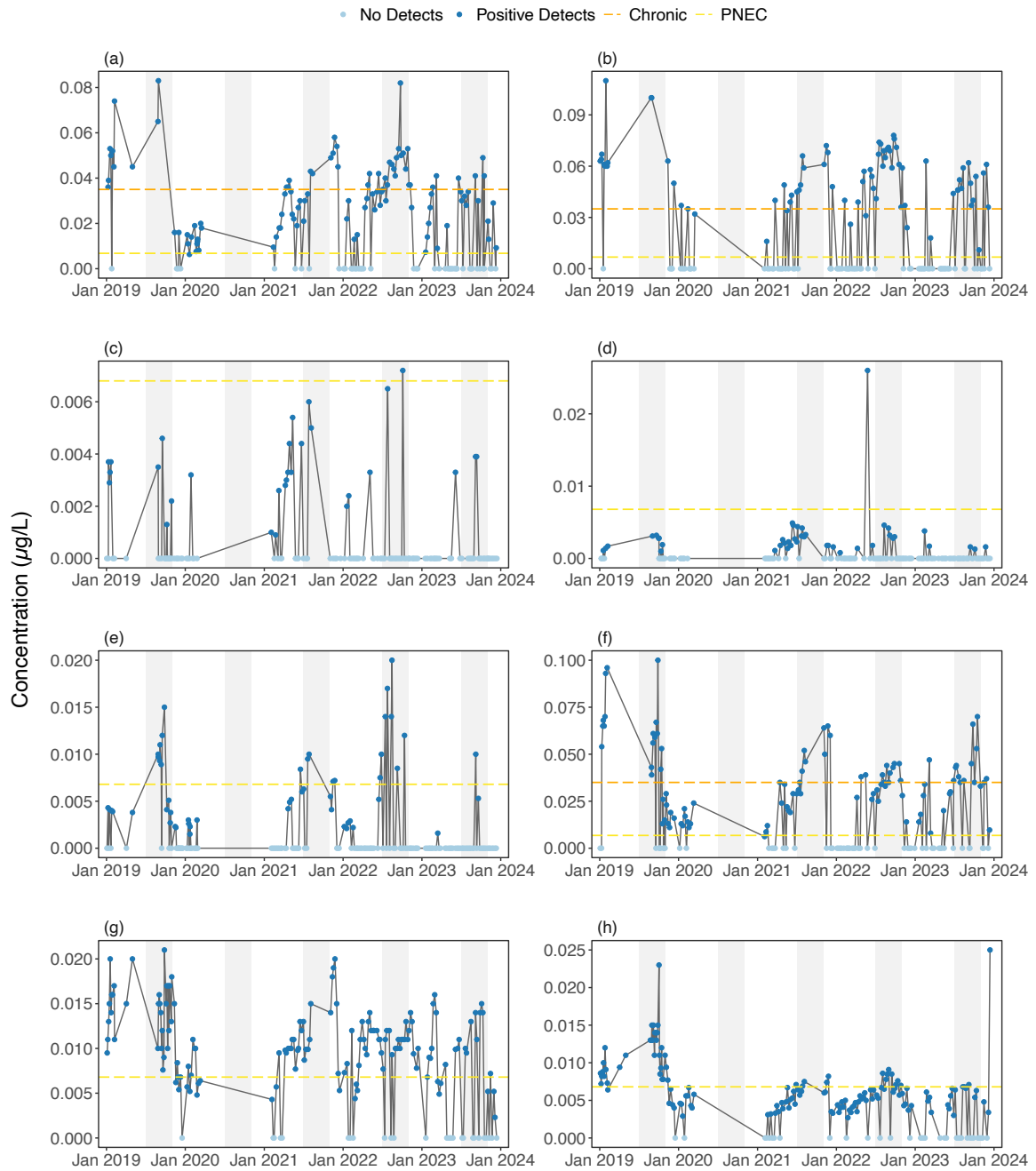


Fig 4: Time series of imidacloprid concentrations at the eight most intensively monitored sites from 2019 to 2023. Dark/light blue = positive/no (below LOD) detections of imidacloprid. Yellow and orange dashed lines = PNEC and chronic toxicity limit, respectively. Shaded background = high flea infestation period. See Fig. 1a for sample locations: a = 12M08, b = 19M07, c = 49100488, d = 50050, e = ANCN5, f = NENE550W, g = WAV120, h = WEN250.

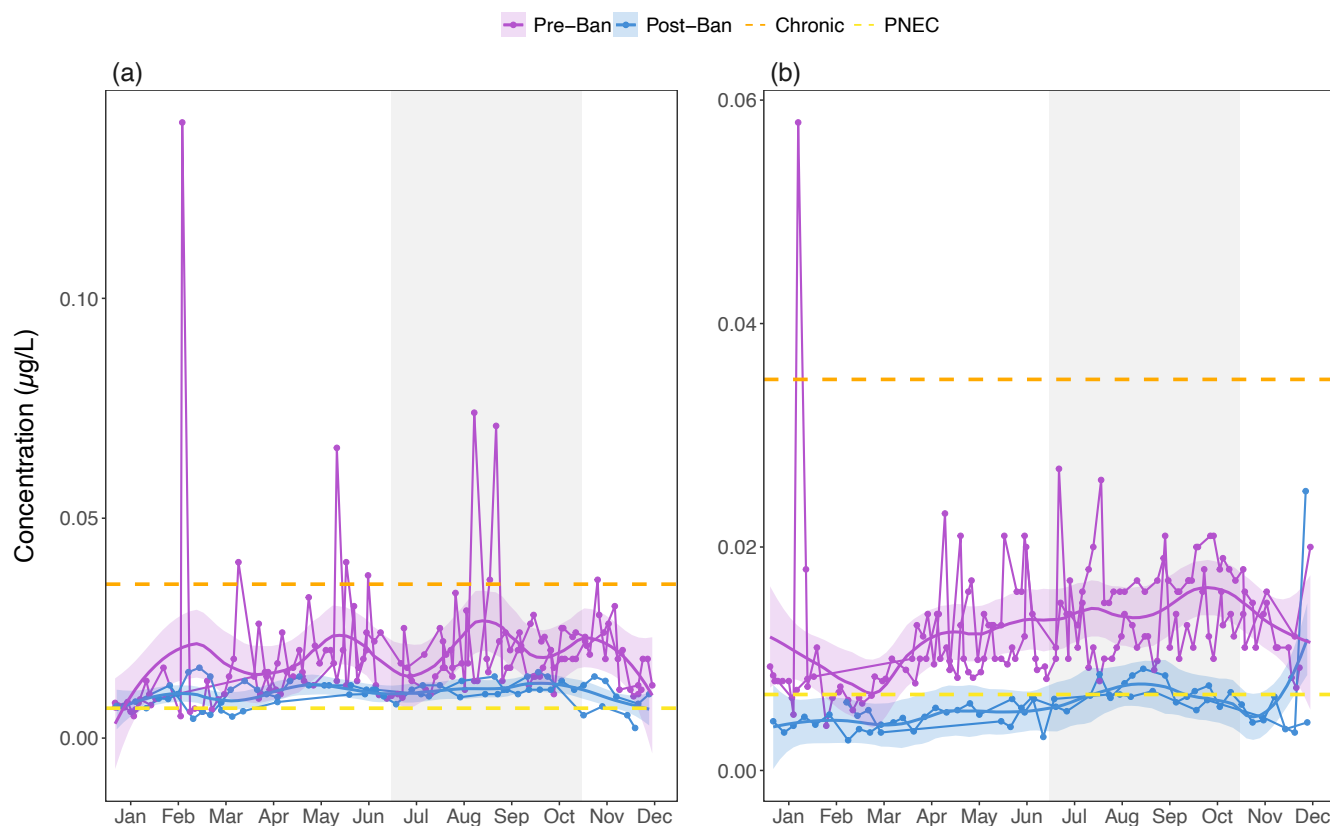


Fig. 5: Comparison of imidacloprid concentrations pre- and post-ban in two agriculturally dominated catchments. See Fig. 1a for site locations: a = WAV120, b = WEN250. Circles and thin lines: purple = pre-ban samples (2015 & 2016); blue = post-ban samples (2022 & 2023). Thick curves and transparent polygons = LOESS of the monthly data and the standard error (span 0.3 and degree 2). Note only concentrations above the limit of detection are included in this figure. Yellow and orange dashed lines = PNEC and chronic toxicity limit, respectively. Shaded background = high flea infestation.

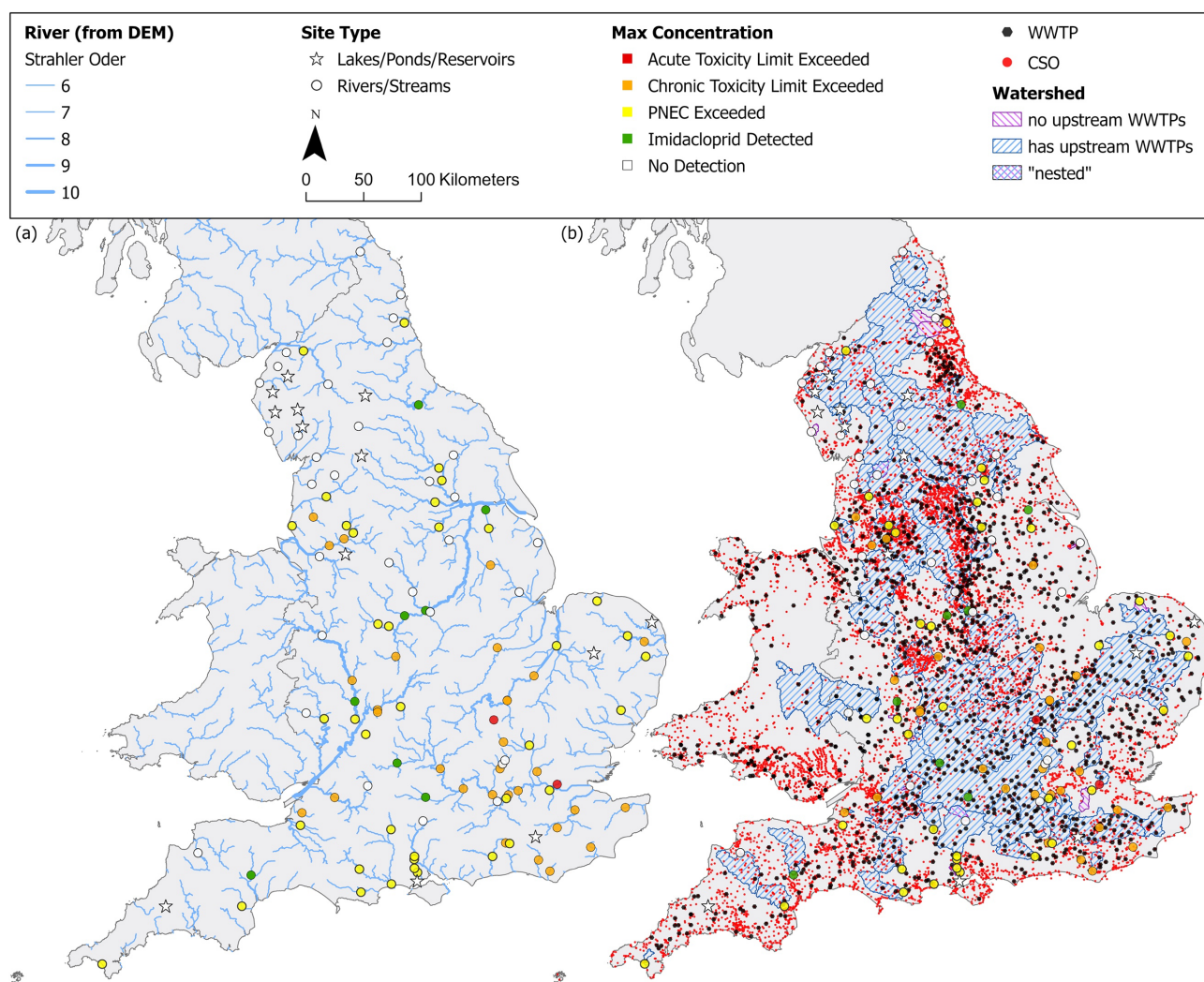


Fig. 6: Post-ban (2019 to 2023) imidacloprid at Environment Agency monitoring sites with delineated watersheds, WWTPs and CSOs. (a) Light blue lines = drainage network extracted from UK DEM (see body text and Methods for details). Note: Maximum imidacloprid concentration recorded at each site is used for classification (circles); limit of detection (LOD) = 0.001 $\mu\text{g/L}$; PNEC: 0.0068 $\mu\text{g/L}$; chronic toxicity: > 0.035 $\mu\text{g/L}$; acute toxicity: > 0.2 $\mu\text{g/L}$. (b) Blue/pink hatched polygons = watersheds with/without upstream WWTPs (black hexagons), cross-hatching polygons = nested watersheds. Red solid dots = combined sewer overflow (CSO).

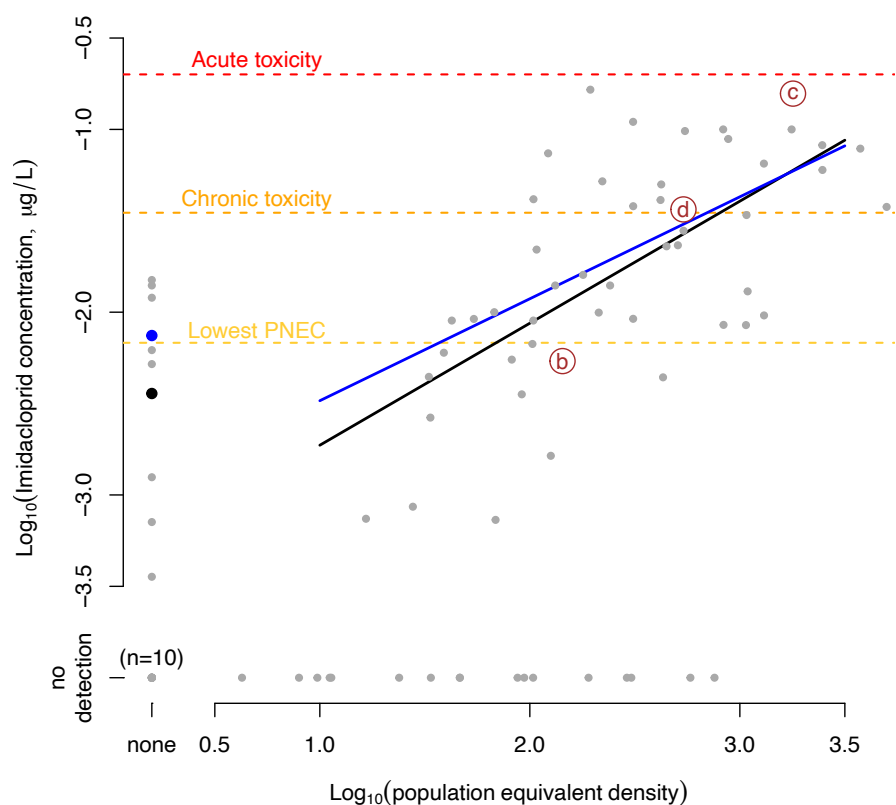


Fig. 7: Imidacloprid concentrations at Environment Agency monitoring sites on running freshwater sampled post-ban (2019–2023) compared to a proxy for pet population (*population equivalent density*) in catchments of upstream WWTP. Imidacloprid concentration increases with population-equivalent density. Site-level means of imidacloprid concentration (vertical axis; broken to represent non-detections) is plotted against population equivalent density (horizontal axis; broken to represent ‘none’ separately). Black line = relationship between mean imidacloprid concentration and *population equivalent density* across all sites; blue line = relationship incorporating all individual samples (taken from the lognormal model component; see Methods and Supporting Information). Black and blue points on the left = estimates of concentration when reported *population equivalent density* (and thus estimated population) is zero upstream, taken from the lognormal component of the averaged and individual sample models, respectively. Some sites had no imidacloprid ($n=27$), some had no population upstream ($n=17$), and some had no imidacloprid or population ($n=10$; marked on the figure, as the over-plotting of data makes it impossible to otherwise see). See Fig. 6 for site locations; contextual hydrological and land use data for three labelled sites (b: 45400013, c: 16M08, d: PTHR0107) is given in Fig. S3. Note: LOD = 0.001 $\mu\text{g/L}$ (not shown); lowest PNEC = 0.0068 $\mu\text{g/L}$ (yellow dashed line); chronic toxicity: > 0.035 $\mu\text{g/L}$ (orange dashed line); acute toxicity: > 0.2 $\mu\text{g/L}$ (red dashed line).

“Sources and Mitigation of Nationwide Parasiticide Water Pollution”

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1. Introduction

A timeline of imidacloprid use and its contamination of wastewater and surface waters in the UK since 1999 is given in Fig. S1.

In Section 2 this document expands the calculation of doses of imidacloprid delivered to companion animals in the UK from 2014 to 2024 given in the main text.

In Section 3 we extend the explanation of our approach to locating samples in running water (e.g., rivers, streams) on the digital drainage network. The samples we make use of were collected between 2014 to 2023 and reported in the Environment Agency’s (EA) dataset, providing information about imidacloprid in England’s waterways before and after the total ban on its use in outdoor agricultural settings in 2018; see the main manuscript and its Data Availability Statement for details of the EA dataset used. Note that we focus our analysis on samples collected from 2019 to 2023, after the ban. Examples of mapping watersheds upstream of sample sites and the mapping of potential upstream sources of imidacloprid are then given, expanding the explanation of the mapping procedures described in the main manuscript.

In Section 4, for completeness and context, we discuss imidacloprid concentrations reported at standing water monitoring sites in the EA dataset, which are briefly referred to in the main manuscript but not included in further analysis, which is focused on the far more extensively sampled running water sites.

In Section 5 the EA data used in this study are compared to recently published data from Welsh rivers.

In Section 6 a discussion of imidacloprid at two focal running water sites that have acute toxicity ($>0.2 \mu\text{g/L}$) and low upstream population—apparent outliers in our study—are considered in more depth.

In Section 7 an extended description of our approach to exploring the relationship between

imidacloprid concentrations at EA monitoring sites and pets in their upstream catchments is given. This includes our approach to working with samples in which imidacloprid was not detected because it was below the limit of detection (LOD) or was not present: 'non-detect' data.

In Sections 8 and 9 we extend the presentation of data and discussion of concentrations of imidacloprid in the influent and effluent of wastewater treatment plants (WWTP) referred to in the main manuscript. This section includes a description of the analytical methods used to produce the 423 measurements of influent imidacloprid at a major UK WWTP that are presented here for the first time.

In Section 10 we expand the methodology used to assess the impact of hypothesized mitigation scenarios discussed in the main manuscript.

The document contains the nine Supplementary Tables referred to in the main manuscript. Supplementary Tables 1 and 2 summarize the distributions of observed imidacloprid concentrations by River Basin District (RBD) with concentrations below LOD set to zero or removed. Supplementary Tables 3 and 4 provides statistics summarizing measured concentrations of imidacloprid annually from 2014 to 2023 in the EA dataset, again with concentrations below LOD set to zero or removed. Supplementary Table 5 presents relationships between imidacloprid detections or 'no detections' at sampling sites and presence or absence of upstream wastewater treatment plants. Supplementary Table S6 gives the percentage reduction in number of samples collected from 2019 to 2024 that would have exceeded LOD, lowest predicted no effect concentration (PNEC), and chronic and acute toxicity thresholds if the samples only exceeded the limits in a single month during the peak infestation period. Supplementary Tables S7-S9 presented the results from the Bayesian hurdle models developed to explore the relationships between imidacloprid concentrations at EA monitoring sites and pet populations in their upstream catchments.

Twelve Supplementary Figures are provided and discussed in the following sections.

2. Imidacloprid dosing of pets in the UK

The number of doses of imidacloprid delivered to pets in the UK from 2015 to 2024 given in the main manuscript was estimated as follows. First, sales data for veterinary medical products (VMPs) were obtained from Brown et al.¹: between 2014 to 2024 the masses, m , of annual VMP usage varied between 2998 kg and 4237 kg (their supplementary table and Fig. 5); $m = 3618 \pm 620$ kg/year. Their reported values are broadly consistent with those given by Anthe et al.² and Perkins et al.³. Secondly, the mean manufacturer recommended monthly dose of imidacloprid per animal were obtained from Wells and Collins⁴: 190 mg, giving an annual dose per animal, $d = 2280$ mg. Finally, following Wells and Collins⁴, the approximate number of doses delivery annually, $m/d = 1.59 \pm 0.27$ million, consistent with Wells and Collins estimate for 2017 (1.85 million doses). We explore alternative approaches to estimating the number of doses in Section 10.

3. Watershed mapping

In the Methods section of the main manuscript a brief description of the steps taken to produce a digital drainage network from the UKCEH Integrated Hydrological Digital Terrain Model (IHDTM), and an overview of the steps performed to locate ('snap') EA monitoring sites on the network, are given. This approach allows us to explicitly link measurements of imidacloprid to potential sources

upstream. It is a considerable advance beyond coarse-grained approaches that, for instance, consider land use adjacent to the sample site. Here, Fig. S2 is included to provide an example of 'snapping' one of the EA sites to the network. It shows how the coordinates provided in the EA dataset (purple pin) are offset from the nearest vertex on the digital network by < 50 m (the resolution of the IHDTM). In most cases the automated snapping tools in ArcGIS, which were used to snap the site onto the nearest vertex (orange pin), were sufficient to accurately position sites on the drainage network. All snapped sites were manually inspected prior to mapping of upstream catchments to ensure that they were correctly positioned on the digital network honouring the hydrological conditions on the ground, with special attention paid to ensuring connectivity to correct branches of the network. In some cases, as we describe in the main manuscript, snapping required manual intervention to correctly locate them on the network. Following inspection, 85 out of the 110 fresh running water sites in the EA dataset could be used to map upstream catchments with confidence. The remaining 25 had equivocal positions on the network and so were excluded from further detailed analyses, but feature in the statistical summaries given in Tables S1-S4. With the 85 sites located on the network it was straightforward to use automated catchment mapping tools in ArcGIS to map catchments upstream, as described in the main manuscript. The resultant catchment maps were used to identify possible upstream sources, e.g. WWTP and combined sewer overflows (CSO), and map land use in the catchments (see Fig. 6 & S2-S3).

Fig. S3 shows four diverse examples of mapped watersheds from across England demonstrating the varied spatial locations, concentration levels, watershed areas, and land use included in this study. This figure also demonstrates the distribution of CSOs upstream of those sites, which may represent an additional input pathway that could be considered when characterising imidacloprid loading within studied catchments in future studies. In theory, it should be relatively straightforward to incorporate information about CSO discharge data upstream of monitoring sites if/when it becomes available (see the main manuscript and Fig. 6 for extended information on CSOs).

4. Standing water sites and running water sites with no imidacloprid detected

In the main manuscript our analysis concerns only the sites in the EA dataset that are located on running waters. Here, for context, we also provide Supplementary Information about the subset of 13 additional sites that are reported in the dataset as being located on standing waters, e.g., lakes, ponds, and reservoirs, after the ban on imidacloprid use in outdoor agricultural settings in 2018 (Fig. S4; which also shows the location of running water sites in the EA dataset where imidacloprid was not detected). From 2019 to 2023, a total of 19 samples were collected at these 13 standing water monitoring sites across England, with none detecting imidacloprid. Twelve of the standing water sites were only sampled once, the other standing water site (Windermere south basin) was sampled seven times. We suggest that the consistent absence of recorded imidacloprid at these sites may be a consequence of larger water volumes and longer residence times in lakes diluting imidacloprid concentrations below the detection limit. However, it is also plausible that the low sampling frequencies were not adequate to observe contamination. Fig. 2 of the main manuscript shows their locations, but they are not considered further here.

5. Imidacloprid concentrations in Welsh rivers and at EA freshwater monitoring sites

Fig S6 compares the distributions of imidacloprid concentrations in samples collected from Welsh rivers reported by Hadley et al.⁵ in their recently published study and of the data from EA monitoring sites on running freshwater used in this study (see e.g. Fig 7 in the main manuscript). The Welsh data was digitized from Fig 2c in Hadley et al.⁵. The violin plots and histograms further emphasize

contamination of waterways across urban and rural areas of England and Wales, with widespread exceedance of lowest PNEC (0.0068 µg/L) and the chronic toxic limit (0.035 µg/L), with occasional exceedance of the acute toxic limit (0.2 µg/L) in the available data.

6. Acute toxicity, and sites with high concentrations and low upstream population

There are two sites, MD02 and 16M08, where concentrations of imidacloprid were very high but upstream *population equivalent density* was low, which is inconsistent with our hypotheses—we consider these sites with large residuals further here (see Fig. 1 for site locations and the main manuscript for details). The highest recorded concentrations in the entire dataset (0.22 µg/L) were detected on two occasions at site 16M08 in running water located approximately 400 m downstream of the Flitwick sewage treatment works in Bedford, and once at site MD02 on Mardyke river near South Ockendon on the outskirts of Greater London. Site MD02 is a significant outlier, exhibiting the highest mean concentration for all sites (0.165 µg/L) despite having a small watershed (91 km²). Given the low sampling density (two samples) at this site and the relatively high sampling frequency (samples were collected within two weeks of each other), we tentatively suggest these concentrations may be related to short-lived spills and a manifestation of acute contamination that requires very frequent, reactive, or opportune sampling to be identified. Another consideration for future work is the contribution from misconnected sewers, which have recently been suggested as a potential source of ecoparasiticides in Welsh rivers⁵.

7. Correlations between pet population and measured downstream imidacloprid

Tables S7-S9 and Fig. S6-S9 summarise the calculated relationships between imidacloprid concentrations measured at EA monitoring sites and the proxy for pet population upstream calculated with Bayesian hurdle and non-hurdle models (see Methods). They summarise results generated from fitting all measured concentrations, and from fitting average concentrations at each monitoring site, including the different approaches we used to work with samples with concentrations below the limit of detection (LOD = 0.001 µg/L). Imidacloprid concentration values below LOD were treated with three commonly applied substitution approaches: replacement with 0, 1/2 LOD, or LOD, and by removal (e.g. Wood et al.⁶).

Fig. S6 extends the results shown in Fig. 7 by showing the concentrations of all samples at each monitoring site with a mappable catchment. The mean values shown in this figure were calculated following replacement of values below LOD with zero (as presented in Fig. 7). All substitution strategies yielded models that were consistent whether they were calculated with mean or median concentrations (see Fig. S7-S9). These results suggest that the specific substitution and averaging approaches explored do not materially influence the overall findings. It is probably good practice to avoid removing values below the detection limit because doing so reduces sample size and distorts the exposure distribution. Nonetheless, we retain them here for completeness and note that the models produced from fitting only values above LOD are broadly similar to the results for zero substitution.

8. Imidacloprid in influent wastewater from an exceptionally high-frequency temporal study at a large WWTP

In this section we extend the description of data used to assess contamination of imidacloprid in untreated, down-the-drain, wastewater given in the main manuscript. It presents previously unpublished concentrations of imidacloprid from 423 samples collected from the influent of

wastewater at a major wastewater treatment plant in the UK serving a *population equivalent* of >1 million people. The samples were collected from October 2021 to January 2023 on separate days over a total period of 462 days. Confidentiality agreements preclude us from naming or precisely locating the WWTP.

8.1 Sampling

Wastewater influent was taken using a chilled (4 °C) automated composite sampler over 24-hours (Glacier Composite Sampler, Teledyne ISCO, Glasgow, UK) based on a time-proportional sampling frequency (< 60 min intervals). Duplicate samples were taken each day in pre-rinsed 30 mL Nalgene bottles (Thermo Fisher Scientific, Rochester, NY, USA). After collection, samples were immediately stored at -20°C on site and in the dark, to minimise transformation and/or degradation of the compounds. Samples were stored, batched and shipped frozen on a monthly frequency. Upon reaching the laboratory, samples were stored at -20°C in the dark until analysis within one week.

8.2 Chemical analysis of wastewater

For semi-quantitative targeted analysis of imidacloprid in wastewater, a previously published direct injection liquid chromatography tandem mass spectrometry (LC-MS/MS)-based analytical method was used employing a Shimadzu LCMS-8060 instrument (Shimadzu Corp., Kyoto, Japan) published by Ng et al.⁷. Briefly, 900 µL of unfiltered sample was added to 100 µL of stable-isotope internal standard (SIL-IS) solution in methanol (d₆-venlafaxine at 500 ng/L final concentration, ordered from QMX, Essex, UK). Following vortexing and filtration using Whatman™ 0.2 µm PTFE membrane filters (GE Healthcare Life Science, Little Chalfont, UK) and 1 mL Plastipak™ syringes (BD, Berkshire, UK), a 10 µL injection of the filtered water sample was injected onto the LC-MS/MS. Gradient elution using mobile phases of ultrapure water with 0.1% formic acid (v/v) (mobile phase A) and 1:1 acetonitrile:methanol with 0.1% formic acid (v/v) (mobile phase B) over 5.5-minutes on a short 3 x 5 mm Raptor™ biphenyl guard cartridge (Thames Restek, Saunderton, UK) housed within an EXP® Direct Connect Holder at 0.5 mL/min. Imidacloprid was detected in samples using two MS/MS transitions of 256>175 (quantifier) and 256>209 (qualifier). Semi-quantitative determination was based on the ion ratio approach between the peak areas for imidacloprid and SIL-IS in the sample, compared to peak area ion ratios in an external calibration curve prepared in ultrapure water over the range 10-7,500 ng/L imidacloprid and with 500 ng/L SIL-IS. Quality control was performed as per the recommendations of Hernández et al.⁸ using two spiked QC solutions (125 and 1,250 ng/L) run at the start, middle, and end of each batch of samples and peak area consistency and instrumental drift were checked and considered acceptable if inaccuracy was <15% when quantified from the calibration curves. Before the beginning of each sequence, test injections of the mobile phases as well as a standard (N = 500 ng/L) were performed to ensure that no mobile phase was contaminated, and that both imidacloprid and SIL- IS were detected. All standards, prepared samples, matrix-matched standards, blanks and controls were kept in 1.5 mL silanised amber vials (Fisher Scientific, Loughborough, UK). HPLC-MS-grade methanol, isopropanol, acetonitrile and formic acid (>95%, v/v) were bought from Sigma-Aldrich (Steinheim, Germany). Ultrapure water (UP) was generated with a resistivity of 18.2 mΩ at 25°C using a Millipore Milli-Q water purification system (Bedford, MA, USA).

8.3 Supplementary Results and Discussion

Fig. S10 shows the time series of imidacloprid concentration in influent wastewater at the WWTP. All samples contained concentrations exceeding the lowest PNEC threshold reported on the Norman Network Ecotoxicology Database. Among the 423 samples, 92% had concentrations exceeding the chronic toxicity limit (35 ng/L). Concentrations in four samples exceeded the acute

toxicity limit (200 ng/L), three of which were recorded within a month during Summer 2022 (see Table S3 for PNEC, chronic and acute toxicity limits).

Despite the complete agricultural ban on imidacloprid since 2018, the concentrations in influent wastewater remained consistently high throughout the sampling period (October 2021 to January 2023), suggesting that significant urban sources persist. Given the size of the catchment, this is likely dominated by household inputs, which is consistent with the widespread use of imidacloprid-containing domestic pet parasiticides. This down-the-drain pathway is further supported by the observation that three of the highest concentrations were recorded between July and August 2022, coinciding with the peak period of pet infestation treatments⁹. These findings indicate that, even in the absence of agricultural inputs, urban domestic activities contribute substantially to environmental imidacloprid loading, posing both chronic and occasional (additional) acute risks to aquatic ecosystems. See main manuscript for further discussion of these and related results.

9. Measurements of imidacloprid in wastewater effluent

An obvious extension of the work presented in Section 8 of this document is to consider measurements of imidacloprid concentrations in treated wastewater, i.e., WWTP effluent. Such measurements, which are currently extremely sparse, would provide direct information about imidacloprid discharges into the receiving waters, and thus quantitative data that could be used to relate concentrations at sources to sites downstream.

The UK Water Industry Research (UKWIR) Chemical Investigation Programme (CIP) has been monitoring imidacloprid in wastewater effluent across the UK for the past decade¹⁰. We extracted all available imidacloprid effluent data from the UKWIR CIP database between 2019 and 2023 and located them in watersheds mapped upstream of the 85 EA sites that form the core of the analyses presented in the main manuscript (see Fig. 4 & S11). Unfortunately, overlapping data coverage is extremely limited and insufficient for further rigorous statistical analysis. Only one CIP sample site can be linked hydrologically to a downstream EA monitoring site: Bedford STW in the watershed of EA site 12M08 (Fig. S11). For this pair of observations, the lack of overlap in timing of CIP and EA monitoring means that it is not obvious how the measured effluent concentrations can be linked to the observed downstream imidacloprid levels in a meaningful way (Fig. S12). Moreover, no effluent data were available for the remaining WWTPs discharging within the catchment upstream of site 12M08, therefore wastewater-derived imidacloprid inputs at this site cannot be directly quantified and thus we did not pursue this analysis further. Such information, if it were to become available in the future, could facilitate significant improvement to the accuracy and precision of source apportionment when compared to the reliance on proxies for sources such as the *population equivalent* values at WWTPs used in this study (see the main manuscript for details). We make recommendations for future monitoring efforts to address this knowledge and data gap in the main manuscript.

CIP monitoring provides valuable information but is insufficient in temporal or spatial resolution to fully capture imidacloprid loading patterns across river catchments. Given that this resource is the most comprehensive and spatiotemporally aligned UK dataset to date, further efforts are required improve chemical surveillance frameworks to enable improved understanding of the influent-effluent-receiving water axis. Wastewater monitoring is typically conducted as periodic screening resulting in sparse datasets that are often temporally misaligned with downstream river sampling and limited to a subset of treatment works within larger catchments. Future research would benefit from co-ordinated effluent and downstream sampling at a higher temporal resolution. We expand on

this discussion in the main manuscript, and provide recommendations for future work.

10. Hypothetical mitigation strategies

In this section we extend the methodology and discussion of three hypothesized mitigation strategies briefly referred to in the main paper. We present potential hypothetical scenarios based on the limited data that are available, to explore the scale of the challenge and possible options. First, we calculate the number of samples that would have exceeded lowest PNEC in the EA data if samples only exceed PNEC in one month of the year. This approach is designed to assess the potential impact of reducing the dosing of pets to one of the peak infestation months, which could, for instance, be related to treatment following clinically diagnosed need. Second, we perform a more complex analysis in which potential reductions in total imidacloprid load used to treat pets with dosing strategies different to those that are currently used are estimated. This approach starts with an estimation of the number of doses of imidacloprid currently given to cats and dogs in the UK. Finally, we perform a simple assessment of how load could be reduced if dosing was reduced 2- and 10-fold. Each of the assessments below would benefit from having information about the number and mass of imidacloprid doses applied to cats and dogs in the UK. In the absence of such data, we develop insight by making the assumptions described below.

10.1 Reductions that could have been possible if samples only exceeded PNEC in one month

The reduced number of samples that would have been taken at the 85 EA monitoring sites on running water if they only exceeded lowest PNEC during one of the peak infestation months are estimated. This approach is designed to assess how a strategy of only dosing in a single month during the peak infestation period, rather than dosing year-round, could impact toxicity of waterways. It assumes that the only sources of imidacloprid were VMPs and instantaneous transfer of imidacloprid from application to waterways. The values calculated below provide a guide to the scale of potential reductions in toxicity possible with modified dosing strategies. We note that following calculated reductions in imidacloprid would be lower if contributions from other sources could be quantified and incorporated into these calculations, e.g. legacy from outdoor agricultural use, biocides, residuals in soil and sediment, amenity usage, horticulture, food residues, etc.¹. Given that contributions from many of these additional sources are poorly understood and lack quantification we do not include them here. These assumptions could be relaxed if knowledge about the transfer time from pet dosing to EA monitoring sites in waterways downstream and the longevity of imidacloprid in the environment becomes available.

First, the number of samples at the 85 EA monitoring sites with mappable upstream catchments with concentrations that exceeded PNEC throughout the year, $N_p = 596$, and in the months of July ($N_7 = 54$), August ($N_8 = 55$), September ($N_9 = 87$) and October ($N_{10} = 66$) from 2019 to 2024 were determined. Percentage reductions in imidacloprid concentrations exceeding PNEC were then estimated. For example, if only samples above PNEC in July are included, the percentage reduction in samples above PNEC year-round is $100 - N_7/N_p \times 100 = 91\%$. See the main manuscript and Table S6 for extended results including estimated reductions in the number of samples with imidacloprid above LOD, and chronic and acute toxicity limits that could have been possible with dosing in a single month during the peak infestation period.

10.2 Possible reductions in imidacloprid load for hypothesized dosing strategies

In this subsection we estimate reductions in total annual imidacloprid load required to treat the cats and dogs currently treated with imidacloprid if different dosing strategies were adopted. For

simplicity we assume that cats and dogs treated with imidacloprid follow one of two treatment plans. We assume that all cats and dogs on treatment plans A and C have full, year-round, coverage, and that all cats and dogs on treatment plans B and D have partial coverage. This scenario is designed to mimic reported practice of cat and dog owners who either treat their pets prophylactically year-round, or in response to infestation or perhaps to provide coverage prophylactically only during peak infestation months¹¹. Given that there is no data to directly quantify the number of doses of imidacloprid given to cats and dogs each year in the UK as far as we are aware, we must first estimate them.

We start by estimating total mass of imidacloprid, I , used to treat cats and dogs annually,

$$I = N[p_c(d_{cA}p_{cA} + d_{cB}p_{cB}) + p_d(d_{dC}p_{dC} + d_{dD}p_{dD})], \quad (1)$$

where N is the total number of doses given each year, noting that as one pet receives one annual dose per year, N is also total number of pets treated each year. p_c and p_d are the respective proportions of cats and dogs (lower case subscripts) in the total population of dogs and cats. d_{cA} and d_{cB} are the annual doses of imidacloprid given to cats on treatment plans A or B, d_{dC} and d_{dD} are the annual doses of imidacloprid given to dogs on treatment plans C or D (uppercase subscripts). p_{cA} and p_{cB} are the proportions of cats on treatment plans A or B, p_{dC} and p_{dD} are the proportions of dogs on treatment plans C or D.

With regards to number of doses, $N = N_c + N_d = N_{cA} + N_{cB} + N_{dC} + N_{dD}$, where N_c and N_d are the respective number of doses given to cats and dogs receiving treatment with imidacloprid, and N_{cA} , N_{cB} , N_{dC} and N_{dD} are the respective numbers receiving one of the four treatment plans. Noting that $N_c = p_c N$ and $N_d = p_d N$, and that $N_c = N_{cA} + N_{cB}$, $N_d = N_{dC} + N_{dD}$, and $N_{cA} = p_{cA} N_c$, $N_{cB} = p_{cB} N_c$, $N_{dC} = p_{dC} N_d$, and $N_{dD} = p_{dD} N_d$. With these quantifies defined it is straightforward to estimate the number of doses cats and dogs on each of the treatment plans received,

$$N_{cA} = N p_{cA} p_c, \quad N_{cB} = N p_{cB} p_c, \quad N_{dC} = N p_{dC} p_d, \quad N_{dD} = N p_{dD} p_d. \quad (2)$$

With values for the parameters defined for current annual imidacloprid load from VMPs, the proportion of dogs and cats in the population treated with imidacloprid, and the annual dosing of pets in a treatment plan, it is then straightforward to rearrange Eq. (1) to estimate the total number of doses of imidacloprid N , and thence the number of doses and pets receiving treatment for each plan with Eq. (2)

Reported imidacloprid usage in VMPs¹, $I = 3618 \pm 620$ kg/year. In the calculations that follow we assume that it is all used to treat only cats and dogs, if fact a small proportion of it is used to treat other pets (e.g. rabbits⁹). Consequently, our estimates of reduction based on changes to dosing of cats and dogs are upper limits with regards to the entire pet population. The population of cats and dogs in the UK is estimated to be 11 million and 12 million¹². If we assume that the proportion of cats and dogs treated with imidacloprid are the same as the proportions of cats and dogs across their whole population, $p_c = 0.478$ and $p_d = 0.522$. Single doses of imidacloprid administered to pets vary depending on the size of the animal being treated. The dose for a large cat (> 4 kg) is 80 mg and a small cat (< 4 kg) is 40 mg, so we have assumed an average single dose for a cat of 60 mg. We use the single dose recommended for a medium dog, 100 mg, in the following calculations. Single doses are usually administered monthly.

Annual dosing of pets on the different treatment plans can now be estimated. For instance, if cats and dogs are dosed every month of the year ('full coverage') then the calculated respective annual doses are 720 and 1200 mg. If they are dosed four times in the year the annual doses for cats is 240 and for dogs it is 400 mg. A recent survey of pet owners indicated that 34% of pets are treated year-round¹¹. In the calculations that follow we assume that the remaining 66% of pets are treated with a partial treatment plan.

If we assume that the partial treatment plans for cats and dogs give coverage for four months of the year, we define $d_{cA} = 720$ mg /year, $p_{cA} = 0.34$, $d_{cB} = 240$ mg /year, $p_{cB} = 0.66$, and $d_{dC} = 1200$ mg /year, $p_{dC} = 0.34$, $d_{dD} = 400$ mg /year and $p_{dD} = 0.66$. We refer to this approach as *treatment strategy 1*. Inserting these values into Eq. (1), indicates that $N = 6.7$ million annual doses of imidacloprid are given each year to 6.7 million pets: 3.2 million cats and 3.5 million dogs. If the 1.1 million cats that we estimate currently have full coverage (12 single doses in a year) had their dosing reduced to the partial treatment plan (4-months of the year), the amount of imidacloprid load would be reduced to 86% of its current value. If treatment for dogs receiving full coverage also followed this strategy the imidacloprid load would be reduced to 60% of its current load.

If we assumed that pets receiving partial coverage only receive once dose in the year ($d_{cB} = 60$ mg /year, $d_{dD} = 100$ mg /year; *treatment strategy 2*) the total number of doses delivered per year is estimated to be 9.4 million (to 4.5 million cats and 4.9 million dogs). If both the cats and dogs that are estimated to currently have full coverage under *treatment strategy 2* were moved onto this partial treatment plan, we estimate that the annual imidacloprid load would be 21% of its current value (from 3618 kg /year to 764 kg /year).

We note that instances of fleas reported from consultations at veterinary practices across Great Britain indicate that a small proportion (1.17% cats, 0.24% dogs) of the total number of pets clinically assessed (1.9 million cats, 4.8 million dogs) were infested between March 2014 and July 2020⁹. The numbers of cases vary seasonally with highest number of recorded flea cases in the third quarter of the year, with approximately three times as many cases reported for cats than dogs. Whilst these values estimate incidence, not annual prevalence, they suggest that testing the impact of modifying *treatment strategy 1* so that only cats are treated in July, August, September and October could be informative. We estimate that such a dosing strategy would reduce imidacloprid load to 21% of its current value.

The strategies explored so far assume that all cats and dogs currently being treated with imidacloprid would continue to receive at least a single dose per year. If treatment was instead based on clinically diagnosed need (e.g. to respond to observed infestation) the reductions could be even greater. However, information about the prevalence of infestation in the cat and dog population is currently lacking. Thus, we cannot estimate reductions in load that could be produced with this hypothesized approach with confidence at this stage.

10.3 Estimates of load reduction for M -fold reductions in dosing

It is straightforward to estimate the impact of reducing dosing on total imidacloprid load. If the amount of dosing is reduced M -fold across all treatment plans within a *treatment strategy*; the total annual load of imidacloprid is reduced by a factor of M . For instance, if $M = 2$, mean annual imidacloprid load is reduced by 50% from its current value of 3618 kg /year to 1809 kg /year. If $M = 10$, the load is reduced from 3618 kg /year to 362 kg /year.

Supplementary References

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Supplementary Tables

Table S1: Summary statistics for Environment Agency imidacloprid concentration data from 2019–2023, separated by River Basin District (RBD). The number of samples (N), detection rate, and mean, median, and maximum concentrations are shown. Detection (%) = number of detects / N; mean and median are calculated with non-detects substituted with 0.

RBD	N	Detection (%)	Mean (µg/L)	Median (µg/L)	Max (µg/L)
Anglian	853	68.5	0.019	0.008	0.22
Humber	441	27.9	0.004	0	0.15
North West	97	42.3	0.009	0	0.1
Northumbria	47	6.4	0	0	0.01
Severn	239	27.2	0.003	0	0.1
Solway Tweed	31	3.2	0.001	0	0.02
South East	76	52.6	0.01	0.002	0.18
South West	51	76.5	0.008	0.006	0.042
Thames	55	76.4	0.067	0.073	0.22
All	1,890	49.6	0.013	0	0.22

Table S2: Summary statistics for Environment Agency imidacloprid concentration data from 2019–2023, separated by River Basin District (RBD). The number of samples (N), detection rate, and mean, median, and maximum concentrations are shown. Detection (%) = number of detects / N; mean and median are calculated with non-detects removed.

RBD	N	Detection (%)	Mean (µg/L)	Median (µg/L)	Max (µg/L)
Anglian	584	68.5	0.0273	0.015	0.22
Humber	123	27.9	0.0142	0.0052	0.15
North West	41	42.3	0.0211	0.015	0.1
Northumbria	3	6.4	0.0054	0.0054	0.0095
Severn	65	27.2	0.0095	0.0031	0.1
Solway Tweed	1	3.2	0.02	0.02	0.02
South East	40	52.6	0.0184	0.0047	0.18
South West	39	76.5	0.0099	0.009	0.042
Thames	42	76.4	0.0883	0.094	0.22
All	938	49.6	0.0257	0.012	0.22

Table S3: Summary statistics for annual EA imidacloprid concentration data pre- (2014–2018) and post-ban (2019–2023) in running water sites. The number of sites monitored each year is included, along with the mean, median, and maximum concentrations. Mean and median values are calculated with non-detects substituted with 0. The percentage of samples equal to or exceeding lowest PNEC (0.0068 µg/L), chronic (0.035 µg/L) and acute (0.2 µg/L) toxicity limits are given.

Year	No. Sites	No. Samples	Mean (µg/L)	Median (µg/L)	Max (µg/L)	Acute Exceed. (%)	Chronic Exceed. (%)	PNEC Exceed. (%)
2014	6	171	0.007	0.006	0.02	0	0	47.4
2015	7	524	0.011	0.007	0.2	0.2	4.6	51.9
2016	20	583	0.024	0.013	0.18	0	27.8	61.6
2017	19	291	0.021	0.010	0.19	0	24.7	58.4
2018	25	668	0.021	0.005	0.36	0.7	24.3	48.7
2019	93	396	0.02	0.008	0.22	0.3	20.5	51.5
2020	22	111	0.007	0.003	0.062	0	3.6	31.5
2021	21	361	0.011	0.001	0.22	0.6	10.8	31.0
2022	34	531	0.013	0.000	0.22	0.6	13.9	34.3
2023	22	491	0.009	0.000	0.15	0	10.6	25.9

Table S4: Summary statistics for annual EA imidacloprid concentration data pre- (2014–2018) and post-ban (2019–2023) in running water sites. The number of sites monitored each year is included, along with the mean, median, and maximum concentrations. Mean and median values are calculated with non-detects removed. The percentage of samples equal to or exceeding lowest PNEC (0.0068 µg/L), chronic (0.035 µg/L) and acute (0.2 µg/L) toxicity limits are given.

Year	No. Sites	No. Samples	Mean (µg/L)	Median (µg/L)	Max (µg/L)	Acute Exceed. (%)	Chronic Exceed. (%)	PNEC Exceed. (%)
2014	6	171	0.01	0.01	0.02	0	0	47.4
2015	7	524	0.016	0.011	0.2	0.2	4.6	51.9
2016	20	583	0.032	0.02	0.18	0	27.8	61.6
2017	19	291	0.031	0.023	0.19	0	24.7	58.4
2018	25	668	0.04	0.032	0.36	0.7	24.3	48.7
2019	93	396	0.031	0.014	0.22	0.3	20.5	51.5
2020	22	111	0.012	0.008	0.062	0	3.6	31.5
2021	21	361	0.022	0.01	0.22	0.6	10.8	31.0
2022	34	531	0.027	0.012	0.22	0.6	13.9	34.3
2023	22	491	0.025	0.014	0.15	0	10.6	25.9

Table S5: Imidacloprid detections or no detections at monitoring sites in running freshwater from 2019 to 2023 in relation to the presence or absence of upstream WWTPs. The corresponding 95% Wilson confidence intervals (CI) for imidacloprid detection probability are provided.

Upstream WWTP(s)	Imidacloprid detected	No detection	Total no. sites	Imidacloprid detection, %	95% Wilson CI
Yes	49	18	67	73.1	61.5–82.3
No	8	10	18	44.4	24.6–66.3
Total	57	28	85	67.1	

Table S6: Estimated reductions in number of samples with imidacloprid concentrations exceeding LOD (0.001 µg/L), lowest PNEC (0.0068 µg/L), chronic (0.035 µg/L) and acute (0.2 µg/L) limits possible with reduced, single-month, dosing strategy. The second column gives the number of samples that exceeded the limit given in the first column year-round from 2019 to 2023 at the 85 EA monitoring sites with mappable upstream catchments. The third to sixth columns give the number of samples that exceeded the thresholds in the named month from 2019 to 2023, and the percentage decrease in the total number of samples that would have exceeded the annotated limits if samples only exceeded the limit in that month.

	Year-round	July	August	September	October
LOD	831	68 / 92%	65 / 92%	106 / 87%	81 / 90%
PNEC	596	54 / 91%	55 / 91%	87 / 85%	66 / 89%
Chronic	232	25 / 89%	33 / 86%	42 / 82%	26 / 89%
Acute	6	0 / 0%	0 / 0%	1 / 83%	0 / 0%

Table S7. Summary of site-averaged, mean, model of imidacloprid, showing strong effect of population (p) on its presence; values below LOD were set to zero prior to averaging. Each row represents a different parameter within the site-averaged hurdle model, grouped according to whether they are in the lognormal (of imidacloprid's concentration, i) or logit (of detection) component. Each section contains an estimate for the overall intercept, the slope of $\log_{10}(\text{population equivalent units})$, and the dummy-coded variable for regions with no population (see Methods). Each row describes, in turn, the mean estimate of the coefficient across the posterior distribution, the re-scaled estimate so that it is in units of $\log_{10}$ (to convert, for ease of interpretation, from a model fit with a natural-base lognormal to a $\log_{10}$ -base), the standard deviation of the posterior coefficient estimate, the 95% credibility intervals (not a confidence interval, since this is a Bayesian model), the Gelman-Ruben convergence statistic¹³, and two commonly-used metrics of the Effective Sample Size of the posterior distribution. The model, indicated by the black lines in Fig. 7 and S6 was produced using the Lognormal values: $\log_{10}(i) = 0.67 \log_{10}(p) - 3.4$, where i is calculated imidacloprid concentration and p is population equivalent density, noting that the intercept is calculated by addition of 'Intercept' and 'Dummy-pop'; converting back into linear space: $i = p^{0.67} / 2512$. That imidacloprid concentration is greater in samples downstream of higher population density is supported by the positive coefficient for $\log(p)$ in the lognormal component, as can be seen in Fig. 7 and S6 where the positive trend is plotted against the data. The logit component shows the same trend; note that the negative coefficient for the slope of population is because this component model the probability that a sample *does not* contain imidacloprid, which decreases with increasing population.

		Estimate	Estimate (rescaled)	SD	Lower 95% CI	Upper 95% CI	$\hat{r}$	Bulk ESS	Tail ESS
Lognormal	Intercept	-5.62	-2.44	0.40	-6.40	-4.81	1	4614	3133
	Log(pop)	1.54	0.67	0.27	0.98	2.05	1.00	2892	2604
	Dummy- pop	-2.20	-0.96	0.79	-3.72	-0.61	1.00	3094	2594
Logit	Intercept	0.25		0.48	-0.70	1.23	1.00	2892	2604
	Log(pop)	-1.86		0.56	-3.04	-0.83	1.00	2844	2478
	Dummy -pop	2.59		1.25	0.27	5.16	1.00	3111	2761

Table S8. Summary of individual samples across sites, showing strong effect of population on its presence. This table should be interpreted as Table S1, but with additional terms for the hyper-parameter estimated as part of the more complex model structure as described in the methods at the bottom of the table. The lognormal components are used to produce the blue line in Fig S6: $\log_{10}(i) = 0.56 \log_{10}(p) - 3.05$; in linear space: $i = p^{0.56} / 1122$.

		Estimate	Estimate (rescaled)	SD	Lower 95% CI	Upper 95% CI	$\hat{r}$	Bulk ESS	Tail ESS
Lognormal	Intercept	-4.89	-2.12	0.32	-5.52	-4.26	1.00	5665	5452
	Log(pop)	1.29	0.56	0.21	0.89	1.69	1.00	2078	3029
	Dummy- pop	-2.13	-0.93	0.62	-3.34	-0.90	1.00	2225	3983
Logit	Intercept	2.48		0.57	1.42	3.63	1.00	7619	6867
	Log(pop)	-1.47		0.45	-2.44	-0.63	1.00	3401	4823
	Dummy -pop	0.28		1.21	-2.01	2.78	1.00	3526	4646
Hyper- parameters	$\sigma_{\text{lognormal}}$	0.80		0.10	0.63	1.01	1.00	2636	3759
	σ_{hurdle}	1.64		0.36	1.07	2.48	1.00	2349	4437
	σ_{overall}	0.51		0.01	0.48	0.53	1.00	14308	6455

Table S9. Summary of site-averaged, median, model of imidacloprid, showing strong effect of population, values below LOD were set to zero prior to averaging. This table should be interpreted as Table S1 but for median averages. See Fig. S7. The fitted model has the form $\log_{10}(i) = 0.51 \log_{10}(p) - 2.95$; in linear space: $i = p^{0.51} / 891$.

		Estimate	Estimate (rescaled)	SD	Lower 95% CI	Upper 95% CI	$\hat{r}$	Bulk ESS	Tail ESS
Lognormal	Intercept	-4.67	-2.03	0.42	-5.47	-3.84	1.00	4725	2942
	Log(pop)	1.17	0.51	0.25	0.68	1.67	1.00	3225	2524
	Dummy- pop	-2.11	-0.92	0.76	-3.62	-0.61	1.00	3325	3042
Logit	Intercept	1.03		0.54	0.04	2.15	1.00	3978	2791
	Log(pop)	-2.39		0.64	-3.73	-1.23	1.00	2771	2120
	Dummy -pop	3.27		1.44	0.58	6.21	1.00	2809	2284

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Supplementary Figures

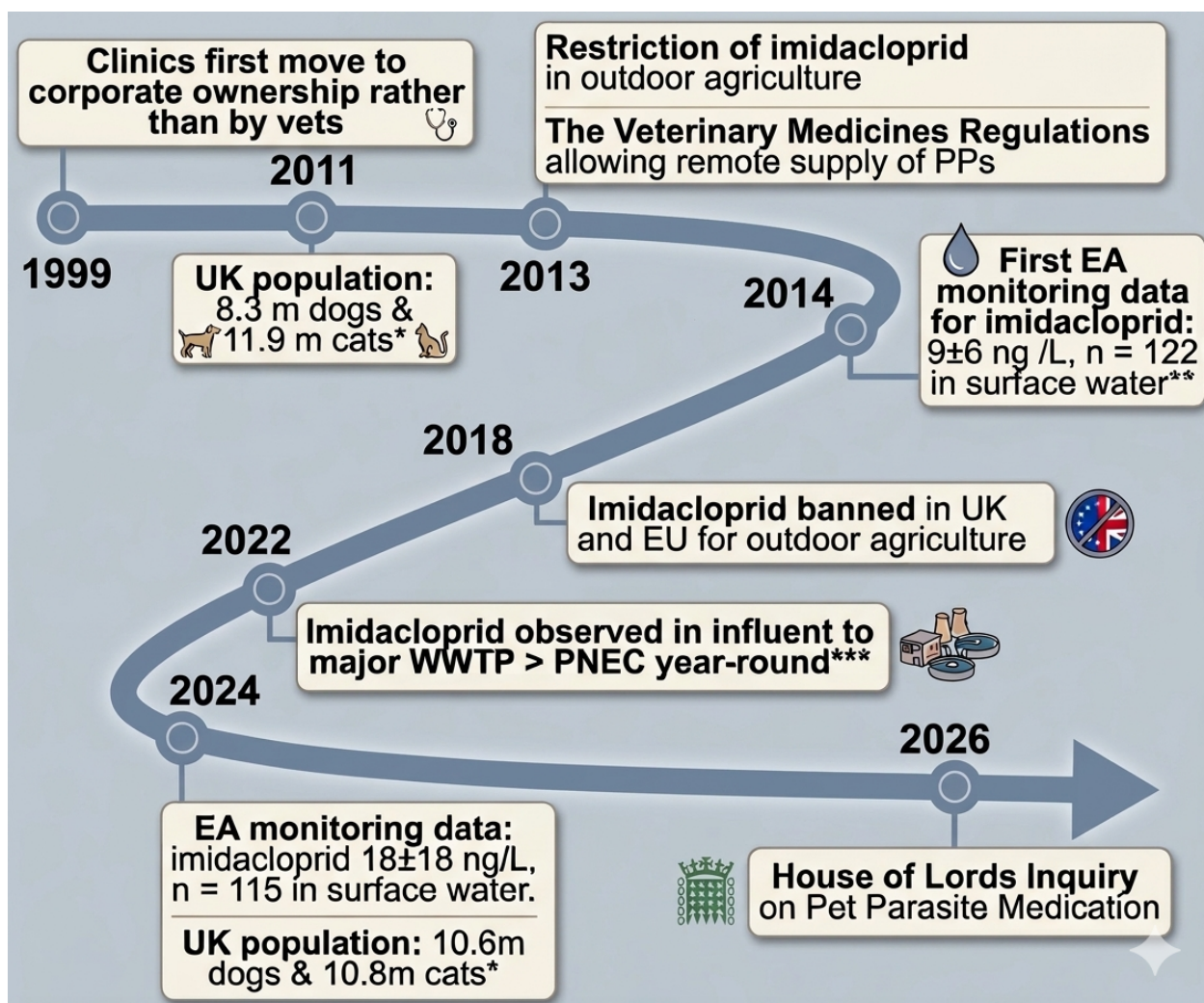


Fig S1: Timeline of key events relevant to UK waste and surface water contamination with imidacloprid. *data from the Pet Animal Wellbeing (PAW) reports 2011 and 2024 published by the People's Dispensary for Sick Animals (PDSA). **Environment Agency Water GC-MS and LC-MS semi-quantitative screen dataset**. ***this study.

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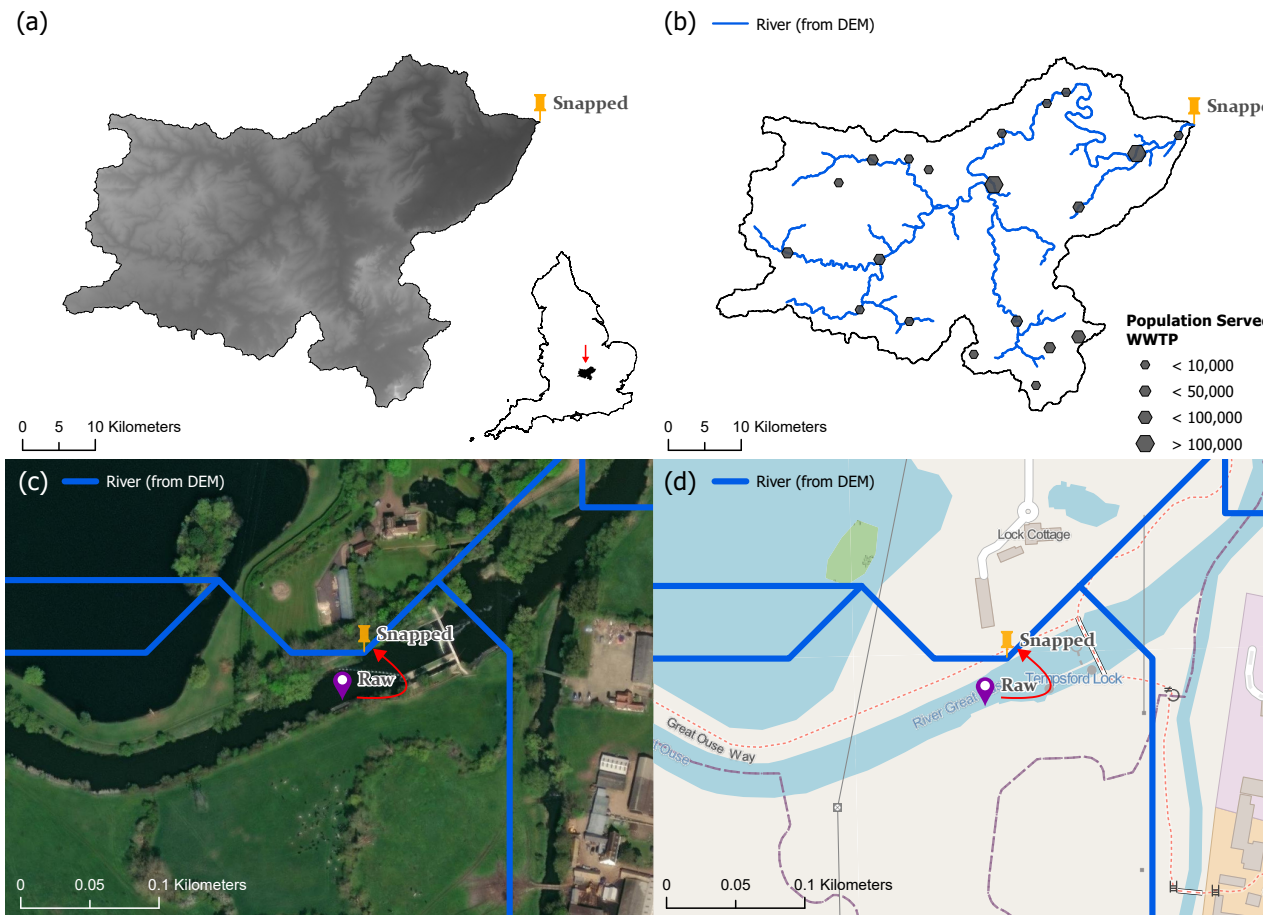
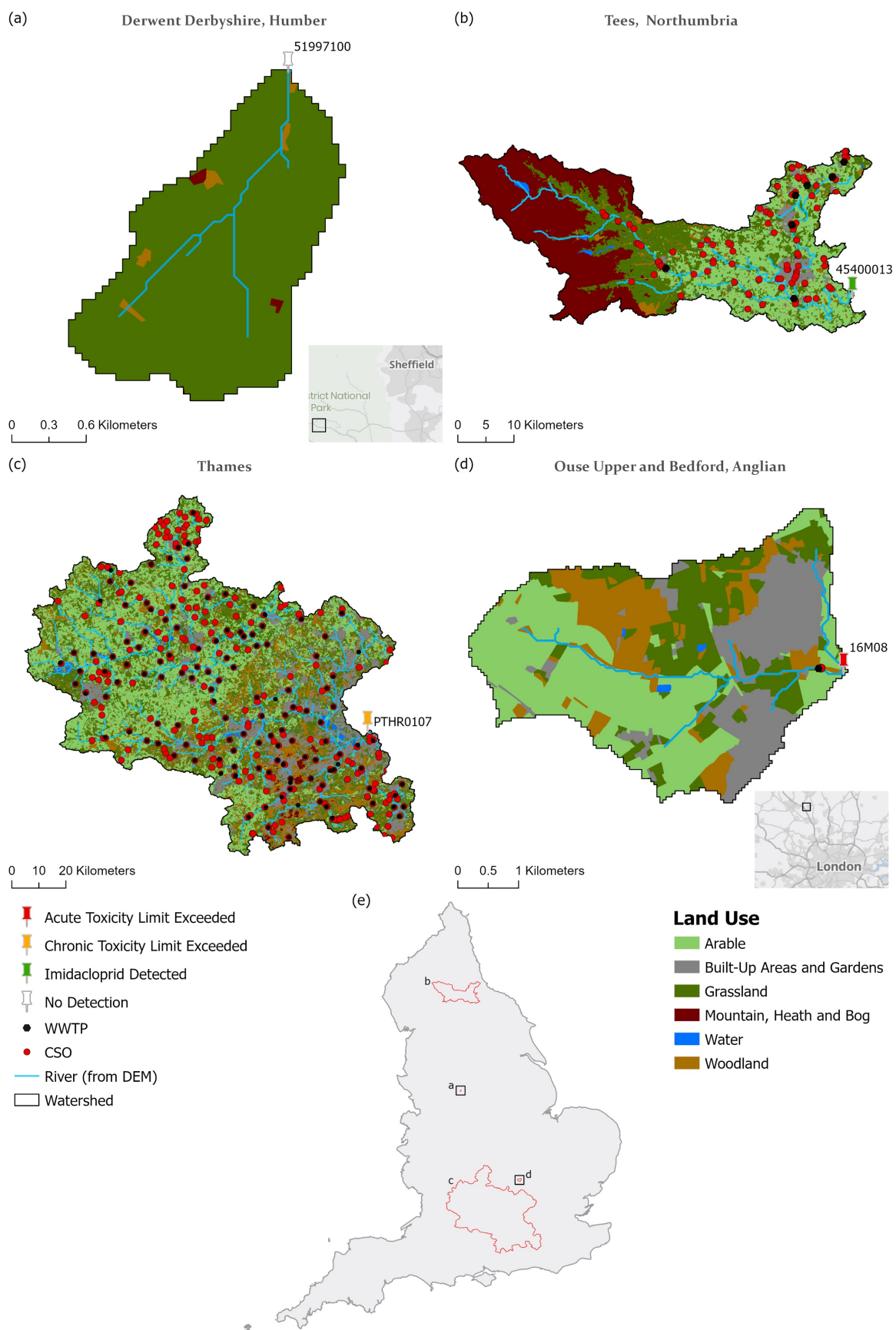


Fig. S2: Example ‘snapping’ of EA sample site (12M08, River Ouse in Roxton, Bedford) to the digital drainage network and mapping of its upstream catchment. (a) Gray scale = DEM (UKCEH IHDTM; 50 m resolution) with hydrological ‘sinks’ filled (see Methods); black outline = catchment boundary; annotated pin = location of snapped EA site generated as shown in panels (c-d). (b) Catchment boundary, drainage network, and WWTP upstream of EA sample site. (c-d) Satellite imagery and Open Street Map around the site. Purple pin = coordinates provided by the EA; orange pin = snapped location on the drainage network.

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579 **Fig. S3: Demonstration of watershed, WWTP, CSO and land use diversity upstream of select**
580 **EA sites in this study.** Pins = snapped site locations colored by measured toxicity; maximum
581 imidacloprid concentration recorded at each site is used for classification; limit of detection = 0.001
582 µg/L; chronic toxicity: > 0.035 µg/L; acute toxicity: > 0.2 µg/L. Blue lines = mapped drainage
583 networks; black = catchment boundaries; see key for additional annotations and inset maps for
584 locations of sample sites with small upstream catchments. (a) Site 51997100: Blackwell Dale Brook.
585 (b) Site 45400013: Tees at Low Worsall. (c) Site PTHR0107: Thames at Teddington Weir. (d) Site
586 16M08: Running Waters Ruxox Bridge. (e) Location of catchments shown in panels (a)-(d).

Site

- ★ Lakes
- 📌 Rivers (no WWTPs)
- 📌 Rivers (has WWTPs)

River (from DEM)

Strahler Order

- 7
- 8
- 9
- 10

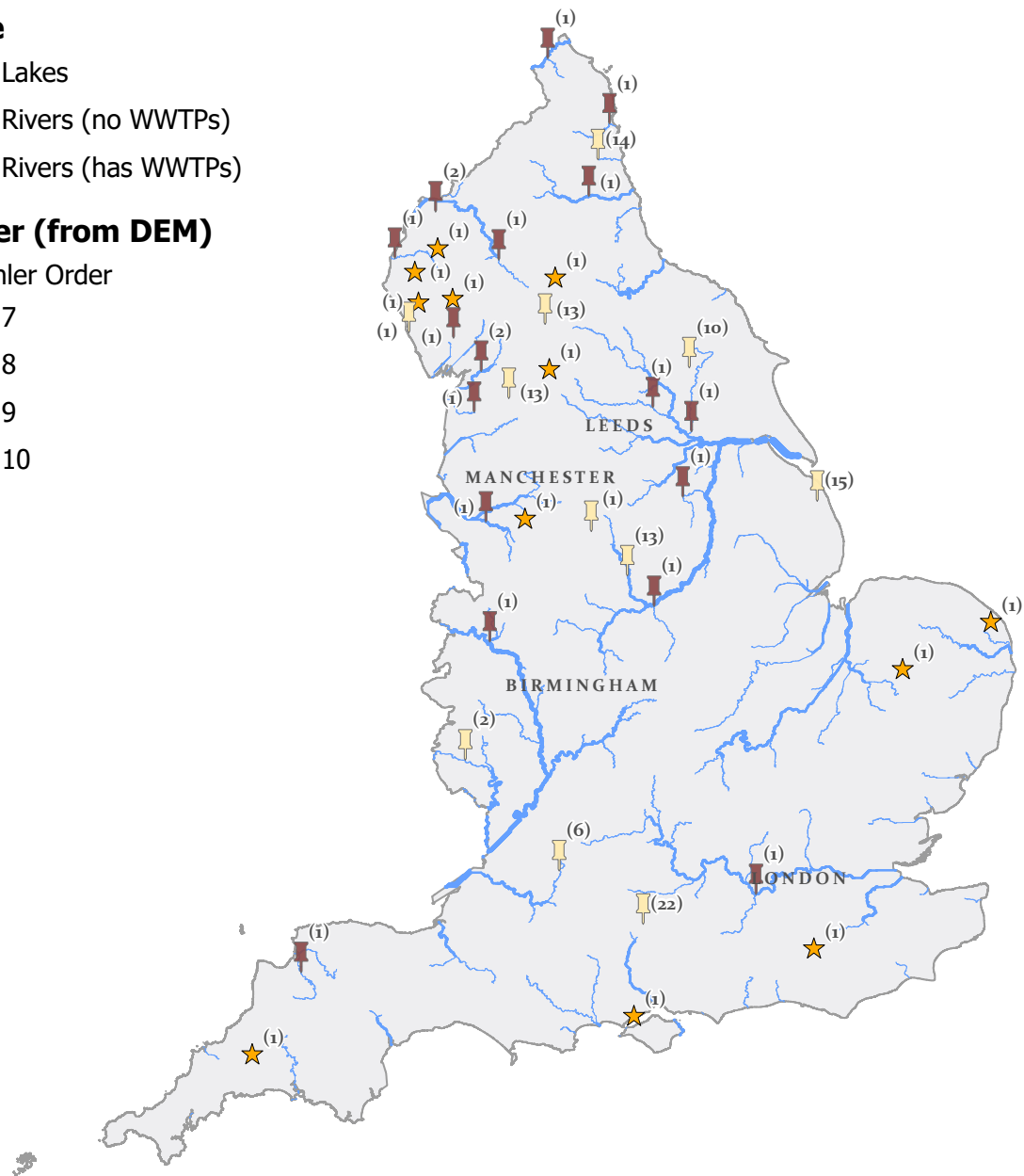


Fig. S4: Sites with no imidacloprid detections from 2019 to 2023 in the EA database. This subset of the entire database of 123 sites includes 28 sites with running water and 13 sites from standing water (lake, pond, reservoir). The number of samples at each site is annotated adjacent to the sites on the map. Blue = river network.

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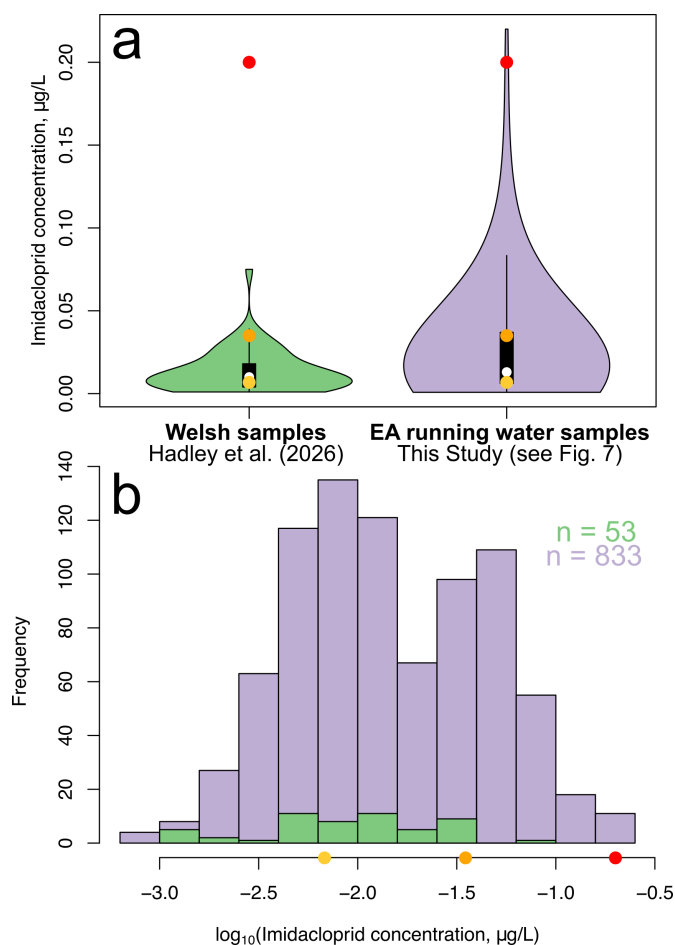


Fig. S5. Comparison of imidacloprid in Welsh rivers reported by Hadley et al. (2026) and from the EA fresh running water sites used in this study (see Fig. 7). (a) Violin plots showing median (white circles, interquartile range (black boxes), 1.5x IQR (lines), and the density of samples in both datasets. (b) Histograms of the samples in the two datasets transformed into log-space. Red, orange and yellow circles = acute, chronic, and lowest PNEC toxicity limits.

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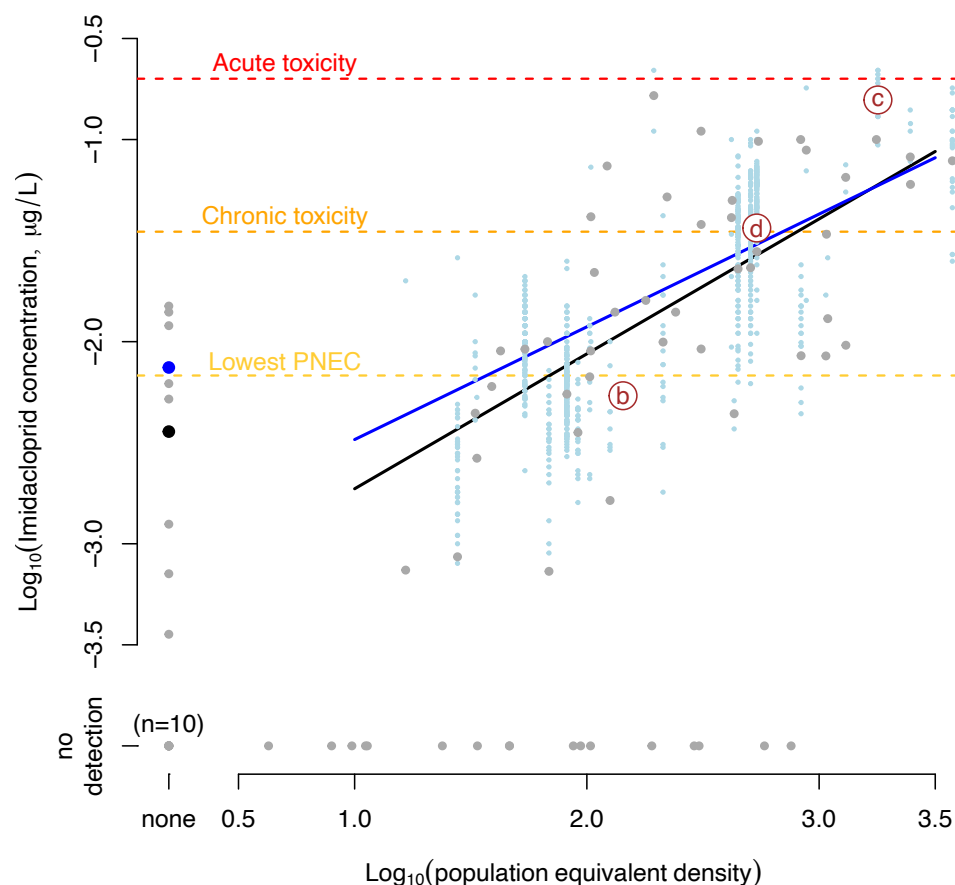


Fig S6. Relationships between imidacloprid concentrations at EA monitoring sites and pet populations in their upstream catchments. Extending the results shown in Fig 7. Light blue circles and gray circles = all measured concentrations above LOD and mean concentrations, respectively at each site; mean values calculated with LOD values set to zero; *population equivalent density* is used as a proxy for upstream pet population. Black line = relationship between mean imidacloprid concentration and *population equivalent density* across all sites; blue line = fit to all individual samples (taken from the lognormal model component; see Methods). See Fig. 6 for site locations; contextual hydrological and land use data for three labelled sites (b: 45400013, c: 16M08, d: PTHR0107) is given in Fig. S3. See Fig. 7 in main manuscript for extended description of annotations.

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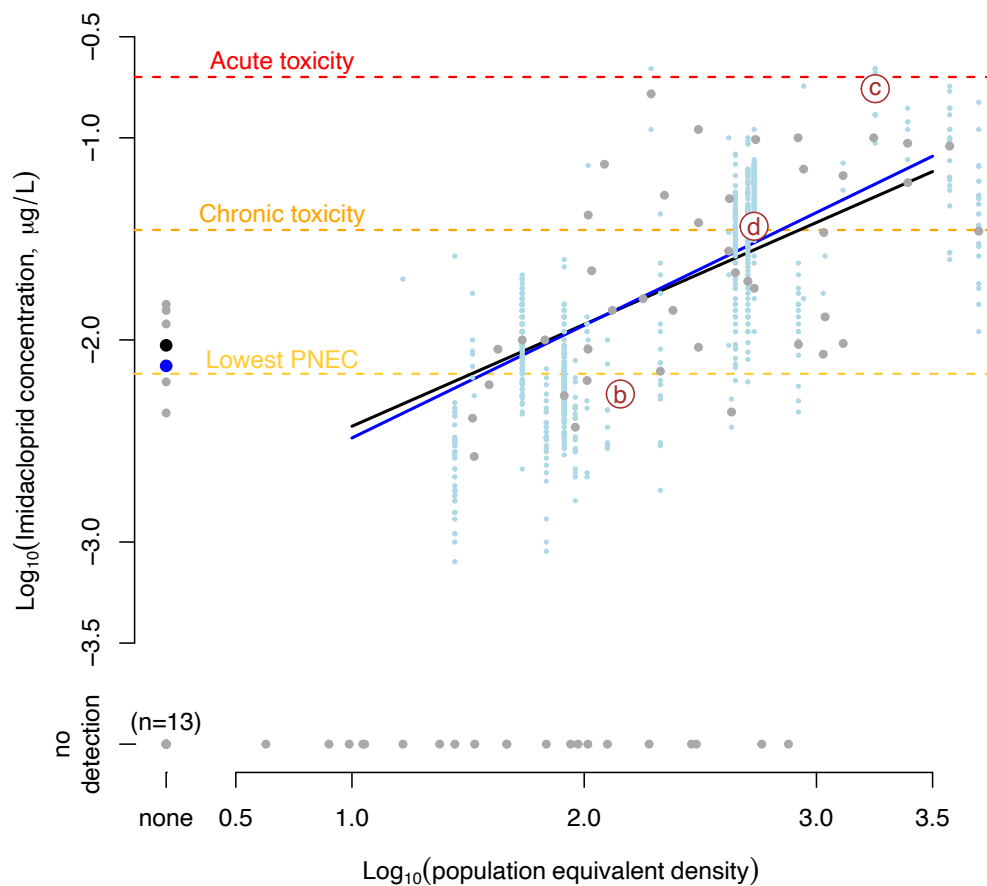


Fig S7. Relationships between imidacloprid concentrations at EA monitoring sites and pet populations in their upstream catchments. Extending the results shown in Fig 7. Light blue circles and gray circles = all measured concentrations above LOD and median concentrations, respectively at each site; median values calculated with LOD values set to zero; See Fig. S6 and 7 for extended description of annotations.

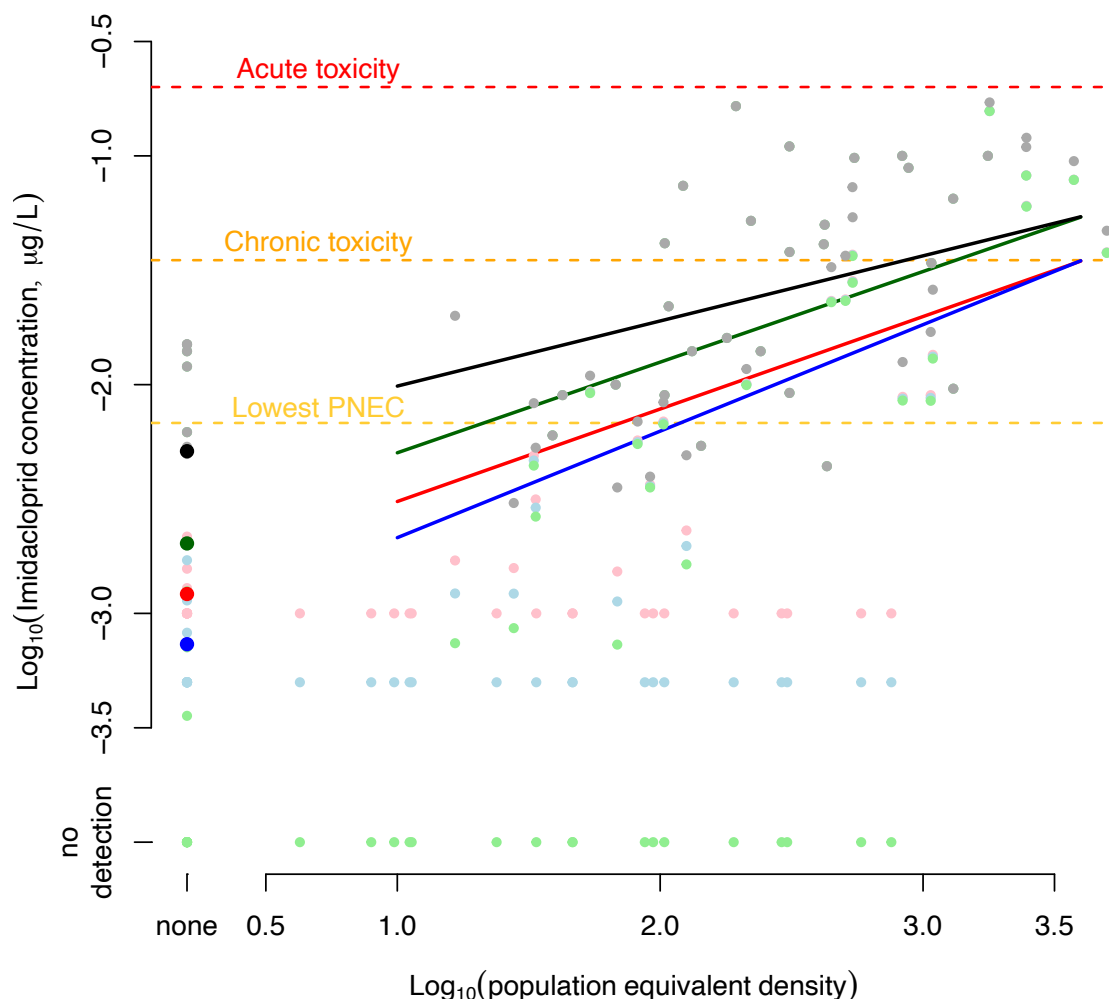


Fig. S8: Assessing impact of censored data on comparison of mean imidacloprid concentrations at freshwater monitoring sites and a proxy for pet population upstream. Linear regressions in log-log space of mean contaminant concentrations at each running water site with a mappable upstream catchment (N=85; samples collected 2019 to 2023 in English rivers and streams) are produced for different below-LOD substitute values; *population equivalent density* is used as a proxy for upstream pet population (see Methods). In the analysis, the 'non-detect' values are replaced by zero (green), 1/2 LOD = 0.0005 µg/L (blue), LOD = 0.001 µg/L (red) or are deleted from the analysis (black). Filled circles = mean concentrations at each site. Solid lines = Bayesian, non-hurdle, models fit to mean concentrations using. See Fig. 7 of the main manuscript and Fig. S6 for extended explanation of annotations.

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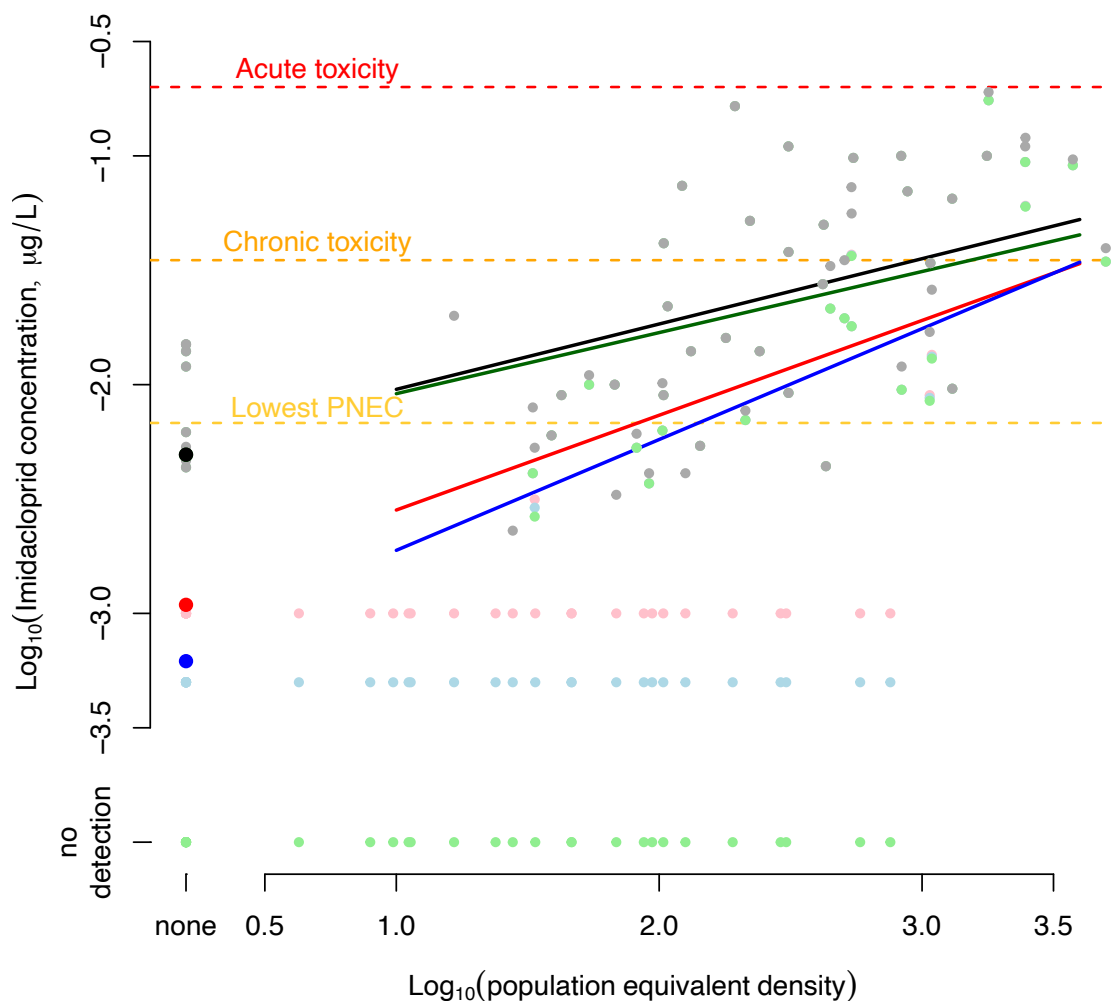


Fig. S9: Assessing impact of censored data on comparison of median imidacloprid concentrations at freshwater monitoring sites and a proxy for pet population upstream. See Fig. S6-S8 for extended explanation of annotations.

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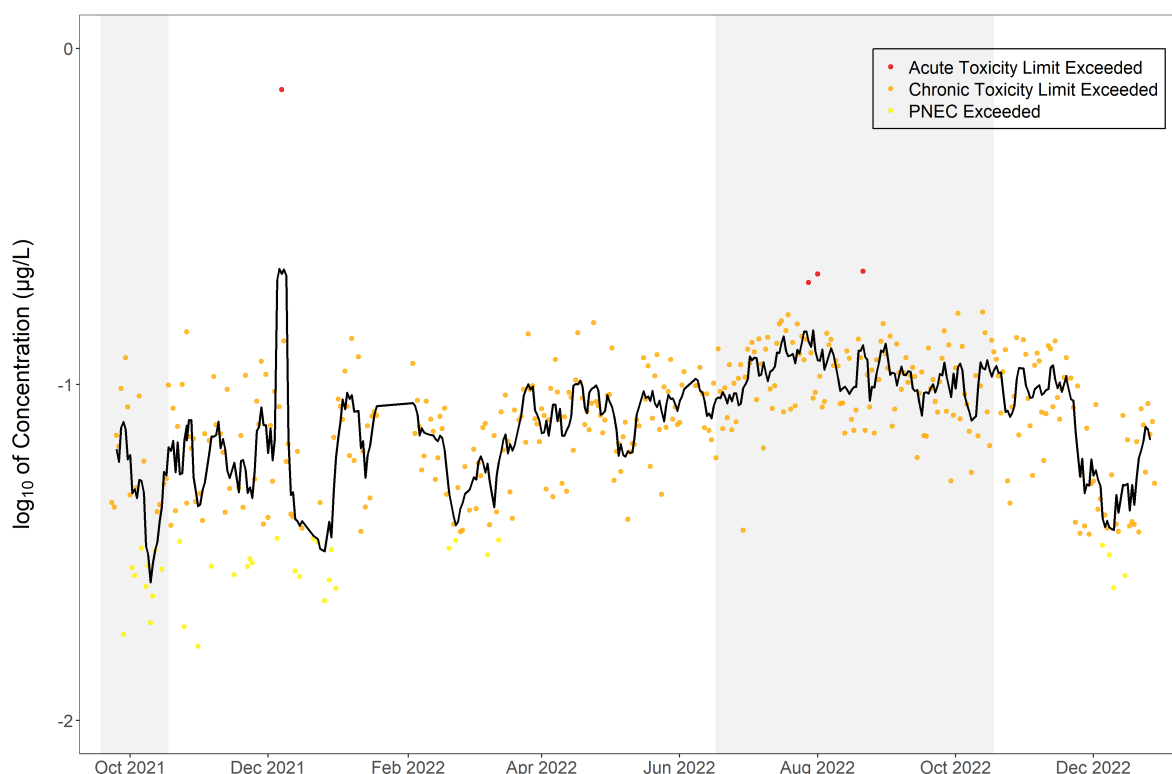


Fig. S10: Imidacloprid in untreated WWTP influent wastewater. Time series of 423 log-transformed imidacloprid concentrations (circles) in influent wastewater from a major wastewater treatment plant serving > 1 million people in the UK. Colors = toxicity limit exceedance: yellow = concentrations exceeding PNEC and below the chronic toxicity limit (8% of the 423 samples); orange = concentrations exceeding chronic limit and below the acute toxicity limit (91% of samples); red = concentrations above acute toxicity limit (1% of samples). Black line = moving average of concentrations. Shaded background = high flea infestation period.

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- ▲ CIP Sampling Point
- EA Site
- River (from DEM)
- Watershed

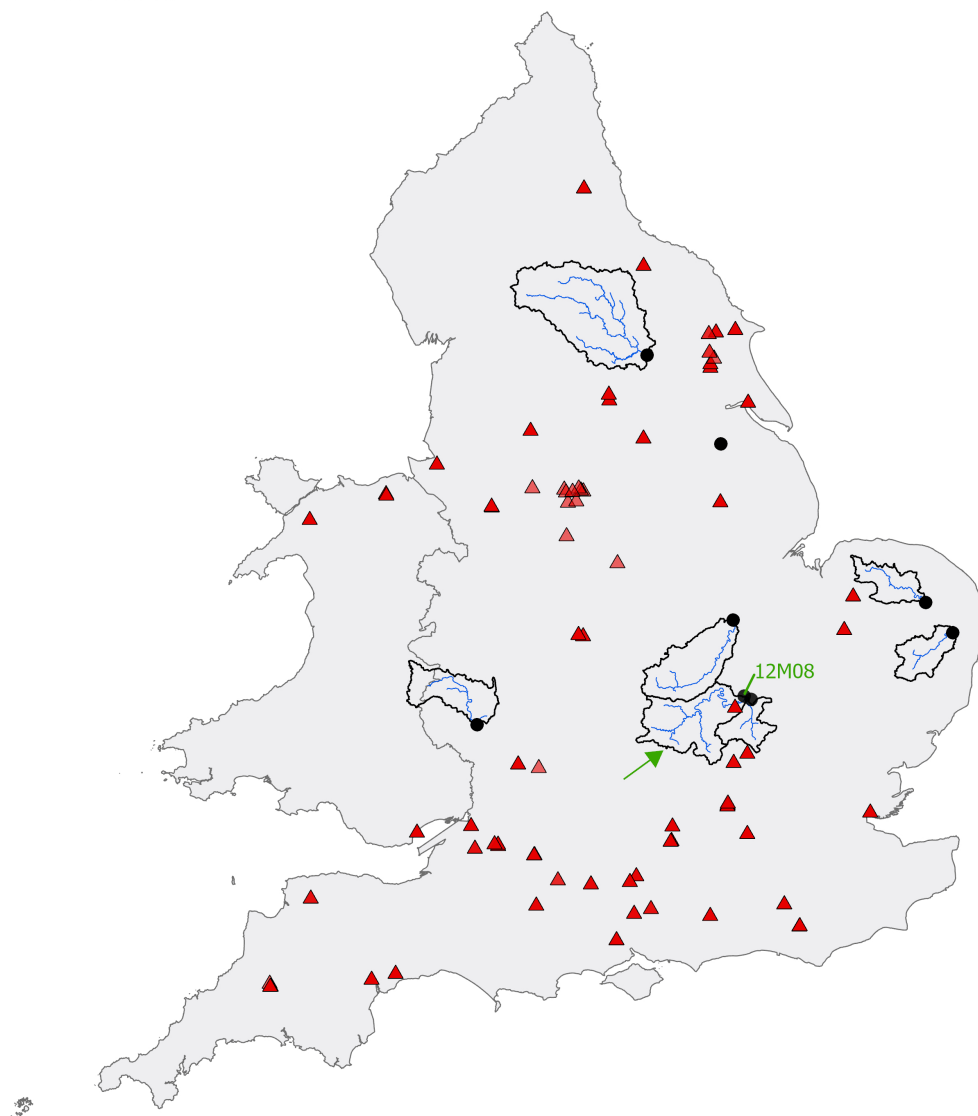


Fig. S11: Locations of imidacloprid measurements in WWTP effluent. Triangles = UKWIR CIP monitoring sites from 2019 to 2023. Circles = densely sampled EA monitoring sites shown in Fig. 1 & 4 in the main document, except site ANCN5 for which no watershed could be produced; blue lines and black polygons = upstream river networks and watersheds. Site 12M08 and its upstream watershed are labelled; see Fig. S12 for time series of concentrations.

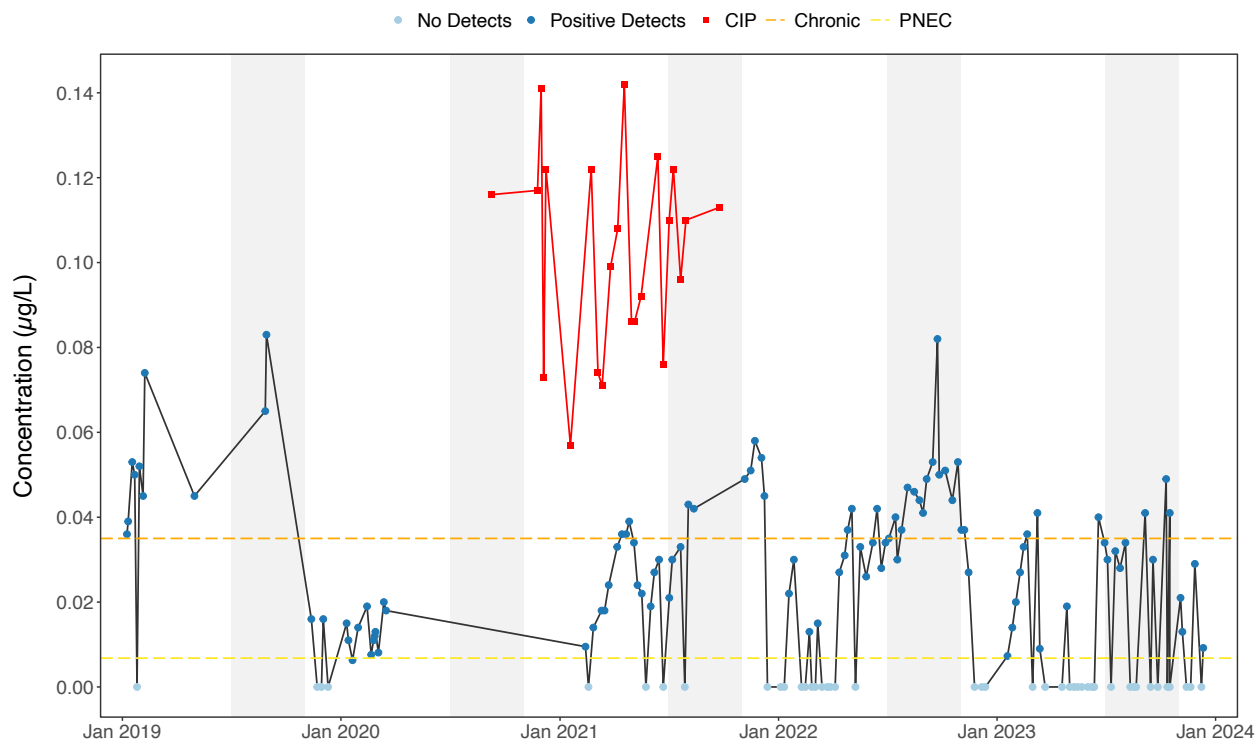


Fig. S12: Time series of imidacloprid concentrations at EA running water site 12M08 (blue) and in the effluent (red) of the upstream Bedford WWTP (see Fig. S11). Dark/light blue = positive/no (below LOD) detections of imidacloprid at the EA site. Yellow/orange dashed lines = PNEC/chronic toxicity limits. Shaded background = high flea infestation period.

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