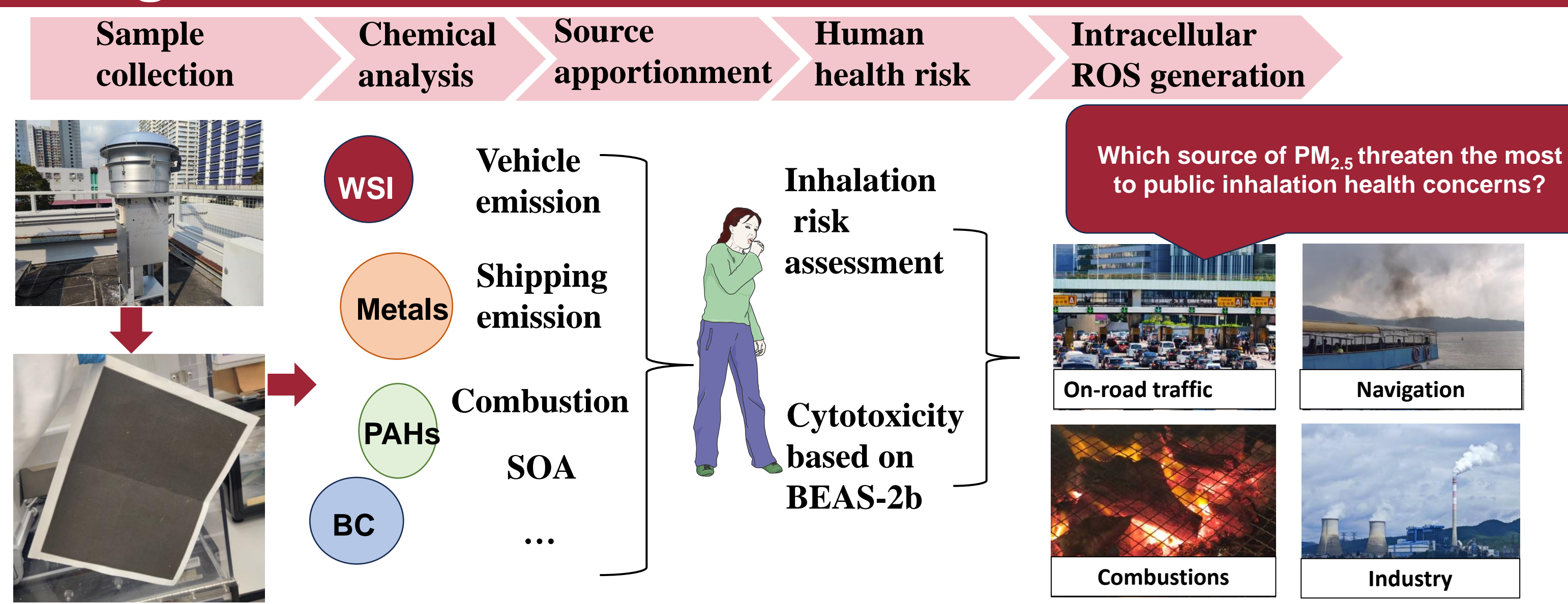


# Traffic-related PM<sub>2.5</sub> pollution in Hong Kong: source- and component-resolved health risks and cytotoxicity

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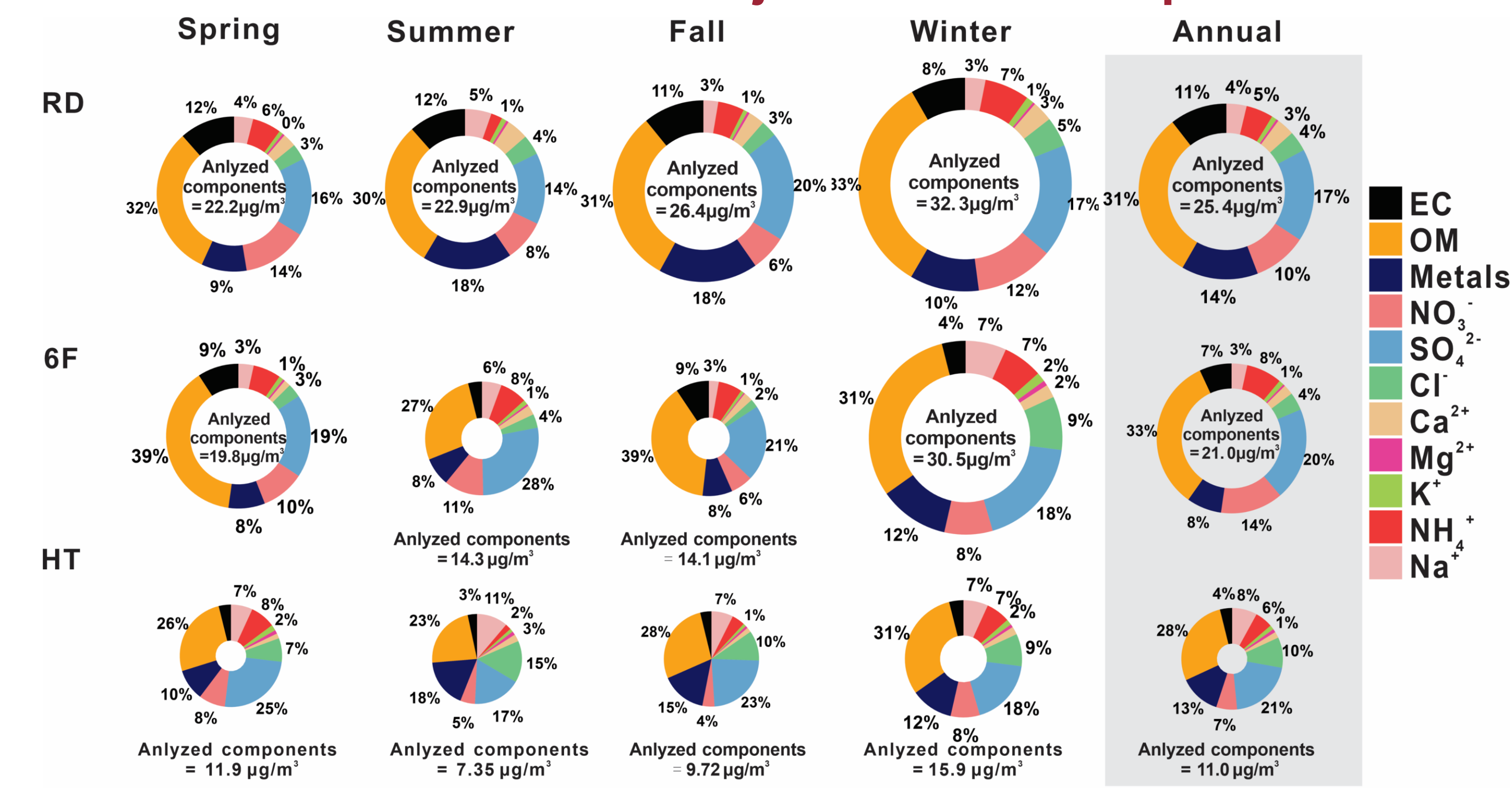
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## 1. Figure abstract



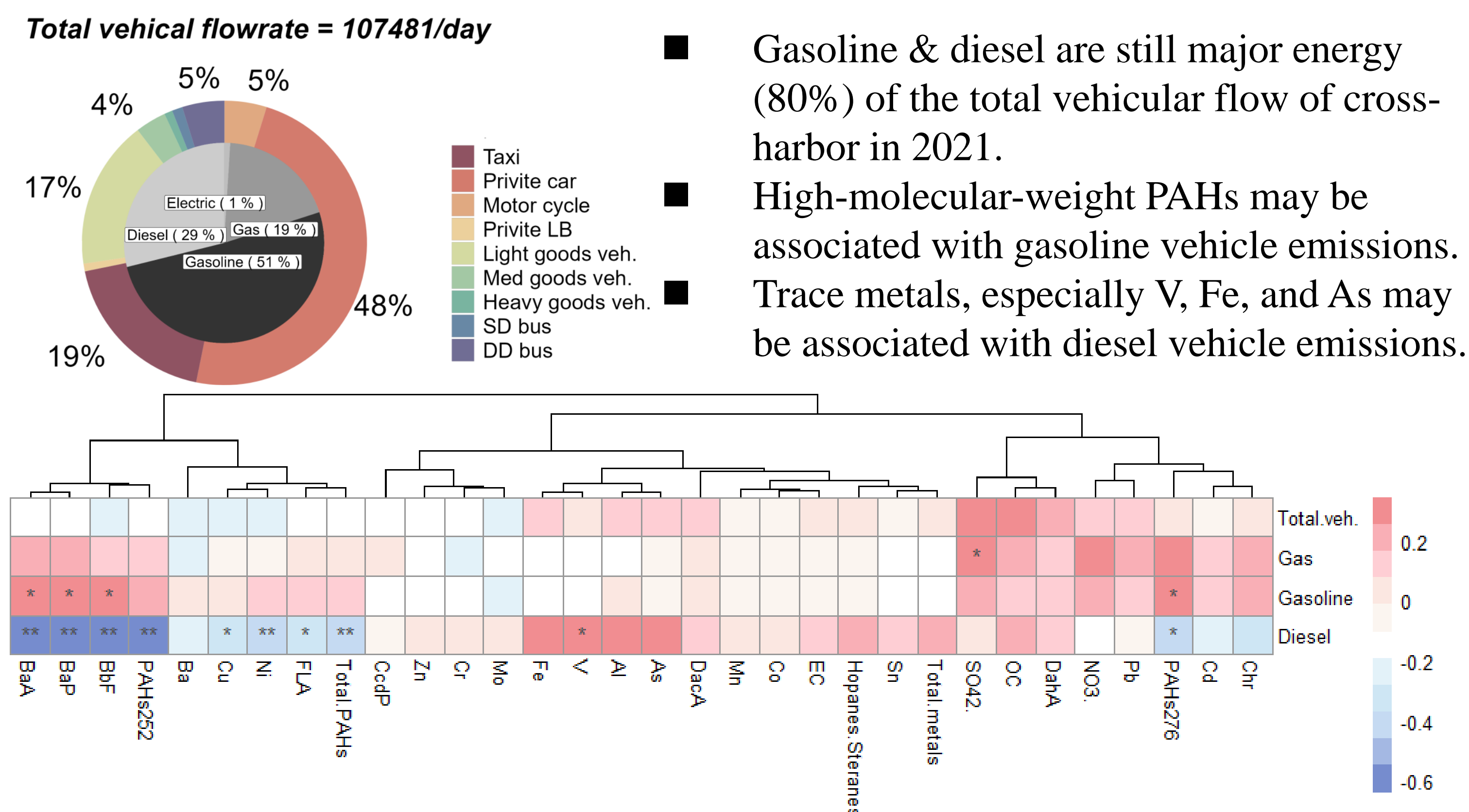
## 2. Chemical analysis

### 2.1 Seasonal variation of major chemical composition

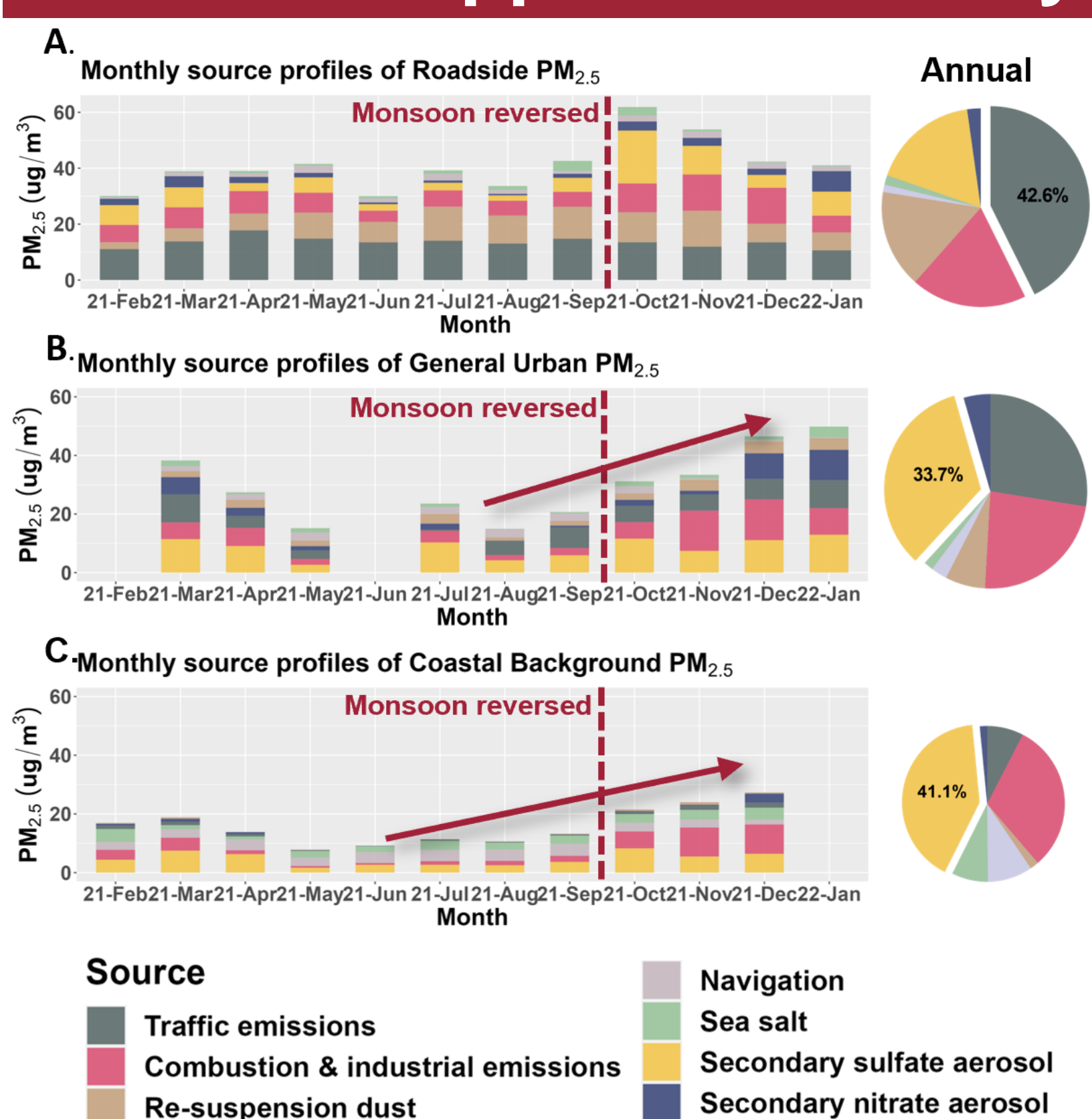


- Significant seasonal trend in total mass concentration: **Winter > Summer**
- Highest trace metals and elemental carbon (EC) contents in roadside samples.
- Secondary nitrate and sulfate contributed more to urban PM<sub>2.5</sub>.

### 2.2 Correlation analysis of vehicular characteristic with key toxic components in PM<sub>2.5</sub>



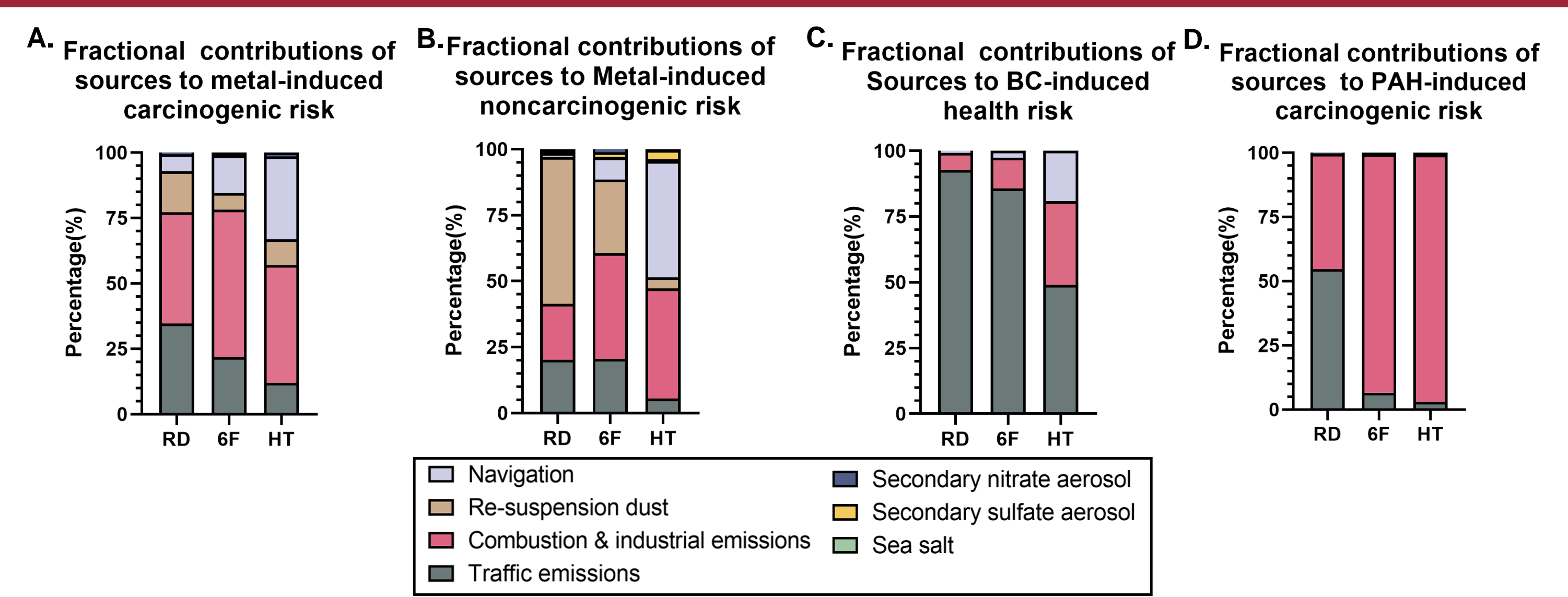
## 3. Source apportionment by PMF model



Site	1 <sup>st</sup> trace metals contributor	1 <sup>st</sup> PAHs contributor	1 <sup>st</sup> OC contributor
Roadside	Traffic emissions (38.7%)	Traffic emissions (57.6%)	Traffic emissions (54.7%)
Urban	Combustions (39.0%)	Combustions (52.3%)	Combustions (44.2%)
Background	Combustions (49.3%)	Combustions (82.5%)	Combustions (60.3%)

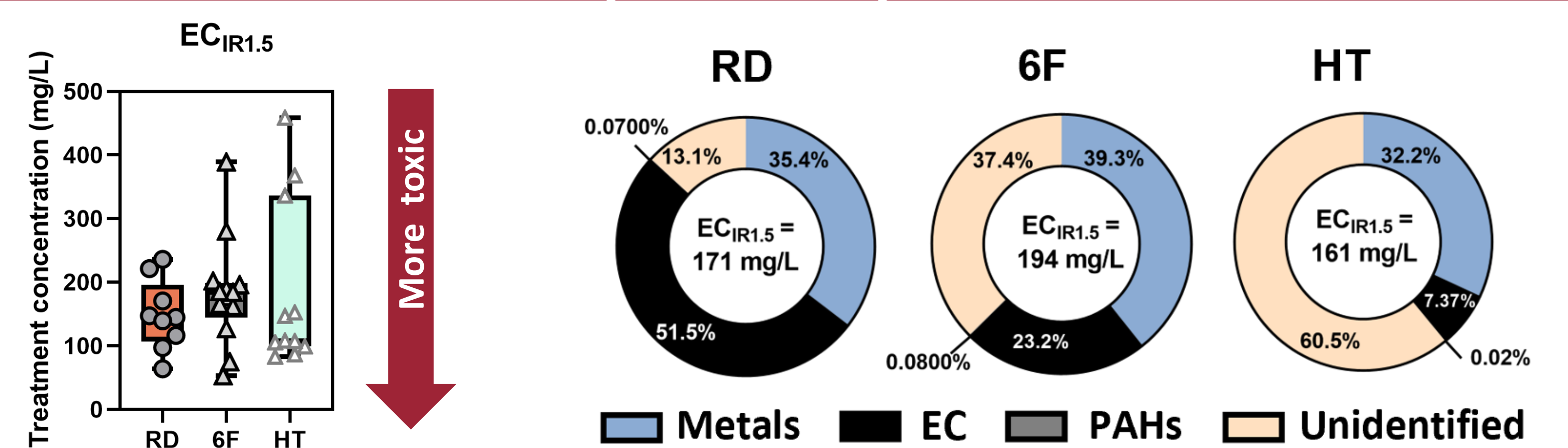
- EPA PMF 5.0. (Factors = 7, runs = 100) Tracers: OCEC, metals, ions, NPOCs
- Stationary variation → local source (e.g., Traffic emissions, Re-suspension dust, Navigation, and Sea salt.)
- Significant variation → regional source (e.g., Combustion & industrial emissions, SSA, SNA)

## 4. Source-resolved health risk assessment

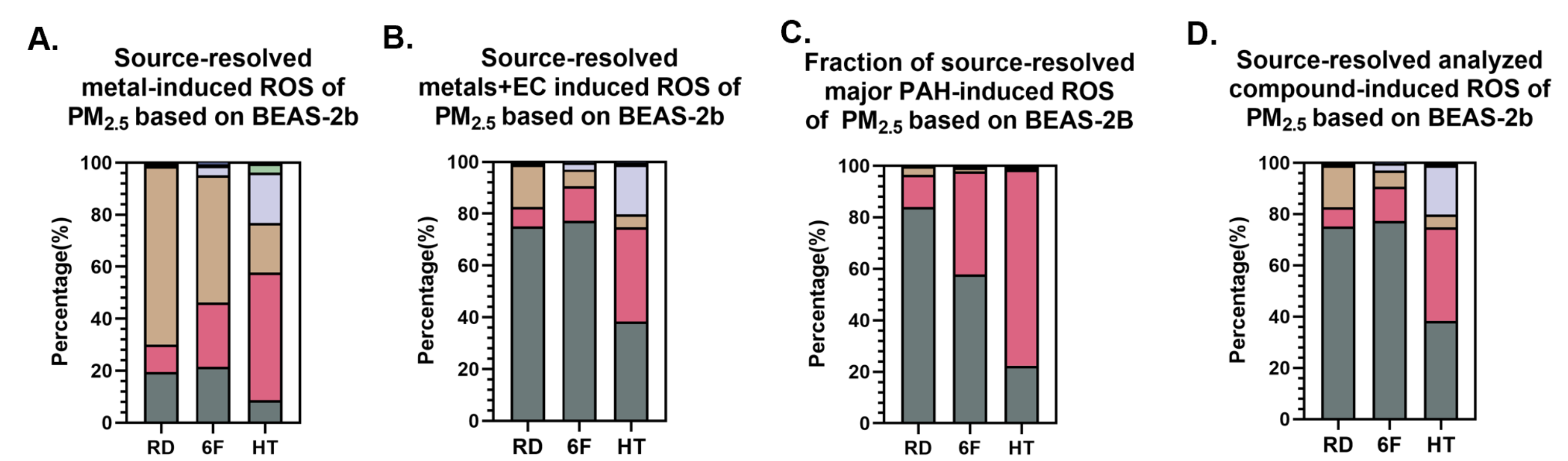


Site	1 <sup>st</sup> metal-induced CR contributor	1 <sup>st</sup> metal-induced NCR contributor	1 <sup>st</sup> BC-induced CR contributor	1 <sup>st</sup> PAH-induced CR contributor
Roadside	Combustions (42.6%)	Re-suspension dust (55.6%)	Traffic emissions (92.6%)	Traffic emissions (54.7%)
Urban	Combustions (56.5%)	Combustions (40.1%)	Traffic emissions (85.7%)	Combustions (93.0%)
Background	Combustions (45.1%)	Navigation (44.0%)	Traffic emissions (48.9%)	Combustions (96.2%)

## 5. Source-resolved cytotoxicity



- Trace metals are the dominating contributors to HK PM<sub>2.5</sub>-induced cytotoxicity, with an output of 20~45% in urban samples, and up to 40~80% in roadside samples, followed by **EC and PAHs**.



Site	1 <sup>st</sup> metal-induced intracellular ROS	1 <sup>st</sup> PAHs-induced intracellular ROS	1 <sup>st</sup> contributor for intracellular ROS induced by Metals+EC+PAHs
Roadside	Re-suspension dust (68.6%)	Traffic emissions (83.9%)	Traffic emissions (75.1%)
Urban	Re-suspension dust (48.8%)	Traffic emissions (57.7%)	Traffic emissions (77.3%)
Background	Combustions (49.0%)	Combustions (76.2%)	Combustions (36.4%)

- Combining all analyzed components, **traffic emissions** emerged as the predominant source of intracellular ROS induced by PM<sub>2.5</sub> in Hong Kong, in which importance should be attached to **non-exhaust emissions**.

## 6. Conclusions

- This investigation **first** linked source profiles with component-specific cytotoxicity of PM<sub>2.5</sub>, putting forward the **source-resolved cytotoxicity** assessment.
- The **local fresh sources were more dominant** in the ROS induction, comparing with the regional sources under long-term transport.
- Our health risks assessment identified **traffic-related sources** as the primary contributors to health risks and intracellular ROS caused by **trace metals, EC** in urban areas.
- Attention should also be paid to non-exhaust traffic-related emissions. Fe, Zn, Mn, and Cu, which are typically linked to friction and road dust, contribute the major part to the non-carcinogenic risk and cell ROS induction.
- Our study firstly estimated the **EC-specific contribution** in intracellular ROS, which draws further attention to the toxicity induced by the insoluble fractions in PM<sub>2.5</sub>.