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Epidemiological Patterns of Chromosomal Congenital Anomalies: A Geospatiotemporal and Causal Inferential Study of Cannabis and Substance Use

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Abstract

Laboratory research connects chromosomal missegregation defects to cannabis exposure. Recent epidemiological studies support this association and raise concerns about elevated chromosomal congenital anomaly rates (CCAR) in Europe, where cannabis use, daily use intensity, and cannabinoid potency are all increasing. Methods: Eurocat's CCAR data. statistics from the European Monitoring Center for Drugs and Drug Addiction on drug use. earnings from the World Bank. Analysis of panel, geotemporal, bivariate, and multivariate regressions. E-values and inverse probability panel model weighting are the two main quantitative causal inferential methods.

Keywords: Mutagenesis • Transgenerational inheritance • Cannabinoid • Cancerogenesis.

Introduction

A number of recent epidemiological investigations investigating the connections and causes of various prenatal drug exposures, including cannabis, have focused on chromosomal congenital abnormalities (CCAs). Congenital chromosomal defects have been frequently included on long lists of congenital anomalies enhanced following prenatal or community marijuana exposure in publications from Hawaii, Colorado, Canada, Australia, and the United States. It was interesting to find out if trends seen elsewhere might be reflected in European data given that most of Europe has recently experienced a triply convergent rise in the prevalence of cannabinoid use, the intensity of daily use, and the 9-tetrahydrocannabinol (THC) content of available cannabis products. It is likely that cannabis exposure has increased given that all three prongs of this triple convergence tend to increase cannabinoid exposure increased on the ground more than any of these widely used measures may convey. In fact, more recent research have shown that combined metrics of cannabis exposure accurately reflect the patterns of genotoxic and congenital abnormality problems than do individual, more widely used metrics [1].

Data

From the European Network of Population-Based Registries for the Epidemiological Surveillance of Congenital Anomalies (EUROCAT) website, data on all accessible congenital anomaly rates were downloaded by each individual year for each of 14 countries and examined. In order to provide a comprehensive picture across all types of births, the EUROCAT total congenital abnormality rate combines anomaly rates from live births, stillbirths, and instances when early termination for anomaly was used. The countries were picked based on the availability of their congenital anomaly data for the majority of the years 2010–2019. Data on national alcohol and tobacco use, including percent daily tobacco usage prevalence and pure alcohol drank per capita yearly, were downloaded from the World Health Organization use of drugs [2].

Data Imputation

Linear interpolation was used to fill in the missing data. This was especially true for regular cannabis use. For these 14 countries throughout this time period, 59 EMCDDA data points on daily cannabis use were available. This dataset's 129 datapoints were added by linear interpolation (further details provided in Results section). There were no data on the THC content of cannabis resin for Sweden. However, it was discovered that the ratio of resin to herb THC concentration in neighbouring Norway was practically constant at 17.7, so this ratio was used to calculate estimations of the THC content of Swedish cannabis resin using data on the herb's THC concentration. Similarly, there were no data on the THC content of cannabis resin in Poland. Germany's neighbouring resin to herb THC content ratio was utilised to extrapolate the known Polish plant THC concentrations to the resin THC content in Poland. For Croatia in 2018 and 2019, and The Netherlands in 2010, the dataset was completed by the latest observation carried forward or backward because geospatial analytical tools do not accept missing data. For this dataset, multiple imputation techniques were inappropriate since they cannot be used with panel or spatial multivariable regression methods [3].

Discussion

In a significant recent whole genome DNA methylation screen, 163 differentially methylated regions (DMRs) involving hundreds of genes across the genome were shown to be affected by cannabis dependency, and 127 DMRs then emerged following 11 weeks of cannabis withdrawal. This process involved chromosomal assembly (CDK1), Philadelphia-chromosome-negative acute lymphoblastic leukaemia, homologous pairing of chromosomes (MSH5, RAD21L1, SMC1B, and SYCP3), and three functional annotations from the Ingenuity Pathway Analysis discovered for chromosomal genes (CD3D, EPHA2, FYN, KMT2A). DNA mismatch repair and DNA crossing over during meiosis are both facilitated by MSH5, MutS homolog 5. Its abnormalities have been linked to infertility in both sexes as well as azoospermia and early ovarian failure. Male prophase I of the cell's RAD21L1 Double Strand Break Repair Protein Meiosis, sister chromatid paring, crossing over, creation of synaptonemal complexes, and beginning of synapse [4].

It results in spermatogenic failure when mutated. A vital component of the meiosis-specific cohesin complex that holds sister chromatids together and

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participates in DNA recombination events is SMC1B, Structural Maintenance of Chromosomes. It manifests in the testicles, heart, and brain. Gonadal dysgenesis and corneal dystrophy are caused by mutatio. In the course of meiosis I, SYCP3, or Synaptonemal Complex Protein 3, which is expressed in the germ cells of the human testis, regulates chromosomal segregation, recombination activities, and the creation of synaptonemal complexes. Infertility, testicular degeneration, spermatogenic failure, and azoospermia are the results of its mutations in males, whereas pregnancy loss is the result in females. At the very edge of cell cycle control, CDK1 is the serine/threonine cyclin dependent kinase I. In addition to controlling cell cycle entrance, mitotic spindle formation and disintegration, centromere assembly, and many other aspects of cell cycle progression, CDK1 phosphorylates over 75 binding partners. It is also tightly controlled by a number of interconnected control systems, including positive feedback loops that serve like switches to abruptly activate or deactivate their functions. It regulates entry into crucial cell cycle phases such the G1/S and G2/M transitions [5-10].

Conclusion

Data offer significant evidence demonstrating that various metrics of cannabis use, notably daily usage, are strongly linked with all seven of the chromosomal diseases evaluated in inverse probability weighted and causal inferential models. These results support decades of in vitro laboratory research, many recent epidemiological series from Hawaii, Colorado, Australia, Canada, and the United States, as well as a previous, less complex analysis of the same dataset. As previously mentioned, the field is open for genotoxic and epigenotoxic mechanistic studies to look into the epigenetic mechanisms of chromosomal mis-segregation and mitotic spindle dysfunction, cannabinoid-induced chromosomal scission, and the gross disorders of meiosis I that result in an increase in whole genome doubling events and are a part of the testicular cancer oncobiogenesis pathway and cellular pathology of epigenomic aging.

Acknowledgement

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Conflict of Interest

None.

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