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Original Article

Clinicodemographic Profile of BCR: ABL1-Positive Chronic Myeloid Leukemia in a Tertiary Care Hospital

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ABSTRACT

Background: The BCR:ABL1 fusion gene resulting from the Philadelphia chromosome translocation is the hallmark of chronic myeloid leukemia (CML), a myeloproliferative neoplasm. Early diagnosis through hematological and molecular investigations is essential for prompt initiation of targeted therapy and improved outcomes. This study aimed to evaluate the demographic and hematological characteristics of BCR:ABL1 (p210)-positive CML patients attending a tertiary care hospital in Dhaka, Bangladesh. **Methods & Materials:** This cross-sectional study was conducted among 45 patients with confirmed BCR:ABL1-positive CML attending the Department of Microbiology and Immunology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh from January 2023 to June 2023. **Results:** A total of 45 patients with BCR-ABL (p210 kDa) positive CML were evaluated, with the highest frequency observed in the 35–44 years age group. Hemoglobin levels ranged from 12.2 g/dL to 16.9 g/dL, with a mean value of 10.44 ± 2.48 g/dL. The majority of patients (57.79%) had hemoglobin levels between 7 g/dL and 11 g/dL. Leukocyte counts exceeding 100×10⁹/L were observed in 48.89% of cases. Platelet counts ranged from 16×10⁹/L to 2100×10⁹/L, with a mean value of 409.13 ± 380.52×10⁹/L. Most patients presented during the chronic phase of the disease with less than 10% peripheral blood blasts. **Conclusion:** The study demonstrated that CML in Bangladesh is more commonly diagnosed among younger and middle-aged adults compared with Western reports. Patients showed variable hematological abnormalities, particularly leukocytosis and moderate anemia. Molecular detection of BCR:ABL1 transcripts by qPCR remains essential for accurate diagnosis and characterization of CML.

Keywords: *Chronic myeloid leukemia, BCR:ABL1, p210 transcript, hematological profile.*

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INTRODUCTION

Chronic myeloid leukemia (CML) is a clonal myeloproliferative neoplasm characterized by uncontrolled proliferation of myeloid precursor cells within the bone marrow, leading to excessive accumulation of mature and immature granulocytic cells in the peripheral blood. It represents one of the most extensively studied hematologic malignancies due to its distinct molecular pathogenesis and characteristic cytogenetic abnormality [1]. The hallmark of CML is the presence of the Philadelphia (Ph) chromosome, which originates from a reciprocal translocation between the long arms of chromosomes 9 and 22, designated as t(9;22)(q34;q11) [2–4]. This chromosomal rearrangement results in the formation of the BCR::ABL1 fusion gene, a constitutively active tyrosine kinase responsible for leukemogenesis and uncontrolled cellular proliferation [5].

The Philadelphia chromosome can be identified in approximately 90–95% of patients diagnosed with CML and is considered a major diagnostic feature of the disease [6]. However, a small proportion of patients may demonstrate a normal karyotype despite exhibiting clinical and hematological characteristics suggestive of CML. Among these individuals, some harbor cryptic or occult BCR::ABL1 rearrangements that cannot be detected by conventional cytogenetic methods but can be identified through molecular diagnostic techniques [7]. Therefore, molecular detection of the BCR::ABL1 fusion transcript plays a crucial role in confirming the diagnosis, particularly in Philadelphia chromosome-negative cases [8].

The ABL1 proto-oncogene, located on chromosome 9, encodes a non-receptor tyrosine kinase involved in cellular differentiation, division, and adhesion. The breakpoint cluster region (BCR) gene, situated on chromosome 22, fuses with ABL1 following chromosomal translocation, resulting in the formation of an abnormal chimeric oncogene [9]. Depending on the location of the breakpoint within the BCR gene, different fusion transcripts may be produced. The major breakpoint cluster region (M-BCR) is most commonly associated with CML, whereas minor (m-BCR) and micro (μ-BCR) breakpoints are less frequently encountered [10]. In the majority of CML patients, fusion of exon 13 (e13/b2) or exon 14 (e14/b3) of the BCR gene with exon 2 (a2) of the ABL1 gene generates the e13a2 (b2a2) or e14a2 (b3a2) transcripts, respectively. These transcripts encode the p210 BCR:ABL1 oncoprotein, which is strongly associated with classical CM [11].

Molecular identification of BCR::ABL1 transcripts has become an essential component in the diagnosis and monitoring of CML. Reverse transcriptase polymerase chain reaction (RT-PCR) is recognized as one of the most sensitive and reliable techniques for detecting BCR::ABL1 fusion transcripts [12]. Among the available molecular approaches, multiplex RT-PCR has gained widespread acceptance because of its ability to simultaneously detect multiple transcript variants, including both major and minor breakpoint rearrangements [13]. This method not only facilitates accurate diagnosis but also assists in disease classification, therapeutic monitoring, and evaluation of treatment response.

The demographic and hematological characteristics of CML patients may vary across different populations and geographical regions. Previous studies have reported that the disease predominantly affects middle-aged and older adults, with a median age ranging between 40 and 65 years, although

pediatric and adolescent cases have also been documented [14,15]. A slight male predominance has consistently been observed in several studies [16]. Hematological parameters such as total leukocyte count, hemoglobin concentration, platelet count, and differential leukocyte count are important determinants for assessing disease burden and prognostic stratification. Several prognostic scoring systems, including Sokal, Hasford, and EUTOS scores, utilize demographic and hematological variables to categorize patients into different risk groups and predict treatment outcomes [17,18].

In developing countries such as Bangladesh, environmental and socioeconomic factors may contribute to the increasing burden of hematological malignancies. Rapid urbanization, widespread exposure to environmental pollutants, pesticide use, smoking, radiation exposure, and food adulteration have been considered possible contributors to genetic instability and carcinogenesis [19]. Despite the growing number of CML cases in Bangladesh, limited data are available regarding the hematological and demographic profiles of patients with BCR:ABL1-positive CML. Moreover, information concerning the distribution of specific transcript variants among Bangladeshi patients remains inadequate.

Therefore, the present study was conducted to evaluate the hematological and demographic characteristics of patients with BCR:ABL1-positive chronic myeloid leukemia and to determine the distribution of p210 BCR:ABL1 transcript variants among patients attending a tertiary care hospital in Dhaka, Bangladesh.

METHODS & MATERIALS

This cross-sectional observational study was conducted in the Department of Microbiology and Immunology at Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh from January 2023 to June 2023 to evaluate the hematological and demographic characteristics of patients with BCR::ABL1-positive chronic myeloid leukemia (CML). A total of 45 patients with established CML, irrespective of age and gender, attending Bangladesh Medical University (BMU) during the study period were recruited. Diagnosis of CML was based on hematological findings, peripheral blood examination, bone marrow findings where available, and molecular detection of BCR:ABL1 transcripts.

Approximately 10 mL of peripheral venous blood was collected aseptically from each participant by clean venipuncture into sterile ethylenediaminetetraacetic acid (EDTA) tubes. Of this, 3 mL was preserved for molecular analysis and stored at 4°C, while processing was completed within three days to maintain RNA integrity. Another 2 mL was used for complete blood count (CBC) analysis and hematological profiling using an automated hematology analyzer (Sysmex XN-1000™, Sysmex Corporation, Japan), including hemoglobin concentration, total leukocyte count, platelet count, and differential leukocyte count.

Molecular detection and characterization of BCR:ABL1 transcripts were performed using RNA extraction, cDNA synthesis, and quantitative real-time PCR (qPCR). Total RNA was extracted from peripheral blood leukocytes using the QIAamp nucleic acid extraction kit following manufacturer instructions, and cDNA synthesis was performed using reagents from the TRUPCR® BCR-ABL1 kit (Ref: 3B1267, 3B BlackBio Biotech India Ltd., India). qPCR was conducted to detect major (M-BCR), minor (m-BCR), and micro (μ-BCR) transcripts using the same kit. Each sample was analyzed in four reactions with a total volume of 20 μL, containing 10 μL of 2× PCR master mix, 4 μL of RNase-free water, 5 μL of cDNA template, and 1 μL of primer-probe mix specific for each target, with ABL1 used as an internal control. Six serial standards ranging from STD1 (1.08×10^6 copies) to STD6 (1.08×10^1 copies) were included for standard curve generation and quantification.

Data were analyzed using SPSS v25.0, with qualitative variables expressed as frequency and percentage and quantitative variables as mean ± standard deviation. Ethical approval and informed consent were exempted as anonymized laboratory data were used in accordance with institutional policy and IVI IRB SOP D-RB-4-003.

RESULTS

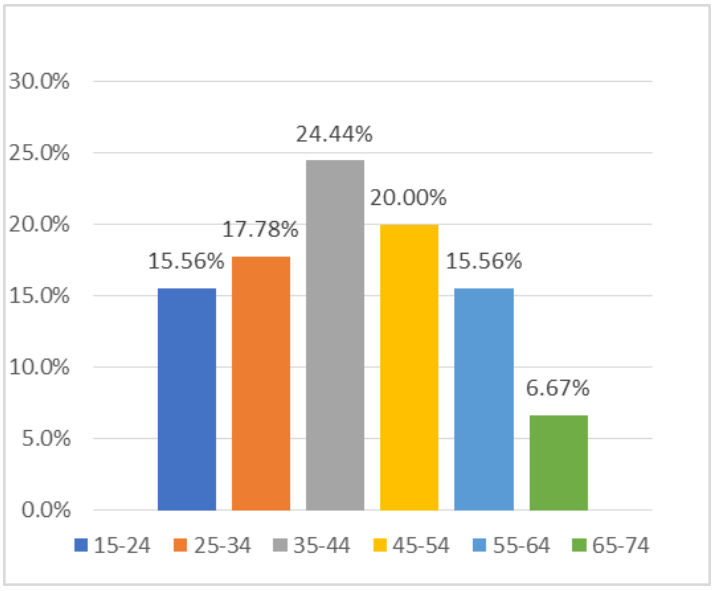

Figure 1 Age Distribution of Our Study Participants.

A total of 45 patients with confirmed chronic myeloid leukemia (CML) were included in the study. The age distribution of the study population demonstrated that the highest proportion of patients belonged to the 35–44 years age group (24.44%), followed by the 45–54 years age group (20.00%). Patients aged 25–34 years constituted 17.78% of cases, while both the 15–24 years and 55–64 years age groups accounted for 15.56% each. The lowest frequency of CML was observed among

patients aged 65–74 years (6.67%) (*Figure 1*).

The demographic and hematological characteristics of the study participants are summarized in *Table I*. A total of 45 patients with confirmed chronic myeloid leukemia (CML) were enrolled in the study. The age of the patients ranged from 15 to 74 years, with a mean age of 38.73 ± 16.90 years. Male predominance was observed, as 28 (62.2%) patients were male and 17 (37.8%) were female, resulting in a male-to-female ratio of 1.64:1.

The mean total leukocyte count was 149.88 ± 147.28 ×10⁹/L, indicating marked leukocytosis among the study population. The mean hemoglobin level was 10.44 ± 2.48 g/dL, reflecting varying degrees of anemia. In addition, the mean platelet count was 409.13 ± 380.52 ×10⁹/L, with a wide variation observed among the patients.

Table I
Clinical and laboratory details of CML patients in study population.

Number of patients (n)	45
Mean age (years)	38.73± 16.90
Range of age (years)	15–74
Male/female (n)	28/17
Male-to-female ratio	1.64:1.0
Mean leukocytes count (×109/L)	149.88 ± 147.28
Mean hemoglobin levels (g/dL)	10.44 ± 2.48
Mean platelets count (×109/L)	409.13 ± 380.52

Hematological data:

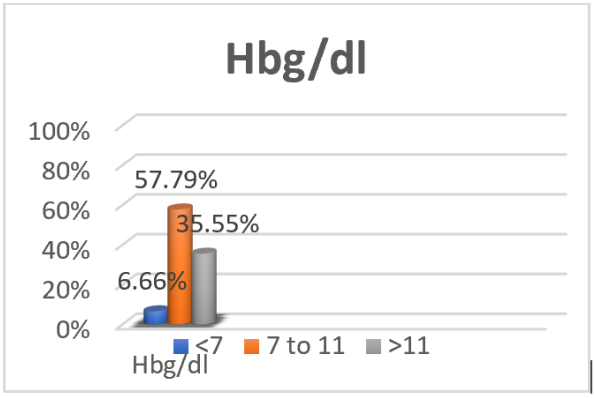

Figure 2(a): Represents the frequency of all study patients with different ranges of hemoglobin concentration.

About (64.45%) of patients were anemic. Hemoglobin value ranged from 12.2 to 16.9 with a mean value of 10.44 ± 2.48g/dL. Most of the patients (57.79%) had a hemoglobin level between 7 to 11 g/dL. The anemia was moderate for 57.79% and severe for 6.66% of the study group. (*Figure 2a*).

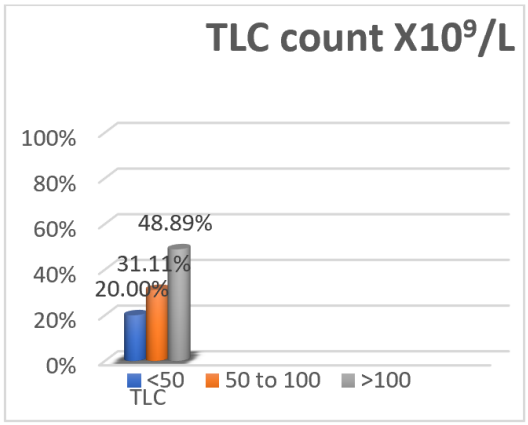

Figure 2(b): Represents the frequency of all study patients with different ranges of leucocyte count.

All patients presented the marked leukocytosis involving the granulocytic lineage, associated with early myeloid cells. Leukocytosis ranged from 5.37 to 500.13×109/L with a mean value of 149.88 ± 147.28 ×109/L. Most of the patients (48.89%) had their leukocyte count >100×109/L. Twenty percent patients had leukocyte values <50×109/L. (*Figure 2b*).

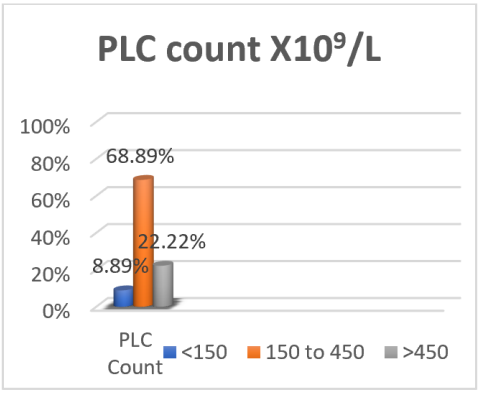

Figure 2(c): Represents the frequency of all study patients with different ranges of platelet count.

Platelet count ranged from 16 to 2100×109/L with a mean value of 409.13 ± 380.52×109/L. Most of patients (68.89%) had platelets count between 150 to 450×109/L, while 8.89% had thrombocytopenia and 22.22% patients had thrombocytosis (*Figure 2c*).

Table II
Salient Hematological Profile of patients in CML at the time of diagnosis.

Parameters	(n=45)	
	n	%
Age (years)		
<45	32	71.11
>45	13	28.89
Gender		
Male	28	62
Female	17	38
Haemoglobin levels (g/dL)		
<7 g/dL	3	6.66
7-11 g/dL	26	57.79
>11 g/dL	16	35.55
Total Leukocyte count 109/L		
<50	9	20
50-100	14	31.11
>100	22	48.89
Platelet count x 109/L		
<150	4	8.89
150-450	31	68.89
>450	10	22.22
Blast (in number)		
1-9	34	75.7
10-19	03	6.6
≥20	08	17.7

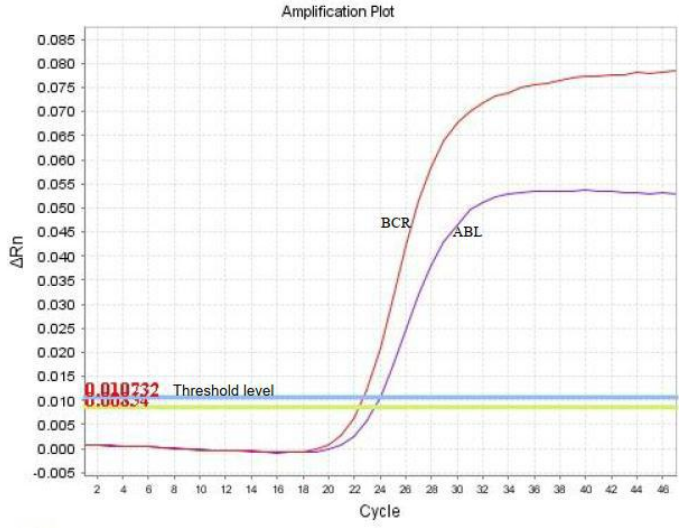

Figure 3: Real-time RT-PCR amplification plots of the BCR/ABL fusion transcripts.

The representative real-time quantitative PCR (qPCR) amplification plot demonstrating BCR:ABL1 fusion transcript detection in a patient with chronic myeloid leukemia (CML) is illustrated in *Figure 3*. The horizontal line parallel to the X-axis represents the fluorescence threshold level used for determination of cycle threshold (Ct) values. Amplification curves crossing the threshold line indicate positive amplification of the target gene.

In the present study, all analyzed samples (45/45, 100%) demonstrated amplification of the typical major BCR:ABL1 (p210) fusion transcript, confirming the molecular diagnosis of CML. Distinct amplification curves were observed for both the BCR::ABL1 target gene and the ABL1 internal control gene, indicating adequate RNA quality, successful cDNA synthesis, and efficient PCR amplification.

DISCUSSION

A total of 45 patients with confirmed chronic myeloid leukemia (CML) were included in the present study. The study was conducted to evaluate the demographic and hematological characteristics of patients with BCR::ABL1 p210-positive CML attending a tertiary care hospital in Dhaka, Bangladesh. Among the enrolled patients, 28 (62.22%) were male and 17 (37.78%) were female, yielding a male-to-female ratio of 1.64:1.0, which indicates a clear male predominance in the study population.

A previous study conducted in Bangladesh also reported a higher frequency of CML among males, where 67% of the patients were male and 33% were female, with a male-to-female ratio of 2.1:1.0 [19]. Comparable findings have been documented in neighboring countries. Studies from India and Pakistan demonstrated male-to-female ratios of 1.9:1.0 and 1.7:1.0, respectively [20,21]. Similarly, studies conducted by Chang et al. and Kumar et al. observed a predominance of male patients among individuals with CML, reporting male-to-female ratios of 1.4:1 and 1.6:1, respectively [22,23]. Another study from India and the United Kingdom also demonstrated similar demographic distributions [20].

The predominance of male patients observed in the present study and in previous reports may be associated with greater occupational and environmental exposure among males to potential leukemogenic factors such as pesticides, industrial chemicals, smoking, and ionizing radiation. In addition, sociocultural and healthcare-seeking behaviors may also contribute to the comparatively higher detection rate of CML among males in developing countries.

In the present study, the mean age of the patients was 38.73 ± 16.9 years, with an age range of 15–74 years. Similar findings were reported by Kumar et al. from Uttar Pradesh, India, where the mean age of CML patients was 38.6 years, which closely corresponds with the current observation [23]. In Iraq, the reported mean age of patients with CML was comparatively higher at 50.4 years [24]. Studies conducted in several African countries also demonstrated comparable age distributions among CML patients. Chetcha et al. in Cameroon, Segbena et al. in Togo, Oyekunle et al. in Nigeria, Silué et al. in Ivory Coast, and Mupepe et al. in the Democratic Republic of Congo reported mean ages of 39 years, 40 years, 38 years, 39 years, and 40 years, respectively [25–29].

In contrast, studies from high-income countries reported comparatively higher mean ages at diagnosis. Furthermore, the reported mean age in France and the United States was 55 years and 66 years, respectively, which is considerably higher than that observed in the present study [30,31].

These epidemiological variations suggest that CML tends to be diagnosed at a younger age in low- and middle-income countries compared with developed nations [32–34]. Several factors may contribute to this disparity, including differences in population demographics, environmental exposures, genetic susceptibility, healthcare accessibility, life expectancy, and patterns of disease detection. In developing countries, a larger proportion of the population belongs to younger age groups, which may also influence the observed lower mean age at diagnosis of CML.

In countries with high population density such as Bangladesh, continual exposure to various environmental and lifestyle-related carcinogenic factors, including chemical pollutants, food adulterants, excessive pesticide application, cigarette smoking, and radiation, may play a significant role in increasing the risk of developing chronic myeloid leukemia (CML), particularly among younger populations. Uncontrolled urbanization and inadequate regulation of environmental hazards may further contribute to the growing burden of hematological malignancies in developing nations. In contrast, these potential risk factors are relatively less common or more strictly regulated in Europe and North America, which may partly account for the differences in the age of presentation between developed and developing countries.

In the current study, most patients were observed within the fourth and fifth decades of life. This observation is in agreement with the findings of Gupta et al., who reported that 45.94% of their Indian CML patients belonged to the fifth decade of age [35].

Complete blood count (CBC), peripheral blood film examination, and bone marrow aspiration were performed in 100%, 58.8%, and 2.6% of the study patients, respectively. In the present study, the mean total leukocyte count, hemoglobin concentration, and platelet count were 149.8 ×10⁹/L, 10.44 g/dL, and 409.1 ×10⁹/L, respectively. Similar hematological findings were reported in an Indian study, where the mean white blood cell count, hemoglobin level, and platelet count were 153.3 ×10⁹/L, 9.7 g/dL, and 448 ×10⁹/L, respectively [35]. However, Chang et al. reported comparatively different hematological parameters, with mean white blood cell count, hemoglobin level, and platelet count of 121 ×10⁹/L, 9.5 g/dL, and 285 ×10⁹/L, respectively [22].

In the current study, hemoglobin levels ranged from 4.7 to 16.9 g/dL, and anemia was observed in 64.45% of patients. Most patients had hemoglobin concentrations between 7 and 11 g/dL. Moderate anemia was identified in 57.7% of cases, whereas severe anemia was present in 6.6% of the study population. These findings are comparable to those reported by Kueviakoe et al., who documented anemia in 68.9% of CML patients, with the majority exhibiting moderate anemia [36].

In the present study, platelet counts ranged from 42 ×10⁹/L to 500 ×10⁹/L, with a mean platelet count of 409 ×10⁹/L. The majority of patients (68.89%) had platelet counts within the normal range (150–450 ×10⁹/L), whereas thrombocytosis was observed in 22.2% of cases. Similar observations were reported by Mupepe et al. and Mukiibi et al., who also found that most CML patients had normal platelet counts at presentation [29,37].

Analysis of peripheral blood findings demonstrated that 75.7% of the patients had less than 10% blast cells in peripheral blood, suggesting that most cases were diagnosed during the chronic phase of the disease. Blast percentages ranging from 10% to 19% were identified in 6.6% of patients, while eight patients exhibited ≥20% blasts in peripheral blood, indicating progression toward advanced disease phases. Among the immature myeloid cells observed, myelocytes and metamyelocytes were the predominant forms detected in peripheral blood smears. Comparable findings were reported in a study conducted in Pakistan, where myelocytes were identified as the most prominent immature white blood cells in patients with CML [22].

LIMITATIONS

This study has several limitations. It was conducted as a single-center study with a relatively small sample size, which may limit the generalizability of the findings to the broader population of Bangladesh. The study was also cross-sectional in design, preventing assessment of longitudinal changes in hematological or molecular parameters over time or response to therapy. In addition, limited clinical and prognostic data were available, restricting correlation between molecular subtypes and disease outcomes. Furthermore, cytogenetic analysis and advanced molecular risk stratification tools were not incorporated, which could have provided a more comprehensive understanding of disease heterogeneity.

CONCLUSION

This study provides a concise overview of the demographic and hematological characteristics of patients with BCR::ABL1-positive chronic myeloid leukemia (CML) confirmed by molecular techniques in a tertiary care hospital in Bangladesh. The findings indicate that CML predominantly affects middle-aged individuals, particularly those aged 35–54 years, with a clear male predominance. The majority of patients presented with classical hematological abnormalities, including marked leukocytosis, anemia of varying severity, and altered platelet counts, with moderate anemia being the most frequent finding. The p210 BCR::ABL1 transcript was identified as the predominant molecular variant among the study population, reinforcing its diagnostic significance. Overall, the study highlights the importance of early molecular detection using real-time PCR for accurate diagnosis, disease characterization, and improved clinical management of CML.

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CONFLICTS OF INTEREST

There are no conflicts of interest.

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