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Jaíne Cocco Uliana

CONDIÇÃO BUCAL EM PACIENTES COM TRANSTORNO DO ESPECTRO AUTISTA (TEA): UMA REVISÃO SISTEMÁTICA DE ESTUDOS OBSERVACIONAIS COM GRUPO CONTROLE

Santa Maria, RS
2022

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Dissertação de Mestrado apresentada ao Curso de Mestrado do Programa de Pós-Graduação em Ciências Odontológicas, Área de Concentração em Odontologia, ênfase em Periodontia, da Universidade Federal de Santa Maria (UFSM, RS), como requisito parcial para obtenção do título de **Mestre em Ciências Odontológicas**.

Orientadora: Prof.ª Dr.ª Karla Zanini Kantorski

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Jaíne Cocco Uliana

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Aprovado em 25 de agosto de 2022:

_____

Karla Zanini Kantorski, Dr.ª (UFSM)
(Presidente/Orientador)

_____

Cristine Miron Stefani, Dr. ª (UnB)

_____

Fabiana Vargas Ferreira, Dr.ª (UFMG)

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2022

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"É justo que muito custe o que muito vale"

Santa Tereza D'Avila

RESUMO

CONDIÇÃO BUCAL EM PACIENTES COM TRANSTORNO DO ESPECTRO AUTISTA (TEA): UMA REVISÃO SISTEMÁTICA DE ESTUDOS OBSERVACIONAIS COM GRUPO CONTROLE

AUTORA: Jaíne Cocco Uliana
ORIENTADORA: Karla Zanini Kantorski

Indivíduos com transtorno do espectro autista (TEA) podem ser mais suscetíveis a desenvolverem doenças bucais crônicas não transmissíveis quando comparados aos indivíduos sem outras condições ou neuroatipias. Isso pode ser devido à seletividade alimentar, uso de medicamentos e padrões comportamentais associados a comportamentos orais prejudiciais. O objetivo desse estudo foi revisar sistematicamente a literatura a fim de comparar cárie, higiene bucal, doenças periodontais, bruxismo, má oclusão, perda dentária e alterações salivares entre indivíduos com TEA e indivíduos controle sem outras condições ou neuroatipias. Pesquisas eletrônicas foram realizadas nas bases de dados EMBASE, Latin American and Caribbean Health Sciences (LILACS), PubMed, PsycINFO, Scopus, Web of Science, além de literatura cinzenta (Google Scholar e ProQuest) sem restrição de período de publicação. Busca manual por estudos adicionais também foi realizada. Os softwares EndNote e Rayyan foram usados para gerenciar referências e auxiliar na remoção de duplicados. Foram incluídos somente estudos observacionais (transversal, coorte e caso-controle), que avaliaram as condições bucais na população com TEA em comparação com os indivíduos controle sem TEA ou qualquer outra neuroatipia apresentando medidas clínicas dos desfechos orais. Dois revisores independentes realizaram a seleção dos estudos em duas fases, a extração dos dados com formulário padronizado e a avaliação da qualidade metodológica dos estudos por meio da escala Newcastle-Ottawa. Metanálises de diferenças médias padronizadas (SMD) e risco relativo/razão de prevalência (RR/RP) foram realizadas. 42 estudos compreendendo um total de 7217 indivíduos foram incluídos na revisão. As metanálises revelaram que indivíduos autistas apresentaram significativamente maior severidade de cárie em dentes decíduos (SMD 0.29, 95% CI 0.04-0.53), maior severidade de lesões de cárie não tratadas (SMD 0.27, 95% CI 0.06 to 0.48, I^2 88%), maior prevalência (RR 2.46, 95% CI 1.23 to 4.91, I^2 83%) e severidade (SMD 0.59, 95% CI 0.24 to 0.94) de pior condição de higiene bucal e de gengivite (RR 1.31, 95% CI 1.02 to 1.70) (SMD 0.45, 95% CI 0.02 to 0.88, I^2 95%), pH salivar significativamente mais baixo (SMD -0.62, 95% CI -0.99 to -0.26, I^2 46%), maior prevalência de bruxismo (RR 4.52, 95% CI 2.07 to 9.86, I^2 85%), sobressaliência aumentada (RR 2.16, 95% CI 1.28 to 3.64, I^2 89%), sobremordida aumentada (RR 1.62, 95% CI 1.02 to 2.59, I^2 80%), mordida cruzada (RR 1.48, 95% CI 1.02 to 2.13, I^2 57%) e mordida aberta (RR 2.37, 95% CI 1.46 to 3.85, I^2 54%) quando comparados aos indivíduos controle neurotípicos. Maiores médias de dentes e superfícies restauradas foram significativamente encontradas nos indivíduos controle (SMD -0.30, 95% CI -0.49 to -0.10, I2 85%). Em geral, as análises de subgrupo envolvendo estudos com alto risco de viés e ausência de variáveis de pareamento fortaleceram as associações. Nossos achados sugerem que os indivíduos autistas apresentam pior condição de saúde bucal quando comparados a controles neurotípicos.

Palavras-chave: Transtorno do Espectro Autista. Cárie Dentária. Placa Dentária. Gengivite. Bruxismo. Má Oclusão. Saliva. Revisão Sistemática.

ABSTRACT

ORAL STATUS IN PATIENTS WITH AUTISM SPECTRUM DISORDER (ASD): A SYSTEMATIC REVIEW OF OBSERVATIONAL STUDIES WITH CONTROL GROUP

AUTHOR: Jaíne Cocco Uliana
ADVISOR: Karla Zanini Kantorski

Individuals with autism spectrum disorder (ASD) may be more susceptible to developing chronic non-communicable oral diseases when compared to individuals without other conditions or neuroatypia. This may be due to food selectivity, medication use, and behavioral patterns associated with harmful oral behaviors. The aim of this study was to systematically review the literature in order to compare caries, oral hygiene, periodontal diseases, bruxism, malocclusion, tooth loss and salivary alterations between subjects with ASD and control subjects without other conditions or neuroatypia. Electronic searches were performed in EMBASE, Latin American and Caribbean Health Sciences (LILACS), PubMed, PsycINFO, Scopus, Web of Science databases, in addition to gray literature (Google Scholar and ProQuest) without restriction of publication period. Manual search for additional studies was also performed. EndNote and Rayyan software were used to manage references and assist in removing duplicates. Only observational studies (cross-sectional, cohort and case-control) that evaluated oral conditions in the ASD population compared to control subjects without ASD or any other neuroatypia presenting clinical measures of oral outcomes were included. Two independent reviewers carried out the selection of studies in two phases, extracting data with a standardized form and evaluating the methodological quality of the studies using the Newcastle-Ottawa scale. Meta-analyses of standardized mean differences (SMD) and relative risk/prevalence ratio (RR/PR) were performed. 42 studies comprising a total of 7217 subjects were included in the review. Meta-analyses revealed that autistic subjects had significantly greater caries severity in primary teeth (SMD 0.29, 95% CI 0.04-0.53), greater severity of untreated caries lesions (SMD 0.27, 95% CI 0.06 to 0.48, I^2 88%) , higher prevalence (RR 2.46, 95% CI 1.23 to 4.91, I^2 83%) and severity (SMD 0.59, 95% CI 0.24 to 0.94) of worse oral hygiene and gingivitis condition (RR 1.31, 95% CI 1.02 to 1.70) (SMD 0.45, 95% CI 0.02 to 0.88, I^2 95%), significantly lower salivary pH (SMD -0.62, 95% CI -0.99 to -0.26, I^2 46%), higher prevalence of bruxism (RR 4.52, 95 % CI 2.07 to 9.86, I^2 85%), increased overjet (RR 2.16, 95% CI 1.28 to 3.64, I^2 89%), increased overbite (RR 1.62, 95% CI 1.02 to 2.59, I^2 80%), crossbite (RR 1.48, 95% CI 1.02 to 2.13, I^2 57%) and open bite (RR 2.37, 95% CI 1.46 to 3.85, I^2 54%) when compared to neurotypical control subjects. Higher means of filled teeth and surfaces were significantly found in control subjects (SMD -0.30, 95% CI -0.49 to -0.10, I2 85%). In general, subgroup analyzes involving studies with a high risk of bias and absence of matching variables strengthened the associations. Our findings suggest that autistic individuals have worse oral health status when compared to neurotypical controls.

Keywords: Autism Spectrum Disorder. Dental Caries. Dental Plaque. Gingivitis. Bruxism. Malocclusion. Saliva. Systematic Review [Publication Type].

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LISTA DE ABREVIATURAS E SIGLAS

CAOD	*Total number of carried, absent and obturated teeth*
CAOS	*Total number of carried, absent and obturated surface*
CPITN	*Community periodontal index of treatment needs*
DAI	*Dental aesthetic index*
DMFS	*Total number of permanent surfaces Decayed, Missing, Filled*
DMFT	*Total number of permanent teeth Decayed, Missing, Filled*
dmfs	*Total number of deciduous surfaces Decayed, Missing, Filled*
dmft	*Total number of deciduous teeth Decayed, Missing, Filled*
DS	*Total number of permanent decayed surfaces*
ds	*Total number of deciduos decayed surfaces*
DT	*Total number of permanent decayed teeth*
dt	*Total number of deciduos decayed teeth*
FS	*Total number of permanent filled surfaces*
fs	*Total number of deciduos filled surfaces*
FT	*Total number of filled permanent teeth*
ft	*Total number of filled primary teeth*
GBI	*Gingival Bleeding Index*
GI	*Gingival Index*
MGI	*Modified Gingival Index*
MS	*Total number of permanent missing surfaces*
ms	*Total number of deciduous missing surfaces*
MT	*Total number of permanent missing teeth*
mt	*Total number of deciduous missing teeth*
OHI-S	*Simplified Oral Hygiene Index*
PI	*Plaque Index*

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1 INTRODUÇÃO

O transtorno do espectro autista (TEA) é uma condição persistente do neurodesenvolvimento, caracterizado frequentemente por limitações nas interações sociais, dificuldade na comunicação verbal e não verbal, comportamentos repetitivos ou estereotipados e dissonância cognitiva, que se manifestam precocemente na vida (AMERICAN PSYCHIATRIC ASSOCIATION, 2013; WHO, 2021). Automutilação, agressão, birras e transtornos mentais, convulsões, hipersensibilidade aos estímulos sensoriais e determinados hábitos orais podem ser observados em crianças com TEA (FRIEDLANDER et al., 2006; STEIN et al., 2011). A prevalência de TEA é muito difícil de estabelecer devido à sua natureza altamente variável e às dificuldades inerentes ao diagnóstico da doença (WHO, 2021), mas houve aumento no número de diagnósticos de TEA nas últimas décadas (MELDRUM et al., 2013); segundo Maenner et al. (2021), a prevalência geral de TEA foi de 23,0 por 1.000 (uma em 44) crianças de 8 anos.

Indivíduos com transtorno do espectro autista (TEA) parecem ser mais suscetíveis a desenvolverem doenças bucais crônicas não transmissíveis quando comparados aos indivíduos sem outras condições ou neuroatipias (FERRAZANO et al., 2020; JABER, 2011; VAJAWAT; DEEPIKA, 2012). Maloclusões (ALJUBOUR; AL-SEHAIBANY, 2018; ALMUSAWI; AL-DABAGH, 2019; FONTAINE-SYLVESTRE et al., 2017), pior estado de higiene oral e doenças periodontais (AL-MAWERI et al., 2014; BHANDARY; NARY, 2017; VAJAWAT; DEEPIKA, 2012), cárie (JABER, 2011; LEIVA-GARCÍA et al., 2019; SUHAIB et al., 2019), perda dentária (NAMAL; VEHIT; KOKSAL, 2007; ONOL; KIRZIOGLU, 2018; ORELLANA et al., 2012), hábitos parafuncionais como o bruxismo (DANESHVAR et al., 2019; EL KATHIB, 2014; LEIVA-GARCÍA et al., 2019) alterações salivares de fluxo salivar, pH e capacidade tampão (BHANDARY; NARY, 2017; BLOMQVIST; BEJEROT; DAHLLÖF, 2015; DIAB et al., 2016) têm se mostrado mais prevalentes em indivíduos com TEA quando comparados aos indivíduos neurotípicos.

Associação entre TEA e cárie pode estar relacionada a comportamentos comumente observados neste perfil de indivíduos. Indivíduos com TEA apresentam seletividade a certos tipos de alimentos em função da textura e cor (CERMAK; CURTIN; BANDINI, 2010), preferência por alimentos adocicados e tendência de manter o alimento na boca ao invés de engoli-lo, devido à deficiência motora oral (KLEIN; NOWAK, 1998). A oferta de alimentos com alto teor de açúcar é muitas vezes utilizada por pais e educadores especiais como recompensa para prevenir hábitos de automutilação e reforçar comportamentos direcionados

(ONOL; KIRZIOGLU, 2018), podendo resultar em aumento significativo de cárie (KOTHA et al., 2018).

Ainda, estima-se que 64% das crianças com TEA têm prescrição de pelo menos um medicamento psicotrópico (SPENCER et al., 2013), classe de droga mais prescrita para esse perfil de indivíduos (HSIA et al., 2014). Crianças com TEA, mais velhas e com alguma comorbidade psiquiátrica, apresentam maior prevalência de polifarmácia psicotrópica (JOBSKI et al., 2007; SPENCER et al., 2013). Essas drogas estão fortemente associadas ao fluxo salivar reduzido e à capacidade tampão prejudicada (WOLFF et al., 2017), o que favorece o desenvolvimento da cárie (FLINK et al., 2019; QUILICI; ZECH et al., 2019; CUNHA-CRUZ et al., 2013). Revisão sistemática de da Silva et al. (2016), observou que a prevalência de cárie e de doença periodontal foi de 60.6% (IC 95% 44.0-75.1) e 69.4% (IC 95% 47.6-85.0), respectivamente, em indivíduos com TEA.

Indivíduos com TEA frequentemente apresentam padrões comportamentais restritos e repetitivos, como movimentos estereotipados, interesses restritos, hiper ou hiporreatividade (AMERICAN PSYCHIATRIC ASSOCIATION, 2013). Comportamentos orais prejudiciais, como bruxismo, também são comumente relatados (KUTER; GULER, 2019). Padrões comportamentais podem afetar a coordenação motora e prejudicar a higiene oral auto executiva (PILEBRO; BÄCKMAN, 2005). Maloclusão pode ser uma consequência desses comportamentos, favorecendo a respiração bucal e tendo implicações na cárie, gengivite e halitose (KOLAWOLE; FOLAYAN, 2019).

Ainda, contribuindo para o cenário de uma condição bucal prejudicada nesse perfil de indivíduos, resistência ao tratamento e dificuldade de adaptação ao ambiente odontológico, leva a um grande número de dentes não tratados e perdidos (ELMORE; BRUHN; BOBZIEN, 2016).

Revisões sistemáticas têm comparado as diferentes condições bucais entre indivíduos com TEA e controles e têm demonstrado resultados conflitantes. Em 2016, Bartolomé-Villar e colaboradores não observaram diferenças quanto à cárie dentária ou maloclusões, mas detectaram pior higiene bucal e condição periodontal em indivíduos com TEA. Em 2020, Corridore e colaboradores observaram que os estudos primários incluídos em sua revisão não foram totalmente consistentes em demonstrar que crianças autistas têm maior prevalência de cárie, mas foram unânimes em demonstrar maior prevalência de doença periodontal. Em contrapartida, Lam e colaboradores (2020) identificaram maior prevalência de bruxismo e mais baixo pH salivar em indivíduos autistas, mas falharam em detectar diferenças quanto a prevalência e gravidade da cárie, higiene oral, condição periodontal, maloclusões e demais alterações salivares. Mais recentemente, Ningrum et al. (2021) verificaram que crianças com

TEA apresentaram significativamente mais cáries quando comparadas a crianças sem a condição. Neste ano de 2022, Granja et al. relataram maior chance de ter bruxismo em indivíduos com TEA quando comparados aos controles; e Barros et al. mostraram resultados inconsistentes na associação entre TEA e má oclusão.

Considerando que as revisões sistemáticas anteriores reportaram achados conflitantes, nossa proposta aqui foi realizar uma nova revisão sistemática utilizando uma ampla estratégia de busca com alvo na maior sensibilidade de identificação dos estudos primários e sem limite de idade para inclusão dos participantes e de período de publicação. Adicionalmente, nossa proposta foi comparar e sumarizar diversos desfechos relacionados a saúde bucal, no intuito de construir um panorama geral da condição de bucal em indivíduos com TEA incluindo cárie, padrão de higiene bucal, doenças periodontais, bruxismo, má oclusão, perda dentária e alterações salivares.

2 REFERENCIAL TEÓRICO

2.1 TRANSTORNO DO ESPECTRO AUTISTA (TEA)

O transtorno do espectro autista (TEA) representa um grupo diversificado de condições e transtornos do desenvolvimento, que se caracterizam por algum grau de limitação de interação social, comunicação e restrição de atividades e interesses, as quais podem ou não acompanhar deficiências intelectuais e de linguagem. Epilepsia, depressão, ansiedade, transtorno de déficit de atenção e hiperatividade podem acompanhar o autismo como doenças concomitantes (WHO, 2021).

As manifestações mais comumente observadas do TEA compreendem padrões atípicos de comportamentos e atividades, como a dificuldade de transição de uma atividade para outra, foco em detalhes e reações incomuns às sensações, diminuição no desejo de compartilhar interesses com outras pessoas, dificuldade de apreciar emoções próprias e dos outros, aversão em manter contato visual, exposição e oratória afetadas, dificuldades de socialização, inflexibilidade de comportamento, hipersensibilidade sensorial e movimentos estereotipados (AMERICAN PSCHIATRIC ASSOCIATION, 2021). Essas características podem variar entre os indivíduos com TEA, em termos de combinações e níveis de gravidade dos sintomas e, na maioria dos casos, persistem ao longo da vida e podem também evoluir com o tempo (AMERICAN PSCHIATRIC ASSOCIATION, 2021; WHO, 2021).

Distúrbios do sono podem ser mais comuns em crianças com autismo de alto funcionamento em comparação com controles saudáveis (ALLIK; LARSSON; SMEDJE,

2006). Alterações na coordenação motora, atraso no desenvolvimento da dominância da mão e tônus muscular deficiente também pode estar presentes (FRIEDLANDER et al., 2006). Ademais, é relatado pelos pais maior sensibilidade tátil e seletividade alimentar (VALICENTI-MCDERMOTT et al., 2006).

As características do TEA podem ser detectadas pelos pais/cuidadores ou pediatras na primeira infância, antes da criança completar um ano de idade (AMERICAN PSCHIATRIC ASSOCIATION, 2021). Contudo, muitas vezes, o autismo só é diagnosticado mais tarde (WHO, 2021), ou quando a criança atinge 2 ou 3 anos de idade, que é quando os sintomas apresentam maior visibilidade ou então exibem algum grau de comprometimento funcional (AMERICAN PSCHIATRIC ASSOCIATION, 2021).

Universalmente, um número crescente de crianças está sendo diagnosticado com TEA (WHO, 2021). Nas últimas décadas, houve aumento na conscientização e notificação sobre o TEA (DOVER; COUTEUR, 2007). Estima-se que, a nível mundial, aproximadamente uma em 160 crianças apresenta um TEA (MAYADA et al., 2012). A prevalência de TEA varia muito entre os estudos sendo provável que muitas pessoas permaneçam não identificadas ou incorretamente diagnosticadas (WHO, 2021). O TEA ocorre em todos os grupos raciais, étnicos e socioeconômicos (MAENNER et al., 2021). A prevalência de TEA ocorre em uma proporção de 5 indivíduos do sexo masculino para 1 do sexo feminino (NAMAL; VEHIT; KOKSAL, 2007; SAMPEDRO-TOBÓN; GONZALEZ-GONZALEZ; VELEZ-VIERA, 2013), embora essa proporção esteja mudando com o tempo (AMERICAN PSCHIATRIC ASSOCIATION, 2021).

O diagnóstico de TEA é normalmente baseado em um exame clínico, muitas vezes apoiado por outras informações e testes (AMERICAN PSCHIATRIC ASSOCIATION, 2021). Pediatras do desenvolvimento, neurologistas, psicólogos ou psiquiatras são os profissionais que realizam esse diagnóstico (CDC, 2020).

A etiologia do TEA tem sido associada com fatores ambientais, biológicos e genéticos. Predisposição genética, medicamentos utilizados na gestação como ácido valpróico e talidomida (DIETERT; DIETERT; DEWITT, 2011), idade aumentada dos pais na gestação (DURKIN et al., 2008), e ter um irmão com autismo (MUHLE; TRENTACOSTE; RAPIN, 2004; HALLMAYER et al., 2011) foram associados com maior probabilidade de uma criança ser diagnosticada com TEA. Ressalta-se que vacinas não foram associadas com maior probabilidade de diagnóstico de autismo, assim como raça, etnia ou status socioeconômico (WHO, 2021).

Indivíduos com TEA apresentam os mesmos problemas de saúde que a população em geral e também têm necessidades específicas de cuidados de saúde que estão relacionados propriamente ao TEA ou às outras condições concomitantes. Não existem doenças bucais especificamente associadas ao TEA, mas esses pacientes sofrem com a falta de atendimento odontológico e higiene bucal. Nesse sentido, são mais suscetíveis a desenvolverem doenças crônicas não transmissíveis como cárie e/ou doença periodontal (KOTHA et al., 2018; QIAO et al., 2018). Isso se dá devido a fatores de risco advindos de comportamentos e preferências, como inatividade física e propensões alimentares inadequadas (WHO, 2021). Sabe-se que indivíduos com TEA têm taxas mais altas de necessidades de saúde não atendidas quando comparados a população em geral. Isso parece consequência de limitado conhecimento e compreensão dos profissionais de saúde e dos serviços odontológicos, inclusive pela discriminação; ao gerar privações em termos de cuidados de saúde (WHO, 2021), e ainda representar uma prioridade baixa (WALDMAN; PERLMAN; WONG, 2008).

Crianças com TEA representam desafios para pais e profissionais da odontologia (GHADI, 2010), pois o ambiente odontológico é um ambiente desafiador (MARSHALL et al., 2008). É de extrema relevância incluir serviços de promoção, prevenção e tratamento de doenças agudas e crônicas, pois pessoas com autismo precisam de serviços de saúde acessíveis para suas necessidades gerais de saúde (WHO, 2021).

2.2 TEA E CÁRIE DENTÁRIA

Cárie é uma das doenças crônicas evitáveis mais comuns e prevalentes da infância; os indivíduos permanecem suscetíveis ao longo da vida (PITTS, 2004). É a principal causa de dor e perda de dentes na população (KIDD; GIEDRYS-LEEPER; SIMONS, 2000) e é caracterizada pela destruição localizada de tecidos duros dentários por subprodutos ácidos de bactérias na fermentação de carboidratos dietéticos (FEJERSKOV; KIDD, 2003).

Por ser uma doença de caráter multifatorial, a cárie está relacionada ao estilo de vida e fatores comportamentais. Fatores associados com cárie incluem hábitos de higiene bucal; dieta, como o consumo frequente de carboidratos refinados, uso regular de medicamentos orais que contenham açúcar, exposição ao flúor (ANDERSON, 2002), métodos inadequados de alimentação de bebês (FEATHERSTONE et al., 2002; KROL, 2003; WINN, 2001; TOUGER-DECKER; VAN LOVEREN, 2003), fatores relacionados à microbiota oral (ZHANG et al., 2018), fatores salivares (LENANDER-LUMIKARI; LOIMARANTA, 2000), incluindo medicamentos que interferem com fluxo da saliva (WOLFF et al., 2017), susceptibilidade do

hospedeiro (FEATHERSTONE, 2000), condição socioeconômica (GIBSON; WILLIAMS, 1999), entre outros.

O autismo é frequentemente associado como possível fator de risco para cárie, considerando medicações que alteram fluxo salivar, seletividade alimentar e higiene bucal precária devido à baixa habilidade para realizar escovação adequada (NAMAL; VEHIT; KOKSAL, 2007). Sabe-se que hábitos alimentares caracterizados por rejeição e/ou preferência a certos tipos de alimentos são frequentemente encontrados em crianças com TEA (CHISTOL et al., 2018; GRAY; CHIANG, 2017). Os padrões de comportamento restritos e repetitivos, também presentes nesses indivíduos, podem estar relacionados a algumas barreiras no momento das refeições, bem como na hiperseletividade alimentar (LEIVA-GARCÍA et al., 2019), preferências por alimentos baseadas nas suas cores, formas e texturas (BANDINI; CURTIN; PHILLIPS, 2017; CHISTOL et al., 2018) e também padrões atípicos de deglutição (VIVIERS et al., 2020). Essas escolhas estão muito relacionadas ao seguimento de rotinas e as reações à mudanças desses indivíduos com TEA (KRAL et al., 2013) ou também pela sensibilidade sensorial oral (CERMAK; CURTIN; BANDINI, 2010).

Geralmente, há uma preferência das crianças com TEA por alimentos doces, macios e pegajosos, indicando uma maior relação e maior susceptibilidade à cárie dentária (LU; WEI; HUANG, 2013; SUHAIB et al., 2019). Para Suhaib et al. (2019) e Jaber (2011), foi encontrada uma maior prevalência de cárie em autistas em comparação a indivíduos controle. Segundo os autores, o principal motivo dessa maior prevalência foi a falta ou irregularidade da escovação e a incapacidade da criança de realizar a escovação de maneira adequada e independente. Uma vez que muitas crianças com TEA apresentam reações de hipersensibilidade, elas podem manifestar esse comportamento durante a escovação (GANDHI; KLEIN, 2014). Além das próprias dificuldades em praticar uma higiene bucal adequada devido à distúrbios comportamentais (DEMATTEI; CUVO; MAURIZIO, 2007), como dificuldade na escovação de forma autônoma e o correto uso do fio dental.

Concomitantemente a isso, alimentos doces são os alimentos preferidos usados como recompensa pelos pais pelo bom comportamento (MARSHALL; SHELLER; MANCL, 2010). A má função mastigatória e dificuldades de deglutição também podem afetar nessas escolhas alimentares de seus pais (KLEIN; NOWAK, 1998). Ainda, alguns centros que oferecem cuidados a indivíduos autistas utilizam doces e outros alimentos cariogênicos para estimular um comportamento mais cooperativo, reforçar comportamentos direcionados (CHARLES, 2010). Também é relatado que crianças com autismo são mais propensas a “procurar comida” para se confortarem quando estão ansiosas ou confusas (O’BRIEN; WHITEHOUSE, 1990).

Hábito de “segurar” por mais tempo a comida na boca ao invés de engoli-la, devido à má coordenação muscular da língua e menor capacidade mastigatória é observado em autistas (BHANDARY; NARY, 2017; JABER, 2011). Consequentemente, o pH salivar reduz, aumentando a suscetibilidade à cárie (KLEIN; NOWAK, 1998; STOOKEY, 2008). O manejo farmacológico de pacientes com TEA geralmente inclui medicamentos que afetam o fluxo salivar, como tratamentos para transtornos de humor, déficit de atenção, agressividade, ansiedade e insônia, o que pode contribuir para um risco aumentado de cárie (HSIA et al., 2014). Alguns desses medicamentos podem causar xerostomia aumentando também a susceptibilidade à cárie (CHANDRASHEKHAR; BOMMANGOUDAR, 2018). Maiores taxas de cárie podem levar à perda dentária precoce e, posteriormente, contribuir para má oclusão (THARAPIWATTANANON, 1994).

Apesar das premissas teóricas que tornam indivíduos com TEA mais suscetíveis à cárie, os estudos não têm demonstrado consistência nessa direção. Algumas evidências demonstram, inclusive, menor prevalência de cárie em autistas quando comparados a controles (AL-MAWERI et al., 2014; DU et al., 2015; FAKROON et al., 2014; KUTER; GULER, 2019; LOO et al., 2008; MORALES-CHÁVEZ et al., 2019; NAMAL; VEHIT; KOKSAL, 2007; ORELLANA et al., 2012; SARNAT et al., 2016; VAJAWAT; DEEPIKA, 2012), mesmo na presença de fatores predisponentes à cárie em que os autistas estão expostos, como os relatados anteriormente, o que sugere que existem outros fatores envolvidos, fatores intrínsecos que podem impactar nessa menor prevalência de cárie, portanto, não se tem evidências suficientes de que o TEA verdadeiramente representa um fator de risco para cárie dentária (NAMAL; VEHIT; KOKSAL, 2007).

Segundo Sarnat et al. (2016), menor prevalência de cárie em autistas pode ser consequência da supervisão dos pais ou cuidadores quanto à higiene bucal, ao uso de dietas com restrição de carboidratos, e comportamento mais regular nas refeições. Em termos de fatores salivares, Bassoukou, Nicolau e dos Santos (2009) em seu estudo, relataram que crianças autistas têm pH e capacidade de tamponamento salivar semelhantes às crianças sem TEA, ocasionando uma atividade de cárie semelhante entre autistas e controles. Ademais, no estudo de Orellana et al. (2012), o uso de medicamentos como antipsicóticos e ansiolíticos, os quais podem afetar a salivação e por conseguinte favorecer a formação de placa bacteriana e cárie, não foi associado ao desenvolvimento de cáries. Para Loo et al. (2008), também a prevalência e gravidade de cárie não foram associadas ao uso de medicação psicotrópica.

Outros autores por sua vez, demonstraram uma prevalência semelhante de cárie dentária entre autistas e controles, sugerindo uma suscetibilidade semelhante à cárie (BASSOUKOU et

al., 2009; DE MOOR; MARTENS, 1997, EL KHATIB et al., 2014; FAHLVIK-PLANEFELDT; HERRSTRÖM, 2001; KLEIN; NOWAK, 1999; LOWE; LINDEMANN, 1985), com baixa atividade de cárie (KLEIN; NOWAK, 1999), diminuindo ao longo do tempo (MORINUSHI; UEDA; TANAKA, 2001). Em relação à dentição, El Khatib et al. (2014) encontraram na dentição decídua, mais dentes cariados não tratados nas crianças com TEA, enquanto que na fase de dentição mista, as crianças com TEA tiveram menos dentes obturados do que as crianças controle.

Em Onol e Kirzioglu (2018), Namal et al. (2007), Daneshvar et al. (2019), Bhandary e Hary (2017), o número de dentes restaurados foi encontrado muito baixo nas crianças com autismo. Quanto à cárie dentária não tratada, crianças autistas tiveram médias maiores de cárie não tratada quando comparado aos controles saudáveis (DANESHVAR et al., 2019). Esses resultados podem ser devidos à baixa conscientização odontológica e à pouca cooperação dessas crianças com os dentistas. Além disso, o treinamento inadequado de dentistas e especialistas em odontologia e a alta sensibilidade dessas crianças a sons, luzes, odores e cores desconhecidos são as barreiras para o acesso ao atendimento odontológico (EL KHATIB et al., 2014). Isso indica que crianças com autismo necessitam de muitos cuidados dentários restauradores, mas não os estão recebendo (JABER; SAYYAB; ABU FANAS, 2011).

2.3 TEA, HIGIENE ORAL E ESTADO PERIODONTAL

A placa dentária é a comunidade de microrganismos encontrados na superfície do dente como um biofilme, incorporado em uma matriz de polímeros de origem bacteriana e hospedeira (SOCRANSKY; HAFFAJEE, 2002; MARSH, 2004). Através de uma sequência ordenada de eventos, a placa dentária é formada, se tornando rica em espécies estrutural e funcionalmente organizada (MARSH, 2004). A partir de bactérias compatíveis com saúde, se desenvolvem bactérias de maior patogenicidade, conforme um maior acúmulo de biofilme e esse acúmulo de biofilme ocorre de modo diferente entre os indivíduos (HAFFAJEE et al., 2009). Após formada, a placa dentária possui um certo grau de estabilidade ou equilíbrio entre as espécies componentes, apesar de pequenos estresses ambientais regulares, como, componentes dietéticos, higiene oral, defesas do hospedeiro, mudanças diurnas no fluxo salivar, entre outros (MARSH et al., 1989). Sendo assim, indivíduos que consomem regularmente componentes da dieta com um alto teor de açúcar têm maiores proporções de microrganismos, como estreptococos mutans e lactobacilos, na placa (MARSH, 1989).

Na ausência de higiene bucal, a resposta a um biofilme de placa dental madura é a gengivite, uma forma reversível de doença periodontal caracterizada pela inflamação das

gengivas (KISTLER et al., 2013; LOE; THEILADE; JENSEN, 1965), considerada uma doença de alta prevalência global (ALBANDAR; KINGMAN, 1999; LI et al., 2010; MURRAY; VERNAZZA; HOLMES, 2015). Na presença de susceptibilidade individual, essa gengivite persiste e evolui para a periodontite, uma destruição irreversível dos tecidos periodontais (SCHATZLE et al., 2003), podendo resultar em eventual perda dentária (ABUSLEME et al., 2013). O principal meio de prevenção para essas doenças são as práticas adequadas de higiene bucal, como a escovação dental e limpeza interdental (KISTLER et al., 2013). Nesse sentido, a placa dentária é indispensável para o desenvolvimento das doenças periodontais e sua remoção é um componente essencial na prevenção e na conformidade do autocuidado bucal (NEWBRUN, 1992).

A higiene oral faz parte das habilidades sociais. Sabe-se que indivíduos com TEA podem ter menos habilidades de aprendizado do que indivíduos saudáveis (MORGAN et al., 2002), o que pode comprometer sua higiene bucal (PILEBRO; BACKMAN, 2005; DEMATTEI; CUVO; MAURIZIO, 2007). Alta prevalência de má higiene bucal e doença periodontal foram encontradas entre crianças e adultos autistas (DANESHVAR et al., 2019; FAKROON et al., 2014; RAI et al., 2012; EL KHATIB et al., 2014; ÖNOL; KIRZIOGLU, 2018; LUPPANAPORNLARP et al., 2010; ORELLANA et al., 2012; SUHAIB et al., 2019; VAJAWAT; DEEPIKA, 2012) quando comparados a controles. O que pode explicar essa pior higiene bucal e estado periodontal inclui: uma escovação irregular; dificuldades de comportamentos direcionados para executar os procedimentos de limpeza; conscientização dos pais e/ou cuidadores no estabelecimento e manutenção da higiene bucal (JABER, 2011; EL KHATIB et al., 2014), falta de destreza manual necessária para a escovação, a qual é diminuída devido à musculatura oral enfraquecida (KLEIN; NOWACK, 1999) e má coordenação das mãos (LUPPANAPORNLARP et al., 2010), uma vez que crianças autistas podem ter limitações em suas habilidades manuais (VAJAWAT; DEEPIKA, 2012). Relatos de pais evidenciam que seus filhos autistas possuem uma aversão ao sabor e textura dos cremes dentais, reflexo de engasgo na escovação, o que pode caracterizar a escovação como uma tarefa difícil a ser executada (STEIN; POLIDO; CERMAK, 2013). A própria higiene bucal dispensada em ambiente de consultório é dificultada devido a fatores comportamentais e sensibilidade oral desses pacientes (STEIN et al., 2011).

Além disso, muitas crianças diagnosticadas com TEA fazem uso de drogas psicoativas ou anticonvulsivantes, e a presença de gengivite generalizada pode ser um dos efeitos colaterais desses medicamentos (THARAPIWATTANANON, 1994; MURSHID, 2005). Os altos custos de tratamento odontológico dificultam o atendimento dos pais às necessidades de saúde bucal

de seus filhos (STEIN et al., 2011). A dificuldade e baixa prioridade dada aos cuidados bucais em comparação com outros problemas diários também tornam complexos os procedimentos de higiene bucal em crianças com TEA (DIAS et al., 2010). Foi relatado que indivíduos autistas com melhores habilidades diárias apresentaram uma melhor saúde bucal (WEIL; INGLEHART, 2012), enquanto que os que tinham habilidades mais baixas demonstraram pior higiene bucal (SARNAT et al., 2016; WEIL; INGLEHART, 2012).

Em contraste com grande parte dos estudos, Sarnat et al. (2016) encontraram que a higiene bucal caseira não se apresentou de maneira diferente entre autistas e controles, ambos apresentaram boa higiene bucal e boa saúde gengival. Alguns autores explicam que, em crianças de pouca idade, a reação gengival à placa pode não ser tão grave quanto em adolescentes e adultos, e não apresenta, por consequência, uma reação inflamatória grave na presença de placa (MOORE; HOLDEMAN; SMIBERT, 1984). Du et al. (2015) mostraram uma melhor saúde gengival para indivíduos autistas, mas, ao considerar que, nesse estudo, quase metade de todos os dentes apresentavam evidências de acúmulo de placa, isso necessita ser reconhecido e requer cuidado, uma vez que crianças com TEA demandam um manejo mais complexo no ambiente odontológico.

2.4 TEA E PERDA DENTÁRIA

É relatado que o nível de tratamento odontológico é menor em crianças com TEA. Indivíduos autistas têm mais necessidades odontológicas não atendidas e apresentam mais dificuldades no tratamento devido a limitações comportamentais de cooperação (FAHLVIK-PLANEFELDT; HERRSTROM, 2001). Isso também pode ser atribuído à baixa conscientização odontológica e treinamento inadequado de profissionais que representam barreiras para o acesso ao atendimento odontológico (EL KHATIB et al., 2014).

No estudo de Onol e Kirzioglu (2018), 11,1% das crianças com TEA tinham ao menos um dente perdido, enquanto que as crianças do grupo controle não tinham nenhum dente permanente perdido. Isso pode refletir ao fato dos pais não levarem seus filhos ao dentista regularmente, até que eles tenham problemas de maior complexidade, ao ponto de tanto os pais como os dentistas preferirem a extração aos tratamentos prévios restauradores, devido às dificuldades nos procedimentos de tratamento e na própria obtenção do tratamento odontológico (BARRY; O'SULLIVAN; TOUMBA, 2014). Mesmo o atendimento odontológico habitual, de rotina, pode ser dificultado, por problemas de comunicação e comportamentais (KLEIN; NOWAK 1998), gerando um maior estresse para paciente e dentista. Sabe-se que indivíduos com TEA são mais propensos a não cooperarem e necessitarem de

tratamento odontológico sob anestesia geral, o que pode complicar ainda mais o tratamento (LOO; GRAHAM; HUGHES, 2009).

Daneshvar et al. (2019), Namal, Vehit e Koksal (2007), Luppanapornlarp et al. (2010), Fakroon et al. (2014), também relataram que crianças com TEA tiveram mais perda dentária em comparação com crianças saudáveis. De acordo com esses resultados, acredita-se que a extração em crianças com TEA possa ser preferida devido à natureza desafiadora de seu manejo (NAMAL; VEHIT; KOKSAL, 2007). Contrastando com esses achados, outros estudos não encontraram diferenças estatisticamente significativas entre autistas e controles em relação à perda dentária (ALAKI et al., 2016; AL-MAWERI et al., 2014; BLOMQVIST; BEJEROT; DAHLLÖF, 2015; BHANDARY; NARY, 2017).

2.5 TEA E MÁ OCLUSÃO

A má oclusão é um distúrbio dento-esquelético, que se deve principalmente a um desequilíbrio no complexo craniofacial em desenvolvimento (KAWALA; ANTOSZEWSKA; NECKA, 2007). Ela pode impactar negativamente na qualidade de vida e causar limitação funcional, dor e incapacidade social, que afeta o bem-estar emocional e as interações sociais (BHATIA; WINNIER; MEHTA, 2016; SUN; WONG; MCGRATH, 2017). Esse impacto negativo na qualidade de vida começa a ser percebido quando as crianças têm 11 a 14 anos e piora à medida que envelhecem (KRAGT et al., 2016). A OMS estima as más oclusões como o terceiro problema de saúde bucal mais prevalente, após cárie dentária e doenças periodontais (GUO et al., 2016).

A diversidade na oclusão dentária é resultado de um padrão hereditário multifatorial no qual fatores genéticos, ambientais e étnicos desempenham papeis importantes (HEIMER; TORNISIELLO; ROSENBLATT, 2008). A detecção precoce da má oclusão na dentição decídua com intervenção pode prevenir a má oclusão que provavelmente se desenvolveria na dentição permanente (MOSLEMI; NADALIZADEH; SARSANGHIZADEH, 2015; KIRZIOGLU; SIMSEK; YILMAZ, 2013). As más oclusões podem afetar negativamente na capacidade dos indivíduos de processar e quebrar os alimentos prejudicando o desempenho mastigatório (MAGALHÃES et al., 2010). Problemas de fala e mastigação interferem e restringem a escolha dos alimentos (ZHANG; MCGRATH; HÄGG, 2006; JERYL; BUSCHANG; THROCKMORTON, 2002).

Quando não tratada, a má oclusão pode afetar outros problemas de saúde bucal, aumentando o risco de cárie (FELDENS et al., 2015), prejudicando a qualidade do auto controle de placa tendo impacto na saúde gengival (KUKLETOVA et al., 2012), e causando dor e

limitação funcional. Apinhamento e mordida cruzada bucal foram associados à cárie (KOLAWOLE; FOLAYAN, 2019) por dificultar a higiene bucal e aumentar retenção de placa (HASHIM; AL-JASSER, 1996). Sobressaliência aumentada e mordida aberta anterior também foram associados à gengivite (KOLAWOLE; FOLAYAN, 2019) e estão intimamente associadas à incompetência labial (JONES; OLIVER, 2000). Mutuamente, a cárie pode levar à perda precoce de dentes e, posteriormente, a más oclusões (THARAPIWATTANANON, 1994).

Hábitos orais como sucção digital e de chupeta, mordedura labial, roer unhas, respiração bucal, bruxismo, hábitos auto lesivos, e interposição de língua (GARDE et al., 2014) estão associados com má oclusão (DOS SANTOS, et al., 2012). Adicionalmente, hábitos alimentares como consumo de alimentos de consistência mole com redução das forças mastigatórias (VIGGIANO et al., 2004), também podem levar à ocorrência de má oclusão (DOS SANTOS, et al., 2012).

Crianças com TEA tendem a apresentar hábitos orais deletérios, como bruxismo, morder a língua, chupar o polegar, beliscar a gengiva e morder objetos, além do comportamento auto lesivo (MEDINA et al., 2003; AL-SEHAIBANY, 2017; MURSHID, 2005), sendo mais propensas a apresentar má oclusão como mordida aberta anterior, mordida cruzada posterior e sobressaliência excessiva (FONTAINE-SYLVESTRE et al., 2017; LUPPANAPORNLARP et al., 2010; ORELLANA et al., 2012; WARREN et al., 2001).

A literatura tem demonstrado que indivíduos com TEA apresentam maior prevalência de má oclusão quando comparados a controles (AL MUSAWI; AL-DABAGH, 2019; ALJUBOUR; SEHAIBANY, 2018; FONTAINE-SYLVESTRE et al., 2017; LEIVA-GARCÍA et al., 2019; ONOL; KIRZIOGLU, 2018; ORELLANA et al., 2012). Entretanto, muitos estudos mostram resultados inconsistentes, os quais podem ser consequência de ausência de nomenclatura padronizada, falta de critérios diagnósticos uniformes e/ou diferenças em idade e origem étnica da amostra.

No estudo de Al Musawi e Al-Dabagh (2019), as maloclusões com prevalência significativamente maior em crianças com TEA foram sobressaliência aumentada, sobremordida profunda, espaçamento, mordida cruzada posterior e mordida aberta. Fontaine-Sylvestre et al. (2017) observaram maior prevalência de mordida cruzada posterior, sobressaliência aumentada e apinhamento maxilar grave em autistas. Esses achados podem estar relacionados aos hábitos parafuncionais frequentemente vistos nos indivíduos com TEA, tais como uso de chupeta, bruxismo, interposição de língua, morder os lábios, comportamento de autolesão e de morder objetos não nutritivos (SARNAT et al., 2016; EL KHATIB et al.,

2014). Uma maior tendência para um palato alto e estreito, que é frequentemente associado à mordida cruzada posterior, também tem sido observada na população com TEA (ORELLANA et al., 2012).

De acordo com Aljubour e Al-Sehaibany (2018), a maioria das crianças autistas exibiu uma relação canina de classe II, seguida por relações de classe I e classe III, enquanto que a maioria das crianças do grupo controle exibiram uma relação canina de classe I, seguida de relações de classe II e classe III, o que é consistente com estudos anteriores (BHAYYA; SHYAGALI, 2011; DE ALMEIDA et al., 2008), também uma prevalência de mordida cruzada posterior significativamente maior em autistas do que nos controles, sobressaliência aumentada, provavelmente devido aos hábitos orais anormais em crianças com TEA. Leiva-García et al. (2019) encontraram mordida aberta e apinhamento mais frequentes no grupo TEA do que nos controles, neste estudo, os problemas de oclusão mostraram-se diretamente associados à rejeição alimentar, com maiores escores de mordida cruzada e mordida aberta no grupo TEA. Essa relação pode estar associada à hiposensibilidade, com diminuição do tônus muscular, problemas de mastigação e padrões mastigatórios atípicos e má oclusão (BEN-SASSON et al., 2008; NADON et al., 2011).

Luppanapornlarp et al. (2010) ao avaliar o índice de estética dentária (DAI), obtiveram pontuações semelhantes para crianças autistas e não autistas. No entanto, as porcentagens de perda de dentes, espaçamento, palato profundo, sobressaliência reversa, mordida aberta e tendências de relacionamento molar de Classe II foram maiores nas crianças com autismo. Já para Kuter e Guler (2019) foi encontrado diferenças significativas entre as crianças autistas e as saudáveis quanto ao palato profundo e interposição de língua, mas em termos de mordida aberta e mordida cruzada não foram encontradas diferenças, concordando com Farmani et al. (2020). Além disso, em Kuter e Guler (2019), o apinhamento dentário nas crianças saudáveis foi mais comum do que nas crianças autistas neste estudo. Assim, os resultados deste estudo parecem corroborar e ao mesmo tempo contrastar com os resultados de outras pesquisas. Du et al. (2015) documentaram maior prevalência de sobremordida, sobressaliência e mordida cruzada anterior em crianças com TEA em relação aos controles, embora não tenha sido alcançada significância estatística. Farmani et al. (2020) não mostraram diferença significativa na prevalência geral de má oclusão entre os grupos TEA e controle. No entanto, a prevalência de sobressaliência aumentada e relação molar de Classe II foi maior em pacientes com TEA.

2.6 TEA E BRUXISMO

O bruxismo é uma atividade repetitiva dos músculos da mandíbula caracterizada por apertar ou ranger dos dentes ou empurrar a mandíbula, podendo levar a traumas oclusais. Apresenta duas manifestações circadianas, pode ocorrer durante o sono (indicado como bruxismo do sono) ou durante a vigília (indicado como bruxismo acordado) (LOBBEZOO et al., 2013). O bruxismo é definido como uma atividade parafuncional (DE LEEUW, 2008).

O bruxismo, por ser uma atividade repetitiva da musculatura mastigatória, pode ser fator de risco para diversas complicações de saúde (LOBBEZOO et al., 2018), sendo associado com dor orofacial, desgaste dentário e falhas nos tratamentos restauradores (LAVIGNE; MANZINI; KATO, 2005; PAESANI, 2010). Em crianças, causa desgaste dentário, dores de cabeça, dores musculares faciais, desconforto durante a mastigação e limitação da abertura da boca (BULANDA et al., 2021). A prevalência de bruxismo do sono em crianças varia e tem se mostrado mais comum em crianças em comparação com adultos (BEDDIS; PEMBERTON; DAVIES, 2018), com taxas de prevalência variando de 13% a 49% (ALFANO; BOWER; MEERS, 2018). Há estudos que relatam uma prevalência de bruxismo de 20% na população adulta e indicam que ele ocorre predominantemente no sexo feminino (LAVIGNE et al., 2008).

A etiologia do bruxismo é complexa (OLIVEIRA et al., 2015). Fatores associados ao bruxismo do sono incluem estado emocional, estresse e a ansiedade (OLIVEIRA et al., 2015), além disso, é descrito na literatura que certas drogas e substâncias químicas, como os inibidores seletivos da recaptação de serotonina (paroxetina, fluoxetina, sertralina), inibidores seletivos da recaptação de norepinefrina (venlafaxina) e antipsicóticos (haloperidol) podem aumentar o número de episódios de bruxismo do sono (CARRA; HUYNH; LAVIGNE, 2012).

Desgaste dentário detectado clinicamente tem sido associado com bruxismo do sono em crianças, especialmente na dentição decídua devido ao menor grau de mineralização do esmalte quando comparados aos dentes permanentes. No entanto, a observação de desgaste nas superfícies duras dos tecidos dentários não confirma o diagnóstico clínico de bruxismo do sono (GOMES et al., 2018). A polissonografia é considerada o padrão ouro para o diagnóstico de bruxismo (ALFANO; BOWER; MEERS, 2018).

Nos indivíduos autistas, comumente são observados distúrbios comportamentais, comportamentos auto lesivos, agressividade, hiperatividade e respostas exacerbadas a rotinas e demandas (KARANDE, 2006). Bruxismo, hábitos auto lesivos, mastigar objetos não nutritivos, padrões atípicos de deglutição, respiração oral (FONTAINE-SYLVESTRE et al., 2017; SARNAT et al., 2016), podem influenciar em outras doenças bucais (KOPYCKA-KEDZIERAWSKI; AUINGER, 2008), como dano nos tecidos moles, perda e desgaste dentário, também nas má oclusões, como mordida aberta anterior e mordida cruzada posterior

(FONTAINE-SYLVESTRE et al., 2017; LUPPANAPORNLARP et al., 2010; ORELLANA et al., 2012), podendo resultar em distúrbios ortodônticos (JABER, 2011).

O bruxismo é um problema de saúde bucal relativamente frequente em crianças com TEA (GANDHI; KLEIN, 2014; MURSHID, 2011; MUTHU; PRATHIBHA, 2008; SCHRECK; MULICK, 2000). Alguns autores observaram maior prevalência de bruxismo em crianças com TEA quando comparadas aos controles (DANESHVAR et al., 2019; KUTER; GULER, 2019; LEIVA-GARCÍA et al., 2019; ONOL; KIRZIOGLU, 2018; SUHAIB et al., 2019), assim como maior prevalência de facetas oclusais de desgaste dentário (El KHATIB et al. 2014), como indicativo de bruxismo (KNIGHT et al., 1997). Em contrapartida, outras evidências não observaram diferenças no desgaste dentário entre TEA e controles (DU et al., 2015; FAHLVIK-PLANEFELDT; HERRSTROM, 2001; LUPPANAPORNLARP et al., 2010; ORELLANA et al., 2012).

O diagnóstico precoce do autismo, com abordagens sociais, de comunicação e comportamentais direcionadas e necessárias (AMERICAN PSYCHIATRIC ASSOCIATION, 1994), pode contribuir para efeitos positivos no controle do bruxismo e outros hábitos.

2.7 TEA E ALTERAÇÕES SALIVARES

Capacidade tampão e pH salivar são importantes na suscetibilidade à cárie dentária (DIAB et al., 2016). Baixo pH salivar pode resultar em rápida desmineralização do esmalte (RAI; HEGDE; JOSE, 2012), e a capacidade de tamponamento é um fator de proteção para os dentes frente ao ataque ácido promovido pela dieta e bactérias cariogênicas (BASSOUKOU; NICOLAU; DOS SANTOS, 2009).

A secreção salivar em indivíduos com TEA pode ser afetada negativamente em decorrência da disfunção do hipotálamo- sistema pituitário-adrenocortical (MARINOVIC-CURIN et al., 2008), que está relacionado com estresse psicossocial (JANSEN et al., 2000; JANSEN et al., 2003).

Vários estudos compararam características da saliva entre indivíduos autistas e controles. Alguns autores verificaram menor capacidade tampão da saliva em autistas (BHANDARY; HARI, 2017), enquanto outros não identificaram diferenças (BASSOUKOU; NICOLAU; DOS SANTOS, 2009; DIAB et al., 2016). Em relação ao pH salivar também há divergência na literatura com alguns estudos apontando pH mais baixo nos autistas (BHANDARY; HARI, 2017; DIAB et al., 2016) e outros não encontrando diferenças (BASSOUKOU; NICOLAU; DOS SANTOS, 2009; RAI; HEGDE; JOSE, 2012; MORALES-CHÁVEZ; VILLARROEL-DORREGO; SALAS, 2019). No contexto da taxa de fluxo salivar

resultados conflitantes também são reportados entre os estudos (BASSOUKOU; NICOLAU; DOS SANTOS, 2009; BLOMQVIST; BEJEROT; DAHLLÖF, 2015; BHANDARY; HARI, 2017).

Quanto ao uso de medicamentos, crianças autistas não tomam medicamentos específicos, no entanto, eles são prescritos para condições concomitantes, como hiperatividade, ansiedade e epilepsia (RAPIN; TUCHMAN, 2008). No estudo de Kuter e Guler (2019), 72,6% dos indivíduos autistas faziam uso de medicamentos. Orellana et al. (2012) também relataram um uso de 77% de medicamentos, os mais comumente utilizados são os anticonvulsivantes, antidepressivos e antipsicóticos (RAPIN; TUCHMAN, 2008). Efeitos adversos orofaciais desses medicamentos podem incluir xerostomia, sialorreia, disfagia, estomatite, gengivite, aumento gengival, glossite, bruxismo, edema e descoloração da língua (FRIEDLANDER et al., 2006). Os efeitos colaterais xerostômicos das drogas psicoativas não foram considerados um fator de risco para cárie em alguns estudos (MARSHALL; SHELLER; MANCL, 2010).

3 OBJETIVO

Comparar cárie, higiene bucal, doenças periodontais, bruxismo, má oclusão, perda dentária e alterações salivares entre indivíduos com transtornos do espectro do autismo (TEA) e controles sem outras condições ou neuroatipias.

4 HIPÓTESE CONCEITUAL

Nossa hipótese é que indivíduos com TEA apresentam maior prevalência e severidade de cárie dentária, presença de placa, doenças periodontais, bruxismo, má oclusão, perda dentária, e alterações salivares quando comparados a controles sem outras condições ou neuroatipias.

ARTIGO – ORAL STATUS IN PATIENTS WITH AUTISM SPECTRUM DISORDER (ASD): A SYSTEMATIC REVIEW OF OBSERVATIONAL STUDIES WITH CONTROL GROUP

Esse artigo será submetido ao periódico *Autism*.
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Oral status in patients with autism spectrum disorder (ASD): a systematic review of observational studies with control group.

Jaíne C Uliana, Catiusse C Del' Agnese, Raquel P Antoniazzi and Karla Z Kantorski

Department of Stomatology, School of Dentistry, Federal University of Santa Maria, Santa Maria, Brazil

Author Note

Jaíne C. Uliana https://.orcid.org/0000-0003-2855-8463

Catiusse C. Del' Agnese

Raquel P. Antoniazzi https://.orcid.org/0000-0003-0282-6896

Karla Z. Kantorski https://.orcid.org/0000-0002-6219-0255

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Correspondence concerning this article should be addressed to Karla Z. Kantorski, School of Dentistry, Universidade Federal de Santa Maria (UFSM), Santa Maria, Brazil. Av Roraima 1000 – Bairro Camobi - 97105-900- Santa Maria - RS / Brazil; Tel: +55(55)3220-9210.

Email: kzkantorski@gmail.com.

Abstract

The current systematic review aimed to compare caries, oral hygiene, periodontal status, bruxism, malocclusion, tooth loss and salivary alterations between individuals with ASD and typically developing individuals from observational studies. Electronic searches were performed in EMBASE, LILACS, PubMed, PsycINFO, Scopus, Web of Science, Google Scholar and ProQuest without restriction of publication period. Two independent reviewers performed study selection, data extraction and methodological quality assessment. Meta-analyses of standardized mean differences (SMD) and risk ratio/prevalence ratio (RR/PR) were performed.42 studies comprising 7217 subjects were included in review. Autistic individuals had significantly higher severity of caries in primary teeth, greater severity of untreated caries lesions, higher prevalence and severity of worse oral hygiene and gingivitis, significantly lower salivary pH, higher prevalence of bruxism, increased overjet, increased overbite, crossbite and open bite when compared to neurotypical control subjects. Higher means of filled teeth and surfaces were significantly found in control subjects. In general, subgroup analyzes involving studies with a high risk of bias and absence of matching variables strengthened the associations. Our findings suggest that autistic individuals have worse oral health status when compared to neurotypical controls. Protocol number registered in PROSPERO: CRD42021284179.

Lay abstract

Individuals diagnosed with autism spectrum disorder (ASD) seem to be more vulnerable to developing oral diseases when compared to individuals without other conditions or neuroatypia. This may be related to dietary habits, such as food selectivity and preference for cariogenic foods, frequent use of medications and behavioral patterns that are associated with harmful oral habits, in addition to difficulty in accessing dental services, resistance to treatment, and difficulty adapting to the dental environment. In this study, we sought to bring together existing studies that compared caries, oral hygiene pattern, gingival inflammation, teeth grinding, abnormal tooth alignment, tooth loss, and salivary changes between autistic

individuals and individuals without other conditions or neuroatypia of any age. Results from a systematic literature search showed that when comparing autistic individuals with individuals without other conditions or neuroatypia, autistic individuals had higher severity of caries in primary teeth, higher severity of untreated caries lesions, worse oral hygiene patterns and gingivitis, lower salivary pH, higher percentage of teeth grinding and abnormal tooth alignment when compared to neurotypical control subjects. More filled teeth were found in control subjects. Our findings suggest that autistic individuals have worse oral health status when compared to neurotypical controls.

Keywords: autism spectrum disorder, oral status, systematic review.

Introduction

Individuals with autism spectrum disorder (ASD) can be more susceptible to developing chronic non-communicable oral diseases when compared to individuals without other conditions or neuroatypia (Ferrazano et al., 2020; Jaber, 2011; Vajawat & Deepika, 2012). Malocclusions (Aljubour & Al-Sehaibany, 2018; Almusawi & Al-Dabagh, 2019; Fontaine-Sylvestre et al., 2017), impared oral hygiene and periodontal diseases (Al-Maweri et al., 2014; Bhandary & Nary, 2017; Vajawat & Deepika, 2012), caries (Jaber, 2011; Leiva-García et al., 2019; Suhaib et al., 2019), tooth loss (Namal et al., 2007; Onol & Kirzioglu, 2018; Orellana et al., 2012), parafunctional habits such as bruxism (Daneshvar et al., 2019; El Kathib et al, 2014; Leiva-García et al., 2019) salivary changes in the flow, pH and buffering capacity (Bhandary & Nary, 2017; Blomqvist et al., 2015; Diab et al., 2016) have been shown to be more prevalent in individuals with ASD when compared to the neurotypical individuals.

Association between ASD and caries may be related to behaviors commonly observed in this profile of individuals. Individuals with ASD show selectivity to certain types of food due to texture and color (Cermak et al., 2010), preference for sweetened foods and a tendency to keep food in the mouth instead of swallowing it, due to oral motor deficiency (Klein & Nowak, 1998). Parents of ASD individuals frequently describe their children as “slow feedrs” (Emond et al., 2010). The offer of high-sugar foods is often used by parents and special educators as a reward to prevent self-harm habits and reinforce targeted behaviors (Onol & Kirzioglu, 2018), which can result in a significant increase in caries (Kotha et al., 2018). Furthermore, it is estimated that 64% of children with ASD receive at least one psychotropic medication (Spencer et al., 2013), the most prescribed drug class in this profile of individuals (Hsia et al., 2014), particularly in older ASD children showing some psychiatric comorbidity (Jobski et al., 2007; Spencer et al., 2013). These drugs are strongly associated with reduced salivary flow and impaired buffering capacity (Wolff et al., 2017), which favors the

development of caries (Flink et al., 2019; Quilici & Zech et al., 2019; Cunha-Cruz et al., 2013). A systematic review from 2016 (da Silva et al.), observed that the prevalence of caries and periodontal disease was more of 60% in individuals with TEA.

Individuals with ASD often present restricted and repetitive behavioral patterns, such as stereotyped movements, restricted interests, hyper or hyporeactivity (American Psychiatric Association, 2013). Harmful oral behaviors such as bruxism are also commonly reported (Kuter & Guler, 2019). Behavioral patterns can affect motor coordination and impair self-executive oral hygiene (Pilebro & Bäckman, 2005). Malocclusion can be a consequence of these behaviors, favoring mouth breathing and having implications for caries, gingivitis and halitosis (Kolawole & Folayan, 2019). Morevoer, contributing to the scenario of an impared oral condition in autistic individuals have the resistance to treatment and difficulty in adapting to the dental environment, leads to a large number of untreated and lost teeth (Elmore et al., 2016); and the fact that only a minority of dental colleagues have expertise to treat ASD individuals hampering them access to an adequate dental treatment with comparable services to the offered to typically developing individuals (Thomas et al., 2018).

Previous systematic reviews have compared the different oral conditions between individuals with ASD and controls and have shown conflicting results. In 2016, Bartolomé-Villar et al. observe no differences regarding dental caries or malocclusions, but detected worse oral hygiene and periodontal condition in individuals with ASD. In 2020, Corridore and colleagues noted that the primary studies included in their review were not fully consistent in demonstrating that autistic children have a higher prevalence of caries, but were unanimous in demonstrating a higher prevalence of periodontal disease. In contrast, Lam et al. (2020) identified a higher prevalence of bruxism and lower salivary pH in autistic individuals, but failed to detect differences in the prevalence and severity of caries, oral hygiene, periodontal condition, malocclusions and other salivary changes. More recently, Ningrum et

al. (2021) found that children with ASD had significantly more caries when compared to children without the condition. In this year, 2022, Granja et al. reported a greater chance of having bruxism in individuals with ASD when compared to controls; and Barros and coworkers showed inconsistent results on the association between ASD and malocclusion.

Considering the conflicting findings reported from previous systematic reviews, our proposal here was to carry out a new systematic review using a broad search strategy to reach higher sensitivity in the primary studies identificiation including grey literature. Differently of the previous systametic reviews, no limit of inclusion regarding participants age, publication period, idioma and geopolitical area was applied. Additionally, our aim was to compare several outcomes such as caries, oral hygiene pattern, periodontal diseases, bruxism, malocclusion, tooth loss and salivary alterations between autistic subjects and controls, and to set an overall picture of the oral status in ASD subjects.

Methods

This systematic review was reported in accordance with the PRISMA guidelines (Page et al., 2021). The systematic review protocol was registered in the International Prospective Registry of Systematic Reviews (PROSPERO, Center for Reviews and Dissemination, University of York, and National Institute for Health Research) through registry CRD42021284179.

Review question

Our systematic review has been guided by the focused question: “Is there a difference in the oral status regarding to the dental caries, oral hygiene, periodontal diseases, bruxism, malocclusion, tooth loss and salivary alterations between subjects with autism spectrum disorders (ASD) and neurotypical controls?”

PECO(S) statement was: (P) subjects any age and sex; (E) autism spectrum disorders; (C) absence diagnosis of autism spectrum disorders or any other disability, (O) clinical

measures of dental caries, periodontal diseases, dental plaque, bruxism, malocclusion, tooth loss and salivary alterations regarding flow rate, pH or buffer capacity; and (S) observational studies (cross-sectional, case-control and cohort).

Eligibility criteria

Eligible studies should meet the following criteria: (a) to evaluate oral conditions in the ASD population compared to the control subjects without ASD or any other disability; (b) to be an observational study (cross-sectional, cohort and case control) without publication period restrictions; (c) to present clinical measures of the oral outcomes.

Studies with self-reported or reported by caregivers' measures of the oral outcomes; letters, book chapters, case reports, personal opinions, posters and case; studies on traumatic injuries or injuries; studies evaluating quality of life measures in oral health; and studies not written in the Latin-Roman alphabet were excluded.

Search strategy

The databases EMBASE, Latin American and Caribbean Health Sciences (LILACS), PubMed, PsycINFO, Scopus and Web of Science were searched without publication period restrictions up July 2022. Grey literature was searched on Google Scholar, limited to the first 100 most relevant articles. ProQuest Dissertations and Theses Database were also searched. Search strategy were customized for each database considering controlled terms and free terms (Appendix 1). The reference lists of the studies included were also investigated to identify additional studies. EndNote (Thomson Reuters, New York, USA) and Rayyan software (http://rayyan. qcri.org/) were used to manage references and to identify and remove duplicate hits.

Selection of studies

The reports were screened independently by two reviewers (J.C.U. and C.C.D). Firstly, the reviewers evaluated all retrieved by reading titles and abstracts for screening.

Secondly, the full-text articles were assessed in cases in which both reviewers considered the abstracts to be potentially relevant to inclusion regarding to elegibility. Disagreements were solved by consensus and discussion with a third reviewer (K.Z.K) in both the phases. Reasons of exclusions were recorded.

Previously, reviewers' agreement for study selection was tested in 10% of the articles. Kappa coefficient inter-reviewers was of 0.82 for titles and abstracts.

Data extraction

Standardized data extraction form was used by two reviewers (J.C.U and C.C.D) independently including information's about author, year of publication, study design, country and setting, sample characteristics (sample size, gender, age), criteria to define an ASD case (tools of diagnosis, professional type), inclusion and exclusion criteria of the studies, clinical criteria to evaluate the different oral conditions (tools, exam protocol), information related to socioeconomic background, medical condition, medication, control matching, adjusted confounding factors, principal findings and main conclusions of the studies.

During the data extraction, if data were missing or unclear, three attempt to contact the corresponding authors were performed to clarify the problem. Three contact attempts were made at an interval of 7 to 10 days via email or Research Gate. Studies with continuous outcomes without variability measures (standard deviation o standard error) were not excluded even without authors response, or if authors provide no data. In this case, the highest standard deviation obtained from an included study was imputed.

Risk of bias assessment (methodological quality):

Two reviewers (J.C.U and C.C.D) independently assessed the methodological quality of studies included by using the Newcastle-Ottawa scale for cohort studies (Wells et al., 2011) and adapted for cross-sectional studies (Moskalewicz & Oremus, 2020). For our research

question, case-control studies were considered cross-sectional studies with control group. Newcastle-Ottawa scale contains eight items distributed among three domains: selection (maximum of four stars); comparability of study groups (maximum of two stars) and outcome assessment (maximum of three stars). The overall score ranges from 0 to 9 points (Wells et al., 2011). Scores of 0-3 were considered indicative of a high risk of bias, 4-6 points were indicative of moderate risk and ≥ 7 points were considered indicate of low risk (Lo et al., 2014).

Data synthesis and analysis

Risk ratio/prevalence ratio (RR/PR) and respective 95% confidence intervals were used as effect measures of categorical variables using the Mantel-Haenszel method and DerSimonian and Laird's random effects model. Standardized mean difference (SMD) and respective 95% confidence intervals were used as effect measures of continuous data using the inverse variance method and DerSimonian and Laird's random effects model. Forest plot were generated for each comparison.

Heterogeneity was assessed using I^2, Q-statistic, Tau^2 e prediction interval. Potential causes and possible association of heterogeneity were explored with subgroup analyzes. Tooth type (deciduous/permanent), clinical exam protocol (partial, full-mouth and not reported), index type (PlI and OHI-S, for example), risk of bias (high, moderate, and low risk), and variables number used to pair the groups were analyzed. We also investigated the Socio-demographic Index (SDI), that represents a composite average, on a scale of 0 to 1, of the rankings of per capita income, average education and fertility rates, identifying where countries or other geographic areas fall on the spectrum of development (Global Burden of Disease Study., 2019). Finally, we conducted sensitivity analysis by removing a study according to the rules of the meta-analysis.

Statistical analyses were performed using the R Statistical software (version 1.3.1093). An alpha of 0.05 was applied as the cutoff point for significance.

Assessment of publication bias

Publication bias was analyze using funnel plots (visually), and if more than ten studies contribute to the outcome, and Egger's statistical test (Higgins et al., 2011) was performed.

Assessment of quality of evidence

The quality of evidence for each outcome was evaluated independently by two reviewrs (J.C.U and C.C.D) by adopting the Grading of Recommendations Assessment Development and Evaluation (GRADE) approach (Ryan & Hill, 2016). In observational studies, this system starts with a low grade and can be either upgraded or downgraded. Risk of bias, inconsistency, indirectness, imprecision, and publication bias are reasons to lower the certainty rating of evidence. The presence of a large effect, dose response gradient and no plausible confounding promote upgrade.

Results

Study selection

In total, 5363 records were initially identified across all electronic databases. After removing duplicates, 3754 screened results remained. Following a comprehensive evaluation of the abstracts, 217 reports were deemed potentially useful and selected for full text reading. Of these 217 reports, 21 could not be evaluated as efforts to access the full-text articles were unsuccessful. Therefore, 196 full-text reports were assessed for eligibility. Of these 196, 154 reports were excluded due to: inappropriate exposure (n=39); unsuitable outcome (n=33); inappropriate study design (n=17); inappropriate language (n=7), no comparison group (n=56) and inappropriate population (n=2). Authorship and exclusion reasons of the 154 reports excluded are presented as supplementary material (Appendix 2). Forty-two articles were

retained for systematic review. Figure 1 presents the study identification and screening process following the PRISMA flow diagram.

Study characteristics

Among the 42 studies included, 31 (73.81%) were cross-sectional studies, 10 (23.81%) were case-control studies, and only one (2.38%) was a retrospective longitudinal cohort study. A total of 7217 participants, including 3472 (48.11%) subjects with ASD and 3745 (51.89%) control subjects were evaluated. Some studies did not provide data separated by gender of the participants (Babu & Roy, 2022; Kuter & Uzel, 2021; Al Musawi & Al-Dabagh, 2019; Frank et al., 2019, Onol & Kirzioglu, 2018; Rai et al., 2012; Vajawat & Deepika, 2012). Considering the studies that provided the gender data, in the ASD group 587 (16.91%) participants were female, while 2086 (60.08%) were male and as for the control group, 1181 (31.53%) were female and 1854 (49.51%) were male. Studies from 22 different countries were included, from different regions of Asia, South America, North America, Europe and Africa. The studies included subjects age range of 2 to 41 years. Seven of the 42 studies included participants over 18 years of age (Meuffels et al., 2022; Blomqvist et al., 2015; Fahlvik-Planefeldt & Herrström, 2001; Leiva-García et al., 2019; Loo et al., 2008; Orellana et al., 2012; Vajawat & Deepika, 2012). 71.43% of participants with ASD were recruited from centers and schools geared towards autistic and/or special needs individuals, while 26.19% were recruited from hospitals, psychiatric clinics or university clinics and 2.38% were recruited from schools in general. Among the control participants, 47.62% were from "regular" schools, 40.48% were from dental or medical clinics, 4.76% were from hospitals, and 7.14% were siblings, relatives or friends of participants with ASD.

The main studies strategy to control plausible confounders was select matched controls for some characteristics of the ASD group. Control participants were matched for age, sex, socioeconomic status and demographic characteristics in 61.90%, 38.09%, 28.57%

and 7.14% of the included studies, respectively. Medication use was reported in 538 participants with ASD versus 10 of the control group. Regarding the ASD diagnosis, 10 studies reported that the diagnosis was established by physicians (Alaki et al., 2016; Bassoukou et al., 2009; Bhandary & Nary, 2017; Blomqvist et al., 2015; Daneshvar et al., 2019; Fahlvik-Planefeldt & Herrström 2001; Farmani et al., 2020; Fontaine-Sylvestre et al., 2017; Jaber, 2011; Loo et al., 2008). 3 studies (Blomqvist et al., 2015; El Khatib et al., 2014; Onol & Kırzıoğlu, 2018) used the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition Text Revision (DSM-IVTR) as a diagnostic tool, 3 studies (Leiva-García et al., 2019; Qiao et al., 2018; Sarnat et al., 2016) used the Diagnostic and Statistical Manual of Mental Disorders- Fifth Edition (DSM-V) tool, one study (Moorthy et al., 2022) used the DSM-IVTR or DSM-V and two studies (Bagattoni et al., 2021; Daneshvar et al., 2019) reported to use the Frank'l behavior rating scale. The other studies did not report how the diagnosis was established.

Risk of bias (methodological quality)

Using the Newcastle-Ottawa for cohort studies and the adapted for cross-sectional studies, the risk of bias is reported in the Table 1. When assessing methodological quality, 4 (9.52%) studies had a low risk of bias, 24 (57.14%) had a moderate risk, and 14 (33.33%) of the studies had a high risk of bias. The only longitudinal study (Frank et al., 2019) included had a high risk of bias, mainly due to the low score on the "Selection" item because the sample was not representative. 14 studies presented a high risk of bias (Babu & Roy, 2022; Meshki et al., 2021; Sam et al., 2021; Tulumbaci et al., 2020; Daneshvar et al., 2019; Morales-Chávez & Villarroel-Dorrego, 2018; Morales-Chávez et al., 2019; Onol & Kirzioglu, 2018; Fontaine-Sylvestre et al., 2017; Sarnat et al., 2016; Rai et al., 2012; Bassoukou et al., 2009; Namal et al., 2007), mainly due to the low score on the item "Selection", since the samples were not representative samples, only a selected group of individuals. Besides, the

studies received a high risk of bias when scoring low in the "Outcome" domain, because no data of examiner calibration have been reported. In addition, some studies not reporting used statistical test to analyze the data, and/or association measures, and/or confidence intervals or probability level (p-value). In general, "Selection" was the least fulfilled domain among all the studies evaluated.

Dental caries experience

A summary of the characteristics of 33 studies that evaluated caries are showed in the Table 2.

Caries experience was evaluated in 33 studies, with a total of 5831 subjects evaluated, 2827 autistic subjects and 3004 controls. Caries prevalence was evaluated in 18 studies. Of these 18, only 6 studies reported higher caries prevalence in autistic subjects than in controls (Bagattoni et al., 2021; Leva-García et al., 2019; Suhaib et al., 2019; Alaki et al., 2016; Al-Maweri et al., 2014; Jaber, 2011). Eight studies appointed to opposite direction (Kuter & Uzel, 2021; Kuter & Guler, 2019; Morales-Chávez et al., 2019; Sarnat et al., 2016; Du et al., 2015; Vajawat & Deepika, 2012; Loo et al., 2008; Namal et al., 2007).

Caries severity was reported in 35 studies. In 11 studies, the caries severity was higher in autistic subjects than controls (Babu & Roy., 2022; Bagattoni et al., 2021; Meshki et al., 2021; Daneshvar et al., 2019; Qiao et al., 2018; Bhandary & Nary, 2017; Alaki et al., 2016; Al-Maweri et al., 2014; Richa et al., 2014; Jaber, 2011; Frank et al., 2019). In 11 studies, lower caries severity was found for ASD when compared to controls (Kuter & Uzel, 2021; Sam et al., 2021; Kuter & Guler, 2019; Morales-Chávez et al., 2019; Sarnat et al., 2016; Du et al., 2015; Fakroon et al., 2014; Orellana et al., 2012; Vajawat & Deepika, 2012; Loo et al., 2008; Namal et al., 2007). Among the studies that evaluated caries prevalence or severity, only 3 showed low risk of bias (Alaki et al., 2016; Du et al., 2015; El Khatib et al. 2013).

Regarding untreated tooth decay, the results were also inconsistent. In 7 studies, autistic individuals had more primary and/or permanent teeth and/or decayed surfaces in the primary, mixed, and/or permanent dentition than controls (Babu & Roy, 2022; Bagattoni et al., 2021; Jaber, 2011; Daneshvar et al., 2019; El Khatib et al., 2013; Onol & Kirzioglu, 2018; Alaki et al., 2016). On the other hand, 4 studies showed findings in opposite direction (Faker et al., 2022; Du et al., 2015; Namal et al., 2007; Fakroon et al., 2014).

About filled teeth or surfaces, the mean number and/or prevalence in primary and/or permanent teeth was lower in subjects with ASD than in controls in 6 studies (Babu & Roy., 2022; Bagattoni et al., 2021; Daneshvar et al., 2019; Onol & Kirzioglu, 2018; El Khatib et al., 2014; Orellana et al., 2012). Also in 4 studies, there was no difference between autistic and controls (Bhandary & Nary, 2017; Alaki et al., 2016; Du et al., 2015; Al-Maweri et al., 2014).

Three studies evaluated the relationship between caries and ASD skills/severity. Among autistic individuals, the mean DMFT/dmft score in children with severe ASD was higher than in children with mild and moderate ASD (Daneshvar et al., 2019). For Sarnat et al. (2016) and Blomqvist et al. (2015) no significant correlation was observed between life skills or motor skills and caries in individuals with ASD.

In the study by Loo et al. (2008), autistic individuals who used psychotropic medication were significantly older than patients who did not use them, and there was no significant difference in terms of caries between them. Autistic individuals who received an additional diagnosis were also significantly older but still did not differ in DMFT scores when compared to individuals who did not receive the additional diagnosis.

In the quantitative analysis, no statistical difference, with considerable heterogeneity (I^2 87%, $p<0.01$, prediction interval of 0.54 to 1.79), was observed between ASD and non-ASD subjects in overall caries prevalence (RR 0.98, 95% CI 0.84 to 1.15) (Figure 2). In the subgroup analysis considering only tooth deciduous, lower proportion of ASD had caried (RR

0.69, 95% CI 0.58 to 0.83) (Appendix 3, Figure 1), but only 2 studies were included in this category. Metanalysis of subgroups of permanent (I^2 82%, p<0.01) or deciduous+permanent (I^2 87%, p<0.01) teeth confirmed the overall findings with no statistical difference between ASD and non-ASD subjects. Subgroups analysis considering the bias risk maintained the high heterogeneity observed in the overall metanalysis, and independently of the risk category, no significant difference was verified between ASD and non-ASD subjects (Appendix 3, Figure 2). A further exploratory subgroup analysis was performed grouping the studies according: a) presence (1 up 2, and 3+ matching variables) and absence of matched controls (Appendix 3, Figure 3); and b) different social demographic index (Appendix 3, Figure 4). In all subgroups, no change in the direction of the overall estimate was observed, with exception of the studies performed from middle SDI countries that demonstrated caries lower risk in ASD subjects. However, all subgroup analysis maintained high heterogeneity. Sensibility analysis also indicated that the effect estimated was not influenced by no study (Appendix 4, Figure 1).

The caries severity pooled estimate showed no statistical difference between ASD and non-ASD subjects (SMD 0.17, 95% CI -0.02 to 0.36, I2 92%, p<0.01, prediction interval -1.07, 1.41) (Figure 3). To further subgroups analysis (Appendix 3, Figures 5, 6 and 7), we reported the caries findings considering data from deciduous, permanent, and grouping deciduous+permanent teeth as described by the authors of the primary studies. In deciduous (SMD 0.29, 95% CI 0.04-0.53, prediction interval -0.79, 1.37) and in deciduous+permanent teeth (SMD 0.43, 95% CI 0.07-0.79 prediction interval -1.20, 2.06), higher caries severity was observed in ASD subjects with considerable heterogeneity (Figure 3). Additional subgroups analysis showed that in deciduous teeth, the caries severity remains higher in ASD subjects from countries with Low/Low-midle SDI, but the difference lost significance in countries ranged from High to Middle SDI. In deciduous+permanent teeth, as only 4 studies were included in overall estimate, the subgroup analysis maintained higher caries severity in

countries ranged from High to Middle SDI, but not in Low SDI because only 1 study was present in this category. Subgroups analysis considering the number of matching variables and bias risk showed that, in general, a higher caries severity in ASD subjects was observed from studies without matched controls and with high bias risk, especially in deciduous teeth. We should highlight that the heterogeneity remained considerable in the subgroups analysis.

Higher mean of non-treated caries lesions was observed in ASD subjects (SMD 0.27, 95% CI 0.06 to 0.48, I^2 88%, p<0.01, prediction interval -0.72 to 1.26) (Figure 4). This finding was maintained only in deciduous teeth (SMD 0.30, 95% CI 0.01 to 0.60), but no in permanent or deciduous+permanent teeth. In deciduous teeth, the higher mean of non-treated lesions in ASD subjects remains in studies with high (SMD 0.63, 95% CI 0.38 to 0.88) and low (SMD 0.42, 95% CI 0.05 to 0.80) bias risk; an in studies without (SMD 0.51, 95% CI 0.25 to 0.78, I2 41%) and with 1 up 2 matching variables (SMD 0.46, 95% CI 0.25 to 0.68, I2 0%). Interestingly, subgroup analysis of studies from countries with Low/low-middle SDI showed higher non-treated caries lesions in ASD subjects in both, deciduous (SMD 0.49, 95% CI 0.21 to 0.77, I^2 25%, p=0.26) and permanent teeth (SMD 0.53, 95% CI 0.12 to 0.95, I^2 64%, p=0.06), while no difference is observed in countries with High/high-middle SDI. Subgroups analysis disponible (Appendix 3, Figure 8, 9 and 10).

Higher mean of filled surfaces and teeth were observed in non-ASD subjects (SMD -0.30, 95% CI -0.49 to -0.10, I^2 85%, p<0.01, prediction interval -1.19, 059) (Figure 5). This estimate remained significant with lower heterogeneity in studies considering primary teeth with high risk of bias (SMD -0.65, 95% CI -0.98 to -0.32, I^2 41%) and without matched controls (SMD -0.52, 95% CI -0.83 to 0.22, I^2 56%). No difference was observed in SDI subgroups. Subgroups analysis disponible (Appendix 3, Figure 11, 12 and 13).

In general, no difference was observed in the caries prevalence between ASD and non-ASD subjects. However, ASD showed higher caries severity in deciduous teeth.

Oral hygiene and periodontal status

The methodological characteristics and main results from 30 studies that evaluated oral hygiene and periodontal status are showed in the Table 2.

Oral hygiene and periodontal status were evaluated in 30 studies, involving 4.377 individuals (2.119 individuals with ASD and 2.258 controls). The index used were simplified oral hygiene index OHI-S (Greene & Vermillion, 1964), community periodontal treatment needs index CPITN (Cutress et al., 1987), oral cleanliness (James et al., 1960), modified gingival index MGI (Lobene et al., 1986), plaque index PlI (Sillness & Löe 1964 and Löe 1967), visual periodontal index (Cappelli & Brown, 2002), gingival index GI (Löe & Sillness 1963 and Loe & Silness 1967) and gingival bleeding GBI (Ainamo & Bay, 1975).

Eleven studies assessed oral hygiene status using OHI-S (Meshki et al., 2021; Moorthy et al., 2022; Sam et al., 2021; Morales-Chávez & Vilarroel-Dorrego, 2018; Morales-Chávez et al., 2019; Bhandary & Nary, 2017; Alaki et al., 2016; Richa et al., 2014; Orellana et al., 2012; Rai et al., 2012; Jaber, 2011). Of these studies, only two of them showed no differences between ASD and controls (Sam et al., 2021; Alaki et al., 2016), while all others showed worse oral hygiene for ASD when compared to controls. Eleven studies evaluated oral hygiene status from PlI, seven of these studies showed higher prevalence and/or mean plaque for individuals with ASD when compared to controls (Bagattoni et al., 2021; Leva-García et al., 2019; Onol & Kirzioglu, 2018; Diab et al., 2016; Al-Maweri et al., 2014; El Khatib et al., 2014; Vajawat & Deepika, 2012). Some studies did not report the index to measure oral hygiene, two of them showed higher plaque values for ASD than controls (Morales-Chávez et al., 2019; Suhaib et al., 2019).

Higher prevalence of excellent/good/fair oral hygiene was observed in non-ASD (RR 0.79, 95% CI 0.64 to 0.96, I^2 88%, $p<0.01$), while ASD subjects showed higher prevalence of poor/very poor oral hygiene (RR 2.46, 95% CI 1.23 to 4.91, I^2 83%, $p<0.01$) (Figure 6A).

Studies that used PlI reduced the heterogeneity when compared to the overall pooled estimates in both categories, excellent/good/fair OH (I^2 61%, p=0.11) and poor/very poor OH (I^2 0%, p=0.00) and ASD subjects (Table 6). Studies with moderate bias also reduced the heterogeneity (I^2 8%, p=0.36) and strengthened that ASD subjects have worst dental plaque control. Findings in the same direction was observed in studies applying 3 or more matching variables (Table 6).

Higher pooled estimate of plaque mean scores was verified in ASD when compared to the non-ASD subjects (SMD 0.59, 95% CI 0.24 to 0.94, prediction interval -0.91, 2.09) (Figure 6B). The heterogeneity was high and did not reduce in subgroups analysis (Table 7). The lowest heterogeneity was observed in the subgroup analysis when 3 matching variables between ASD and controls were applied (I^2 63%, p=0.044). Sensitivity analysis (Figure 7) omitting Du et al (2015) study promoted an increase of the overall estimate size (SMD 0.62, 95% CI 0.54 to 0.71) strengthening the highest plaque scores in ASD.

Gingival inflammation was assessed in 4 studies using the CPITN (Leva-García et al., 2019; Fakroon et al., 2014; Vajawat; Deepika, 2012; Luppanapornlarp et al., 2010), all of these studies showed a higher prevalence of bleeding for ASD. Nine studies assessed the presence and/or severity of gingivitis through the GI (Sam et al., 2021; Daneshvar et al., 2019; Onol & Kirzioglu, 2018; Qiao et al., 2018; Du et al., 2015; Al-Maweri et al., 2014; El Khatib et al., 2014; Vajawat & Deepika, 2012; Jaber, 2011). Of these studies, only Du et al. (2015) demonstrated better gingival health for individuals with ASD when compared to controls and the study of Sam et al. (2021) found no differences. The other studies showed a higher mean and/or prevalence of gingivitis for autistic individuals, indicating a worse gingival condition. Three studies reported data on gingivitis without informing the index used, two of which found no differences between groups (Fahlvik-Planefeldt & Herrström, 2001; Tulumbaci et al., 2020).

Two studies evaluated the relationship between ASD skills/functioning and OH. Sarnat et al. (2016) reported that ASD children with high life skills had significantly better OH; and Rai et al. (2012) found that ASD children with low functioning had significantly worse OH when compared to children with average functioning.

ASD showed higher overall gingivitis prevalence (RR 1.31, 95% CI 1.02 to 1.70, prediction interval 0.54, 3.22) when compared to the non-ASD subjects with considerable heterogeneity (I^2 89%, $p<0.01$) (Figure 8A). Subgroups analysis (Table 8) considering index type (I^2 ranged from 81% to 97%), risk of bias (I^2 ranged from 73% to 93%) and SDI (I^2 ranged from 86% to 95%) maintained the high heterogeneity. Significantly higher gingivitis prevalence with low heterogeneity was found only in subgroup analysis of generalized gingivitis (RR 5.63, 95% CI 2.03 to 15.64, I^2 63%, $p=0.34$) and of those studies with matched controls to 3 or more variables (RR 2.22, 95% CI 1.69 to 2.91, I^2 0%, $p=0.37$), which increased the strength association between ASD subjects and gingivitis when compared to the overall estimate. As most of the studies included in this metanalysis included studies with 1 up 2 matching variables and with moderate risk of bias, we performed sensitivity analyses to evaluate whether the estimate direction/size would be affected by any study omission. We verified that among the 11 studies included, 6 studies when omitted caused statistical significance lost (Leiva-Garcia et al. 2019, Bhandary & Hari 2017, Al-Maweri et al. 2014, Fakroon et al. 2014, Jaber et al. 2011, Luppanapornlarp et al. 2010). However, the omission of no study promoted change of the association direction (Figure 8B).

ASD presented higher gingivitis severity (SMD 0.45, 95% CI 0.02 to 0.88, I^2 95%, $p<0.01$, prediction interval -1.14, 2.04) (Figure 9A) when compared to the non-ASD subjects. Subgroups analysis (Table 9) considering risk of bias, SDI, and number of matching variables maintained the high heterogeneity. Of the 9 studies included in the overall metanalysis, 7 used GI (Löe & Silness 1963 or Löe 1967) as gingivitis index, 7 did not report how the

examination protocol was evaluated, and 7 showed matched controls to 1 up 2 variables. In this context, sensitivity analysis was performed. The omission of Du et al. (2015) study significantly increase the pooled estimate of gingivitis severity (SMD 0.53, 95% CI 0.41 to 0.65), specially highlight the confidence interval inferior limit (Figure 9B).

Tooth loss

A summary of the characteristics of 14 studies that reported tooth loss data showed in the Table 2. Tooth loss was reported from 2337 subjects, 918 autistic subjects and 1419 controls. In 13 studies, the component "missing" of the DMFT index was used to report tooth loss (Babu & Roy., 2022; Bagattoni et al., 2021; Daneshvar et al., 2019; Onol & Kirzioglu, 2018; Bhandary & Nary, 2017; Alaki et al., 2016; Blomqvist et al., 2015; Du et al., 2015; Al-Maweri et al., 2014; Orellana et al., 2012; Fakroon et al., 2014; Jaber, 2011; Namal et al., 2007). Of these 14 studies, 7 studies (Babu & Roy., 2022; Onol & Kirzioglu, 2018; Al-Maweri et al., 2014; Orellana et al., 2012; Jaber, 2011; Luppanapornlarp et al., 2010; Namal et al., 2007) showed higher means of tooth loss and of missing surfaces in autistic subjects when compared to the controls. In 3 studies, no difference was observed between groups (Bagattoni et al., 2021; Bhandary & Nary, 2017; Blomqvist et al., 2015).

No difference in the tooth loss was observed in overall estimate between ASD and non-ASD (SMD 0.12, 95% CI -0.02 to 0.26, I^2 55%, prediction interval -0.35, 0.59). The same finding was observed in primary (I^2 0%, p=0.40) and in permanent teeth (I^2 65%, p<0.01) (Figure 10). Subgroups, sensitivity and publication bias analyses were performed using data from permanent teeth. Studies with high risk of bias and without matched control (Appendix 3, Figure 14 and 15) showed higher significantly estimates of tooth loss in ASD subjects. In the sensitivity analysis, 3 studies have shown to contribute to the statistical significance absence in the tooth loss estimate (Babu & Roy, 2022; Onol & Kirzioglu, 2018; Namal et al., 2007) (Appendix 4, Figure 2).

Malocclusion

A summary of the characteristics of 13 studies that reporting malocclusion data are showed in the Table 3. Thirteen studies evaluated the prevalence of malocclusion including 2925 individuals (1539 ASD vs 1386 controls). Six studies reported Angle classification (Meuffels et al., 2022; Bagattoni et al., 2021; Farmani et al., 2020; Leiva-García et al., 2019; Onol & Kırzıoğlu, 2018; Fontaine-Sylvestre et al., 2017), 1 reported the Foster & Hamilton Index (1969) (Aljubour & Al-Sehaibany, 2018), another one used Orthodontic Treatment Need Index (ALMusawi & Al-Dabagh 2019), and one study used Dental Aesthetic Index (DAI) (Luppanapornlarp et al., 2010). In 3 studies no index was reported (Kuter & Guler, 2019; Du et al., 2015; Orellana et al., 2012).

There was not consistence between studies regarding malocclusion prevalence. Five studies showed a similar overall prevalence between ASD and controls (Meuffels et al., 2022; Bagattoni et al., 2021; Farmani et al., 2020; Du et al., 2015; Luppanapornlarp et al., 2010). The other 7 studies reported a higher overall prevalence of malocclusion in ASD subjects (AlMusawi & Al-Dabagh, 2019; Kuter & Guler, 2019; Leiva-García et al., 2019; Aljubour & Al-Sehaibany, 2019; Onol & Kirzioglu, 2018; Fontaine-Sylvestre et al., 2017; Orellana et al., 2012), regardless of their demographic characteristics (Fontaine-Sylvestre et al., 2017).

Quantitative analysis showed no statistical difference in the prevalence of any type of Angle Class between ASD and non-ASD (Figure 11). The heterogeneity was higher in the Class I and II metanalysis, while to Class III metanalysis the I^2 value was of 0%, p=0.44 and Tau2=0 indicating low heterogeneity. Subgroups analysis considering risk of bias confirmed no difference between ASD and controls, with exception of studies showing high risk of bias and type I of Angle Class (RR 0.79, 95% CI 0.68 to 0.92, I^2 0%) (Appendix 3, Figure 16). Metanalysis of studies without matched controls considering type I of Angle Class showed higher prevalence in non-ASD subjects (RR 0.84, 95% CI 0.73 to 0.97, I^2 0%) (Appendix 3,

Figure 17). SDI subgroups maintained no difference between ASD and controls showing low heterogeneity in High and High-middle countries, independently of the Angle Class type (Appendix 3, Figure 18).

Increased overjet was significantly more prevalent in ASD (RR 2.16, 95% CI 1.28 to 3.64, I^2 89%, prediction interval 0.35, 13.24) with considerable heterogeneity (Figure 12A). The heterogeneity remained high in all subgroups analysis (Table 10). Of the 6 included studies, 4 showed moderate risk of bias, and 4 presented 1 up 2 matching variables between ASD and controls. In this context, we considered that sensitivity analysis would be more important to explain some change in the overall estimate than subgroups analysis. In the sensitivity analysis, no omitted study caused statistical significance lost or change of the direction overall estimate (Appendix 4, Figure 3).

ASD subjects had a higher overall prevalence of overbite (RR 1.62, 95% CI 1.02 to 2.59, I^2 80%, prediction interval 0.38, 7.02) (Figure 12B). Subgroups analysis considering risk of bias and absence of matched controls reduced the heterogeneity and maintained the higher prevalence of overbite in ASD subjects (Table 11). SDI subgroups maintained similar heterogeneity to that observed from overall estimate (Table 11). Studies subgroup with moderate risk of bias strengthened the pooled estimate (RR 2.27, 95% CI 1.46 to 3.54). Four studies (Meuffels et al., 2022; Farmani et al., 2020; AlMusawi & Al-Dabagh 2019; Aljubour & Al-Sehaibany, 2018) when individually omitted promoted the statistical significance loss; although the estimate direction remains favoring the higher overbite prevalence in ASD subjects (Appendix 4, Figure 4).

Crossbite prevalence was higher significantly in ASD subjects (RR 1.48, 95% CI 1.02 to 2.13, I^2 57%, prediction interval 0.54, 4.05) (Figure 12C). In subgroups analysis, the heterogeneity was maitanined similar that observed from overall estimate (Appendix 3, Figure 19 and 20). In the sensitivity analysis, by omitting one by one, 5 studies promoted statistical

significance loss from overall estimate, although the estimate direction remained unchanged (Meuffels et al., 2022; ALMusawi & Al-Dabagh, 2019; Aljubour and Al-Sehaibany, 2018; Fontaine-Sylvestre et al., 2017; Du et al., 2015) (Appendix 4, Figure 5).

Openbite was significantly more prevalent in ASD (RR 2.37, 95% CI 1.46 to 3.85, I^2 54%, prediction interval 0.60, 9.38) (Figure 13). Subgroup analysis (Table 12) considering countries with SDI High/high-middle reduced the heterogeneity and maintained the estimate size and significane (I^2 3%, p=.041). Studies with moderate risk of bias ans applying 1 up 2 matching variables between groups show heterogeneity similar to that verified in overall estimate and maintained the size and significance of the higher openbite prevalence in ASD subjects. Sensibility analysis also indicated that the estimated effect was not influenced by no study (Appendix 4, Figure 6).

Bruxism

The methodological characteristics and findings main of the studies that reported bruxism data comparing ASD and controls are presented in the Table 4. Thirteen studies evaluated the bruxism and of tooth wear prevalence with a total of 2471 individuals, including 1286 ASD and 1185 controls (Bagattoni et al., 2021; Kuter & Uzel, 2021; Daneshvar et al., 2019; Kuter & Guler, 2019; Leiva-García et al., 2019; Suhaib et al., 2019; Onol & Kirzioglu, 2018; Bhandary & Nary, 2017; Sarnat et al., 2016; Du et al., 2015; El Khatib et al., 2014; Orellana et al., 2012; Fahlvik-Planefeldt & Herrström, 2001). Of these 13, 9 studies have shown a higher bruxism prevalence and signs of tooth wear in ASD when compared to controls (Kuter & Uzel, 2021; Daneshvar et al., 2019; Kuter & Guler, 2019; Leiva-García et al., 2019; Suhaib et al., 2019; Onol & Kirzioglu, 2018; El Khatib et al., 2014; Orellana et al., 2012; Fahlvik-Planefeldt & Herrström, 2001); 2 studies have demonstrated a lower prevalence of dental erosion and enamel defects for ASD (Bhandary & Nary 2017; Sarnat et

al., 2016); and two studies reported a similar prevalence of tooth wear between ASD and controls (Bagattoni et al., 2021; Du et al., 2015).

Onol & Kirzioglu (2018) verified no relationship between the use medication use and the bruxism prevalence in ASD. However, the authors observed that the age that ASD subjects started their special education was associated with bruxism prevalence. Lower bruxism prevalence was observed for subjects that started before 3 years of age.

ASD had a higher overall prevalence of bruxism (RR 4.52, 95% CI 2.07 to 9.86, I^2 85%, prediction interval 0.33, 61.07) than non-ASD subjects (Figure 14). In the subgroups analysis (Table 13), there was overlap between studies with high risk of bias and without matched controls (Daneshvar et al., 2019; Onol & Kirzioglu, 2018), and between studies with moderate risk of bias and 1 up 2 matching variables (Orellana et al. 2012, Leiva Garcia et al. 2019, Suhaib et al. 2019). The studies with high risk of bias/without matched control overestimating the association between ASD and bruxism (RR=10.92, 95% CI 5.46 to 21.86, I^2 29%), while studies with moderate risk of bias/1 up 2 matching variables reduced the association strength between bruxism and ASD (RR 2.08, 95% CI 1.10 to 3.94, I2 58%). SDI subgroups failed to explain the heterogeneity, because independently of the SDI category, the heterogeneity was similar than that observed from overall estimate (Table 13). Sensitivity analysis showed that the omission of any studies did not change the statistical significance or the direction of the overall estimate (Appendix 4, Figure 7).

Salivary status: flow rate, pH and buffering capacities

A summary of the characteristics of 9 studies that reported salivary data are showed in the Table 5. Nine studies presented prevalence or mean data of salivary factors, such as flow rate, pH, and buffering capacities (Kuter & Uzel, 2021; Kuter & Guler, 2019; Morales-Chavez et al., 2019; Onol & Kirzioglu, 2018; Bhandary & Nary, 2017; Diab et al., 2016; Blomqvist et al., 2015; Rai et al., 2012; Bassoukou et al., 2009). Two studies (Bhandary &

Nary., 2017; Diab et al., 2016) found a lower salivary pH in participants with ASD when compared to the controls, while another 2 studies (Morales-Chávez et al., 2019; Rai et al., 2012) found no differences between groups.

Six studies reported flow rate data between ASD and control groups. Two studies (Bhandary & Nary., 2017; Bassoukou et al., 2009) reported no difference between groups. Blomqvist and coworkers (2015) found a lower salivary secretion in ASD subjects when compared to the controls, regardless of medication use. In the other hand, Kuter and Guler (2019) and Kuter and Uzel (2021) showed higher salivary secretion in ASD subjects. Onol and Kirzioglu (2018) showed no difference between ASD and controls regarding dry mouth and abnormal swallowing habits. Besides, the authors demonstrated that the relationship between medication usage and dryness of the mouth in children with ASD was not statistically significant.

Three studies reported salivary buffering capacity data (Bhandary & Nary 2017; Diab et al. 2016; Bassoukou et al., 2009). No consensus between the study's findings was observed.

Metanalysis showed no difference in mean salivary flow rate between ASD and non-ASD (SMD -0.48, 95% CI -1.16 to 0.20, I^2 81%) (Figure 15A). Salivary pH was significantly lower in ASD when compared to the non-ASD subjects (SMD -0.62, 95% CI -0.99 to -0.26, I^2 46%) (Figure 15B) when evaluating 4 studies.

Publication bias

Linear regression test of funnel plot asymmetry revealed a symmetrical distribution for the studies of caries prevalence (p-value = 0.7420), severity of tooth loss (p-value = 0.7689) and openbite prevalence (p-value = 0.6179) not indicating publication bias. Funnel plots can be seems in the Appendix 5 (Figure 1 to 3).

Discussion

The present systematic review aimed to compare caries, oral hygiene pattern, periodontal diseases, bruxism, malocclusion, tooth loss and salivary alterations between autistic individuals and neurotypical controls, in addition to presenting an overview of the oral condition in individuals with ASD. Overall, the dental caries experience consistently diverged between the primary studies, both in terms of methodological characteristics and the high statistical heterogeneity, even in subgroup analyses. Caries prevalence did not differ statistically. Caries severity in primary teeth was significantly higher in ASD, with an estimate increase in countries with low/low-middle SDI; a higher estimate was also noted in studies without matched controls and with a high risk of bias. ASD showed higher untreated caries lesions means, in primary teeth the highest means remained in studies without any/with matching variables. For the "filled" component, higher means were found in non-autistic subjects. Tooth loss was also inconsistent across studies, regardless of tooth type. As observed in the caries outcome, high risk of bias and lack of matching presented higher estimates in ASD subjects.

On the other hand, oral hygiene and periodontal status findings were statistically convergent for worse oral hygiene status and higher prevalence and severity of gingival inflammation in autistic individuals, with a high heterogeneity that was partially explained by subgroup and sensitivity analysis. For plaque prevalence, the analysis of the risk of bias and the number of paired variables reduced the heterogeneity and strengthen the overall significance. When evaluating the dental plaque and gingivitis scores, heterogeneity was better explained when the study by Du et al. (2015) was omitted, thus increasing the strength of the combined estimate. Autistic individuals had a higher prevalence of gingivitis and generalized gingivitis, with significantly reduced heterogeneity in studies with a greater number of matching variables.

For the outcome of malocclusion, the results differed greatly between the primary studies, which used different measurement indices, and there was no standardized nomenclature. There was no statistical significance in the prevalence of any type of Angle Class between ASD and non-ASD. Only studies with high risk of bias, without any matching variables and Angle Class type I differed between autistics and controls. Regarding the specific characteristics of malocclusion, ASD subjects showed higher overall prevalence of overjet, overbite, crossbite and openbite. High heterogeneity in all analysis subgroups was verified to overjet and overbite. Crossbite subgroups analysis reduced the heterogeneity and caused statistical significance lost. Openbite subgroups analysis reduced the heterogeneity and maintained a higher statistically prevalence in ASD subjects. Bruxism was the outcome most strongly associated with autistic individuals, studies with high risk of bias/no matched control overestimated the association, while studies with moderate risk of bias/1 to 2 corresponding variables reduced the strength of association between bruxism and ASD. For this outcome, the omission of any study did not change the statistical significance or direction of the overall estimate.

Salivary flow rate, pH and buffering capacity, were evaluated in a few studies, with contrasting results among them. There was no consensus on the buffering capacity between the studies, the mean salivary flow rate did not differ between ASD and non-ASD and with data referring to 4 studies the mean salivary pH in ASD was significantly lower than in non-ASD.

The inconsistent and conflicting findings regarding the caries outcome disagree with Ningrum et al. (2021), who evaluated the DMFT index in only 3 studies, but agree with the systematic reviews by Bartolomé-Villar et al. (2016), and Corridore et al. (2020). We believe that this inconsistency is largely due to the etiology and multifactorial character of caries disease, which is related to lifestyle and behavioral factors as a characteristic of the diet,

including high consumption of carbohydrates (Fejerskov & Kidd, 2003), drugs that interfere with saliva flow (Wolff et al., 2017), host susceptibility (Featherstone, 2000), socioeconomic status (Gibson & Williams, 1999), among others; and not only to the standard of oral hygiene. According to the studies included in the review, few of them provided information about diet, and few studies matched their controls to socioeconomic conditions, important factors in the development and involvement of oral diseases. In addition, the studies practically did not provide information on the level of support of autistic individuals in relation to caries and other oral conditions, or even many studies excluded participants with a more severe involvement of the condition. ASD can show several commitment levels, this ends up generating difficulties in analyzing and interpreting the data quantitatively, when presenting a single data without stratifying it, which can bias the results. Another possible explanation for the inconsistent results between the studies is due to the caries measurement method of the included studies, using the DMFT/dmft index. This classification system considers caries only as an already cavitated lesion extending to dentin (Reddy et al. 2017), does not include enamel lesions and does not differentiate between the severity of caries lesions. In this sense, it ignores the presence of pre-cavitated lesions (Borse et al., 2016) that progress more slowly compared to cavitated lesions (Gomez, 2015). Thus, perhaps many early caries lesions in autistic individuals were not considered.

When stratified by tooth type, deciduous teeth had higher means of dental caries in autistic individuals. Caries affecting primary teeth is the 10th most prevalent disease (Folayan, et al., 2015) and is especially high in many low-income countries (Reisine & Douglass, 1998; Prakash et al., 2012) and in disadvantaged socioeconomic groups (Tomar & Reeves, 2009), as noted in our review. Caries in primary teeth is also a risk factor for caries in permanent dentition (Li & Wang, 2002), due to increasing age and increasing exposure time of teeth to the oral environment, as it represents a continuous and cumulative process

(DevDutt et al., 2015); although we did not find this repercussion in permanent teeth in this review. Higher prevalence of caries in primary compared to permanent dentition may be due to the lower calcium content of primary teeth and structural differences that may increase susceptibility to caries (Saravanan, et al., 2008; Reddy et al., 2017). We assume that this statistically significant difference found in primary teeth and not in permanent teeth may also be a reflection of the age characteristics of the sample of the present review, although age was not an eligibility criterion, most autistic individuals came from studies that included mostly young children and teenagers. In addition to the fact that autistic children who are in the deciduous dentition phase may have greater difficulty in efficiently brushing autonomously and this may contribute to providing an environment more susceptible to greater severity of caries. When considering only the "decay" component of the DMFT/dmft index, autistic individuals presented higher means than non-autistic individuals, while non-autistic individuals presented higher means of filled teeth/surfaces. This reflects the high need for restorative dental management that these individuals are not receiving. Low awareness and inadequate training of dentists, as well as little cooperation of these children in care and their high sensitivity to the oral environment represent barriers to adequate access to dental care (El Khatib et al., 2014), as well as to obtaining dental treatment (Barry et al., 2014), which may justify these findings. These issues can summarize that autistic individuals do not go to the dentist regularly, until they have more complex problems, to the point that both their parents and dentists prefer extraction to previous restorative treatments (Barry et al, 2014), due to the challenging nature of its management (Namal et al., 2007). This situation may result in greater tooth loss, which was not statistically significant in our study; although the included studies show a trend towards greater tooth loss in autistic individuals.

The worst oral hygiene condition, with a greater amount of dental plaque and gingivitis found in autistic individuals, is justified by the absence of an adequate standard of

oral hygiene, which allows plaque accumulation and, in this way, will generate a gingival inflammatory response, resulting in in gingivitis (Loe et al., 1965). Conditions inherent to autism can often hinder behaviors aimed at performing an appropriate dental cleaning, thus contributing to irregular brushing due to limitations in manual dexterity (Klein & Nowak, 1999). In addition to high oral sensitivity (Stein et al., 2013) as well as a low awareness of parents and/or caregivers regarding the maintenance of oral hygiene (Jaber, 2011; El Khatib et al., 2014) also can impair the periodontal health. These findings are in agreement with previous systematic reviews by Bartolomé-Villar et al. (2016) and Corridore et al. (2020).

Higher prevalence of increased overjet, overbite, open bite and crossbite observed in autistic individuals is due to their own behavioral patterns. In the oral environment, they are characterized by harmful oral behaviors manifested as bruxism, habit of biting the tongue, sucking the thumb, pinching the gum and biting objects, in addition to self-injurious behavior (Medina et al., 2003; Al-Sehaibany, 2017; Murshid, 2005), being more susceptible to malocclusion such as anterior open bite, posterior crossbite and excessive overjet (Warren et al., 2001). A greater tendency towards a high and narrow palate was also observed, which is often associated with posterior crossbite (Orellana et al., 2012). Crossbite and open bite may even be related to chewing problems, decreased muscle tone, which provide atypical chewing patterns (Ben-Sasson et al., 2008; Nadon et al., 2011), which are often found in autistic individuals (Leiva-García et al., 2019). In agreement with Barros et al. (2022), in our study, no significant differences were found between the Angle Class categories in autistic individuals and controls, but greater involvement of overjet was found in autistic individuals. Significantly higher prevalence of bruxism in autistic individuals can be understood by the fact that bruxism is designated as a parafunctional activity (De Leeuw, 2008). In autism, behavioral disorders, self-injurious behaviors, aggression, hyperactivity and exacerbated responses to routines and demands are commonly observed (Karande, 2006), including

bruxism is related as an oral manifestation of these habits (Al-Sehaibany, 2017). Bruxism may also be related to ASD due to anxiety and stress (Gillot & Standen, 2007) and effects of medication use for conditions concomitant with ASD (Sarnat et al., 2016). Our bruxism finding ratified results from previous systematic review (Granja et al. al., 2022; Lam et al., 2020).

Salivary pH was significantly lower in autistic individuals, with a limited number of studies, its repercussion is of that a low salivary pH can favor enamel demineralization (Rai et al., 2012) and thus create a favorable environment for greater susceptibility to caries (Diab et al., 2016). Lower salivary pH in autistic individuals was also found in Lam et al. (2020).

Subgroup analyzes for several evaluated outcomes revealed that the findings were overestimated in the presence of some methodological limitations, such as studies that had a high risk of bias and those with no or few paired variables to try to circumvent possible confounding factors. Another point is the sociodemographic factors evaluated in the most diverse countries included, there was a tendency for a low to middle SDI compared to a high/high-middle SDI to have favored statistical significance. This is in agreement with what can be observed worldwide in developed countries, which have better sociodemographic conditions, a reduction in the prevalence of caries and other diseases, due to greater control in the diet, better oral hygiene habits and adequate consumption of fluorides, in addition to focusing on preventive care. Countries with worse sociodemographic conditions, on the other hand, pay attention to more curative health policies and devote little attention to preventive care and oral health promotion (Sudha et al., 2005). Thus, autistic individuals located in more developed countries, with better sociodemographic indices, can also benefit from more preventive oral health care.

Our systematic review compiled a series of outcomes strongly related to autism, through a highly sensitive search strategy in six main databases and gray literature, which

allowed reaching a high number of results from different designs of observational studies. Furthermore, there were no restrictions regarding the age of the participants, period of publication, language and geopolitical area, which is a strong point of our study. On the other hand, methodological limitations of the included studies were observed, which hindered a better understanding and exploratory analysis of the findings, as well as the high value of heterogeneity that even in subgroup and sensitivity analyzes were not correctly explained. Limited samples with an age group predominantly of children and young adults made it difficult to assess and compare the impact of oral conditions that affect autistic individuals in adulthood. In terms of risk of bias assessment, little or no clear description was observed of how outcome measurements were made, little information was provided about the reliability of evaluators, and strategies to control confounders were very limited in terms of variables that play a determining role in the occurrence of diseases, such as socioeconomic conditions. This allowed for a greater decrease in methodological quality, with a greater number of studies at moderate to high risk of bias. In addition, the included studies practically did not investigate associated factors inherent to autism, such as stratification in levels of support/commitment, association with medication use, diet, which may have made it difficult, underestimated and resulted in a greater possibility of biasing the findings. Future studies are needed to try to overcome these methodological difficulties and increase the reliability of the results in order to propose health actions and policies that aim to meet the oral needs of autistic individuals.

Conclusion

Autistic individuals had significantly higher severity of caries in primary teeth, higher means of untreated caries lesions, higher prevalence and severity of worse oral hygiene, plaque and gingivitis, significantly lower salivary pH, higher prevalence of bruxism, overjet,

overbite, crossbite and openbite when compared to neurotypical control subjects. Higher means of restored teeth and surfaces were significantly found in control subjects. In general, studies with a high risk of bias and absence of matching variables strengthened the associations.

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Figure 1. *Flow diagram of screening process.*

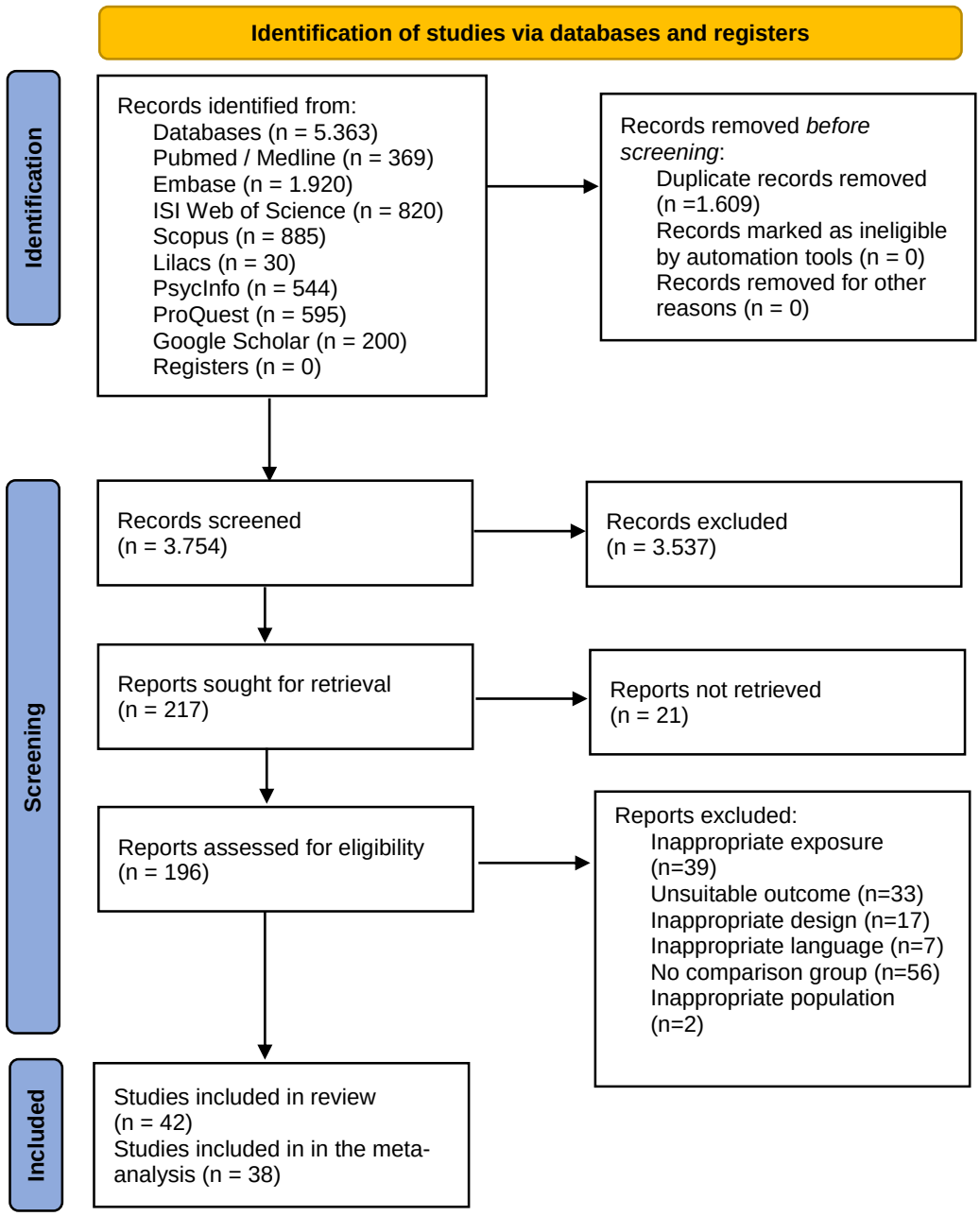


Figure 2. *Forest plot for caries prevalence comparison between ASD and non-ASD.*

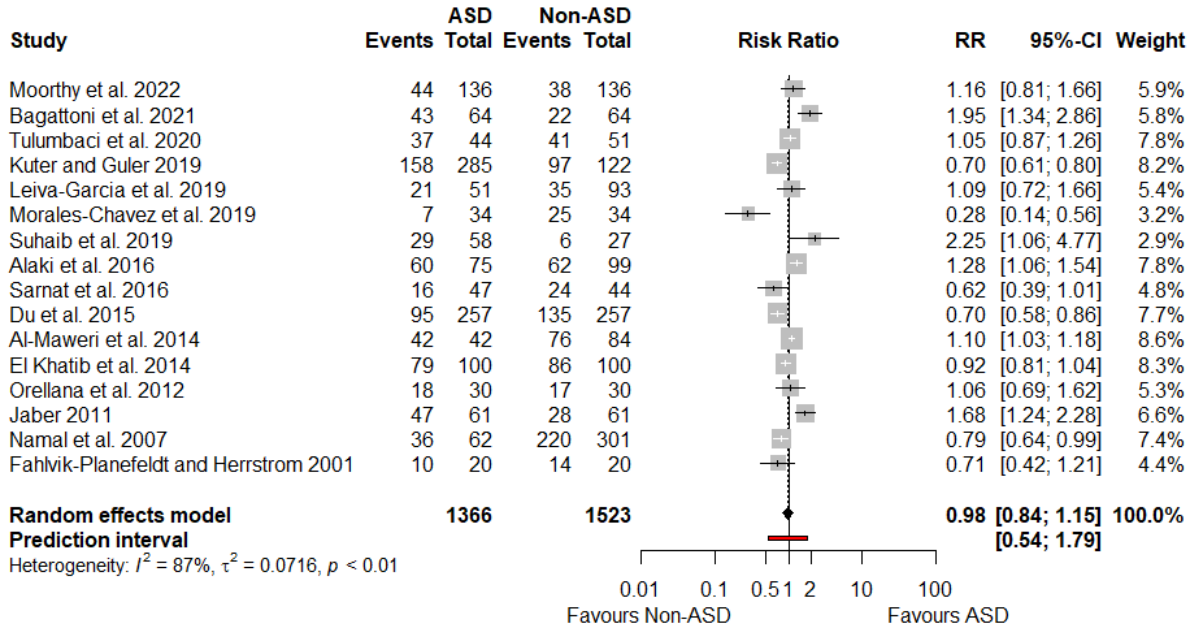


Figure 3. *Forest plot for caries severity (DMFT/dmft/DMFT+dmft means) comparison between ASD and non-ASD according tooth type.*

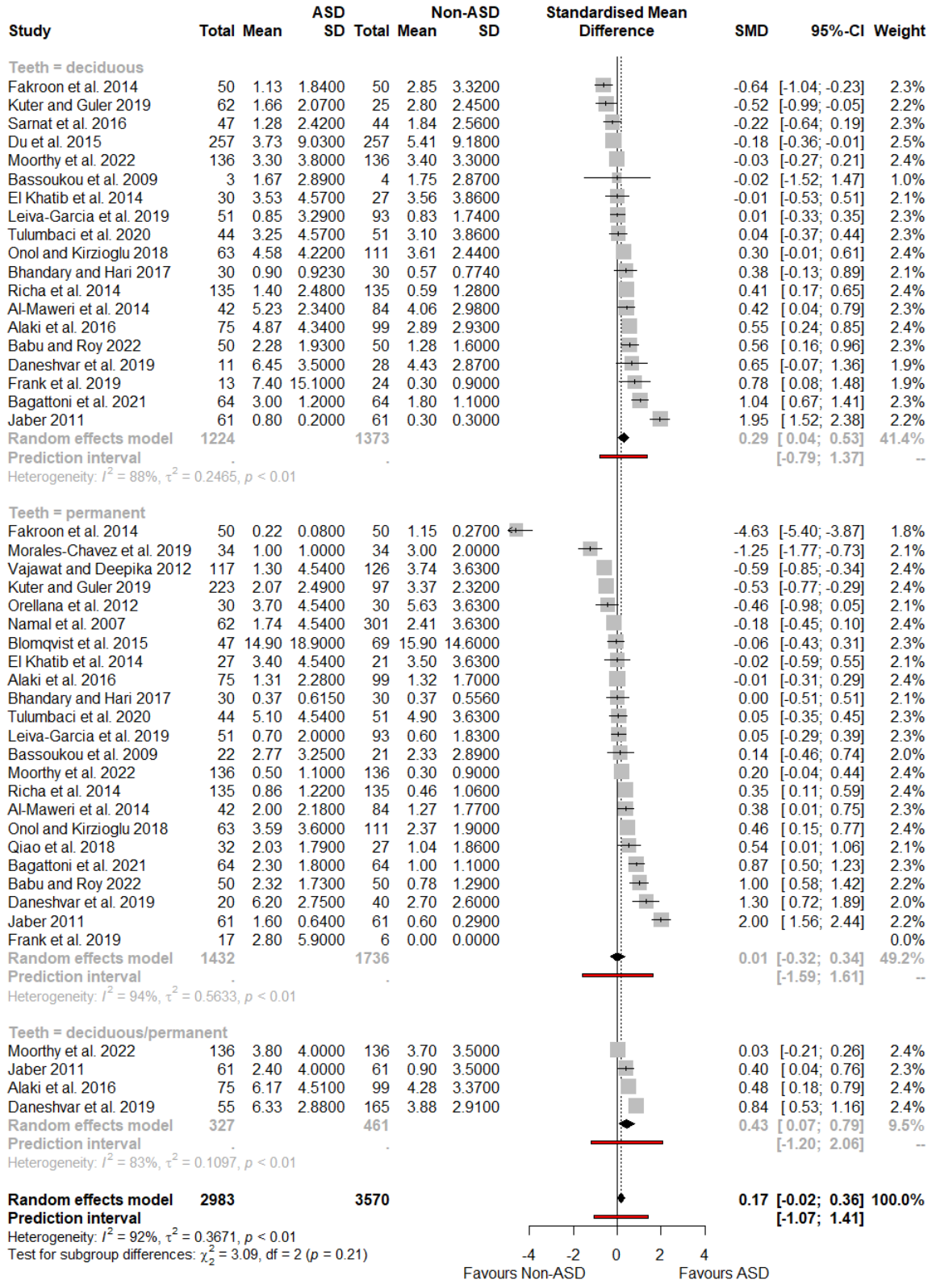
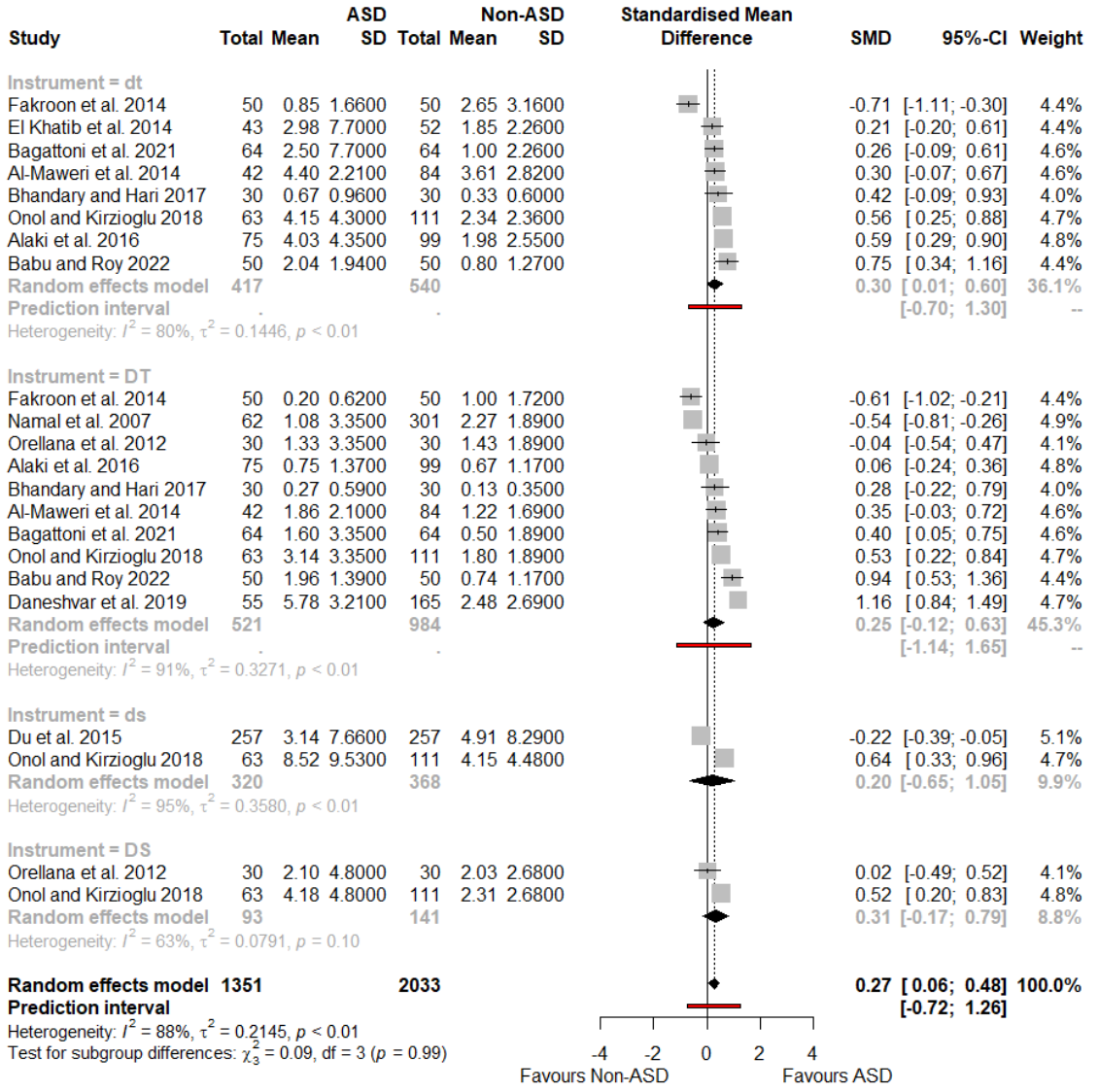
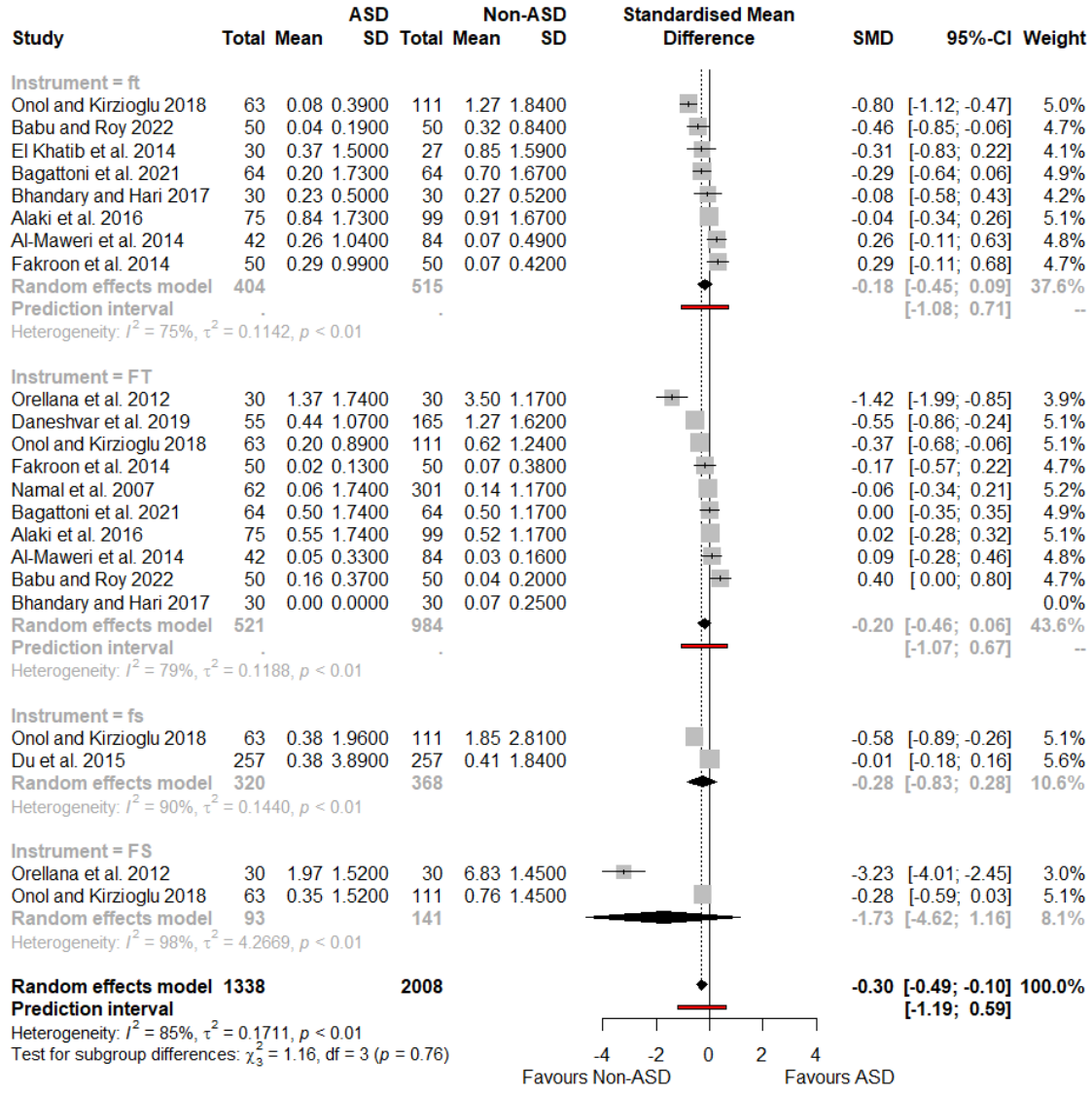


Figure 4. *Forest plot for non-treated caries (DT/dt/DS/ds means) comparison between ASD and non-ASD according Instrument.*



dt: deciduous teeth; DT: permanent teeth; ds: deciduous tooth surface, DS: permanent tooth surface.

Figure 5. *Forest plot for filled caries lesions (FT/ft/FS/fs means) comparison between ASD and non-ASD according Instrument.*



ft: filled deciduous teeth; FT: filled permanent teeth; fs: filled deciduous surface, FS: filled permanent surface.

Figure 6A. *Forest plot for oral hygiene prevalence comparison between ASD and non-ASD according oral hygiene (OHR) categories.*

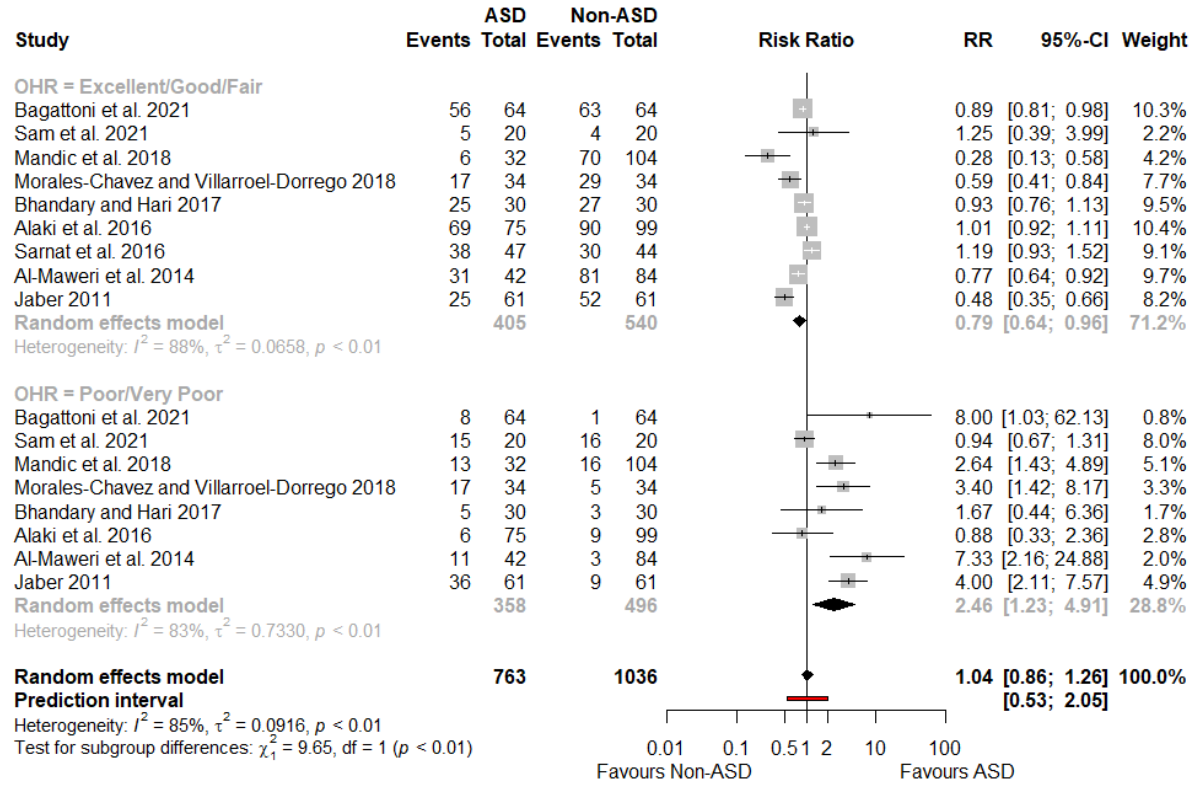


Figure 6B. *Forest plot for plaque severity comparison between ASD and non-ASD.*

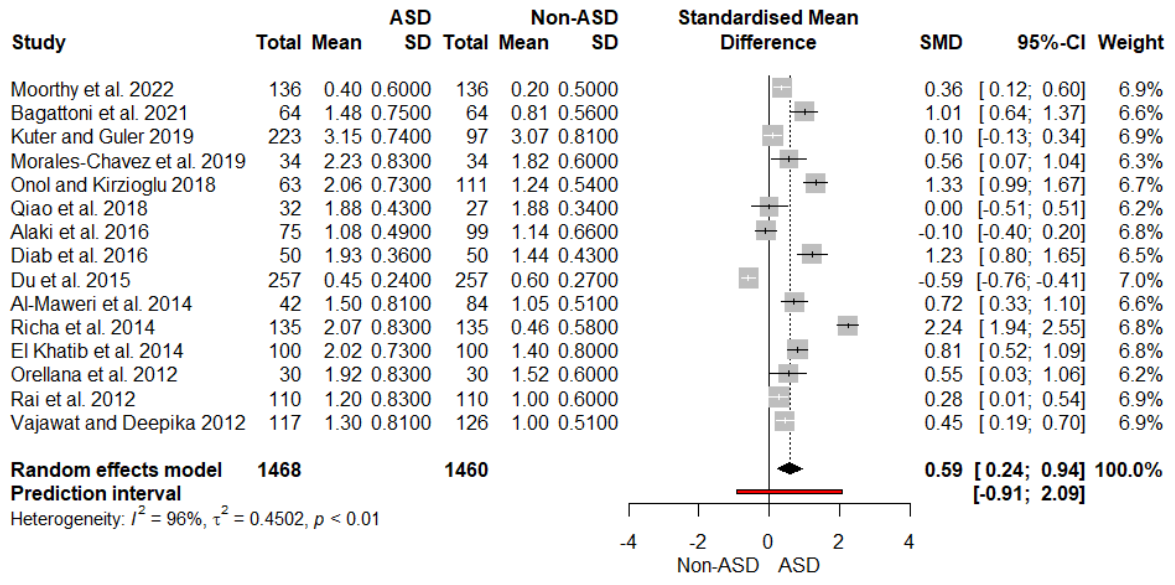


Figure 7. *Sensitivity analysis of plaque severity. SMD and 95% CI were determined by omitting each study according to the rules of the metanalysis.*

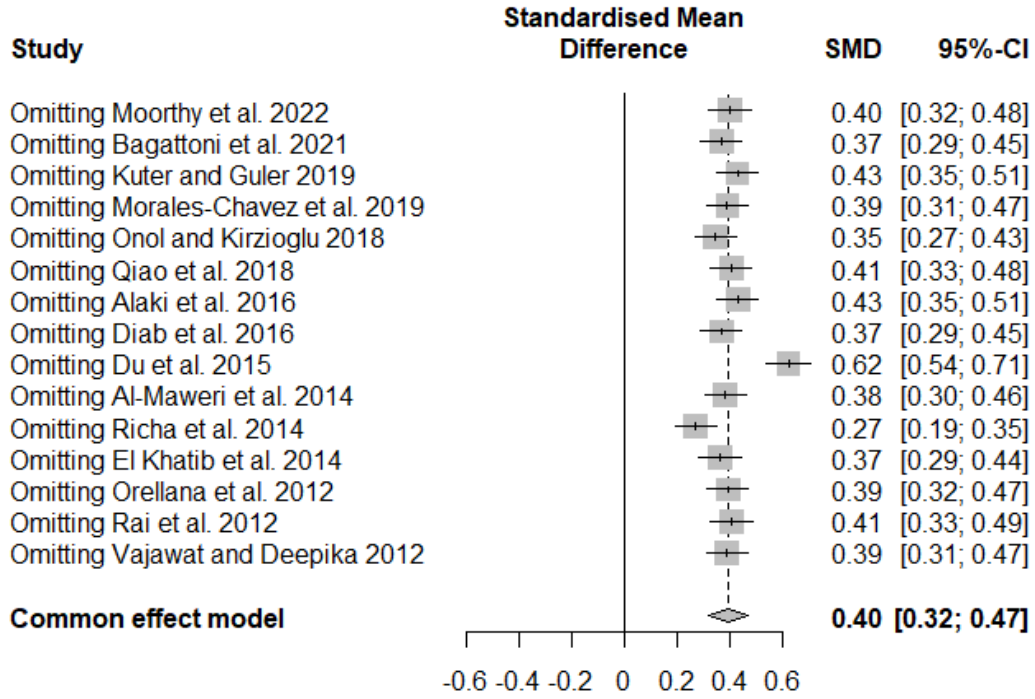


Figure 8A. *Forest plot for gingivitis prevalence comparison between ASD and non-ASD.*

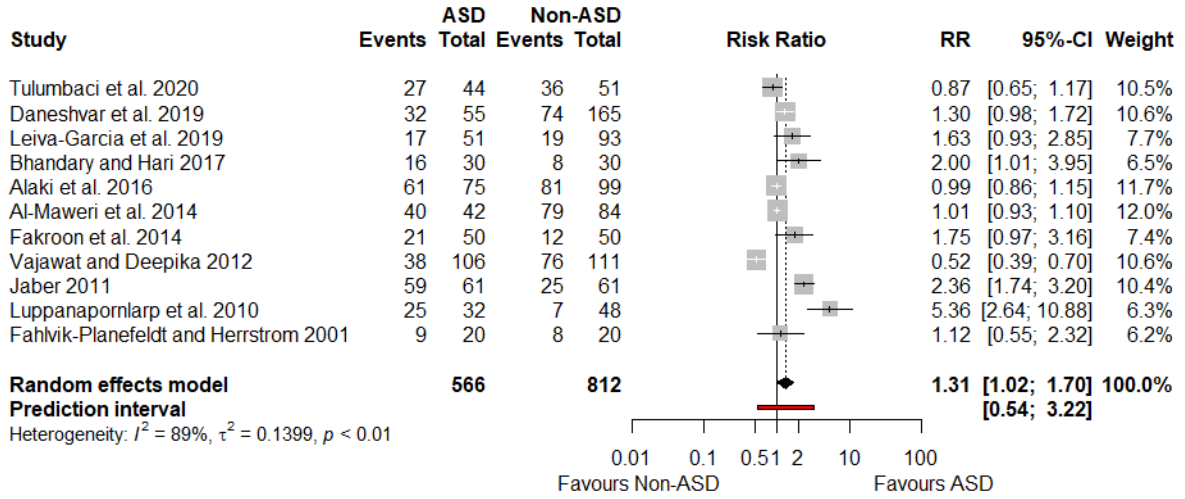


Figure 8B. *Sensitivity analysis of gingivitis prevalence. SMD and 95% CI were determined by omitting each study according to the rules of the metanalysis.*

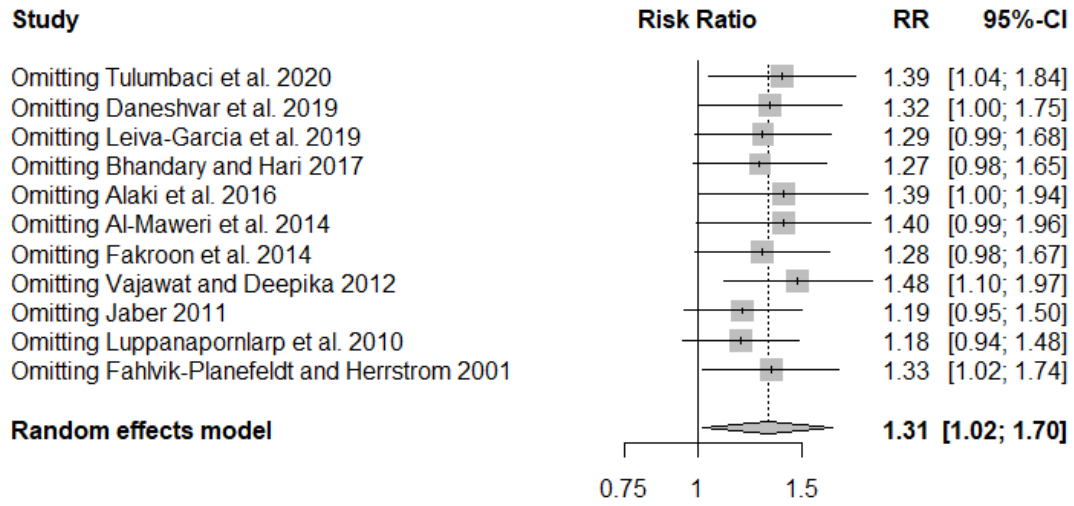


Figure 9A. *Forest plot for gingivitis severity comparison between ASD and non-ASD.*

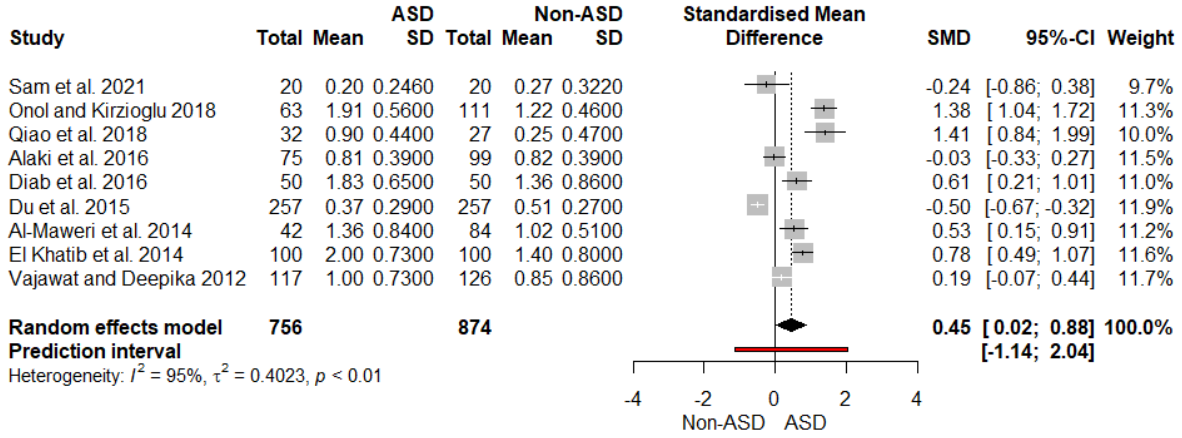


Figure 9B. *Sensitivity analysis of gingivitis severity. SMD and 95% CI were determined by omitting each study according to the rules of the metanalysis.*

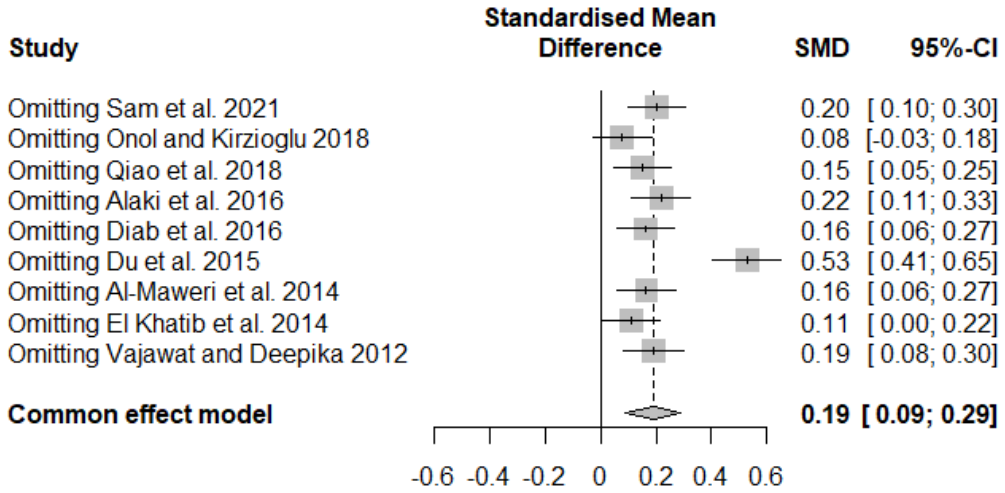


Figure 10. *Forest plot for tooth loss severity comparison between ASD and non-ASD according tooth type.*

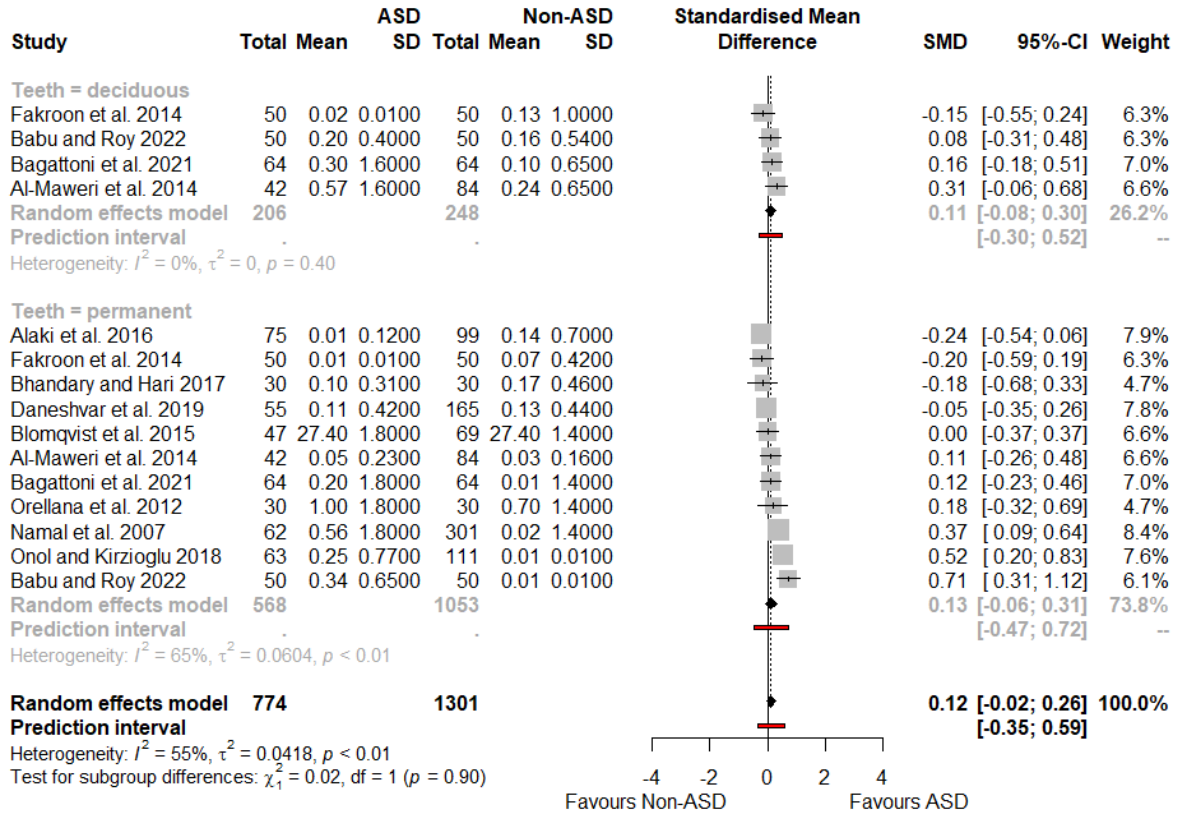


Figure 11. *Forest plot for malocclusion prevalence according Angle Class comparison between ASD and non-ASD.*

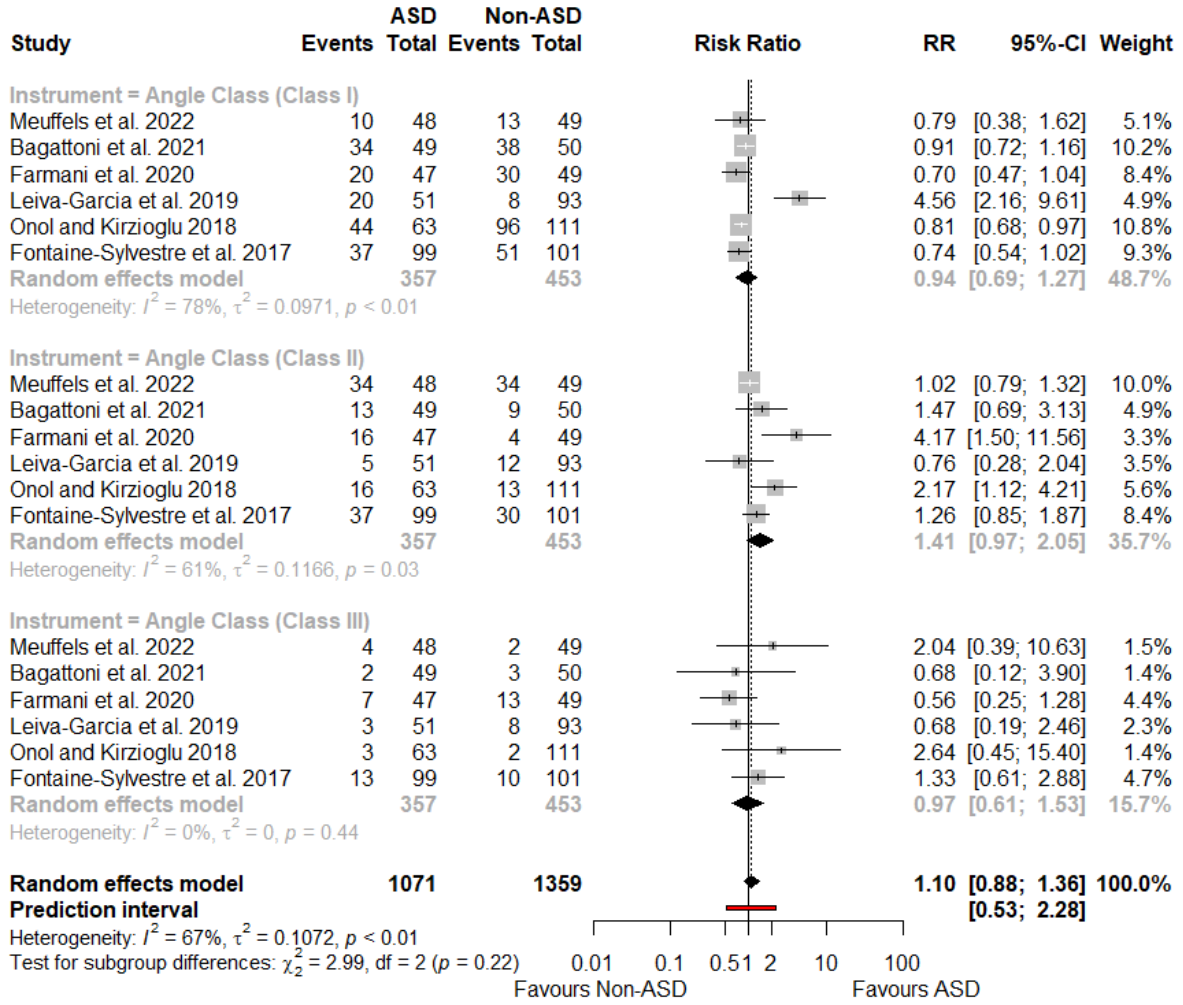


Figure 12A. *Forest plot for overjet prevalence comparison between ASD and non-ASD.*

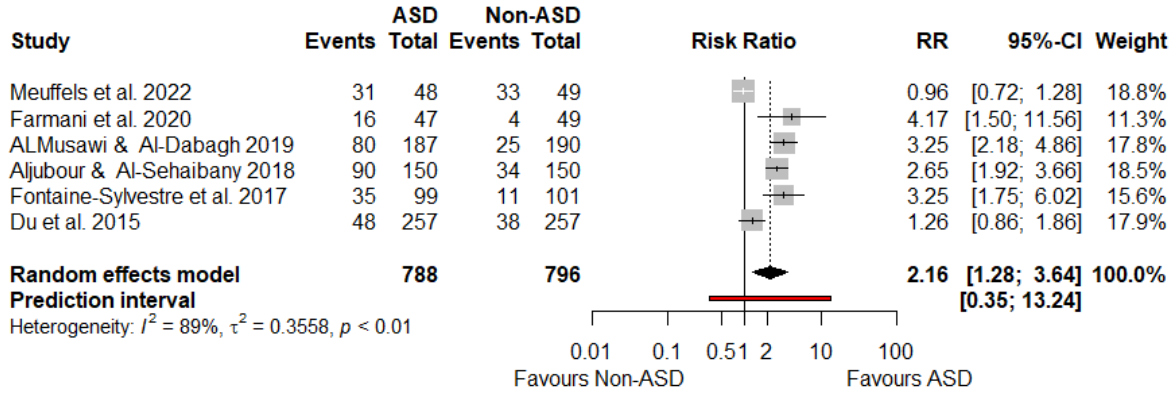


Figure 12B. *Forest plot for overbite prevalence comparison between ASD and non-ASD.*

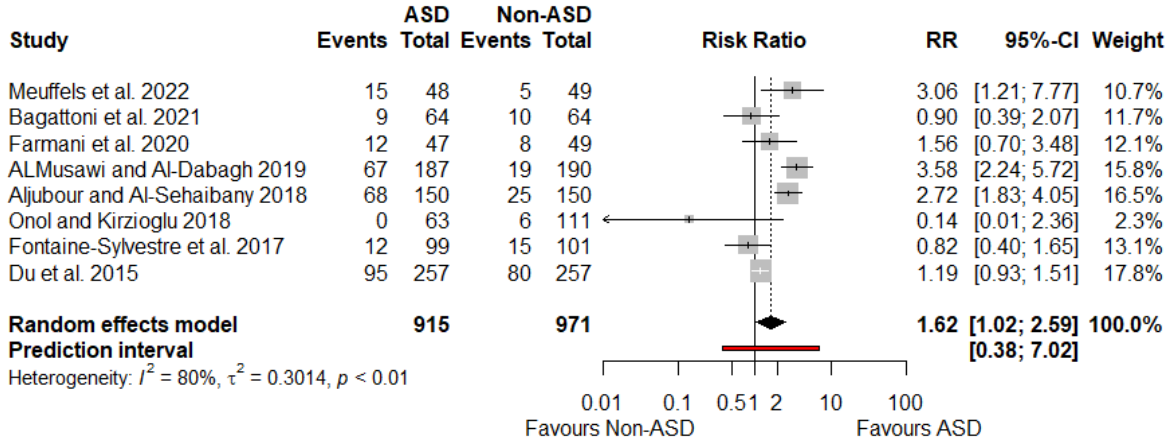


Figure 12C. *Forest plot for crossbite prevalence comparison between ASD and non-ASD.*

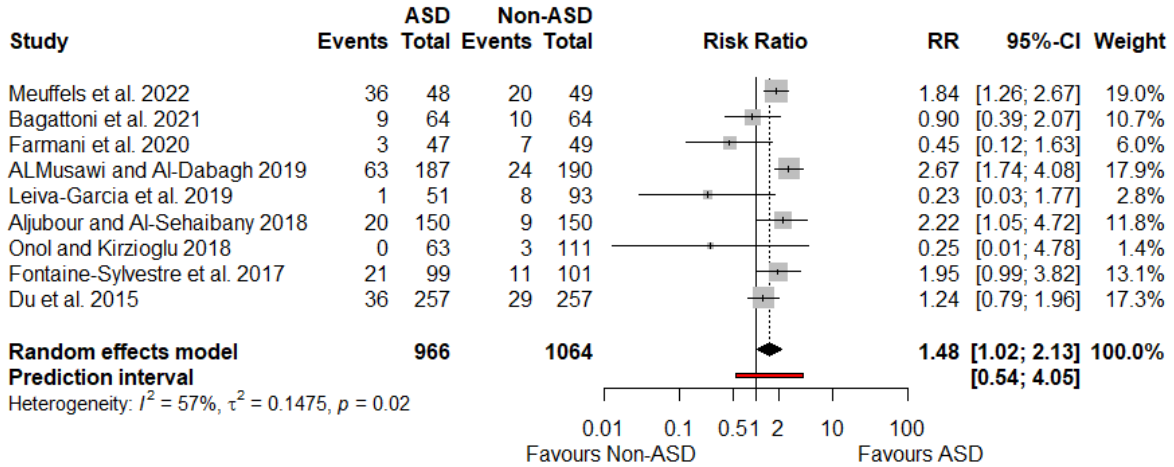


Figure 13. *Forest plot for openbite prevalence comparison between ASD and non-ASD.*

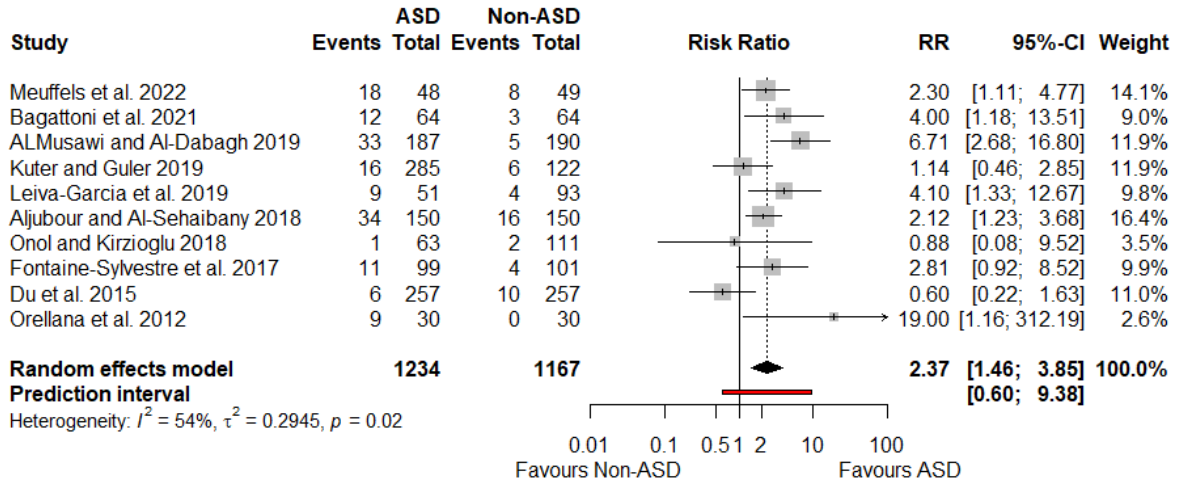


Figure 14. *Forest plot for bruxism prevalence comparison between ASD and non-ASD*

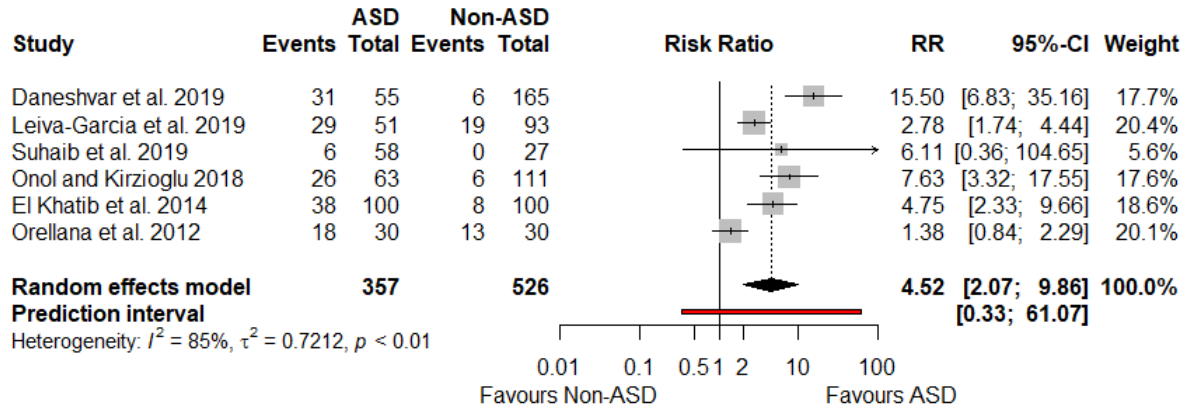


Figure 15A. *Forest plot for flow rate mean comparison between ASD and non-ASD.*

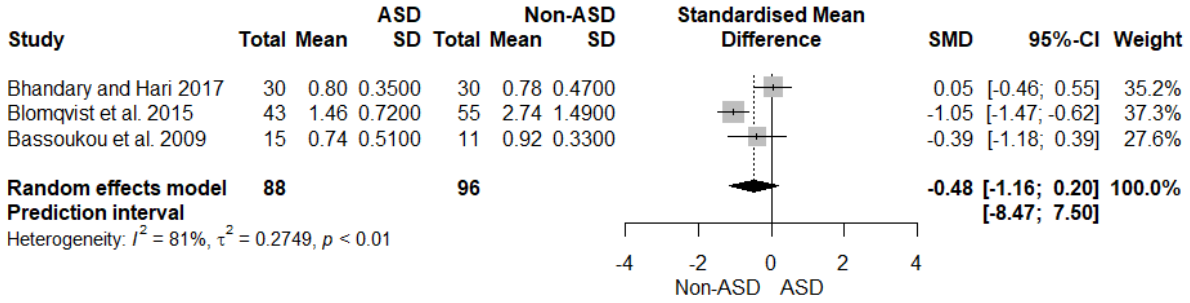


Figure 15B. *Forest plot for pH mean comparison between ASD and non-ASD.*

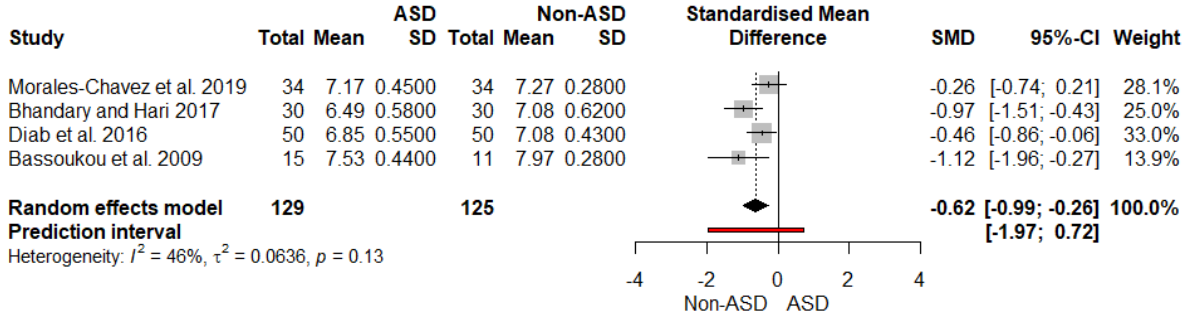


Table 1. *Quality assessment of included studies (n = 42)*

Cross-sectional Study	Selection	Comparability	Outcome	Max 9 *	Max 100%	Risk of bias
Babu & Roy 2022	**	--	*	3/9	33.33%	High
Faker et al. 2022	*	--	***	4/9	44.44%	Moderate
Meuffels et al. 2022	*	--	***	4/9	44.44%	Moderate
Bagattoni et al. 2021	**	--	***	5/9	55.55%	Moderate
Kuter & Uzel 2021	**	**	*	5/9	55.55%	Moderate
Meshki et al. 2021	**	--	--	2/9	22.22%	High
Moorthy et al. 2021	***	**	*	6/9	66.66%	Moderate
Sam et al. 2021	*	--	*	2/9	22.22%	High
Farmani et al. 2020	*	*	***	5/9	55.55%	Moderate
Tulumbaci et al. 2020	*	*	*	3/9	33.33%	High
ALMusawi & Al-Dabagh 2019	**	**	*	5/9	55.55%	Moderate
Daneshvar et al. 2019	*	--	*	2/9	22.22%	High
Kuter & Guler 2019	*	**	*	4/9	44.44%	Moderate
Leiva-García et al. 2019	*	*	***	5/9	55.55%	Moderate
Morales-Chávez et al. 2019	*	**	--	3/9	33.33%	High
Suhaib et al. 2019	*	**	*	4/9	44.44%	Moderate
Aljubour & Al-Sehaibany 2018	**	**	**	6/9	66.66%	Moderate
Mandić et al. 2018	*	**	***	6/9	66.66%	Moderate
Morales-Chávez & Villarroel-Dorrego 2018	*	**	--	3/9	33.33%	High
Onol & Kırzıoglu 2018	**	--	*	3/9	33.33%	High
Qiao et al. 2018	*	**	*	4/9	44.44%	Moderate
Bhandary & Nary 2017	*	*	***	5/9	55.55%	Moderate
Fontaine-Sylvestre et al. 2017	*	*	*	3/9	33.33%	High
Alaki et al. 2016	***	*	***	7/9	77.77%	Low
Diab et al. 2016	**	**	***	7/9	77.77%	Low
Sarnat et al. 2016	*	--	--	1/9	11.11%	High
Blomqvist et al. 2015	*	**	***	6/9	66.66%	Moderate
Du et al. 2015	***	**	***	8/9	88.88%	Low
Al-Maweri et al. 2014	**	**	*	5/9	55.55%	Moderate
Fakroon et al. 2014	*	**	***	6/9	66.66%	Moderate
Richa et al. 2014	*	--	***	4/9	44.44%	Moderate
El Kathib et al. 2013	**	**	***	7/9	77.77%	Low
Orellana et al. 2012	*	**	*	4/9	44.44%	Moderate
Rai et al. 2012	*	*	--	2/9	22.22%	High
Vajawat & Deepika 2012	*	**	*	4/9	44.44%	Moderate
Jaber 2011	*	**	***	6/9	66.66%	Moderate
Luppanapornlarp et al. 2010	*	*	***	5/9	55.55%	Moderate
Bassoukou et al. 2009	*	--	*	2/9	22.22%	High
Loo et al. 2008	*	**	*	4/9	44.44%	Moderate
Namal et al. 2007	**	--	*	3/9	33.33%	High
Fahlvik-Planefeldt & Herrström 2001	*	**	*	4/9	44.44%	Moderate
Cohort Study	**Selection**	**Comparability**	**Outcome**	**Max 9 ***	**Max 100%**	**Risk of bias**
Frank et al. 2019	*	*	*	3/9	33.33%	High

Table 2. *Characteristics of included studies and comparison of caries prevalence and severity (n=33), tooth loss (n=14), oral hygiene and periodontal status (n=30) between ASD and controls.*

Author **Country** **Study design**	**Place recruitment**	**ASD group** Sample (N), gender (%); age (mean±sd) Socioeconomic status ASD diagnosis Tools/professional type Medications	**Control group** Sample (N), gender (%); age (mean±sd) Socioeconomic status	**Inclusion (I) and exclusion (E) criteria**	**Control matching**	**Outcomes measures** Tools Full-mouth/partial	**Results** ASD vs control group	**Main findings**
Babu & Roy 2022 India Cross-sectional.	ASD: various autistic institutions across Bengaluru city. Controls: department of Pediatric and Preventive dentistry at VS Dental College, Bengaluru.	n: 50 Gender: Out of the 100 children who participated in the study, 56 (56%) were males and 44 (44%) were females. Mean age 8.94±2.28y. Age range: 3 -13y.	n:50 Mean age 9.38±2.11y.	(I) children between age group of 3–13 years diagnosed with autism. (E) children with other systemic disorders, excluding autism.	Not reported.	Mean±SD of caries according DMFT/dmft index for primary (dmft) and permanent (DMFT) dentition (WHO, 2013).	Mean±SD of: d (p= 0.003) ASD 2.04±1.94 vs 0.8±1.27 control. f (p= 0.02) ASD 0.04±0.19 vs 0.32±0.84 control. dmft (p= 0.005) ASD 2.28±1.93 vs 1.28±1.60 control. Mean±SD of: D (p= 0.0001) ASD 1.96±1.39 vs 0.74±1.17 control. F (p= 0.04) ASD 0.16±0.37 vs 0.04±0.197 DMFT (p= 0.0001) ASD 2.32±1.73 vs 0.78±1.29 control. Mean±SD of missing tooth: m (p= 0.67) ASD 0.2±0.40 vs 0.16±0.54 control. M (p= 0.0005) ASD 0.34±0.65 vs 0±0 control.	The mean of DMFT/dmft was found to be significantly higher in autistic patients compared to controls.

Faker et al 2022 Brazil Cross-sectional	ASD: Acolher Project/PNE of the Pediatric Dentistry Outpatient Clinic, both at Fluminense Federal University in Niteroi, Rio de Janeiro. Controls: Pediatric Dentistry Outpatient Clinic of same local.	n:34 Gender: 27 (79.4%) M 7 (20.6%) F Mean age 4.47±1.26y. Age range: 2-6y.	n:34 Gender: 9 (26.5%) M 25 (73.5%) F Mean age 4.21±1.07y.	(I) age between 2 and 6 years and parent/caretaker living with the child for at least 12 hours/ day. (I) ASD diagnosis with medical information on ASD no associated comorbidities. (E) children who were current orthodontic treatment, systemic disease, uncooperative during clinical examination, lack of a written informed consent signed by parents or caretakers, and incomplete questionnaires (parents who failed to answer more than two child items and one family item were excluded from the analysis.	Not reported.	Untreated dental caries according component 'decayed – d/D' of the DMFT/dmft index (decayed, missing, and filled teeth). The participants were categorized into two groups: children without untreated dental caries (d + D = 0) and children with untreated dental caries (d + D > 0).	Prevalence of untreated dental caries (p= 0.040): ASD 41.1% (14) vs 64.7% (22) control.	The untreated dental caries in ASD was significantly less than in control.
Moorthy et al. 2022 India Case control. Study was conducted during the 10-month time frame between	ASD: diagnostic and treatment centers for autistic children and special needs schools selected	n: 136 Gender: 96 (70.6%) M 40 (29.4%) F Mean age 7.7±2.1y Age range: 5-12y. Tool: DSM 4 or DSM 5 criteria. Socio-economic status:	n: 136 Gender: 97 (71.3%) M 39 (28.7%) F Mean age 7.9±2.1y. Socio-economic status: Upper 29 (21.3%) Upper middle	(I) ASD Children between age 5 and 12 years, diagnosed with Autism Spectrum Disorder (ASD) based on the DSM 4 or DSM 5 criteria and with positive	Age, sex and socio-economic status.	Mean±SD of deft for primary teeth and DMFT for permanent teeth (DMFT, WHO 2013). To evaluate caries experience of	Mean±SD of: deft (p = 0.49) ASD 3.3±3.8 vs 3.4±3.3 control. DMFT (p = 0.53) ASD 0.5±1.1 vs 0.3±0.9 control. Caries-affected primary and permanent teeth (p = 0.74) 3.8±4.0 vs 3.7±3.5 control.	Caries experience and caries status were found to be similar in children with or without ASD. Autistic

2018 and 2019. Two-stage Sampling method was used for the study.	randomly from Mumbai, Navi Mumbai and Thane regions. Controls: schools in the vicinity of facilities from where the children with ASD were selected.	Upper 39 (28.7%) Upper middle 64 (47.1%) Lower middle 21 (15.4%) Upper lower 12 (8.8%) Mother's working status: Home maker 118 (86.8%) Working 18 (13.2%) Mother's educational level ($p < 0.001$): Post Graduate 46 (33.8%) Graduate 45 (33.1%) Higher secondary School 21 (15.4%) High school 5 (3.7%) Middle school 15 (11.0%) Literate 2 (1.5%) Illiterate 2 (1.5%)	66 (48.5%) Lower middle 28 (20.6%) Upper lower 13 (9.6%) Mother's working status: Home maker 121 (89.0%) Working 15 (11.0%) Mother's educational level ($p < 0.001$): Post Graduate 2 (1.5%) Graduate 13 (9.6%) Higher secondary school 34 (25.0%) High school 32 (23.5%) Middle school 42 (30.9%) Literate 9 (6.6%) Illiterate 4 (2.9%)	parental consent were included. (E) ASD Children with known nutritional disorders or on special diets for management of ASD or its comorbid conditions. (I) Controls with no relevant medical history and children who were able to understand simple verbal commands were selected from the schools in the vicinity of facilities from where the children with ASD were selected. (E) All Children who were extremely uncooperative or having any known nutritional disorder and children currently undergoing antibiotic/ anti-inflammatory therapy were excluded from both the groups.	children, the ones with both deft and DMFT of zero were considered as cariesfree and children with either deft or DMFT more than zero were considered as caries-affected. Mean±SD of Oral Hygiene Index-Simplified (OHI-S) by Greene and Vermilion (1964).	Caries experience ($p = 0.51$) ASD 32.4% (44) vs 27.9% (38) control. Mean±SD of: OHI-S ($p < 0.001$) ASD 0.4±0.6 vs 0.2±0.5 control.	children had poorer oral hygiene status as compared to the controls.

Bagattoni et al 2021 Italy Cross-sectional.	ASD: Paediatric Units of St. Orsola-Malpighi Polyclinic, Department of Medical and Surgical Sciences, University of Bologna, Italy. Controls: Italian healthy children.	n: 64 Gender: 42 (66%) M 22 (34%) F Mean age 9.0±2.9y. Tool: Frankl scale into four categories from definitely negative (grade 1) to definitely positive (grade 4) (Frankl et al., 1962).	n: 64 Gender: 37 (58%) M 27 (42%) F Mean age 8.4±3.0y.	(E) medical condition associated with oral diseases; unable to cope with an oral examination; dental prophylaxis in the previous 6 months; history of orthodontic treatment.	Not reported.	Mean of decay, filled teeth Index for dental caries of the primary dentition (dmft) and of the permanent dentition (DMFT) according WHO, 2013. Prevalence of caries. Mean of missing tooth. Mean±SD of plaque and prevalence of oral hygiene status according Silness and Loe (1964) Plaque Index (PlI).	Mean±SD of dmft (p<0.001): ASD 3.00±1.2 vs 1.8±1.1 control. Mean of: d ASD 2.5 vs 1.0 control. f (p= 0.034): ASD 0.2 vs 0.7 control. Mean±SD of DMFT (p<0.001): ASD 2.3±1.8 vs 1.0±1.1 control. Mean of: D ASD 1.6 vs 0.5 control. F (p=0.021): ASD 0.5 vs 0.5 control. Prevalence of caries (p<0.001): ASD 67.19% (43) vs 34.37% (22) control. Mean of missing tooth: m ASD 0.3 vs 0.1 control. M ASD 0.2 vs 0 control. Mean±SD of PlI (p=0.001): ASD 1.48±0.75 vs 0.81±0.56 control. Prevalence of oral hygiene status (p= 0.013): Good (PI 0-1) ASD 42% (27) vs 73% (40) control. Fair (PI 1–2) ASD 45% (29) vs 36% (23) control.	Children with ASD have a poorer oral health status than healthy children, ASD had a higher prevalence of caries than controls. A significantly higher number of healthy children had fillings in the primary teeth.

							Poor (PI 2-3) ASD 13% (8) vs 1% (1) control.	
Kuter & Uzel 2021 Turkey Cross-sectional retrospective study.	ASD: Moris Bencuya Special Education Public School for Children with Autism Spectrum Disorder in Izmir, Turkey. Controls: their own public school in Izmir, Turkey.	n: 226 randomly selected. Age range: 5-15y. Mixed dentition (5-12y) Permanent dentition (13-15y)	n: 122 randomly selected. Age range: 5-15y. Mixed dentition (5-12y) Permanent dentition (13-15y)	(I) ASD: Children with a diagnosis of autism spectrum disorders, aged between 5 and 15 years and with dental records in the hospital files were included. (E) ASD: Children who had undergone dental prophylaxis in the past 6 months and children who did not cooperate during the oral examination were excluded. Children using medication for any systemic diseases that could influence dental caries or the severity of periodontal disease were excluded from the study. Children who had systemic diseases were excluded from the study. (I) Controls: Healthy children who met the aforementioned criteria were	Age, sex, and socioeconomic status.	Mean±SD of dmft (decayed missed filled permanent tooth in primary dentition), DMFT (decayed missed filled permanent tooth in permanent dentition), plaque index (Silness & Loe, 1964) and caries prevalence scores.	Mean±SD of: dmft ASD boys 5-12y 1.17±2.19 vs 2.66±2.18 control. ASD girls 5-12y 1.16±1.34 vs 2.87±2.65 control. DMFT ASD boys 5-12y 0.63±1.30 vs 1.2±1.60 control. ASD girls 5-12y 0.00±0.00 vs 1.09±1.37 control. ASD boys 13-15y 2.17±2.32 vs 2.77±2.36 control. ASD girls 13-15y 4.10±2.75 vs vs 3.54±2.30 control. Caries prevalence: ASD boys 5-12y 58.62% vs 85.71% control. ASD girls 5-12y 66.66% vs 66.66% control. ASD boys 13-15y 57.40% vs 57.14% control. ASD girls 13-15y 77.77% vs 84.21% control.	ASD boys had lower mean of dmft and DMFT than control. ASD girls 13-15y had higher mean of DMFT than control. ASD boys and girls had equal prevalence or lower than control.

				chosen. All children were healthy and none were undergoing antibiotic or anti-inflammatory medication therapy.				
Meshki et al. 2021 Iran Case control.	ASD and Control: selected by stratified random sampling from different schools placed in the city of Ahvaz, Iran.	n: 51 Gender: 48 (94.11%) M 3 (5.88%) F Mean age 8.6±1.5y. Age range: 7-12y.	n: 51 Gender: 29 (56.9%) M 22 (43.1%) F Mean age 8.39±1.37y. Age range: 7-12y.	(I) Frothy-eight patients, whose parents or guardians were awareness of dental evaluation.	Not reported.	Mean of decayed, missing, and filled teeth (DMFT; both permanent and primary) and also OHI-S index (simplified oral hygiene index). The OHI-S was make on the buccal and lingual surfaces of every of three parts of each dental arch according to 12 numerical determinations.	Mean dmft (p=0.001): ASD 65.74 vs 34.53 control. Mean DMFT ($p \leq 0.001$): ASD 59.16 vs 40.60 control. Mean dmft/DMFT ASD 48.03 vs 31.76 control. Mean OHI-S (p=0.000): ASD 65.21vs 35.02 control.	Autistic children had higher mean of caries and OHI-S. Also, children with autism showed more needs in primary dentition (P=0.002).
Sam et al. 2021 India Cross-sectional.	ASD: Autism Sisu Kshema Kendram, Thrissur, Kerala, India. Control: randomly selected from a government school in the same area.	n: 20 Gender: 14 (70%) M 6 (30%) F Mean age 9.2±1.1y. Age range: 6-12y.	n: 20 Gender: 12 (60%) M 8 (40%) F Mean age 9.45±1.0y. Age range: 6-12y.	(I) The children with autism who had the ability to follow simple instructions such as “sit down”, “open your mouth and lower your hands”, who allowed touching his/her face or mouth,	Not reported.	Median values of DMFT/dmft. Mean±SD of gingival status was recorded as no inflammation (0), or mild (1), moderate (2) or severe (3) inflammation depending on qualitative changes in	Median values (Q3, Q1) of: DMFT (p=0,757) ASD 0 (1.25, 0) vs 0 (1.00, 0) control. dft (p=0,035) ASD 0 (1.25, 0) vs 2 (4.00, 0) control. Mean±SD of: Gingival index GI (p=0,811) ASD 0.20±0.246 vs 0.27±0.322 control.	Caries experience in the permanent dentition in the autistic children and healthy children were comparable while autistic children had a lower caries experience in

				had not received dental prophylaxis in the past six months, having no disorder such as Down's syndrome and other medical conditions which could affect his/her oral status were included in the study. (E) Mentally challenged children, children on longterm medication such as anti-anxiety drugs, anti-psychotic drugs, anticonvulsants and children showing aggressive/hostile behaviour were excluded from the study.		gingiva and bleeding on probing according Loe, 1967. Prevalence of the oral hygiene was assessed using the Simplified oral hygiene index (Greene & Vermillion , 1964) and recorded as good when oral hygiene score was 0-1.2, fair when it was 1.3-3 and poor when it was 3-6.	Prevalence of Oral hygiene status according OHI-S (p=0,833): Good ASD 2 (10%) vs 1 (5%) control. Fair ASD 3 (15%) vs 3 (15%) control. Poor ASD 15 (75%) vs 16 (80%) control.	the primary dentition. Both autistic children and healthy children exhibited mild gingival inflammation.
Tulumbaci et al 2020 Turkey Cross-sectional.	ASD: Individual Training Center in Selcuklu, Konya. Controls: Department of Pediatric Dentistry, Faculty	n: 44 Gender: 37 (84%) M 7 (16%) F Mean age 9.09±4.38y. Age range: 3 -17y.	n: 51 Gender: 31 (61%) M 20 (39%) F Mean age 9.09±3.82y. Age range: 3-17y.	(I) to understand basic verbal instructions; autism diagnosis (ASD); signed parental/legal guardian agreement to the informed consent forms. (E): antibiotics 2 week prior	Age	Prevalence of caries of gingivitis. Mean values of Dmft and of plaque index (index not reported).	Caries prevalence: ASD 83.1% (37) vs 81.10% (41) control. Dmft mean (p=0.430): ASD 3.25 vs 3.10 control. Score of DMFT: ASD 5.10 vs 4.90 control. Gingivitis prevalence: ASD 61.4% (29) vs 70.6% (36) control.	No significant difference was found between the ASD and control in terms of caries, plaque and periodontal diseases.

	of Dentistry, NEU, Konya.			to the study; having a systemic disease.			Values of plaque index: No significant differences between groups (p=0.357).	
Daneshvar et al.2019 Iran Cross-sectional.	ASD: autism rehabilitation center of Rasht city. Controls: children referred to the Dentistry faculty of Guilan University of Medical Science, Rasht, Iran.	n: 55 Gender: 49 (89.1%)M 6 (10.9%) F Mean age 9.32±2.33y. Father's education: Elementary: 18.2% (10) High school: 50.9% (28) University degree: 30.9% (17) Mother's education: Elementary: 21.8% (12) High school: 43.6% (24) University degree: 34.5% (19) Tool: ASD severity mild, moderate and severe based on the impairments in communication, social interaction and repetitive behaviors by a physician. Frankl's behavior rating scale./Professional type: physician.	n:165 Gender: 83 (50.3%) M 82 (49.7%) F Mean age 8.71±1.97y. Father's education: Elementary: 6.7% (11) High school: 52.7% (87) University degree: 40.6% (67) Mother's education: Elementary: 7.9% (13) High school: 45.5% (75) University degree: 46.7% (77)	(I) 6–12 y (E): poor cooperation, having other disorders and parental dissatisfaction.	NR.	Mean±SD of DMFT/dmft and of missing tooth. Prevalence of gingivitis (GI).	DMFT+dmft (p<0.001): ASD 6.33±2.88 vs 3.88±2.91control. Decayed teeth (D+d) (p<0.001): ASD 5.78±3.21 vs 2.48±2.69 control. Filled teeth (F+f) (p<0.001): ASD 0.44±1.07 vs 1.27±1.62 control. DMFT (p<0.001) in permanent dentition: ASD 6.20±2.75 (n=20) vs 2.7±2.6 (n=40) control. Dmft in primary dentition: ASD 6.45±3.50 (n=11) vs 4.43±2.87 (n=28) control. DMFT+dmft in mixed dentition: ASD 6.38±2.81 (n=24) vs 4.22±2.93 (n= 97) control. Mean score of DMFT/dmft in the children with severe ASD was higher than that in the children with mild and moderate ASD (p=0.007)**:** Mild: (n=17) 5.59±2.74 Moderate: (n=27) 5.78±2.71 Severe: (n=9) 8.78±2.05 Missing tooth (MT+mt) (p<0.001): ASD 0.11±0.42 vs 0.13±0.44 control. Gingivitis prevalence (p=0. 014): ASD 58.2% (32) vs 44.8% (74) control. Localized: ASD 41.8% (23) vs 40% (66) control.	ASD had higher DMFT/dmft scores compared with control. The mean score of DMFT/dmft in the children with severe ASD was higher than that in the children with mild and moderate ASD. The mean number of missing teeth in the ASD was lower than that in control. Gingivitis prevalence (generalized or localized) in ASD was significantly higher than control.

							Generalized: ASD 16.4% (9) vs 4.8% (8) control	
Frank et al. 2019 USA Retrospective Longitudinal cohort. Follow-up mean of 6.9+12.5y in ASD and 2.5±4.9y in control.	ASD: CSHCN Group. Controls: private pediatric dentistry clinic in an urban center in Durham, N.C., USA.	n: 30 Mean age Start of follow-up: 6.4±3.8y. End of follow-up: 13.1 ±4.9y. Family income level (Inferred by insurance): Public insurance only 70% (21) Private insurance only 20% (6) No insurance 7% (2) Patient taking xerostomia inducing medications 50% (15). Caregiver/patient have difficulty performing oral hygiene: 20% (6). Patients has visible cavities or fillings: 43% (13).	n: 30 Mean age Start of follow-up: 3.4+23y. End of follow-up: 10.9±5.3y. Family income level (inferred by insurance) Public insurance only 10% (3) Private insurance only 83% (24) No insurance 3% (1) Patient taking xerostomia inducing medications 10% (3). Caregiver/patient have difficulty performing oral hygiene: 3% (1). Patients has visible cavities or fillings: 27% (8).	(I) Cases: patients had to have one of the diagnoses (not multiple diagnoses) under study:autism spectrum disorder; cerebral palsy; congenital heart disease; Down syndrome (I) Controls: not have any of the studied diagnoses or other significant medical histories. (E):patients who were followed for less than six months.	Age	Mean±SD of dmfs and DMFS (patients who were at least 71 months old).	Mean±SD of DMFS increments between first and last clinic visit: ASD (n=29) 6.9±12.5 vs 2.5±4.9 control (n=23) considering a follow-up mean, in years, of 6.9+12.5y in ASD and 2.5±4.9y in control. Mean±SD of DMFS increment adjusted (annual): ASD 2.5±8.9 vs 0.2±0.4 control. Incidence rate ratio (95% CI) using control as reference: ASD 1.85 (0.70-4.9)	ASD had high permanent dentition increment (DMFS, annual) than control. Children with ASD had not a risk ratio significantly higher for caries when compared to control.
Kuter & Guler 2019 Turkey Cross-sectional.	ASD: Autistic children attending schools. Control: regular schools.	n: 285 Gender: 223 (78.35%) M 62 (21.7%) F Age of 5–11y: 21.8% (62) mixed dentition. Age of 12–16y: 78.2% (223) permanent dentition. Mother's education Primary school: 47.2% (135) Secondary: 8.5% (24)	n: 122 Gender: 96 (78.3%) M 26 (21.7%) F Age of 5–11y: 20.6% (25) mixed dentition. Age of 12–16y: 79.4% (97) permanent dentition. Mother's education Primary school: 55.9% (68)	NR.	Age and Socioeconomic status	Mean±SD of Dmft and DMFT and prevalence of caries according age 5-11y and 12-16y. Mean±SD of plaque (PlI).	dmft: 5-11y: ASD 1.66±2.07 vs 2.8±2.45 control. DMFT 5-11y: ASD 0.52±1.21 vs 1.14±1.48 control. DMFT 12-16y: ASD 2.07±2.49 vs 3.37±2.32 control. Prevalence of caries: 5-11y:	Children with ASD had lower DMFT/dmft means values and caries prevalence than controls. No difference was found in the PlI mean values between ASD and control.

		High school: 27.3% (78) University: 17.0% (48) Father's education Primary school: 30.2% (86) Secondary: 14.1% (40) High school: 28.3% (81) University: 27.4% (78) Family income Low 41.5% (118) Moderate: 44.3% (126) High 14.2% (41) 72.6% (207) of autistics used medications, 27.4% of them took only risperidone and 0.9% took risperidone, sodium valproate and centraline hydrochloride or fluoxetine, 3.8% took aripiprazole.	Secondary: 12.8% (16) High school: 17.6% (21) University: 13.7% (17) Father's education Primary school: 42.2% (51) Secondary: 15.7% (19) High school: 23.5% (29) University: 18.7% (23) Family income Low 31.4% (38) Moderate 58.8% (72) High 9.8% (12)				ASD 50% (31) vs 80.47% (20) control. 12-16y: ASD 57.14% (127) vs 79.06% (77) control. Plaque Index: 5-11y: ASD 2.60±0.48 vs 2.66±0.56 control. 12-16y: ASD 3.15±0.74 vs 3.07±0.81 control. Prevalence of the different PlI scores: Score 1: ASD 25.5% (73) vs 28.4% (35) control. Score 2: ASD 45.3% (129) vs 41.2% (50) control. Score 3: ASD 29.2% (83) vs 29.4% (36) control.	
Leiva-García et al. 2019 Spain Cross sectional.	ASD: different ASD special education centers. Controls: schools in the same area and children of similar	n: 51 Gender: 37 (74.0%) M 13 (26.0%) F Mean±SE of age 12.84±3.67y. Age range: 6-18y. Tool: DSM-V criteria.	n: 93 Gender: 50 (53.8%) M 43 (46.2%) F Mean±SE of age 9.56±1.67y.	(E): special diets (e.g., casein and gluten free), those with food allergies or medications capable of modifying dietary intake and which could alter oral health, non-	Area and socioeconomic status.	Mean±SE of DMFT and dmft and prevalence of caries. Prevalence of periodontal status according CPI. Prevalence of plaque (PlI).	DMFT:ASD 0.70±0.28 vs 0.60±0.19 control. dmft:ASD 0.85±0.46 vs 0.83±0.18 control. Caries prevalence: ASD 42.0% (21) vs 7.60% (35) control.	Children with ASD had higher prevalence of caries when compared to control. ASD had higher prevalence of

	socioeconomic status as the special schools attended by the ASD group were selected.			collaboration in the oral examination, failure to report for the oral examination, or failure to receive or complete the questionnaires.			Periodontal health prevalence (p=0.011): ASD 24.0% (12) vs 43.0% (40) control. Inflammation without bleeding prevalence: ASD 18.0% (9) vs 26.9% (25) control. Inflammation with bleeding prevalence: ASD 34.0% (17) vs 20.4% (19) control. Plaque prevalence" No plaque: ASD 24.0% (12) vs 50.5% (47) control. Plaque presence only detected with probe: ASD 16.0% (8) vs 8.60% (8) control. Plaque visible: ASD 40.0% (20) vs 22.6% (21) control. Abundance plaque (> 1/3): ASD 20.0% (10) vs 18.3% (17) control.	moderate and visible plaque, periodontal disease when compared to control.
Morales-Chávez et al. 2019 Venezuela Cross-sectional.	ASD: two private schools for patients with autism in the city of Caracas. Controls: private school and Pediatric Dental Clinic of the Faculty of Dentistry of the Santa María University, Caracas.	n: 34 Gender: 34 (100%) M Mean age 8.12±1.92y. Age range: 4-13y.	n: 34 Gender: 34 (100%) M Mean age 8.12±1.92y. Age range: 4-13y.	(I): Autism diagnosis grade 1 and 2. (E): gastrointestinal disorders, patients taking medications that could alter salivary flow, which contained dyes or causing gingival hyperplasia, patients with other concomitant syndromes, such as Down syndrome and grade 3 of autism.	Age, social stratum and gender.	Prevalence of caries and mean±SD of DMFT. Prevalence of dental plaque and mean of OHI-S.	Caries prevalence (p≤0.001): ASD 20.60% (7) vs 73.52% (25) control. DMFT (p≤0.001): ASD 1±1 vs 3±2 control. Plaque prevalence (p=0.042): ASD 64.70% (22) vs 61.80% (21) control. OHI-S (p=0.008): ASD 2.23±0.83 vs 1.82±0.60 control.	Caries prevalence was significantly lower in ASD than control. Mean±SD of DMFT was higher significantly in controls than ASD children. Oral hygiene index was higher in ASD when compared to control.

Suhaib et al. 2019 Pakistan Cross-sectional.	Autism Resource Centre (ARC), Rawalpindi, Pakistan.	n: 58 Gender: 46 (79.3%) M 12 (20.7%) F Mean age 5.4y. Age range: 2-10y.	n: 27 health siblings. Gender: 17 (62.96%) M 10 (37.04%) F Mean age 5.6y. Age range: 2-10y. Out of the 58 mothers surveyed: 41% had a professional degree; 45% were graduates and 12% had primary-level education.	(I): Children in the age bracket 2–10 y and whose mothers consented to be included were enrolled.	Age and Socioeconomic status.	Prevalence of caries. Presence of plaque (anterior teeth), partial exam.	Caries prevalence ($p<0.05$): ASD 50% (29) vs 22.2% (6) control. Presence of plaque*: ASD 25.9% (15) vs 14.8% (4) control.	Caries and dental plaque were significantly more common in children with ASD as compared to their siblings.
Mandić et al. 2018 Serbia Case-control.	ASD: children referred to the Clinic of Pediatric and Preventive Dentistry in the period of one year. Faculty of Dental Medicine, University of Belgrade, Serbia. Control: healthy school children in Belgrade.	n:32 Gender: 19 (59.37%) M. 13 (40.63%) F. Mean age: 11.19±3.36y. Age range: 6-16y. 6-11y (n=19). 12-16y (n=13).	n: 104 Gender: 51(49%) M. 53 (51%) F. Mean age: 10.83±3.30y. Age range: 6-16y. 6-11y (n=51, mixed dentition). 12-16y (n=53, permanent dentition).	(I) ASD: sufficient cooperation level to be examined in a dentist chair. (I) Controls: Non user of any medication that could affect oral health. (E): institutionalized patients; patients whose primary medical condition also includes: blood dyscrasia, congenital heart disease, diabetes, autoimmune conditions, kidney diseases, chemo- or radiation therapy; previously dental treatment under general	Age, Gender and type of dentition (mixed/permanent).	Prevalence of oral cleanliness categories according James et al. 1960 (no plaque-good cleanliness; some plaque or food accumulation-fair; marked presence of plaque and/or food-poor cleanliness). Partial exam.	Good hygiene: ASD 18.6% (6) vs 67.3% (70) control. Fair hygiene: ASD 39.5% (13) vs 17.3% (18) control. Poor hygiene: ASD 41.9% (13) vs 15.4% (16) control.	Children with ASD had higher fair and poor hygiene when compared to control.

				anaesthesia; fluorosis.				
Onol & Kırzıoğlu 2018 Turkey Cross-sectional.	Seven Special Education and Rehabilitation Centers.	n: 63 Gender: NR. Mean age: 10.5±2.9 y Epilepsy accompanied ASD in 22.2% (14). Tool: Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition Text Revision Criteria.	n: 111 Gender: NR. Mean age: 10.2±2.5y	(I) ASD: Ability to follow instructions such as "sit down, open your mouth, and lower your hands." Allows touching his/her face or mouth, no professional prophylaxis in the last 6 months, no disorder such as Down syndrome and diabetes.	No.	Mean±SD of decayed and filled tooth and surfaces in the primary and permanent dentition. Mean±SD of missing tooth and surfaces in the permanent dentition. Mean±SD of PlI and GI.	Primary dentition: dft:ASD 4.58±4.22 vs 3.61±2.44 control. dfs: ASD 8.58±9.34 vs 5.94±4.55 control. Decayed tooth: ASD 4.15±4.3 vs 2.34±2.36 control. Filled tooth (p= 0.000): ASD 0.08±0.39 vs 1.27±1.84 control. Permanent teeth: DMFT: ASD 3.59±3.6 vs 2.37±1.9 control. DMFS (p=0.027): ASD 5.8±6.55 vs 3.08±2.79 control. Decayed tooth (p=0.027): ASD 3.14±3.35 vs 1.8±1.89 control. Filled tooth (p=0.001): ASD 0.2±0.89 vs 0.62±1.24 control. Missing tooth (p=0.001): ASD 0.25±0.77 vs 0 control. Missing surfaces (p=0.000): ASD 1.27±3.85 vs 0 control. PlI (p=0.000):ASD 2.06±0.73 vs 1.24±0.54 control. GI (p=0.000):ASD 1.91±0.56 vs 1.22±0.46 control	Children with ASD had higher values of DMFT and dft but was not found to be significant. Children with ASD had higher mean of missing tooth and surfaces than control. Children with ASD had significantly higher PlI and GI values in comparison to control.
Qiao et al. 2018 China Cross-sectional.	ASD: Shanghai Children's Medical Center.	n:32 Gender: 27 (84.37%) M 5 (15.63%) F Mean age: 10.02±1.43y. Age range: 7-14y.	n:27 Gender: 21 (77.77%) M 6 (22.22%) F Mean age: 10.19±0.59y.	(I) ASD: absence of systemic disease. (I) control: Age, gender and socioeconomic status comparable	Age and gender.	Mean±SD of caries according DMFT and DMFS. Mean±SD of PlI, BOP, GI,	DMFT (p<0.01): ASD 2.03±1.79 vs 1.04±1.86 control. DMFS (p<0.01): ASD 3.91±3.83 vs 1.96±3.86 control.	ASD showed higher statistically means of caries and gingival inflammation

	Control: primary schools.	Tool: DMS-5 Diagnosis was further confrmed at Shanghai Mental Health Center with the criteria for the International Classifcation of Diseases and Related Health Problems, Tenth Revision (ICD-10).		with ASD; good mental and physical health. (E) antibiotics within 3 months before the study; local antimicrobial treatment within 2 weeks; previous medical treatment for ASD or usage of sedatives; gluten-free/casein-free diet or probiotics use, oral mucosal lesions such as lichen planus; periodontal pockets >4 mm; acute oral infection; oral candidiasis.		sulcus bleeding index (SBI), probing depth (PD). All participants had mixed dentition.	PI (p=0.88): ASD 1.88 ±0.43 vs 1.88±0.34 control. GI (p<0.01): ASD 0.90± 0.44 vs 0.25±0.47 control. SBI (p<0.01): ASD 1.02±0.72 vs 0.25± 0.47 control. BOP (p=0.01): ASD 0.69±0.56 vs 0.31±0.45 control. PD (p=0.53): ASD 1.92±0.37 vs 1.81±0.47 control.	(GI, BOP, SBI) when compared to the controls.
Bhandary & Nary 2017 India Cross- sectional.	ASD: Special schools, hospitals of Mangalore, India.	n:30 Gender: 20 (66,6%) M. 10 (33.3%) F. Age range: 6–12y. Professional type: neurologist.	n: 30 Gender: 8 (26,6%) M. 22 (73.3%) F Age range: 6–12y.	(I): ASD diagnosis by a neurologist. (I): Control healthy siblings of ASD children. (E): Children extremely aggressive, children with underlying medically compromised conditions.	Age.	Mean±SD of DMFT/dft and of missing teeth. Prevalence of oral hygiene status according OHI-S categorized by good, fair, poor. Prevalence of gingival bleeding.	Primary tooth: Dft (p=0.13): ASD 0.90±0.92 vs 0.57±0.77 control. Permanent tooth DMFT (p=1.0): ASD 0.37±0.62 vs 0.37±0.56 control. Missing tooth (p=0.51): ASD 0.10±0.31 vs 0.17±0.46 control. Prevalence of good oral hygiene: ASD 30% (9) vs 56.6% (17) control. Fair oral hygiene: ASD 53.3% (16) vs 33.3% (10) control.	No significative difference regard caries or missing teeth was observed between ASD and controls. Higher prevalence of gingival bleeding was observed in ASD children when compared to the controls.

							Poor oral hygiene: ASD 16.6% (5) vs 10% (3) control. Prevalence of gingival bleeding: ASD 53.3% (16) vs 26.6% (8) control.	
Alaki et al. 2016 Saudi Arabia Cross-sectional.	ASD: Ten centers for autism and/or mental disability (one public and nine private). Control: five elementary schools (two public and three private) in the city of Jeddah, Saudi Arabia.	n: 75 Gender: 58 (77.3%) M. 17 (22.7%) F. Age range: 6-12y. Father's level of education: School 38.7%; Diploma 13.3%; College 48%. Type of school/center (p=0.016): Public: 26.7% (20). Private: 73.3% (55). Medical reports or center specialist.	n: 99 Gender: 59 (59.6%) M. 40 (40.4%) F. Father's level of education: School: 25.3; Diploma: 9.1%; College: 65.7%. Type of school/center (p=0.016): Public: 44.4% (44) Private: 55.5% (55)	(I): the center offered an established regular rehabilitation program for children with autism, it enrolled autistic children with ages 6-12 years, it had more than five enrolled autistic children. (E) ASD: Children diagnosed with other ASDs or chronic medical problems.	Age.	Prevalence of caries. Mean±SD of dft/DMFT, decay tooth and filling tooth, and missing teeth. Prevalence of oral hygiene according OHI-S and gingival status according GI. Mean±SD of OHI-S and Visual periodontal index.	Caries prevalence (p=0.013): ASD (75) 80% (60) vs 62.6% (62) control. dft + DMFT (p=0.003): ASD 6.17±4.51 vs 4.28±3.37 control. Primary dentition: Dft (p=0.001): ASD 4.87±4.34 vs 2.89±2.93 control. Decayed tooth (p=0.000): ASD 4.03±4.35 vs 1.98±2.55 control. Filled tooth: ASD 0.84±1.73 vs 0.91±1.67 control. Permanent dentition DMFT: ASD 1.31±2.28 vs 1.32±1.70 control. Decayed tooth: ASD 0.75±1.37 vs 0.67±1.17 control. Filled tooth: ASD 0.55±1.74 vs 0.52±1.17 control. Missing tooth: ASD 0.01±0.12 vs 0.14±0.70 control. Prevalence of oral hygiene status: Good: ASD 37.3% (28) vs 26.3% (26) control. Fair: ASD 54.7% (41) vs 64.6% (64) control. Poor: ASD 8.0% (6) vs 9.1% (9) control.	Children with ASD have significantly higher caries prevalence and severity, and lower missing tooth number than controls. There was no difference between ASD and control in oral hygiene status, mean OHI-S, gingival health status and mean Visual Periodontal Index score.

							Prevalence of gingival status: Healthy: ASD 18.7% (14) vs 18.8% (18) control. Mild to moderate Gingivitis: ASD 81.3% (61) vs 81.8% (81) control. OHI-S:ASD 1.08 ±0.49 vs 1.14±0.66 control. Visual Periodontal Index: ASD 0.81±0.39 vs 0.82±0.39 control.	
Diab et al. 2016 Saudi Arabia Case-control.	ASD: Azzam Autism School Almorsalat Square, Riyadh city. Controls: from outpatient clinic, Riyadh Colleges of Dentistry and Pharmacy**.**	n: 50 Gender: 37 (74%) M. 13 (26%) F. Mean age 8.5y. Age range: 4-15y.	n: 50 Gender: 37 (74%) M. 13 (26%) F. Mean age 8.7y. Age range: 4-15y.	(I) Case: diagnosis of autism. (E) Case: dental prophylaxis in the last 6 months, patients with systemic disorders that affect the periodontal disease and diabetes and potentially uncooperative patients. (E) Control: undergoing antibiotic or anti-inflammatory therapy or had undergone dental prophylaxis in the past 6 months.	Age and Gender.	Mean±SD of PlI and MGI. Partial exam for PI and MGI: Ramjford teeth.	MGI (p=0.003):ASD 1.83±0.65 vs 1.35±0.86 control. PlI (p=0.000): ASD 1.93±0.36 vs 1.44±0.43 control.	Children with ASD showed scores of plaque and of gingival inflammation higher significantly than controls.
Sarnat et al. 2016 Israel Cross-sectional.	ASD: 3 special kindergartens from three	n: 47 Gender: 39 (83%) M. 8 (17%) F. Mean age 5.53±1.06y.	n: 44 Gender: 10 (22.7%) M. 34 (77.3%) F.	NR.	NR.	Prevalence of caries. Mean±SD of def (number of decayed,	Def (p= 0.059): ASD 1.28±2.42 vs 1.84±2.56 control.	No difference in the caries experience and in the good oral

	towns in Israel. Controls: 4 kindergartens from 2 neighboring towns.	Age range: 3.5-8y. Vinland Adaptive Behavioral Scales: to assess the social competence of handicapped and non-handicapped individuals from birth to age 19 years. The scale measures four domains: communication, daily living skills, socialization and motor skills. Diagnostic Tool: DSM-5.	Mean age 5.63±0.43y. Age range: 4.5-6.5y.			extracted, or filled teeth). In ASD children: correlation between motor or living skills and caries, and between motor or living skills and oral hygiene. Prevalence of good oral hygiene.	Caries prevalemce: ASD 16 (34%) vs 24 (54%) control. No correlation significant was observed between living skills or motor skills and caries among ASD children. Prevalence of good oral hygiene: ASD 80.8% (38) vs 68% (30) control. The correlation between living skills and oral hygiene demonstrated that children who had high living skills demonstrated significant better oral hygiene (p = 0.026).	hygiene prevalence was observed between ASD and control.
Blomqvist et al. 2015 Sweden Cross-sectional.	ASD: Northern Stockholm psychiatric Clinic and a community-based unit for adults with ASD in Stockholm, Sweden. Controls: six dental clinics in the Stockholm area.	n: 47 Gender: 25 (53%) M. 22 (47%) F. Mean age: 33±8y. Educational level (p=0.021): Elementary school: 6% . High school: 60%. University/college: 34%. Working or studying (p<0.001): Full time 23%. Part time 15%. Unemployed/ not studying 62%. Smoking 4%.	n: 69 Gender: 34 (49%) M. 35 (51%) F. Mean age: 34±7y. Educational level (p=0.021): Elementary school: 3%. High school: 36%. University/college: 59%. Working or studying (p<0.001): Full time 91%. Part time 7%. Unemployed/ not studying 1%. Smoking 13%.	(E) ASD/Control**:** diagnosis of intellectual disability, history of brain damage, current or past neurological disorder, epilepsy, alcohol abuse, or dependence, past or present substance abuse and psychosis. (E) Controls: Scores above cut-off for a probable ASD, according to AQ.	Age, gender and area of residence (socio-economic background).	Mean±SD of DMFS and of number of present teeth in the permanent dentition. Mean±SD of the percentage of sites with gingival bleeding. Prevalence of individuals with buccal gingival recession. Mean number of teeth with buccal gingival recession. Prevalence of calculus supragingival.	DMFS: ASD 14.9±18.9 vs 15.9±14.6 control. Number of teeth: ASD 27.4±1.8 vs 27.4±1.4 control. Percentage of sites with gingival GBI (p=0.046): ASD 4.9±6.2 (n=46) vs 10.3±17.2 (n=58) control. Patients with buccal gingival recessions (p<0.001): ASD 72% (34) vs 36% (21) control. Mean number of teeth with buccal gingival recessions (p = 0.001): ASD 6.3±6.2 vs 2.7±4.8 control. Supragingival calculus prevalence: ASD 26% (12) vs 17% (10) control.	The severity of caries and the number of present teeth was equal in ASD and control. No association between ASD severity and DMFS was found (p=.381). ASD group had more buccal gingival recessions than controls.

		AQ single count: 28.7±10. Medications users (p<0.001): 66%. Users of medications associated with hyposalivatin (p<0.001): 47%. Tools: Wechsler Adult Intelligence Scale; neuropsychological tests, and clinical interviews following the DSM-IV-TR criteria for autistic disorder, Professional type: Consensus between psychiatrist and a Psychologist.	AQ single count: 11.4±4.5. Medications users (p<0.001): 10%. Users of medications associated with hyposalivation (p<0.001): 1%.					
Du et al. 2015 China Case control.	ASD: Special Child Care Centres. Controls: mainstream preschools.	n: 347 participants n: 257 (clinical examination) Gender: 217 (84.4%) M. 40 (15.6%) F. Mean age: 59±10 months.	n: 347 participants n: 257 (clinical examination) Gender: 217 (84.4%) M. 40 (15.6%) F. Mean age: 59±10 months.	NR.	Age (±3 months) and Gender.	Prevalence of caries (dmfs>0, ds>0) and mean±SD of dmfs, ds, fs and the number of missing tooth surfaces in the primary dentition. Mean±SD of GI and of plaque presence. Prevalence of sites with plaque and gingivitis. Partial exam: Teeth 55, 52,	Caries prevalence (p <0.001): ASD 37.0% (95) vs 52.5% (135) control. ds>0 (p <0.001): ASD 35.4% (91) vs 51.4% (132) control. dmfs (p= 0.038): ASD 3.73±9.03 vs 5.41±9.18 control. ds (p= 0.012): ASD 3.14±7.66 vs 4.91±8.29 control. fs: ASD 0.38±3.89 vs 0.41±1.84 control. missing surfaces: ASD 0.21±1.85 vs 0.09±1.15 control.	Children with ASD had less caries and missing tooth surfaces, and better gingival health when compared to control.

						63, 64, 75, 72, 83 and 84.	PlI (p <0.001): ASD 0.45±0.24 vs 0.60±0.27 control. GI (p <0.001): ASD 0.37±0.29 vs 0.51±0.27 control. Percentage of sites with presence of plaque (p<0.001): ASD 44.4% (22.8) vs 55.7% (23.1) control. Percentage of sites with gingivitis (p<0.001): ASD 36.7% (27.8) vs 49.8% (25.9) control.	
Al-Maweri et al. 2014 Yemen Case control.	ASD: Al-Yemen special center for rehabilitation and education of children with autism in Sana'a. Control: Two public schools in the same neighborhood.	n: 42 Gender: 33 (78.6%) M. 9 (21.4%) F. Mean age: 8.45y. Age range: 5-16y. Type of dentition: Primary 11.9% (5) Mixed 69.0% (29) Permanent 19.0% (8)	n: 84 Gender: 66 (78.6%) M. 18 (21.4%) F. Mean age: 8.6y. Type of dentition: Primary: 14.5% (12) Mixed: 69.0% (58) Permanent: 16.7% (14)	(I):autism diagnosis, understanding of at least very simple instructions, enough cooperation. (E): Children with any other systemic disease known to cause dental problems and extremely uncooperative children.	Age and Gender.	Prevalence of caries. Mean±SD of dmft/DMFT, decay tooth, missing tooth, filling tooth. Prevalence of gingivitis and oral hygiene status. Mean±SD of GI and PlI.	Caries prevalence (p=0.05): ASD 100% (42) vs 90.5% (76) control. DMFT: ASD 2.00±2.18 vs 1.27±1.77 control. DT: ASD 1.86±2.10 vs 1.22±1.69 control. FT: ASD 0.05±0.33 vs 0.03±0.16 control. Missing tooth (MT): ASD 0.05±0.23 vs 0.03±0.16 control. Dmft (p=0.001): ASD 5.23±2.34 vs 4.06±2.98 control. dt (p=0.001): ASD 4.40±2.21 vs 3.61±2.82 control. ft: ASD 0.26±1.04 vs 0.07±0.49 control. missing tooth (mt): ASD 0.57±1.60 vs 0.24±0.65 control. Status gingival: Healthy: ASD 4.8% (2) vs 6.0% (5) control.	Children with ASD had higher statistically mean of dmft and of missing tooth when compared to controls. No difference was observed between groups regard carie and tooth loss from permanent dentition. Poorer oral hygiene and significantly higher mean PI and GI were observed in ASD when compared to controls.

							Mild: ASD 31.0% (13) vs 41.1% (35) control. Moderate: ASD 40.5% (17) vs 42.9% (36) control. Severe: ASD 23.8% (10) vs 9.5% (8) control. OH status: Excellent: ASD 4.8% (2) vs 0.0% (0) control. Good: ASD 31.0% (13) vs 58.3% (49) control. Fair: ASD 38.1% (16) vs 38.1% (32) control. Poor: ASD 26.2% (11) vs 3.6% (3) control. Mean±SD of: GI (p=0.037): ASD 1.36±0.84 vs 1.02±0.51 control. PI (p=0.002):ASD 1.5±0.81 vs 1.05±0.51 control.	
Fakroon et al. 2014 Libya Cross-sectional.	ASD: Benghazi Centre of Autism, Benghazi, Libya. Controls: schools in same location as children with ASD.	n: 50 Gender: 40 (80%) M. 10 (20%) F. Mean age: 7.29±3.11y.	n: 50 Gender: 40 (80%) M. 10 (20%) F. Mean age: 7.29±3.11y.	(I) ASD: autism diagnosis, written informed consent signed by parents or carers. (E) ASD: extremely uncooperative or diagnosed for any other illness that could have an effect on the occurrence of dental caries.	Age, sex and socio-economic status.	Mean±SD of Dmft (primay dentition) and DMFT (permanent dentition), and of missing tooth. Prevalence of periodontal Status (CPITN). Partial exam.	Permanent dentition: DT (p<0.001): ASD 0.20±0.62 vs 1.00±1.72 control. FT (p= 0.041): ASD 0.02±0.13 vs 0.07±0.38 control. DMFT (p<0.001): ASD 0.22±0.08 vs 1.15±0.27 control. Missing tooth (p=0.011): ASD 0.00±0.00 vs 0.07±0.42 control. Primary dentition: dt (p<0.001): ASD 0.85±1.66 vs 2.65±3.16 control. ft (p=0.002): ASD 0.29±0.99 vs 0.07±0.42 control. dmft (p=0.001): ASD 1.13±1.84 vs 2.85±3.32 control. missing tooth (p<0.001):	Children with ASD showed significantly lower means of DMFT, of dmft and of missing tooth than control. Children with ASD showed significantly higher periodontal treatment need and nearly double of signs of gingival inflammation when compared to the control.

							ASD 0.02±0.00 vs 0.13±1.00 control. Prevalence of periodontal status: Healthy: ASD 9.1% (5) vs 41.8% (23) control. Gingival bleeding: ASD 38.2% (21) vs 21.8% (12) control. Supra/subgengival calculus: ASD 52.7% (29) vs 36.4% (20) control	
Richa et al. 2014 India Cross-sectional.	ASD: Various special schools. Controls: Regular schools.	n: 135 Gender: 108 (80%) M 27 (20%) F Age range: 4-15y. Socio-economic status (modified Kuppuswamy scale): Upper: 34.1% Upper middle: 32.6% Lower middl : 27.4% Upper lower: 5.9% Lower: 0%	n: 135 Gender: 79 (58.5%) M 56 (41.5%) F Age range: 4-15y. Socio-economic status: Uppe: 22.2% Upper middle: 37.1% Lower middle: 38.5% Upper lower: 2.2% Lower: 0%	(I) ASD: austism diagnosis (I) Control: Non-ASD medically fit. (E) oral prophylaxis in the last 6 months; antibiotic or antiinflammatory therapy; Down's syndrome and diabetes.	No.	Mean±SD of DMFT, dmft, DMFS, dmfs. Mean±SD of hygiene status (OHI-S). Partial exam.	DMFT ($p<0.01$): ASD 0.86±1.22 vs 0.46±1.06 control. dmft ($p<0.01$): ASD 1.40±2.48 vs 0.59±1.28 control. DMFS ($p<0.01$): ASD 0.90±1.33 vs 0.59±1.40 control. dmfs ($p<0.01$):ASD 2.65±6.32 vs 1.13±2.81 control. OHI-S ($p<0.01$): ASD 2.07±0.83 vs 0.46±0.58 control.	Caries experience and OHI-S mean were significantly higher among children with ASD when compared to control.
El Khatib et al. 2014 Egypt Case control.	ASD: Private and governmental institutions of intellectually disabled children in Alexandria. Controls: private and governmental schools.	n: 100 Gender: 75 (75%) M 25 (25%) F Mean age: 9.06±4.03y. Dental visit in previous year ($p=0.002$): 44.4%. Ease of finding a dentist ($p<0.0001$): Easy: 23.8% Difficult: 64.3%	n:100 Gender: (73) 73% M (27) 27% F Mean age: 8.88±4.40y. Dental visit in previous year ($p=0.002$): 66.7%. Ease of finding a dentist ($p<0.0001$): Easy: 75.4% Difficult: 24.6%	NR.	Sex; Age and Socio-economic status.	Prevalence of caries. Mean±SD of dmft, dtf and DMFT according dentition type. Mean±SD of PlI and GI.	Caries prevalence: Overhall: ASD 79 (79%) vs 86 (86%) control. Primary dentition: ASD 56.7% (17) vs 66.7% (18) control. Mixed dentition (primary teeth): ASD 76.7% (33) vs 67.3% (35) control. Mixed dentition (permanent teeth): ASD 32.6% (14) vs 34.6% (18) control. Permanent dentition:	No significant differences were found in caries prevalence or experience between ASD and control. Children with ASD had significantly poorer oral hygiene and

		Could: 9.5% Working mothers: 68.5%. Medication: 34% [32% anticonvulsant as phenytoin, and 2% antidepressant as citalopram]. Tool: DSM-IVTR.	Could: 9.5% Working mothers: 66%. Medication (p=0.02): 5.1%.				ASD 55.6% (15) vs 71.4% (15) control. Mean±SD of caries experience: Primary dentition-dmft: ASD 3.53±4.57 vs 3.56±3.86 control. Mixed dentition-dft: ASD 3.33±3.39 vs 2.94±2.94 control. Mixed dentition-DMFT: ASD 0.93±1.58 vs 0.79±1.26 control. Permanent dentition-DMFT: ASD 3.4±4.54 vs 3.50±3.63 control. Mean±SD of: PlI (p<0.0001): ASD 2.02±0.73 vs 1.40±0.80 control. GI (p<0.0001): ASD 2.00 ±0.73 vs 1.40±0.80 control.	gingival condition than control.
Orellana et al. 2012 Spain Case-control.	ASD: Two-day centers for people with autism in the Valencian Community (one in the province of Castellón and the other in the province of Valencia).	n: 30 Gender: 27 (90%) M 3 (10%) F Mean age: 27.7±5.69y. Age range: 20-41y. All patients presented some degree of mental impairment: Mild: 26% Moderate: 37% Severe: 37% Patients institutionalized: 63.33% (19). ASD receiving some kind of medication 77% (anxiolytics 57%,	n: 30 Gender: 23 (76.67%) M 7 (23.33%) F Mean age: 27.83±5.84y.	(I) Cases: autism diagnosis; the understanding at least of very simple instructions; and the obtainment of written informed consent from the caregivers for participation in the study.	Age and Gender.	Prevalence of caries. Mean of CAOD (Caried, absent and obturated teeth) and of CAOS (Caried, absent and obturated surface). Mean of OHI-S and of Index score of PI-S. Partial exam.	Caries prevalence: ASD 60% (18) vs 56.67% (17) control. CAOD (p= 0.032): ASD 3.7 vs 5.63 control. Missing tooth: ASD 1 vs 0.7 control. CAOS: ASD 9.03 vs 12.37 control. Absent tooth surfaces: ASD 5 vs 3.50 control. OHI-S: ASD 1.92 vs 1.52 control. PI-S (p=0.000): ASD 1.57 vs 0.96 control.	ASD group presented less caries experience and more absent tooth surfaces than control. The amount of plaque was significantly greater in the ASD group than control

		antipsychotics 48%, anticonvulsants 39%, neuroleptics 22%; antidepressants 17% and other medications 22%).						
Rai et al. 2012 India Cross-sectional.	ASD: special schools. Controls: healthy siblings of ASD children with no delayed developmental milestones and had good academic performance.	n: 01 Gender: M and F. Age range: 6-12 y. Medium functioning: 48 (IQ between 50 and 70). Low functioning: 53 (IQ<50).	n: 50 Age range: 6-12y.	NR.	Age. A three day diet chart was recorded for both the groups (intake of carbohydrates and refined sugars). The diet was comparable between groups.	Prevalence of caries (index not reported). Median of OHI-S.	Caries prevalence: ASD 65.34% (66). On further subdividing the study group as low and medium functioning, no significant difference (p=0.118) was observed. Median of OHI-S (p< 0.001): ASD 1.2 (fair oral hygiene) vs 1 (good oral hygiene) control. Median of OHI-S was of 1.4 in ASD presenting low functioning vs 1.2 in ASD with medium functioning.	Caries prevalence did not differ statistically in children with autism and their siblings. Children with ASD had significantly poorer oral hygiene condition than control.
Vajawat & Deepika 2012 India Case control.	ASD: Academy for Severely Handicapped and Autistics. Control: regular schools.	n: 117 Gender: NR. Age range: 5-22y. Dentition: Primary (0-5y) n=11; Mixed (6-10y) n=48; Permanent (11-15y): n=58.	n: 126 Gender: NR. Dentition: Primary n=13; Mixed n=58; Permanent n n=53.	(I) Cases: autism diagnosis. (I) Controls: similar age group. (E): dental treatment in the last 6 months, any other systemic disease known to cause dental problems and uncooperative patient.	Age and Socio-economic status.	Prevalence of caries and mean of DMFT. Mean of PlI and GI. Prevalence of periodontal status according CPITN.	Prevalence of caries was lower in ASD (p=0.000) than control. Incidence of caries was increasing with age in both cases and controls. Mean of DMFT: Overhall: ASD 1.30 vs 3.74 control. Primary dentition *: ASD 0.55 vs 2.69 control. Mixed dentition *: ASD 1.33 vs 3.75 control. Permanent dentition*: ASD 1.41 vs 3.98 control. PlI mean (p=0.000): Overall: ASD 1.30 vs 1.00 control. Primary dentition: ASD 0.66 vs 0.69 control.	Caries prevalence and DMFT mean were lower in ASD than control. PlI and GI mean were significantly higher in ASD than control. ASD had a higher rate of periodontal disease compared to controls.

							Mixed dentition: ASD 1.20 vs 0.92 control. Permanent dentition: ASD 1.51 vs 1.18 control. GI mean (p=0.000): Overall: ASD 1.00 vs 0.85 control. Primary dentition: ASD 0.61 vs 0.66 control. Mixed dentition: ASD 0.10 vs 0.79 control. Permanent dentition: ASD 1.25 vs 0.97 control. Periodontal disease prevalence (p=0.000): Mixed dentition: Bleeding: ASD 56.3% (27) vs 83.3% (48) control. Calculus: ASD 41.7% (20) vs 16.7% (10) control. Pocket: ASD 2.1% (1) vs 0% (0) control. Permanent dentition Bleeding: ASD 18.6% (11) vs 54.2% (28) control. Calculus: ASD 61.0% (35) vs 44.2% (23) control. Pocket: ASD 20.3% (11) vs 1.6% (1) control.	
Jaber 2011 United Arab Emirates Cross sectional.	ASD: Centers of emirates of Dubai and Sharjah, that offer an intensive rehabilitation program only for children	n: 61 Gender: 45 (73.7%) M 16 (26.2%) F Mean age: 8.45y. Age range: 6-16y.	n: 61 Gender: 45 (73.7%) M 16 (26.2%) F Mean age 8.6y.	(I) ASD: autism diagnosis; 6-16 y old. (E) ASD: dental prophylaxes in the last 6 months; Down's syndrome and diabetes. Controls:	Age, sex, socio-economic status and general dental care background.	Prevalence of dental caries, oral hygiene status(OHI-S) and gingivitis (GI). . Mean±SD of dmft and DMFT.	Prevalence overall of caries (p<0.05): ASD 77.0% (47) vs 46.0% (28) control. Prevalence overall of caries according gender: Female: ASD 87.5% (14) vs 75% (12) control. Male: ASD 73.3% (33) vs 35.5% (16) control.	Children with ASD had higher significantly prevalence of caries, gingivitis and poor oral hygiene when compared to the control.

	diagnosed with autism. Control: relatives or friends of autistic patients in an attempt to have matched age, sex, socioeconomic status and general dental care background.			all controls were medically fit and none was undergoing antibiotic or antiinflammatory therapy or had undergone such therapy in the previous 6 months.			dmft (p<0.05): ASD 0.8±0.20 vs 0.3±0.3 control. DMFT (p<0.05): ASD 1.6±0.64 vs 0.6±0.29 control. Overall Mean of DMFT/dmft: ASD 2.4 vs 0.9 control. Number of missing tooth: ASD NR (8) vs NR (7) control. Prevalence of: Good oral hygiene (p<0.05): ASD 3.3% (2) vs 59.0% (36) control. Fair oral hygiene (p<0.05): ASD 38.0% (23) vs 26.2% (16) control. Poor oral hygiene (p<0.05): ASD 59.0% (36) vs 14.8% (9) control. Prevalence of: Gingivitis: ASD 97.0% (59) vs 41.0% (25) control. Generalized (p<0.05): ASD 78.0% (46) vs 20.0% (5) control. Localized (p<0.05): ASD 22.0% (13) vs 80.0% (20) control.	
Luppanapornlarp et al. 2010. Thailand. Cross-sectional.	ASD: various areas in Bangkok. Control: pediatric clinic, Faculty of Dentistry, Mahidol University, Bangkok.	n: 32 Gender: 25 (78.12%) M 7 (21.88%) F Mean age 9.7±1.2y.	n: 48 Gender: 19 (39.58%) M 29 (60.42%) F Mean age 9.9±1.1y.	(I): age between 8-12 y and no orthodontic treatment before or during the examination. (E): inability to cooperate in the oral examination.	Age.	Prevalence of periodontal status according CPITN modified (pocket depths and dental caries was not recorded). Missing tooth prevalence according	Periodontal status prevalence (p=0.000): Healthy: ASD 9.4% (3) vs 29.2% (14) control. Calculus: ASD 12.5% (4) vs 56.3% (27) control. Bleeding: ASD 78.1% (25) vs 14.5% (7) control. Prevalence of missing tooth (≥ 1): ASD 6.3% (2) vs 0.0% (0) control.	Chidren with ASD had significantly poorer oral hygiene and significantly more bleeding than control. Missing teeth prevalence was higher in

						Dental Aesthetic Index.		ASD than control.
Bassoukou et al. 2009 Brazil Cross-sectional.	ASD: "Associação de Amigos do Autista" and "Centro Terapêutico Educacional", São Paulo. Controls: individuals attending the Pediatric Dental Clinic at the Unicsul School of Dentistry and individuals attending the Fé Cristã Church, São Paulo.	n: 25 boys. Gender: 25 (100%) M. 10 children, ages 3–8y (6.1±1.5y). 6 children taking medication (methylphenidate n=2, two; risperidone n=4). 15 adolescents, ages 9–13y, (10.5±1.2 y). 11 adolescents taking medication (risperidone, n=6; valproate n=3; sertraline n=2). Medical diagnosis.	n: 25 Gender: 25 (100%) M. 14 children, ages 4–8y (6.8±1.2y). 11 adolescents, ages 9–14y, (10.8±1.4y).	Controls: None of them had any systemic diseases and were not taking any medication for at least 15 days before saliva collection.	N.	Mean±SD of DMFT/dmft categorized by age and medication's use.	Dmft (all sample): ASD (n=3) 1.67±2.89 vs 1.75±2.87 control (n=4). DMFT (all sample): ASD (n=22) 2.77±3.25 vs 2.33±2.89 control (n=21). DMFT aged 3–8 y (p=0.82): ASD 2.00±2.83 (n=10) vs 1.79±3.07 control (n=14). DMFT aged 9–13 y (p=0.57): ASD 2.00±2.20 (n=15) vs 3.00±3.10 control (n=11).	
Loo et al. 2008 United States Cross-sectional.	The non-archived records at the dental department of Franciscan Hospital for Children (FHFC) (Boston).	n:395 Gender: 317 (80.25%) M 78 (19.75%) F Median age of 12y; Age range: 3-28y. 47.3% (187) receiving medication (45.5% taking two or more medications; 41.2% receiving antipsychotic agents. Risperidone 61.6%. Antidepressives 11.6%. Anticonvulsant agents 16.2%).	n: 386 Gender: 193 (50%) M 193 (50%) F Median age of 8y; \Age range 3-20y.	(I) control: healthy, no medical conditions and no medication.	NR/ Multiple logistic regression analysis controlling for age and sex: (1) to evaluate the association between ASD and caries prevalence; (2) to compare the caries prevalence of the ASD and control.	Prevalence of caries.	Caries prevalence (DMFT>0) (p< .0001): ASD 68.1% (269) vs 86% (332) control. Caries severity (p< .0001): ASD (median DMFT, 3; interquartile range, 7; range, 0-30) vs (median DMFT, 5; interquartile range, 7; range, 0-21) control. Multiple logistic regression analysis controlling for age and sex showed a significant association between ASD and caries prevalence (p<.0001): ASD patients were 70.5% less likely to have a positive caries history than control: OR 0.30 (95% CI 0.20-0.44).	Caries prevalence and severity in ASD were lower than control. There was a significative association between ASD and a lower odd of caries when compared to control. Caries prevalence and severity in ASD were

	In the ASD group, 55.2% (218) of patients were uncooperative (either negative or definitely negative behavior), and 9.2% (36) exhibited definitely positive behavior. In the control, 25.4% (98) of the patients were uncooperative, and 46.6% (180) exhibited definitely positive behavior. 31.4% (124) of patients with ASD resided in an institution or group home and 68.6% (271) resided at home. Diagnosis presented in the medical histories, which were completed and updated by their parents or legal guardians during routine dental visits.		Regression analysis controlling for age and sex to compare the caries prevalence of the ASD and control: -Primary dentition (younger than 6y) (p=.0003): ASD were 83.4% less likely to have a positive caries history than control: OR 0.17 (95% CI 0.06-0.44). -Mixed or permanent dentition (age 6-1y) (p< .0001): ASD were 65.9% less likely to have a positive caries history than control: OR 0.34 (95%CI 0.22-0.54). ASD who received psychotropic medication were significantly older (median age, 14y;) than patients who did not receive psychotropic medication (median age 10y) (p=.0002). There was no significant difference in DMFT scores between these two groups (p=.20). Within the ASD group, the 17.2% of patients who had a seizure disorder were significantly older (median age 14y) than those who did not have a seizure disorder (median age 11 y) (p<.0001). We noted no significant difference in DMFT scores between these two groups (p=.3). ASD who had an additional diagnosis (median age 14y)	not associated with institutionalization, presence of seizure disorder or additional diagnosis.

							were significantly older than patients with ASD who did not have an additional diagnosis (median age 12y) (p< .0001), but have no significant difference in DMFT scores between these two groups (p= .075).	
Namal et al. 2007 Turkey Cross-sectional.	ASD: 3 schools (one autistic child center and two elementary schools offering private classes to autistic children). Controls: randomly selecting three elementary schools in the same districts.	n: 62 Gender: 46 (74.19%) M 16 (25.81%) F Mean age: 9.32±1.68y. Age range: 7-12y. -More than half of the mothers 85.4% (53) and of the fathers 71.1% (44) showed a low level of education or no education -More than half of the families of the children with ASD had a high family income 69.4% (43).	n: 301 Gender: 136 (45.18%) M 165 (54.82%) F Mean age: 9.32±1.68y. Age range: 7-12y. -More than half of the mothers 85.4% (257) and of the fathers 71.1% (214) showed a low level of education or no.	NR.	No.	Prevalence of caries (DMFT≥1). Overall mean DMFT, decayed filled and missing tooth.	Caries prevalence (p<0.05): ASD 58.1% (36) vs 73.1% (220) control. Non-ASD children had 3.99 times the odds of having any experience of caries than autistics (p<0.05): OR 3.99 (95% CI 1.56-10.19). DMFT mean: ASD 1.74 vs 2.41 control. D means: ASD 1.08 vs 2.27 control. F mean: ASD 0.06 vs 0.14 control. Missing tooth mean: ASD 0.56 vs 0.02 control.	Younger age, a child of high-family-income, brushing teeth regularly, consuming less sugar and having ASD are factors that led to less caries experience. ASD had higher mean of missing tooth than control.
Fahlvik-Planefeldt & Herrström 2001 Sweden Case control.	ASD: Childhood Habilitation Unit and the Psychiatric outpatients ward for children and adolescents in the primary care area of Kungsbacka, Sweden.	n: 20 Gender: 12 (60%) M 8 (40%) F Mean age: 12y M Mean age: 10y F Age range: 3-19y. 40% (8): regular medication, which could affect oral conditions. Medical diagnosis.	n: 20 Gender: 12 (60%) M 8 (40%) F Mean age: 12y M Mean age: 10y F Age range: 3-19y. None of the controls used such medication.	NR.	Age and Gender.	Prevalence of caries, of fiilings surfaces and of gingivitis (bleeding on probing).	Caries prevalence: ASD 50% (10) vs 70% (14) control. Number mean of ds: ASD 2.25 vs 2.30 control. Number means of FS in primary and permanent teeth: ASD 0.6 vs 1.25 control. Prevalence of fillings surfaces in primary teeth: ASD NR (0) vs NR (12) control. Prevalence of fillings surfaces in permanent teeth: ASD NR (12) vs NR (13) control.	ASD and controls had a similar prevalence of caries, of fillings surfaces and of gingivitis.

	Controls: same clinic.									Gingivitis prevalence: ASD 45% (9) vs 40% (8) control.	

DMFT= total number of permanent teeth Decayed, Missing, Filled; DMFS= total number of surfaces Decayed, Missing, Filled; Dmft = total number of deciduos teeth Decayed, Missing, Filled; dmfs = total number of deciduos surfaces Decayed, Missing, Filled; DS = total number of permanent decayed surfaces; ds = total number of deciduos decayed surfaces; ms = total number of deciduos missing surfaces; fs = total number of deciduos filled surfaces; d = decayed primary teeth; f = filled primary teeth; Dft = decayed and filled primary teeth; D = decayed permanent teeth; M = missing permanent teeth; F = filled permanent teeth; Def = number of decayed, extracted, or filled teeth.
CPINT= community periodontal index of treatment needs; OHI-S = Simplified Oral Hygiene Index; GI = Gingival Index; PlI = Plaque Index; GBI = Gingival Bleeding Index; MGI = Modified Gingival Index.
*= represents statistical significance

Table 3. *Characteristics of included studies and comparison of malocclusion between ASD and controls (n=13).*

Author **Country** **Study design**	**Place recruitment**	**ASD group** Sample (N), gender (%); age. Socioeconomic status ASD diagnosis Tools/professional type Medications	**Control group** Sample (N), gender (%); age (mean±sd) Socioeconomic status	**Inclusion (I) and exclusion (E) criteria**	**Control matching**	**Outcomes measures** Tools Full-mouth/partial	**Results** ASD group vs control group	**Main findings**
Meuffels et al. 2022 Netherlands Cross-sectional.	ASD: subjects referred for orthodontic treatment to Erasmus Medical Center / Sophia Children's Hospital. Controls: a dental clinic.	n:48 Gender: 39 (81.3%) M 9 (18.8%) F Median age: 13.0y. Age range: 10-18y. Reported Comorbidity (intellectual disability, developmental disorder, attention deficit hyperactivity disorder, epilepsy) 30 (62.5%). Prescribed medication 24 (50%).	n:49 Gender: 22 (44.9%) M 27 (55.1%) F Median age: 13.0y. Age range: 10-18y. Reported Comorbidity 0 (0%). Prescribed medication 0 (0%).	(I) ASD (1) children with a confirmed diagnosis of ASD, (2) aged between 9 and 18 years, and (3) patients in the mixed or permanent dentition. (I) Controls (1) children without ASD, (2) aged between 9 and 18 years, and (3) with mixed or permanent dentition. (E) Children with incomplete dental records or who presented with a congenital facial deformity,	Not reported.	Prevalence of malocclusion traits such as Angle's classification (class I, class II, class III), overjet (measured on incisors, in millimeters), overbite (measured on incisors, in millimeters), crossbites (anterior or posterior), and open bites (anterior or posterior).	Prevalence of: Angle Class (p=0.61) Class I ASD 20.8% (10) vs 26.5% (13) control. Class II ASD 70.8% (34) vs 69.4% (34) control. Class III ASD 8.3% (4) vs 4.1% (2) control. Overjet (p=0.0016): < 1mm ASD 25% (12) vs 12.2% (6) control. 1 to ≤ 3mm ASD 10.4% (5) vs 20.4% (10) control. > 3 to ≤ 8 mm ASD 31.3% (15) vs 59.2% (29) control. > 8 mm ASD 33.3% (16) vs 8.2% (4) control. Overbite (p=0.031): ≤ 1 mm (decreased) ASD 20.8% (10) vs 14.3% (7) control. > 1 to ≤ 3 mm ASD 12.5% (6) vs 22.4% (11) control. > 3 to ≤ 7 mm ASD 35.4% (17) vs 53.1% (26) control. Impinging or 100% (extreme increase)	Increased overjet and overbite were more common in children with ASD than those without ASD.

				including any craniofacial anomaly or cleft lip and/or palate.			ASD 31.3% (15) vs 10.2% (5) control. Crossbite (p=0.57): Anterior ASD 29.2% (14) vs 12.2% (6) control. Posterior ASD 45.8% (22) vs 28.6% (14) control. Open bite (p=0.36): Anterior ASD 22.9% (11) vs 14.3% (7) control. Posterior ASD 14.6% (7) vs 2.0% (1) control.	
Bagattoni et al. 2021 Italia Cross-sectional.	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Prevalence of malocclusion according the relationship between the maxillary and mandibular first permanent molars (Angle's classification system); the relationship between the upper and lower dental arch in the three planes of space (sagittal, vertical, transverse).	Prevalence of malocclusion according Angle's classification (p= 0.565): Class 1 ASD 70% (34) vs 76% (38) control. Class 2 ASD 26% (13) vs 18% (9) control. Class 3 ASD 4% (2) vs 6% (3) control. Prevalence of: Posterior Crossbite (p= 0.565): ASD 14% (9) vs 17% (10) control. Anterior open bite (p= 0.013): ASD 19% (12) vs 5% (3) control. Deep bite (p= 0.803): ASD 14% (9) vs 17% (10) control.	Anterior open bite was significantly more prevalent in autistics than controls.
Kuter & Uzel 2021 Turkey Retrospective study.	Vide Table 2.	Vide Table 2.	Vide Table 2.	Vide Table 2.	Vide Table 2.	Prevalence of deep palate.	Deep palate: ASD boys 60.7% vs 16.9% control boys (p=0.000). ASD girls 50.0% vs 21.7% control boys (p=0.017).	ASD girls and boys had higher prevalence of deep palate than controls.
Farmani et al. 2020 Iran	Three main rehabilitation centers in Shiraz, Iran	n: 47 Gender: 36 (76.6%) M 11 (23.4%) F	n: 49 Gender: 28 (57.1%) M 21 (42.9%) F	(I) ASD: age 7–15y, medical diagnosis of	Demogra-phic features.	Prevalence of malocclusion, distribution of malocclusions	Prevalence of Malocclusion: ASD 76.1% (35) vs 79.2% (38) control.	ASD and control had not significant difference in

Cross-sectional.	(seventeen children from Froogh-Asr Rehabilitation Center, fourteen children from Shiraz School of Autism and sixteen children from Autism Care Center).	Mean age 10.74±2.1y. Age range: 8.13 - 13.72y. Medical diagnosis.	Mean age 9.5±1.32y.	ASD, mixed or permanent dentition. (I) Control: aged 7–15y, healthy children in the mixed or permanent dentition . (E) incomplete eruption of incisors, history of orthodontic treatment, anomalies presence, syndromes other than ASDs.	according Angle classification, overjet, overbite, and dental alignment (midline deviation, crossbite and space analysis).	Angle classification: Class I: ASD 55.6% (20) vs 88.2% (30) control. Class II (p= 0.03): ASD 44.4% (16) vs 11.8% (4) control. Class III: ASD 25.9% (7) vs 30.2% (13) control. OR (95%CI) 2.05 (0.905–4.654). Overjet Increased (p=0.03): ASD 44.4% (16) vs 11.8% (4) control. Decreased: ASD 25.9% (7) vs 30.2% (13) control. OR (95%CI) 2.05 (0.905–4.654). Overbite Increased: ASD 33.3% (12) vs 25% (8) control. Decreased: ASD 22.6% (7) vs 35.1% (13) control. OR (95% CI) 0.920 (0.413–2.049). Lip Competent: ASD 80.9% (38) vs 93.8% (45) control. Incompetent: ASD 19.1% (9) vs 6.3% (3) control. OR (95%CI) 3.355 (0.897–14.067). Midline deviation Coincident: ASD 82.6% (38) vs 43.8% (21) control. Deviated (p= 0.001): ASD 17.4% (8) vs 56.3% (27) control. OR (95% CI) 0.178 (0.071–0.446). Crossbite Anterior + Posterior: ASD 6.4% (3) vs 14.3% (7) control. OR (95% CI) 0.409 (0.099–1.687).	the overall prevalence of malocclusion. The ASD presented a higher prevalence of increased overjet and Class II molar relationship than control. The control showed a higher prevalence of midline deviation than ASD.

							Abnormal (crowding +spacing) ASD 13.3% (6) vs 22.4% (11) control. OR (95% CI) 0.709 (0.257–1.954).	
ALMusawi & Al-Dabagh 2019 Iraq Cross-sectional.	ASD: Special institutes for education and rehabilitation in six different governorates in Iraq. Control: Ordinary schools in the same geographical area where ASD institutions found.	n: 187 Gender: most participants in both groups were boys. Mean age 9.9y. Age range: 9-12y.	n: 190 Gender: most participants in both groups were boys. Mean age 9.9y. Age range: 9-12y.	(I): Age 9-12 y. No previous orthodontic treatment.	Age, Gender, same demogra-phic features.	Prevalence of increased over jet; deep bite; spacing; Posterior crossbite; openbite; displacement crowding; anterior crossbite and decreased overjet. Intraoral examination was derived from the orthodontic treatment need index.	Prevalence of malocclusion: Increased over jet (p< 0.00001): ASD 42.7% (80) vs 13.1% (25) control. Deep bite (p < .00001): ASD 35.8% (67) vs 10% (19) control. Spacing (p=.00086): ASD 28.8% (54) vs 14.7% (28) control. Posterior crossbite (p= .00008): ASD 19.25% (36) vs 5.7% (11) control. Openbite (p< .00001): ASD 17.6% (33) vs 2.6% (5) control. Displacement (p=0.00014): ASD 16% (30) vs 4.2% (8) control. Crowding: ASD 16.5% (31) vs 15.2% (29) control. Anterior crossbite (p<.00001): ASD 14.4% (27) vs 6.8% (13) control. Decreased overjet: ASD 13.9% (26) vs 6.3% (12) control.	Prevalence of malocclusion was higher among ASD than control. ASD had significantly higher prevalence of deep bite, open bite, spacing, increased over jet, anterior and posterior cross bite than control group children. Increased over jet was the most common trait in the ASD.
Kuter & Guler 2019 Turkey Cross-sectional.	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Prevalence of dental crowding, open bite, deep palate.	Dental crowding (p<0.05): ASD 32.1% (91) vs 52.0% (63) control. Open bite (p>0.05): ASD 5.7% (16) vs 4.9% (6) control. Deep palate (p<0.05): ASD 52.9% (151) vs 17.9% (22) control.	ASD had lower dental crowding than controls. Children with ASD had higher statistically prevalence of deep palate when compared to control. No significant difference was observed

								between ASD and controls regarding open bite.
Leiva-García et al. 2019 Spain Cross sectional.	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Prevalence of malocclusion as Class I crowding, Class II, Class III, crossbite and openbite.	Presence of malocclusions (p= 0.000): No malocclusion ASD 24.0% (12) vs 49.5% (46) control. Malocclusion: ASD 76.0% (39) vs 50.5% (47) control. Class I crowding: ASD 40.0% (20) vs 8.60% (8) control. Class II: ASD 10.0% (5) vs 12.9% (12) control. Class III: ASD 6.00% (3) vs 8.60% (8) control. Openbite: ASD 18.0% (9) vs 4.30% (4) control. Crossbite: ASD 2.00% (1) vs 8.60% (8) control.	Children with ASD had higher prevalence of malocclusion when compared to control.
Aljubour & Al-Sehaibany 2018 Saudi Arabia Cross sectional.	Saudi preschool children's private kindergartens and ASD centers in Riyadh, Saudi Arabia.	n:150 Gender: 109 (72.7%) M 41 (27.3%) F Mean age 4.9±0.6y. Age range: 3-6y.	n: 150 Gender: 109 (72.7%) M 41 (27.3%) F Mean age 4.6±0.4y.	(I): Occlusal evaluation performed on children with complete primary dentition and without any erupted permanent teeth or extensive caries that would affect the mésio-distal or occluso-gingival dimension.	Age and gender.	Prevalence of primary molar relationship; primary canine relationship; overbite; overjet; posterior crossbite and scissors bite according Foster & Hamilton 1969.	Molar relationship (p= 0.001): Flush Terminal: ASD 35.3% (53) vs 83.3% (125) control. Distal step: ASD 51.3% (77) vs 4.0% (6) control. Mesial step: ASD 13.4% (20) vs 12.7% (19) control. Canine relationship (p= 0.001): Class I: ASD 39.3% (59) vs 77.3% (116) control. Class II: ASD 56.7% (85) vs 15.4% (23) control. Class III: ASD 4.0% (6) vs 7.3% (11) control. Overbite (p= 0.001): Normal: ASD 28.7% (43) vs 52.0% (78) control. Increased: ASD 45.3% (68) vs 16.7% (25) control.	Prevalence of malocclusion was higher in children with ASD than control. Most children with ASD exhibited a distal step molar relationship, a class II canine relationship, and increased overjet and overbite. Most of the control children exhibited a flush terminal molar

							Edge-to-edge: ASD 3.3% (5) vs 20.6% (31) control. Anterior open bite: ASD 22.7% (34) vs 10.7% (16) control. Overjet (p= 0.001): Normal: ASD 36.6% (54) vs 74.0% (111) control. Increased: ASD 59.3% (90) vs 22.7% (34) control. Reversed:ASD 4.1% (6) vs 3.3% (5) control. Posterior crossbite (p=0.012): ASD 13.3% (20) vs 6.0% (9) control. Unilateral posterior crossbite: ASD 85% (127) vs 77.7% (117) control. Unilateral scissors bite: ASD 0.67% (1) vs 0% (0) control.	relationship, a class I canine relationship, normal overbite and overjet. The prevalence of posterior crossbite was significantly higher in the ASD than the control.
Onol & Kırzıoğlu 2018 Turkey Cross-sectional.	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Prevalence of malocclusions, occlusal disharmonies.	Prevalence of malocclusions (p=0.05): Class I: ASD 69.8% (44) vs 86.5% (96) control. Class II division I:ASD 20.6% (13) vs 8.1% (9) control. Class II division II: ASD 4.8% (3) vs 3.6% (4) control. Class III: ASD 4.8% (3) vs 1.8% (2) control. Prevalence of Occlusal disharmonies (p=0.013): Crowded teeth: ASD 22.2% (14) vs 9.9% (11) control. Cross bite: ASD - vs 3.0% (3) control. Open bite: ASD 1.6% (1) vs 1.8% (2) control. Deep bite: ASD - vs 5.4% (6) control.	ASD children had significantly more occlusal disharmonies than control.

							High arch palate: ASD 6.3% (4) vs 0.9% (1) control.	
Fontaine-Sylvestre et al. 2017 Canada Cross-sectional retrospective.	Division of Dentistry of the Montreal Children's Hospital in Canada.	n: 99 Gender: 78 (78.8%) M 21 (21.2%) F Mean age 11.04±3.71y. Socioeconomic status: 1 (high): 11.3% (8) 2: 12.7% (9) 3: 28.2% (20) 4: 33.8% (24) 5 (low): 14.1% (10) Medical diagnosis.	n: 101 Gender: 83 (82.2%) M 18 (17.8%) F Mean age 10.96±3.77y. Socioeconomic status: 1 (high): 12.3% (9) 2: 6.8% (5) 3: 21.9% (16) 4: 45.2% (33) 5 (low): 13.7% (10)	(I) ASD: confirmed medical diagnosis of ASD, healthy children, mixed or permanent dentition. (I) Control**:** Healthy children, mixed or permanent dentition. (E) ASD/Control: children having another disorder or syndrome than ASD; previous orthodontic treatment. Incomplete files with respect to the child's diagnosis and illegible information.	Demogra-phic features.	Prevalence of malocclusion, of Angle classification, midline deviation, crossbite, open bite, overbite, overjet and crowding.	Prevalence of malocclusion (p= 0.002): ASD 58.6% (58) vs 35.6% (36) control. Logistic regression model for the prevalence of malocclusion in ASD children and control (p=0.001): ASD OR 2.60 (IC95% 1.46-4.65). Class I: ASD 42.5% (37) vs 56% (51) control. Class II: ASD 42.5% (37) vs 33% (30) control. Class III: ASD 14.9% (13) vs 11% (10) control. Midline deviation: < 4 mm: ASD 61.1% (55) vs 68.3% (69) control. > 4 mm: ASD 38.9% (35) vs 31.7% (32) control. Crossbite (p= 0.03): Anterior: ASD 8.1% (8) vs 5.9% (6) control. Posterior**:** ASD 13.1% (13) vs 4.9% (5) control. Open bite: Anterior**:** ASD 8.1% (8) vs 3.9% (4) control. Posterior: ASD 3% (3) vs – control. Overbite Normal: ASD 77% (67) vs 79% (79) control. Increased (> 65%): ASD 13.8% (12) vs 15% (15) control. Decreased (< = 0%): ASD 9.2% (8) vs 6% (6) control. Overjet (p<0.0001):	ASD children had a significantly higher prevalence of malocclusion compared with the control. ASD were significantly more likely to have posterior crossbite, increased overjet, and severe maxillary crowding and also to have malocclusion when compared to control, independently of their demographic characteristics.

							Normal: ASD 54.4% (49) vs 85.1% (86) control. Increased (> 4 mm): ASD 38.9% (35) vs 10.9% (11) control. Decreased (< 1 mm): ASD 6.7% (6) vs 4% (4) control. Maxillary crowding (p= 0.006): Minimal: ASD 29.3% (29) vs 19.8% (20) control. Moderate: ASD 4% (4) vs 16.8% (17) control. Severe: ASD 5.1% (5) vs 1% (1) control. Mandibular crowding Minimal: ASD 36.4% (36) vs 22.8% (23) control. Moderate: ASD 8.1% (8) vs 14.9% (15) control. Severe: ASD 4% (4) vs 3% (3) control.	
Du et al. 2015 China Case control.	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Prevalence of malocclusion (deep over-bite, anterior open-bite, increased over-jet, anterior cross-bite and posterior cross-bite).	Deep over-bite (p=0.172): ASD 37.0% (95) vs 31.1% (80) control. Anterior open-bite (p=0.30): ASD 2.3% (6) vs 3.9% (10) control. Increased over-jet (p=0.245): ASD 18.7% (48) vs 14.8% (38) control. Anterior cross-bite (p=0.343): ASD 14.0% (36) vs 10.9% (28) control. Posterior cross-bite (p=0.316): ASD 0% (0) vs 0.4% (1) control.	Children with ASD and control had similar prevalence of malocclusion.
Orellana et al. 2012 Spain Case-control.	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Prevalence of malocclusions (ogival palate, dental crowding, anterior open bite).	Ogival palate (p= 0.021): ASD 33.33% (10) vs 6.67% (2) control. Dental crowding: ASD 46.67% (14) vs 70% (21) control. Anterior open bite (p= 0.002): ASD 30% (9) vs 0% (0) control.	In the ASD group a significantly greater presence of ogival palate and anterior open bite was

								recorded than control.
Luppanapornlarp et al. 2010 Thailand Cross-sectional.	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Vide Table 2	Prevalence of malocclusions according Dental Aesthetic Index (DAI).	Crowding (1 to 2 incisal segments): ASD 59.4% (19) vs 83.3% (40) control. Spacing (1 to 2 incisal segments): ASD 43.8% (14) vs 33.3% (16) control. Midline diastema (≥ 1 mm): ASD 31.3% (10) vs 20.8% (10) control. Anterior maxillary irregularity (≥ 1 mm: ASD 40.6% (13) vs 62.5% (30) control. Anterior mandibular irregularity (≥ 1 mm): ASD 31.3% (10) vs 60.4% (29) control. Anterior maxillary overjet (≥ 3 mm): ASD 56.3% (18) vs 70.8% (34) control. Anterior mandibular overjet (≥ 0 mm): ASD 18.8% (6) vs 2.1% (1) control. Open bite (≥ 0 mm): ASD 6.3% (2) vs 0.0% (0) control. Molar relationship (≥ 1⁄2 cusp): ASD 62.5% (20) vs 52.1% (25) control. Prevalence of malocclusions according DAI scores: ≤ 25: ASD 37.5% (12) vs 29.0% (14) control. 26–30: ASD 25.0% (8) vs 29.0% (14) control. 31-35: ASD 22.0% (7) vs 27.0% (13) control. ≥ 36: ASD 15.5% (5) vs 15.0% (7) control. OR 0.69 (95% CI 0.27 - 1.77).	The DAI was similar in the ASD and control. However, the prevalence of missing teeth, spacing, reverse overjet, open bites, and Class II molar relationship tendencies were higher in children with ASD.

DAI=Dental Aesthetic Index
*= represents statistical significance

Table 4. *Characteristics of included studies and comparison of bruxism between ASD and controls (n=13).*

Author Country Study design	**Methodological characteristics**	**Outcomes measures** Tools Full-mouth/partial	**Results** ASD group vs control group	**Main findings**
Bagattoni et al. 2021 Italia Cross-sectional.	Vide Table 2.	Prevalence of developmental defects of enamel according the modified index of developmental defects of enamel (FDI, 1992).	Prevalence of developmental defects of enamel (hypomineralisation) (p= 0.232): ASD 13% (8) vs 19% (12) control.	There was not signifficance difference in prevalence of developmental defects of enamel (hypomineralisation) between the groups.
Kuter & Uzel 2021 Turkey Retrospective study.	Vide Table 2.	Prevalence of bruxism and tooth wear.	Prevalence of Tooth wear: ASD boys 28.9% vs 0.0% control boys (p=0.001). ASD girls 39.1% vs 9.5% control girls (p=0.001). Prevalence of Bruxism (p=0.017): ASD girls 43.5% vs 18.9% control girls.	ASD girls and boys had higher prevalence of tooth wear and ASD girls had higher prevalence of bruxism than controls.
Daneshvar et al. 2019 Iran Cross-sectional.	Vide Table 2	Prevalence of bruxism that was recorded based on the observation of wear facets as an indication of enamel and dentin attrition.	Bruxism prevalence (p=0.001):ASD 56.4% (31) vs 3.6% (6) control.	Prevalence of bruxism in ASD was significantly higher than that in the control.
Kuter & Guler 2019 Turkey Cross-sectional.	Vide Table 2	Prevalence of bruxism and tooth wear.	Bruxism prevalence (p<0.05): ASD 33% (94) vs 19.6% (24) control. Tooth wear (p<0.05): ASD 31.1% (89) vs 6.9% (8) control.	ASD had higher statistically prevalence of bruxism and tooth wear when compared to control.
Leiva-García et al. 2019 Spain Cross sectional.	Vide Table 2	Prevalence of bruxism.	Bruxism (p=0.000): ASD 58.0% (29) vs 20.4% (19) control.	ASD had higher prevalence of bruxism when compared to control.
Suhaib et al. 2019 Pakistan Cross-sectional.	Vide Table 2	Prevalence of signs of bruxism.	Prevalence of signs of bruxism: ASD 10.3% (6) vs 0% (0) control.	Children with ASD had more signs of bruxism than control.
Onol & Kırzıoğlu 2018 Turkey Cross-sectional.	Vide Table 2	Prevalence of bruxism at nighttime, daytime and all day. Relationship between initiation age of special education and bruxism findings. Relationship between medication usage and bruxism.	Bruxism prevalence (p=0.000): Nighttime: ASD 4.8% (3) vs 1.8% (2) control. Daytime: ASD 22.2% (14) vs 3.6% (4) control. All day: ASD 14.3% (9) vs -control. ASD, who started special education before the age of 3 had lower bruxism prevalence when compared those started the age of 3 (6.4% vs 25.4%) (p=0.012) No significant association between ASD medication and bruxism was observed (p=0.064).	ASD children had significantly more cases of bruxism than control.

Bhandary & Nary 2017 India Cross- sectional.	Vide Table 2	Prevalence of dental erosion.	ASD 6.66% (2) vs 10% (3) control.	Children with ASD had lower dental erosion than controls.
Sarnat et al. 2016 Israel Cross-sectional.	Vide Table 2	Prevalence of enamel defects.	ASD 0.0% (0) vs 43% (19) control ($p < 0.001$).	Enamel defects was lower significantly in ASD than control.
Du et al. 2015 China Case control.	Vide Table 2	Prevalence of teeth with tooth wear (Smith & Knight 1984).	ASD 54.1% (139) vs 48.2% (124) control.	ASD and control had similar prevalence of tooth wear.
El Khatib et al. 2014 Egypt Case control.	Vide Table 2	Prevalence of bruxism/tooth wear (observation of wear facets as an indication of enamel and dentin attrition, and intraoral signs self-injurious behaviors.	($p<0.0001$): No attrition: ASD 62% (62) vs 92% (92) control. Enamel attrition: ASD 38% (38) vs 8% (8) control.	Bruxism was significantly more practiced by children with ASD than control.
Orellana et al. 2012 Spain Case-control.	Vide Table 2	Prevalence of bruxism.	ASD 60% (18) vs 43.33% (13) control.	ASD had higher prevalence of bruxism than controls.
Fahlvik-Planefeldt & Herrström 2001 Sweden Case control.	Vide Table 2	Prevalence of pronounced teeth grinding facets in two or several teeth.	ASD 20% (4) vs 35% (7) control.	ASD had a higher prevalence of pronounced teeth grinding facets than control.

Table 5. *Characteristics of included studies and comparison of salivary changes between ASD and controls (n=9).*

Author **Country** **Study design**	**Methodological characteristics**	**Outcomes measures** Tools Full-mouth/partial	**Results** ASD group vs control group	**Main findings**
Kuter & Uzel 2021 Turkey Retrospective study.	Vide Table 2	Prevalence of drooling of saliva.	ASD boys 21.7% vs 3.6% control (p=0.028). ASD girls 43.5% vs 9.5% control (p=0.000).	ASD boys and girls had higher prevalence of drooling of saliva than control.
Kuter & Guler 2019 Turkey Cross-sectional.	Vide Table 2	Prevalence of drooling of saliva.	ASD 26.4% (75) vs 7.8% (10) contro (p<0.05).	Children with ASD was higher drooling of saliva than control.
Morales-Chávez et al. 2019 Venezuela Cross-sectional.	Vide Table 2	Mean values of salivary pH, and salivary levels of phosphate and calcium.	pH: ASD 7.17±0.45 vs 7.27±0.28 control. Decreased calcium levels (p=0.013) were observed in autistics (0.62±0.35 mmol/L) compared to controls (0.89±0.51 mmol/L), but phosphate levels were similar (6.17±4.22 M, 5.51±4.86 M, respectively).	No difference was observed between ASD and controls regard salivary pH or phposphate levels. ASD presented lowerecalcium levels than control.
Onol & Kırzıoğlu 2018 Turkey Cross-sectional.	Vide Table 2	Dryness of the mouth.	Relationship between medication usage and dryness of the mouth in children with ASD was not statistically significant (p=0.285).	There was no significant difference between ASD and control in terms of abnormal swallowing habits and dryness of the mouth. The relationship between medication usage and dryness of the mouth in ASD was not statistically significant.
Bhandary & Nary/ 2017/ India/ Cross- sectional.	Vide Table 2	Mean±SD of: unstimulated salivary pH (pH indicator strips); salivary flow rate; salivary buffering capacity (Ericsson's Method/1959).	pH (p=0.000): ASD 6.49±0.58 vs 7.08±0.62 control. Buffering capacity (p=0.000):ASD 4.28±0.27 vs 4.56±0.27control. Flow rate (ml/min): ASD 0.8±0.35 vs 0.78±0.47 control.	Children with ASD had salivary pH and buffering capacity lower than control.
Diab et al. 2016 Saudi Arabia Case-control.	Vide Table 2	Mean±SD of salivary ph (unstimulated saliva). Prevalence of	Salivary ph (p=0.021): ASD 6.85±0.55 vs 7.08±0.43 control. Prevalence of Salivary buffering capacity (p=0.544): Very Low: ASD 10.0% (5) vs 6.0% (3) control. Low: ASD 30.0% (15) vs 24.0% (12) control. Normal: ASD 60.0% (30) vs 70.0% (35) control.	Salivary pH was lower significantly in ASD than controls. No statistically significant difference of the salivary buffering capacity among groups.

		salivary buffer capacity of stimulated saliva.		
Blomqvist et al. 2015 Sweden Cross-sectional.	Vide Table 2	Mean±SD of salivary secretion rate.	Stimulated salivary secretion rate (p<0.001): ASD (n=43) 1.46±0.72 vs 2.74±1.49 (n=55) control. There was no correlation in the ASD group between number of medications and salivary secretion rate (p=0.564). Salivary secretion rate between ASD using medication associated with hyposalivation (n=21) 1.34±0.63 vs ASD without medications associated with hyposalivation (n=22) 1.58±0.80; (p=0.297). Un-medicated participants with ASD (n=16) had a lower saliva secretion rate than in the control group (n=51) (1.5±0.7 and 2.7±1.5, p=0.048).	The stimulated saliva secretion was lower in the ASD group, regardless of medication. Adults with ASD had lower saliva flow compared to healthy controls. Futhermore, un-medicated participants with ASD had a lower saliva secretion rate than in the control.
Rai et al. 2012 India Cross-sectional.	Vide Table 2	Unstimulated whole saliva, salivary pH (pH indicating paper), salivary antioxidant levels.	Median value of salivary pH was of 7 for both groups. Total salivary antioxidant levels, in ASD (mean of 8.14 mg/ml) was low compared to their siblings (mean of 43.31 mg/ml). Correlations between caries and salivary antioxidant levels were not statistically significant.	Salivary pH did not differ statistically in children with ASD and their siblings. The salivary antioxidant levels were lower in ASD when compared to the control.
Bassoukou et al. 2009 Brazil Cross-sectional.	Vide Table 2	Mean±SD of unstimulated saliva flow rate, of pH, of buffer capacity (ml acid/ml saliva).	No significant difference in flow rate, pH, buffer capacity of whole saliva was verified between ASD and Controls in ages 3–8 y. Aged 3-8y (ASD n=10, Control n=14): Flow rate (p=0.64): ASD 0.74±0.35 vs 0.67±0.36 control. pH (p=0.54): ASD 7.69±0.40 vs 7.79±0.38 control. Buffer capacity (p=0.20): ASD 0.45±0.26 vs 0.64±0.40. No significant difference in flow rate was verified between ASD and Controls in ages 9–13 y. pH and buffer capacity in the range pHi–pH 7.0, were lower in ASD compared to the controls. Aged 9–13 y (ASD n=15, Control n=11): Flow rate (p=0.32): ASD 0.74±0.51 vs 0.92±0.33 control. pH (p=0.007): ASD 7.53±0.44 vs 7.97±0.28 control. Buffer capacity (p=0.001): ASD 0.35±0.25 vs 0.74±0.29 control. No statistical difference was observed for any parameter between autistic children using or not using medication, independently of the age range.	When comparing ASD children 3-8y and control 4-8y, groups did not differ in flow rate, pH levels and buffer capacity. Patients with ASD 9-13y had lower scores significantly in pH than controls 9-14y, pH and pHi=7.0 and buffer capacity. In ASD individuals aged 3–8 and 9–13, medicated or not, there was no significant statistical difference in flow rate, pH, and buffer capacity. Individuals with ASD had neither a higher flow rate nor a better buffer capacity.

	Aged 3-8y (ASD medication users n=6 vs ASD medication nonusers n=4): Flow rate: users 0.86±0.37 vs 0.56±0.26 nonusers. pH: users 7.67±0.36 vs 7.73±0.51 nonusers Buffer capacity: users 0.56±0.22 vs 0.28±0.24 nonusers. Aged 9-13 y (ASD medication users n=11 vs ASD medication nonusers n=4): Flow rate: users 0.69±0.42 vs 0.89±0.76 nonusers. pH: users 7.50±0.51 vs 7.60±0.15 nonusers. Buffer capacity: users 0.34±0.24 vs 0.37±0.33 nonusers

Table 6. *Subgroup analysis of oral hygiene status prevalence.*

		Number of studies	Pooled estimate (95% CI)	I^2 (%)	p	Tau^2
Excellent/good/fair		9	0.79 (0.64,0.96)	88	<0.01	0.065
Poor/very poor		9	2.46 (1.23,4.91)	83	<0.01	0.733
Excellent/good/fair	OHI-S	5	0.76 (0.52,1.11)	91	<0.01	0.146
Index	PlI	2	0.84 (0.71,0.99)	61	0.11	0.008
Poor/very poor	OHI-S	5	1.80 (0.76,4.22)	85	<0.01	0.761
index	PlI	2	7.50 (2.63,21.43)	0	0.94	0.00
Excellent/good/fair Bias risk	High	3	0.90 (0.50,1.62)	81	<0.01	0.187
	Moderate	5	0.67 (0.48,0.94)	92	<0.01	0.506
	Low	1	1.01 (0.92,1.11)	-	-	
Poor/very poor Bias risk	High	2	1.71 (0.35,8.26)	91	<0.01	1.181
	Moderate	5	3.45 (2.27,5.25)	8	0.36	0.019
	Low	1	0.88 (0.33,2.36)	-	-	
Excellent/good/fair	0	3	1.02 (0.75,1.40)	73	0.02	0.047
Matching variables N	1,2	3	0.90 (0.75,1.09)	76	0.02	0.019
	3+	3	0.47 (0.34,0.67)	48	0.15	0.043
Poor/very poor	0	2	2.39 (0.14,40.18)	87	<0.01	3.65
Matching variables N	1,2	3	2.14 (0.60,7.71)	72	0.03	0.916
	3+	3	3.26 (2.20,4.84)	0	0.64	0.00

Table 7. *Subgroup analysis of mean scores of dental plaque.*

		Number of studies	Pooled estimate (95% CI)	I^2 (%)	p	Tau^2
	All studies	15	0.59 (0.24,0.94)	96	<0.01	0.450
Index	OHI-S	6	0.65 (-0.05,1.35)	96	<0.01	0.729
	PlI	10	0.59 (0.14,1.04)	95	<0.01	0.494
SDI	High/high-middle	6	0.68 (0.16,1.19)	93	<0.01	0.377
	Middle	3	0.07 (-0.91,1.05)	97	<0.01	0.717
	Low/low-middle	6	0.77 (0.16,1.37)	96	<0.01	0.538
Bias risk	High	3	0.72 (0.03,1.41)	91	<0.01	0.337
	Moderate	8	0.68 (0.17,1.19)	95	<0.01	0.506
	Low	4	0.32 (-0.50,1.14)	97	<0.01	0.674
Matching variables N	0	3	1.53 (0.78,2.28)	93	<0.01	0.405
	1,2	9	0.28 (-0.09,0.64)	93	<0.01	0.280
	3+	3	0.57 (0.26,0.87)	63	0.07	0.044

Table 8. *Subgroups analysis of gingivitis prevalence.*

		Number of studies	RR (95% CI)	I^2 (%)	p	Tau^2
All studies		11	1.31 (1.02,1.70)	89	<001	0.139
Extent gingivitis	Localized	2	0.88 (0.56,1.38)	44	0.18	0.050
	Generalized	2	5.63 (2.03,15.64)	63	0.10	0.343
Index	GI	3	1.45 (0.70,2.99)	97	<0.01	0.394
	CPI/CPITN	4	1.63 (0.59,4.54)	94	<0.01	1.006
	VPI	1	0.99 (0.86,1.15)			
	BoP	1	1.12 (0.55,2.32)			
	NR	2	1.25 (0.54,2.92)	81	0.02	0.308
SDI	High/high-middle	6	1.35 (0.92,1.99)	86	<0.01	0.182
	Middle	2	2.54 (0.59,10.90)	93	<0.01	1.028
	Low/low-middle	3	0.33 (0.00,0.66)	95	<0.01	0.406
Bias risk	High	2	1.06 (0.72,1.58)	73	0.05	0.059
	Moderate	8	1.54 (0.97,2.45)	93	<0.01	0.377
	Low	1	0.99 (0.86,1.15)			
Matching variables N	0	1	1.30 (0.98,1.72)			
	1,2	8	1.13 (0.88,1,47)	86	<0.01	0.091
	3+	2	2.22 (1.69,2.91)	0	0.37	0.00

Table 9. *Subgroup analysis of mean scores of gingivitis.*

		Number of studies	Pooled estimate (95% CI)	I^2 (%)	p	Tau^2
	All studies	9	0.45 (0.02,0.88)	95	<0.01	0.403
Index	GI	7	0.50 (-0.88,1.08)	96	<0.01	0.579
	VPI	1	-0.03 (-0.33,0.27)			
	MGI	1	0.61 (0.21,1.01)			
Exam protocol	NR	7	0.57 (0.14,1.00)	90	<0.01	0.294
	Partial	2	0.04 (-1.05,1.13)	96	<0.01	0.592
SDI	High/high-middle	3	0.65 (-0.20,1.51)	95	<0.01	0.536
	Middle	4	0.35 (-0.55,1.26)	96	<0.01	0.796
	Low/low-middle	2	0.33 (0.00,0.66)	54	<0.01	0.032
Bias risk	High	2	0.59 (-0.99,2.18)	95	<0.01	1.243
	Moderate	3	0.66 (0.05,1.27)	87	<0.01	0.243
	Low	4	0.21 (-0.45,0.86)	96	<0.01	0.422
Matching variables N	0	1	-0.24 (-0.86,0.38)			
	1,2	7	0.49 (-0.05, 1.03)	95%	<0.01	0.497
	3+	1	0.78 (0.49,1.07)			

Table 10. *Subgroup analysis of prevalence of overjet.*

		Number of studies	Pooled estimate (95% CI)	I^2 (%)	p	Tau^2
	All studies	6	2.16 (1.28,3.64)	89	<0.01	0.355
SDI	High/high-middle	3	1.98 (0.84,4.67)	94	<0.01	0.529
	Middle	3	2.41 (1.12,5.21)	85	<0.01	0.366
Bias risk	High	1	3.25 (1.75,6.02)			
	Moderate	4	2.30 (1.09,4.86)	93	<0.01	0.506
	Low	1	1.26 (0.86,1.86)			
Matching variables N	0	1	0.96 (0.72,1.28)			
	1,2	4	2.38 (1.43,3.98)	75	<0.01	0.189
	3+	1	3.25 (2.18,4.86)			

Table 11. *Subgroup analysis of prevalence of overbite.*

		Number of studies	Pooled estimate (95% CI)	I^2 (%)	p	Tau^2
	All studies	8	1.62 (1.02,2.59)	80	<0.01	0.301
SDI	High/high-middle	5	1.39 (0.66,2.91)	75	<0.01	0.465
	Middle	3	1.87 (0.85,4.15)	89	<0.01	0.420
Bias risk	High	2	0.55 (0.12,2.51)	36	0.21	0.624
	Moderate	5	2.27 (1.46,3.54)	59	0.04	0.141
	Low	1	1.19 (0.93,1.51)			
Matching variables N	0	3	1.14 (0.32,4.09)	69	0.04	0.706
	1,2	4	1.46 (0.86,2.48)	80	<0.01	0.213
	3+	1	3.58 (2.24,5.72)			

Table 12. *Subgroups analysis of openbite prevalence.*

		Number of studies	Pooled estimate (95% CI)	I^2 (%)	p	Tau^2
	All studies	10	2.37 (1.46,3.85)	54	0.02	0.295
SDI	High/high-middle	8	2.30 (1.64,3.25)	3	0.41	0.007
	Middle	2	2.02 (0.19,22.04)	92	<0.01	2.732
Bias risk	High	2	2.28 (0.83,6.24)	0	0.39	0.00
	Moderate	7	2.89 (1.76,4.75)	47	0.08	0.192
	Low	1	0.60 (0.22,1.63)			
Matching variables N	0	3	2.48 (1.35,4.54)	0	0.50	0.00
	1,2	4	1.92 (1.02,3.62)	56	0.04	0.321
	3+	1	6.71 (2.68,16.80)			

Table 13. *Subgroups analysis of bruxism prevalence.*

		Number of studies	Pooled estimate (95% CI)	I^2 (%)	p	Tau^2
	All studies	6	4.52 (2.07,9.86)	85	<0.01	0.721
SDI	High/high-middle	3	2.92 (1.23,6.93)	85	<0.01	0.487
	Middle	2	8.42 (2.64,26.87)	78	0.03	0.547
	Low/low-middle	1	6.11 (0.36,104.6)			
Bias risk	High	2	10.92 (5.46,21.86)	29	0.23	0.073
	Moderate	3	2.08 (1.10,3.94)	58	0.09	0.160
	Low	1	4.75 (2.33,9.66)			
Matching variables N	0	2	10.92 (5.46,21.86)	29	0.23	0.073
	1,2	3	2.08 (1.10,3.94)	58	0.09	0.160
	3+	1	4.75 (2.33,9.66)			

Appendix 1

Search strategies used in the respective electronic databases

Database	Search strategy	Hits
Pubmed/MEDLINE (https://www.ncbi.nlm.nih.gov/pubmed)	((((((((((((((((((((("Autistic Disorder"[MeSH Terms]) OR ("Autism Spectrum Disorder"[MeSH Terms])) OR ("Asperger Syndrome"[MeSH Terms])) OR ("Kanner's Syndrome")) OR ("Kanner Syndrome")) OR ("Kanners Syndrome")) OR ("Infantile Autism")) OR (Autism)) OR ("Early Infantile Autism")) OR ("Autism Spectrum Disorders")) OR ("Asperger's Disease")) OR ("Asperger's Diseases")) OR ("Aspergers Disease")) OR ("Asperger Disease")) OR ("Asperger Diseases")) OR ("Asperger Disorder")) OR ("Asperger Disorders")) OR ("Asperger's Disorder")) OR ("Asperger's Syndrome")) OR ("Aspergers Disorder")) OR ("Aspergers Syndrome")) AND ((("Tooth loss"[MeSH Terms]) OR ("Oral Health"[MeSH Terms])) OR ("Oral Manifestations"[MeSH Terms])) OR ("Dental Caries"[MeSH Terms])) OR ("Oral Hygiene"[MeSH Terms])) OR ("Oral Hygiene Index"[MeSH Terms])) OR ("Dental Plaque"[MeSH Terms])) OR ("Dental Plaque Index"[MeSH Terms])) OR ("Periodontal Diseases"[MeSH Terms])) OR ("Gingivitis"[MeSH Terms])) OR ("Gingival Diseases"[MeSH Terms])) OR ("Periodontitis"[MeSH Terms])) OR ("Chronic Periodontitis"[MeSH Terms])) OR ("Aggressive Periodontitis"[MeSH Terms])) OR ("Gingival Hemorrhage"[MeSH Terms])) OR ("Periodontal Index"[MeSH Terms])) OR ("Alveolar Bone Loss"[MeSH Terms])) OR ("Malocclusion"[MeSH Terms])) OR ("Malocclusion, Angle Class III"[MeSH Terms])) OR ("Malocclusion, Angle Class II"[MeSH Terms])) OR ("Malocclusion, Angle Class I"[MeSH Terms])) OR ("Bruxism"[MeSH Terms])) OR ("Sleep Bruxism"[MeSH Terms])) OR ("Xerostomia"[MeSH Terms])) OR ("saliva"[MeSH Terms])) OR ("salivation"[MeSH Terms])) OR ("Oral Manifestation")) OR ("Dental Decay")) OR ("Carious Dentin")) OR ("Dental White Spot")) OR ("White Spot")) OR ("Dental White Spots")) OR ("Dental Hygiene")) OR ("Oral Hygiene Indices")) OR ("Oral Hygiene Indexe")) OR ("Dental Plaque Indexes")) OR ("Dental Plaque Indices")) OR ("Periodontal Disease")) OR ("Parodontosis")) OR ("Parodontoses")) OR ("Pyorrhea Alveolaris")) OR ("Gingival Disease")) OR ("Gingivosis")) OR ("Gingivoses")) OR ("Pericementitis")) OR ("Adult Periodontitis")) OR ("Prepubertal Periodontitis")) OR ("Early-Onset Periodontitis")) OR ("Early Onset Periodontitis")) OR ("Juvenile Periodontitis")) OR ("Periodontal Indices")) OR ("Periodontal Indexes")) OR ("Community Periodontal Index of Treatment Needs")) OR ("CPITN")) OR ("DMFT")) OR ("Gingival Bleeding on Probing")) OR ("Gingival Index")) OR ("Gingival Indices")) OR ("Gingival Indexes")) OR ("Alveolar Resorption")) OR ("Alveolar Resorptions")) OR ("Periodontal Bone Loss")) OR ("Periodontal Resorption")) OR ("Periodontal Resorptions")) OR (Malocclusions)) OR ("Tooth Crowding")) OR (Crossbite)) OR (Crossbites)) OR ("Cross Bite")) OR ("Angle's Classification")) OR ("Angle Classification")) OR ("Teeth Grinding Disorder")) OR ("Teeth Grinding Disorders")) OR ("Sleep Bruxisms")) OR ("Nocturnal Teeth Grinding Disorder")) OR ("Nocturnal Bruxism")) OR ("Nocturnal Bruxisms")) OR ("Childhood Sleep Bruxism")) OR ("Sleep-Related Bruxism")) OR ("Sleep Related Bruxism")) OR ("Adult Sleep Bruxism")) OR (Hyposalivation)) OR (Hyposalivations)) OR ("Mouth Dryness")) OR (Sialorrhea)) OR ("white-spot lesion")) OR ("tooth decay")) OR ("carious tooth")) OR ("carious lesions")) OR ("probing depth")) OR ("clinical attachment")) OR ("bleeding on probing")) OR ("gingival bleeding")) OR ("Gum diseases")) OR ("salivary flow")) OR ("salivary flow rate")) OR ("salivary flow rates")) OR ("salivary pH"))	369
Embase (https://www.embase.com)	('autism'/exp OR 'pdd (pervasive developmental disorder)' OR 'autism' OR 'autism spectrum disorder' OR 'autism, early infantile' OR 'autism, infantile' OR 'autistic child' OR 'autistic children' OR 'autistic spectrum disorder' OR 'child development disorders, pervasive' OR 'childhood autism' OR 'classical autism' OR 'infantile autism, early' OR 'pervasive child development disorders' OR 'pervasive developmental disorder' OR 'pervasive developmental disorders' OR 'typical autism' OR 'asperger syndrome'/exp OR 'asperger syndrome' OR 'asperger`s syndrome' OR 'aspergers syndrome' OR 'high functioning autism' OR 'autism-spectrum quotient'/exp OR 'autism quotient' OR 'autism-spectrum quotient' OR 'rett syndrome'/exp OR 'rett syndrome' OR 'rett`s syndrome' OR 'retts syndrome' OR 'morbus rett' OR 'rett disease' OR asperger OR 'autistic disorder' OR 'infantile autism' OR 'early infantile autism' OR 'autism spectrum disorders'/exp OR 'asperger`s disease' OR 'asperger`s diseases' OR 'aspergers disease' OR 'asperger disease' OR 'asperger disorder' OR 'asperger disorders' OR 'asperger`s disorder' OR 'aspergers disorder' OR 'kanner syndrome') AND ('oral health status'/exp OR 'mouth disease'/exp OR 'diagnosis, oral' OR 'edentulous mouth' OR 'leukoedema, oral' OR 'leukooedema, oral' OR 'mouth abnormalities' OR 'mouth disease' OR 'mouth diseases' OR 'mouth submucous fibrosis' OR 'mouth, edentulous' OR 'oral diagnosis' OR 'oral disease'	1920

	OR 'oral leukoedema' OR 'oral leukooedema' OR 'oral manifestations' OR 'oral submucous fibrosis' OR 'stomatognathic diseases' OR 'oral manifestation' OR 'dental caries'/exp OR 'caries' OR 'caries, dental' OR 'cariogenesis' OR 'carious dentine' OR 'carious teeth' OR 'dental caries' OR 'dental caries susceptibility' OR 'dental fissure' OR 'dental fissures' OR 'fissure, tooth' OR 'root caries' OR 'tooth caries' OR 'tooth fissure' OR 'dental decay' OR 'dental white spot' OR 'dental white spots' OR 'mouth hygiene'/exp OR 'hygiene, mouth' OR 'hygiene, tooth' OR 'mouth care' OR 'mouth hygiene' OR 'mouth rinsing' OR 'mouth washing' OR 'mouthwashing' OR 'oral care' OR 'tooth hygiene' OR 'oral hygiene index'/exp OR 'oral hygiene index' OR 'oral hygiene indexes' OR 'oral hygiene indices' OR 'oral hygiene' OR 'dental hygiene' OR 'dental plaque' OR 'dental plaque index' OR 'periodontal disease'/exp OR 'dental loss' OR 'dental migration' OR 'dental mobility' OR 'edentulism' OR 'furcation defects' OR 'mesial movement of teeth' OR 'paradontal disease' OR 'paradontopathy' OR 'paraodontopathy' OR 'parodentopathy' OR 'parodontal disease' OR 'parodontium disease' OR 'parodontive tissue disease' OR 'peridontal disease' OR 'peridontal tissue disease' OR 'peridontium disease' OR 'periodontal atrophy' OR 'periodontal attachment loss' OR 'periodontal cyst' OR 'periodontal disease' OR 'periodontal diseases' OR 'periodontal infection' OR 'periodontium disease' OR 'periodontopathy' OR 'tooth loss' OR 'tooth migration' OR 'tooth mobility' OR 'tooth movement' OR 'gingivitis'/exp OR 'acute gingivitis' OR 'chronic gingivitis' OR 'crevicular fluid' OR 'fluid, gingiva crevice' OR 'gingiva crevice fluid' OR 'gingiva inflammation' OR 'gingiva pocket' OR 'gingival crevicular fluid' OR 'gingival inflammation' OR 'gingival pocket' OR 'gingivitis' OR 'gingivitis syndrome' OR 'gingival disease' OR 'periodontitis'/exp OR 'paradontitis' OR 'parodontitis' OR 'peridontitis' OR 'periodontitis' OR 'chronic periodontitis'/exp OR 'adult periodontitis' OR 'aggressive periodontitis'/exp OR 'aggressive periodontitis' OR 'early onset periodontitis' OR 'juvenile periodontitis' OR 'periodontitis, juvenile' OR 'prepubertal periodontitis' OR 'gingival hemorrhage' OR 'periodontal index'/exp OR 'russell periodontal index' OR 'russell`s periodontal index' OR 'periodontal index' OR 'periodontal indexes' OR 'community periodontal index of treatment needs'/exp OR 'community periodontal index of treatment needs' OR cpitn OR 'gingival bleeding on probing' OR 'gingival index'/exp OR 'loe and silness gingival index' OR 'loe-silness gingival index' OR 'gingival index' OR 'gingival indexes' OR 'alveolar bone loss'/exp OR 'alveolar bone loss' OR 'mandible alveolar bone loss' OR 'alveolar resorption' OR 'periodontal bone loss' OR 'periodontal resorption' OR 'alveolar bone atrophy' OR 'malocclusion'/exp OR 'angle class i malocclusion' OR 'angle class ii malocclusion' OR 'angle class iii malocclusion' OR 'dental malocclusion' OR 'jaw malocclusion' OR 'jaw occlusion disorder' OR 'malocclusion' OR 'malocclusion, angle class i' OR 'malocclusion, angle class ii' OR 'malocclusion, angle class iii' OR 'occlusion disorder, jaw' OR 'occlusion, mal' OR 'open bite' OR 'overbite' OR 'tooth malocclusion' OR malocclusions OR 'tooth crowding' OR 'crossbite'/exp OR 'cross bite' OR 'crossbite' OR 'bruxism'/exp OR 'bruxism' OR 'dental grinding' OR 'grinding, tooth' OR 'teeth clenching' OR 'teeth grinding' OR 'tooth clenching' OR 'tooth grinding' OR 'teeth grinding disorder' OR 'teeth grinding disorders' OR 'sleep bruxism'/exp OR 'night-time bruxism' OR 'nighttime bruxism' OR 'nocturnal teeth grinding' OR 'nocturnal tooth grinding' OR 'sleep bruxism' OR 'sleep teeth grinding' OR 'sleep tooth grinding' OR 'nocturnal teeth grinding disorder' OR 'nocturnal bruxism' OR 'childhood sleep bruxism' OR 'sleep-related bruxism' OR 'sleep related bruxism' OR 'adult sleep bruxism' OR 'xerostomia'/exp OR 'dry mouth' OR 'oral dryness' OR 'xerostomia' OR 'xerostomy' OR 'zerostomiasis' OR 'hyposalivation'/exp OR 'hyposalivation' OR 'hyposialia' OR 'salivation, hypo' OR hyposalivations OR 'mouth dryness' OR 'salivation'/exp OR 'saliva flow' OR 'saliva release' OR 'saliva secretion' OR 'salivary flow' OR 'salivary gland secretion' OR 'salivary secretion' OR 'salivation' OR sialorrhea OR dmft OR 'tooth decay' OR 'carious tooth' OR 'carious lesions' OR 'probing depth'/exp OR 'clinical attachment level'/exp OR 'clinical attachment loss'/exp OR 'bleeding on probing'/exp OR 'gingival bleeding' OR 'gum diseases' OR 'gum disease' OR 'gum inflammation' OR 'salivary flow rate'/exp OR 'salivary ph'/exp)	
Scopus (https://www.scopus.com)	(TITLE-ABS-KEY (* "Autistic Disorder" OR "Kanner's Syndrome" OR "Kanner Syndrome" OR "Kanners Syndrome" OR "Infantile Autism" OR autism OR "Early Infantile Autism" OR "Autism Spectrum Disorder" OR "Autism Spectrum Disorders" OR "Asperger Syndrome" OR "Asperger's Disease" OR "Asperger's Diseases" OR "Aspergers Disease" OR "Asperger Disease" OR "Asperger Diseases" OR "Asperger Disorder" OR "Asperger Disorders" OR "Asperger's Disorder" OR "Aspergers Disorder" OR "Asperger's Syndrome" OR "Aspergers Syndrome" OR "Syndrome, Asperger")) AND (TITLE-ABS-KEY (* "Tooth loss" OR "Oral Health" OR "Oral Manifestations" OR "Dental Caries" OR "Oral Hygiene" OR "Oral Hygiene Index" OR "Dental Plaque" OR "Dental Plaque Index" OR "Periodontal Diseases" OR "Gingivitis" OR "Gingival Diseases" OR "Periodontitis" OR "Chronic Periodontitis" OR "Aggressive Periodontitis" OR "Gingival Hemorrhage" OR "Periodontal Index" OR "Alveolar Bone Loss" OR "Malocclusion" OR "Bruxism" OR "Sleep Bruxism" OR "Xerostomia" OR "saliva" OR "salivation" OR "Oral Manifestation" OR "Dental Decay" OR "Dental White Spot" OR "Dental White Spots" OR "Dental Hygiene" OR "Periodontal Disease" OR "Pyorrhea Alveolaris" OR "Gingival Disease" OR "Adult Periodontitis" OR "Prepubertal Periodontitis" OR "Early-Onset Periodontitis" OR "Early Onset Periodontitis" OR "Juvenile Periodontitis" OR "Community Periodontal Index of Treatment Needs" OR "CPITN" OR "DMFT" OR "Gingival	885

	Bleeding on Probing" OR "Gingival Index" OR "Alveolar Resorption" OR "Periodontal Bone Loss" OR "Periodontal Resorption" OR "Periodontal Resorptions" OR malocclusions OR "Tooth Crowding" OR crossbite OR "Teeth Grinding Disorder" OR "Teeth Grinding Disorders" OR "Sleep Bruxisms" OR "Nocturnal Teeth Grinding Disorder" OR "Nocturnal Bruxism" OR "Childhood Sleep Bruxism" OR "Sleep-Related Bruxism" OR "Sleep Related Bruxism" OR "Adult Sleep Bruxism" OR hyposalivation OR "Mouth Dryness" OR sialorrhea OR "tooth decay" OR "carious lesions" OR "probing depth" OR "clinical attachment" OR "bleeding on probing" OR "gingival bleeding" OR "Gum diseases" OR "salivary flow" OR "salivary flow rate" OR "salivary flow rates" OR "salivary pH"))	
Web of Science (https://login.webofknowledge.com)	(TÓPICO:((((((((((((((((((((("Autistic Disorder" OR "Kanner's Syndrome") OR "Kanner Syndrome") OR "Kanners Syndrome") OR "Infantile Autism") OR Autism) OR "Early Infantile Autism") OR "Autism Spectrum Disorder") OR "Autism Spectrum Disorders") OR "Asperger Syndrome") OR "Asperger's Disease") OR "Asperger's Diseases") OR "Aspergers Disease") OR "Asperger Disease") OR "Asperger Diseases") OR "Asperger Disorder") OR "Asperger Disorders") OR "Asperger's Disorder") OR "Aspergers Disorder") OR "Asperger's Syndrome") OR "Aspergers Syndrome") OR "Syndrome, Asperger") AND TÓPICO:(((" periodontal disease" OR "periodontal diseases") OR "Aggressive Periodontitis") OR "Periodontitis") OR "early onset periodontitis") OR "early-onset periodontitis") OR "juvenile periodontitis") OR "chronic periodontitis") OR "adult periodontitis") OR "Periodontal Index") OR "periodontal indexes") OR "community periodontal index of treatment needs") OR cpitn) OR "gingival bleeding on probing") OR "gingival index") OR "gingival indexes") OR "gingivitis") OR "gingival diseases") OR "Alveolar Bone Loss") OR "alveolar process atrophy") OR "alveolar process atrophies") OR "alveolar resorption") OR "periodontal bone loss") OR "Malocclusion") OR "malocclusions") OR "tooth crowding") OR "crossbite") OR "crossbites") OR "angle classification") OR "angle s classification") OR "teeth grinding disorder") OR "Bruxism") OR "Sleep Bruxism") OR "nocturnal bruxism") OR "childhood sleep bruxism") OR "sleep related bruxism") OR "adult sleep bruxism") OR "Xerostomia") OR "xerostomia") OR "hyposalivation") OR "mouth dryness") OR "sialorrhoea") OR "Dental Caries") OR caries) OR "white spot lesion") OR "dental white spot lesion") OR "DFMT" "dental decay") OR "probing depth") OR "clinical attachment level") OR "bleeding on probing") OR "gingival hemorrhage") OR "gingival bleeding") OR "Gingival Diseases") OR "gum disease") OR "salivary flow rate") OR "hyposalivation") OR "tooth loss") OR "dental caries") OR "carious dentin") OR "dental plaque") OR "dental hygiene") OR "oral health") OR "oral manifestations") OR "oral hygiene") OR "oral hygiene index") OR "dental plaque index") OR "mouth dryness") OR "tooth decay") OR "gum disease") OR "gum diseases") OR "salivary flow") OR "salivary") OR "oral conditions") OR "oral status") OR "mouth"))	820
Lilacs (lilacs.bvsalud.org)	"Autistic Disorder" OR "Autism Spectrum Disorder" OR "Asperger Syndrome" OR "Kanner's Syndrome" OR "Kanner Syndrome" OR Autism OR "Autism Spectrum Disorders" AND "tooth loss" OR "oral health" OR "oral manifestation" OR "dental caries" OR "oral hygiene" OR "oral hygiene index" OR "Dental plaque" OR "periodontal diseases" OR "gingivitis" OR "periodontitis" OR "gingival diseases" OR "malocclusion" OR "bruxism" OR "xerostomia" OR "dental hygiene" OR "community periodontal index of treatment needs" OR "CPITN" OR "DMFT" OR "Gingival index" OR "gingival bleeding" OR "hypo salivation" OR "sialorrhea" OR "salivary flow" OR "salivary flow rate"	30
PsycInfo (https://www.apa.org/pubs/databases/psycinfo)	Any Field: "autism spectrum disorders" OR "Aspergers Syndrome" OR "Autism" OR "Autistic Children" OR "Autistic Psychopathy" OR "Early Infantile Autism" OR "Pervasive Developmental" OR “Kanner Syndrome” OR “Kanners Syndrome” OR “Asperger Disease” OR “Asperger Disorder” OR “Kanners Disorder” OR “Kanners Disease” OR “Asperger's Disease” AND Any Field: "oral health" OR "dental health" OR "BehavioOral Health Services" OR "Preventive Health Behavior" OR “primary health care” OR "saliva" OR "salivation" OR "Bruxism" OR “tooth loss” OR “dental caries” OR “oral hygiene” OR “oral hygiene index” OR “dental plaque” OR “dental plaque index” OR “ periodontal disease” OR “gingivitis” OR “gingival diseases” OR “periodontitis” OR “periodontal index” OR “alveolar bone loss” OR “gingival inflammation” OR “malocclusion” OR “bruxism” OR “salivary flow” OR “DMFT” OR “sleep bruxism” OR “nocturnal teeth grinding disorder” OR “nocturnal bruxism” OR “sleep related bruxism” OR “mouth dryness” OR “hyposalivation” OR “teeth grinding disorder” OR “tooth crowding” OR “gum disease” OR “gum inflammation” or “bleeding gum” or “gingival bleeding” OR “xerostomia”	544
Google Scholar (https://scholar.google.com.br/)	"autism spectrum disorder" OR "asperger syndrome" AND “Tooth loss” OR "Oral Health" OR "Dental Caries" OR "Oral Hygiene" OR "Periodontal diseases" OR "Malocclusion" OR “Bruxism” OR "Xerostomia" filetype:pdf	200

Proquest (https://www.proquest.com/)	("autism spectrum disorder" OR "asperger syndrome" OR "autism") AND ("Tooth loss" OR "Oral Health" OR "Dental Caries" OR "dental plaque" OR "Oral Hygiene" OR "Periodontal diseases" OR "gingivitis" OR "gingival bleeding" OR "periodontitis" OR "Malocclusion" OR "Bruxism" OR "Xerostomia" OR "hyposalivation")	595
Total		5363

Appendix 2

Characteristics of excluded studies through full text reading and reasons for exclusion (n=175)

	Author	Year	Reasons for exclusion
1	AbdAlgabbar, E. H., Abuaffan, A. H.	2015	1
2	Albanese, A. V., Dall'Oppio, L., Venditti, P.	1896	2
3	AlHumaid, J., Gaffar, B., AlYousef, Y., Alshuraim, F., Alhareky, M., El Tantawi, M.	2020	1
4	Alkhabuli, J. O. S., Essa, E. Z., Al-Zuhair, A. M., Jaber, A. A.	2019	1
5	Alkhadra, T.	2017	1
6	Altun, C., Guven, G., Akgun, O. M. Akkurt, M. D., Basak, F., Akbulut, E.	2010	1
7	Alvares, G. A.;, Mekertichian, K., Rose, F., Vidler, S., Whitehouse, A. J. O.	2022	6
8	Al-Yassiri, A. M. H., Abdul-Zahraa Mahdi, K.	2015	1
9	Al-Zaidi, R. R.	2021	2
10	Amrollahi, N., Amouchi, R.	2021	4
11	Ashour, N. A., Ashour, A. A., Basha, S.	2018	1
12	Awasthi, P., Peshwani, B., Tiwari, S., Thakur, R., Shashikiran, N., Singla, S.	2015	3
13	Azimi, S., Lima, F., Slack-Smith, L., Bourke, J., Calache, H., Junaid, M., Leonard, H.	2021	1
14	Bao, X. H., Pan, H., Song, F. Y., Wu, X. R.	2004	4
15	Baumgarten, A., Hilgert, J. B., Rech, R. S., Cunha-Cruz, J., Goulart, B. N. G.	2021	5
16	Bejarano, N. M. P., Ferreira Gaona, M. I., Reissner, C. V. D., Sanabria Vázquez, D. A.	2017	5
17	Bisgaard, A.-M., Schönewolf-Greulich, B., Ravn, K., Rønde, G.	2015	5
18	Bossù, M., Trottini, M., Corridore, D., Giorgio, G. D., Sfasciotti, G. L., Palaia, G., Ottolenghi, L., Polimeni, A., Carlo, S. D.	2020	1
19	Burkhart, N.	1984	2
20	Calderon-Gonzalez, R., Calderon-Sepulveda, R. F., Trevino-Welsh, J.	1999	5
21	Calović, T., Petrović1, B., Perić, T., Radumilo, D., Popov, I., Marković, E., Marković, D.	2022	1
22	Cancio, V., Faker, K., Tostes, M. A.	2019	1
23	Capetillo, G., Esparza, R., Torres, E., Flores, S., Parra, C. L., Leyva, F., Mendez, T., Ortiz, I., Torres, B.	2016	2
24	Carli, E., Pasini, M., Pardossi, F., Capotosti, I., Narzisi, A., Lardani, L.	2022	1
25	Carlsen, W. R., Galluzzi, K. E., Forman, L. F., Cavalieri, T. A.	1994	2
26	Carranza Sotelo, R. M. C.	2018	1
27	Castro, A. M. de., Marchesoti, M. G. N., Oliveira, F. S. de., Novaes, M. S. de. P.	2010	6

28	Chadwick, D., Chapman, M., Davies, G.	2018	5
29	Chen, C.-Y., Chen, Y.-W., Tsai, T.-P., Shih, W.-Y.	2014	1
30	Chen, Ming-Chuan., Kung, Pei-Tseng., Su, Hsun-Pi., Suh-May, Y., Li-Ting, C., Wen-Chen, T.	2016	1
31	Chen-Yi, L., Liu, YC. G., Tien-Yu, S., Yi-Chun, T., Teng, A. YT.	2019	5
32	Chhajed, S., Bhambhani, G., Agarwal, R., Balsaraf, S.	2016	1
33	Chi, D. L., Masterson, E. E., Wong, J. J.	2014	6
34	Chi, D. L., Rossitch, K. C., Beeles, E. M.	2013	5
35	Cocca, S., Viviano, M., Loglisci, M., Parrini, S., Monciatti, G., Paganelli, I. I., Livi, W., Mezzedimi, C.	2018	5
36	Cocchi, R., Lamma, A.	1999a	2
37	Cocchi, R., Lamma, A.	1999b	2
38	Çokpekin, F., Köymen, G., Başak, F., Akbulut, E., Altun, C.	2003	1
39	Couto, P., Pereira, P. A., Nunes, M.,Mendes, R. A.	2018	5
40	Couto, P., Pereira, P. A., Nunes, M.,Mendes, R. A.	2018	5
41	Da Silva Pereira, M. J. M.	2014	3
42	D'Alessandro, G., Cremonesi, I., Alkhamis, N., Piana, G.	2014	2
43	David Amaral, L., Fabiano de Carvalho, T., Barreto Bezerra, A. C.	2016	1
44	Dayakar, M., Shivprasad, D., Dayakar, A., Deepthi, C.	2015	8
45	De Almeida, J. S., Fernandes, R. F., Andrade Á, C. B., Almeida, B. D. C., Amorim, A., Lustosa, J., Mendes, R. F., Prado Júnior, R. R.	2021	1
46	De Pontual, L., Heulin, M., Charles, E., Héron, B., Zylberberg, P.	2015	3
47	Del Machuca Portillo, M. C., Hanke Herrero, R., del López Valle, L., Machuca Portillo, G., Bullón Fernández, P.	2005	5
48	DelRosso, L., Ferri, R.	2019	5
49	DeMattei, R., Cuvo, A., Maurizio, S.	2007	1
50	Echevarría-Goche, A., Munayco-Magallanes, A.	2012	3
51	Ellement, J. K., Virues-Ortega, J.;, Boris, A.	2021	3
52	Escribano Hernández, A., Hernández Corral, T., Ruiz-Martín, E., Porteros Sánchez, J. A.	2007	5
53	Fahad, A. H., Radhi, N. J. M. H.	2019	6
54	FitzGerald, P. M., Jankovic, J., Glaze, D. G., Schultz, R., Percy, A. K.	1990	5
55	FitzGerald, P. M., Jankovic, J., Percy, A. K.	1990	5
56	Florindez, L. I., Como, D. H., Florindez, D. C., Florindez, F. M., Law, E., Polido, J. C., Cermak, S. A.	2022	6
57	Fuertes-González, M. C., Silvestre, F.-J.	2014	5
58	Gaçe, E., Kelmendi, M., Fusha, E.	2014	1
59	Gadiyar, A., Gaunkar, R., Kamat, A., Tiwari, A., Kumar, A.	2018	5
60	Gandhi, R., Ruxmohan, S., Puranik, C. P.,	2021	6
61	Genaro, L. E., Marconato, J. V., Hanai, D., Pawloski, C. L. G., Capote, T. S. D.	2022	6

62	Gepner, B., Godde, A., Charrier, A., Carvalho, N., Tardif, C.	2021	6
63	Gonçalves, L. T. Y. R., Gonçalves, F. Y. Y. R., Nogueira, B. M. L., Fonseca, R. R. de S., de Menezes, S. A. F., da Silva e Souza, P. de A. R.., Menezes, T. O. de A.	2016	1
64	Haddad, A. S., Tagle, E. L., Passos, V. de A. B.	2016	3
65	Hagenmuller, F., Rössler, W., Wittwer, A., Haker, H.	2014	6
66	Hariyani, N., Soebekti, R. H., Setyowati, D., Bramantoro, T., Palupi, L. S., Oktarina., Putriana, E.	2019	1
67	Hegde, A. M., Babu, A. A., Mohammed, A., John, A., Singh, K., Preethi, V. C., Shetty, S.	2015	5
68	Hemmati, S., Soleymani, R., Mousavi, S. J., Nahvi, A.	2021	4
69	Hennequin, M., Moysan, V., Jourdan, D., Dorin, M., Nicolas, E.	2008	5
70	Ho, N. T., Kroner, B., Grinspan, Z., Fureman, B., Farrell, K., Zhang, J., Buelow, J., Hesdorffer, D. C., McDonald, B., Weldon, M., Bradish, J., Vogel-Farley, V., Nues, P., Dixon-Salazar, T., Bliss, G., DeWoody, Y., Nakagawa, J. A., Kroner, B., Harris, M., ... Nye, K.	2018	5
71	Hoshino, Y., Yashima, Y., Ishige, K., Kaneko, M., Kumashiro, H.	1979	4
72	Ibrahim, S. E.-S. M., Bahgat, R. S., Elhendawy, F. A.,	2020	3
73	Igić, M., Kostadinović, L., Janjić, O. T., Stojković, B., Obradović, R.	2017	3
74	Ilona, S., Emese, G., Tamás, P. G., György, S., Zsolt, N.	2019	6
75	Jaber, M. A., Sayyab, M., Abu Fanas, S. H.	2011	1
76	Jockusch, J., Sobotta, B. A. J., Nitschke, I.	2020	5
77	Jokić, N. I., Majstorović, M., Bakarcić, D., Katalinić, A., Szirovicza, L.	2007	5
78	Kamp-Becker, I.	2014	6
79	Kancherla, V., Van Naarden Braun, Kim., Yeargin-Allsopp, M.	2013	5
80	Karadag, G., Bilsin, E.	2016	7
81	Katge, F., Rusawat, B., Shitoot, A., Poojari, M., Pammi, T., Patil, D.	2015	1
82	Kheur, S., Deshpande, R., Mahajan, P., Bagde, K., Dungarwal, P., Rajpurohit, L. S., Panicker, S., Kheur, M.	2016	6
83	Klein, U., Nowak, A. J.	1999	1
84	Kooijman, M. N., Kruithof, C. J., van Duijn, C. M., Duijts, L., Franco, O. H., van IJzendoorn, M. H., de Jongste, J. C., Klaver, C. C. W., van der Lugt, A., Mackenbach, J. P., Moll, H. A., Peeters, R. P., Raat, H., Rings, E. H., Rivadeneira, F., van der Schroeff, M. P., Steegers, E. A. P., Tiemeier, H., Uitterlinden, A. G., … Jaddoe, V. W. V.	2016	8
85	Kopel, H. M.	1977	2
86	Koritsas, S., Iacono, T.	2011	7
87	Koskela, A., Neittaanmäki, A., Rönnberg, K., Palotie, A., Ripatti, S., Palotie, T.	2020	5
88	Kotha, S. B., AlFaraj, N. S. M., Ramdan, T. H., Alsalam, M. A., Al Ameer, M. J., Almuzin, Z. M.	2018	6
89	Lai, Y. Y. L., Wong, K., King, N. M., Downs, J., Leonard, H.	2018	5

90	Latifi-Xhemajli, B., Begzati, A., Kutllovci, T., Ahmeti, D.	2018	1
91	Lavås, J., Slotte, A., Jochym-Nygren, M., van Doorn, J., Engerström, I. W.	2006	7
92	Lefer, G., Bourdon, P., Mercier, C., Cazaux, S. L.	2018	3
93	Lim, J.	2018	2
94	Lim, M. A. W. T., Borromeo, G. L.	2019	1
95	Limeres, J., Martínez, F., Feijoo, J. F., Ramos, I., Liñares, A., Diz, P.	2014	5
96	Liu, Hsiu-Yueh., Hung, Hsin-Chia., Hsiao, Szu-Yu., Chen, Hong-Sen., Yen, Yea-Yin., Huang, Shun-Te., Chen, Chun-Chih., Chen, Ping-Ho., Chen, Cheng-Chin., Lin, Pei-Chen., Lu, Yun-Lin.	2013	3
97	Liu, Z., Yu, D., Luo, W., Yang, J., Lu, J., Gao, S., Li, W., Zhao, W.	2014	5
98	Logrieco, M. G. M., Ciuffreda, G. N., Sinjari, B., Spinelli, M., Rossi, R., D'Addazio, G., Lionetti, F., Caputi, S., Fasolo, M.	2021	7
99	Loo, C. Y., Graham, R. M., Hughes, C. V.	2009	1
100	Lowe, O., Lindemann, R.	1985	1
101	Machado, B. A., Moro, J. S., Massignam, C., Cardoso, M., Bolan, M.	2021	7
102	Magoo, J., Shetty, A. K., Chandra, P., Anandkrishna, L., Kamath, P. S., Iyengar, U.	2015	1
103	Mahendran, R., Subramaniam, M., Cai, Y., Chan, Y. H.	2006	6
104	Makkar, A., Indushekar, K. R., Saraf, B. G., Sardana, D., Sheoran, N.	2019	1
105	Mangione, F., Bdeoui, F., Monnier-Da Costa, A., Dursun, E.	2020	6
106	Marshall, J., Sheller, B., Mancl, L.	2010	1
107	Mbaabu, E. N.	2021	1
108	Mendez, S. V., Rotman, M., Hormazabal, F., Sepulveda, L., Valle, M., Alvarez, E.	2022	6
109	Mirtala Orellana, L., Cantero-Fuentealba, C., Schmidlin-Espinoza, L., Luengo, L.	2019	1
110	Mohinderpal Chadha, G., Kakodkar, P., Chaugule, V., Nimbalkar, V.	2012	1
111	Morais Junior, R. C., Rangel, M. L., de Carvalho, L. G. A., Figueiredo, S. C., Ribeiro, I. L. A., de Castro, R. D.	2019	1
112	Morales-Chávez, M. C.	2017	1
113	Morinushi, T., Ueda, Y., Tanaka, C.	2001	1
114	Morisaki, I.	2017	4
115	Mota, F. S. B., Nascimento, K. S., Oliveira, M. V., Osterne, V. J. S., Clemente, J. C. M., Correia-Neto, C., Lima-Neto, A. B., van Tilburg, M. F., Leal-Cardoso, J. H., Guedes, M. I. F., Cavada, B. S.	2022	6
116	Muppa, R., Bhupathiraju, P., Duddu, M. K., Dandempally, A., Karre, D. L.	2013	1
117	Murshid, E. Z.	2014	7
118	Naidoo, M., Singh, S.	2018	1

119	Naka, S., Yamana, A., Nakano, K., Okawa, R., Fujita, K., Kojima, A., Nemoto, H., Nomura, R., Matsumoto, M., Ooshima, T.	2009	5
120	Nakao, S., Scott, J. M., Masterson, E. E., Chi, D. L.	2015	7
121	Nassif, N.	2019	5
122	Nayak, P., Prasad, K. V. V., Bhat, Y.	2015	5
123	Nelson, L. P.	2011	7
124	Nqcobo, C., Ralephenya, T., Kolisa, Y. M., Esan, T., Yengopal, V.	2019	1
125	Nunes, R., Simões, P. W., Pires, P. D. S., Rosso, M. L. P.	2017	1
126	Oda, G., Karayagmurlu, A., Dagli, I., Aren, G., Soylu, N.	2021	1
127	Öner Özdas, D., Özalp, S., Pamuk, F., Koyuncuoglu, C. Z.	2013	2
128	Orellana, L. M., Martínez-sanchis, S., Silvestre, F. J.	2014	6
129	Paraipan, C., Mihailescu, I., Rad, F., Dobrescu, I.	2015	2
130	Parkhurst, L. L.	2019	1
131	Penmetsa, C., Penmetcha, S., Cheruku, S., Mallineni, S., Patil, A., Namineni, S.	2019	1
132	Permatasari, I., Saskianti, T., Moeharyono, M.	2020	2
133	Petrova, E. G., Hyman, M., Estrella, M. R. P., Inglehart, M. R.	2014	5
134	Picciani, B. L. S., Santos, B. M., Silva-Júnior, G. O., Souza, T. T., Faria, M. B. D., Bastos, L. F.	2018	5
135	Pilebro, C., Bäckman, B.	2005	3
136	Pirela de Manzano, M. A., Salazar, V., Carmen Rosa., Manzano, F., Moisés, A.	1999	1
137	Pisiriciler, R., Emekli-Alturfan, E., Ozbay, G., Caliskan-Ak, E., Akyuz, S., Yarat, A.	2013	2
138	Polimeni, M. A., Richdale, A. L., Francis, A. J. P.	2005	7
139	Prakash, J., Das, I., Bindal, R., Shivu, M. E., Sidhu, S., Kak, V., Kumar, A.	2021	1
140	Puteri, M. M., Saskianti, T., Ananda, R. R.	2019	1
141	Puthiyapurayil, J., Kumar, T. V. A., Syriac, G., Maneesha, R., Raseena, K. T., Najmunnisa.	2022	1
142	Rada, R. E.	2013	6
143	Radha, G., Swathi, V., Jha, A.	2016	5
144	Rajić, A., Dzingalasević, G.	1989	3
145	Ribeiro, R. A., Romano, A. R., Birman, E. G., Mayer, M. P.	1997	5
146	Santosh, A., Kakade, A., Mali, S., Takate, V., Deshmukh, B., Juneja, A.	2021	1
147	Savioli, C., Campos, V. F., Santos, M. T. B. R. dos.	2005	2
148	Schreuder, S., van de Loo-Neus, G. H. H., Broers, D. L. M.	2021	4
149	Shapira, J., Mann, J., Tamari, I., Mester, R., Knobler, H., Yoeli, Y., Newbrun, E.	1989	2
150	Shivakumar, K., Patil, S., Kadashetti, V., Raje, V.	2018	1
151	Slayton, R. L.	2010	3
152	Srivastava, S., Desai, S., Cohen, J., Smith-Hicks, C., Barañano, K., Fatemi, A., Naidu, S. B.	2018	3

153	Standridge, S., Kaul, A., Horn, P., Harvey, J.	2006	2
154	Stein, L. I.	2012	7
155	Stein, L. I., Lane, C. J., Williams, M. E., Dawson, M. E., Polido, J. C., Cermak, S. A.	2014	3
156	Subramaniam, P., Gupta, M.	2011	6
157	Swallow, J. N.	1969	3
158	Tartaglia, N., Davis, S., Hench, A., Nimishakavi, S., Beauregard, R., Reynolds, A., Fenton, L., Albrecht, L., Ross, J., Visootsak, J., Hansen, R., Hagerman, R.	2008	6
159	Temudo, T., Oliveira, P., Santos, M., Dias, K., Vieira, J., Moreira, A., Calado, E., Carrilho, I., Oliveira, G., Levy, A., Barbot, C., Fonseca, M., Cabral, A., Dias, A., Cabral, P., Monteiro, J., Borges, L., Gomes, R., Barbosa, C., … Maciel, P.	2007	5
160	Timans, J. K.	1989	6
161	Tsai, F.-J., Chiang, H.-L., Lee, C.-M., Gau, S. S.-F., Lee, W.-T., Fan, P.-C., Wu, Y.-Y., Chiu, Y.-N.	2012	7
162	Urbanowicz, A., Van Dooren, K., Granich, J., Whitehouse, A., Lennox, N.	2016	2
163	Valendriyani, N.,Wen-Chen, W., Hung-En, L., Abu, B., Yin-Hwa, S.	2020	5
164	Vellappally, S., Gardens, S. J., Al Kheraif, A.-A.A., Krishna, M., Babu, S., Hashem, M., Jacob, V., Anil, S.	2014	1
165	Venkataraghavan, K., Garg, V., Kaur, M., Trivedi, K., Shah, S.	2016	5
166	Vishnu Rekha, C., Arangannal, P., Shahed, H.	2012	1
167	Vittek, J., Winik, S., Winik, A., Sioris, C., Tarangelo, A. M., Chou, M.	1994	2
168	Waldman, H. B., Perlman, S. P.	2012	3
169	Wu, X. R., Xia, B., Ge, L. H., Qin, M., Li, R. Z., Wang, B., Ge, F. Q., Wang, X. J., Chen, X., Song, G. T., Shao, L. Q., Wang, J., Zou, J., Lin, J. J., Zhao, Y. M., Mei, Y. F., Huang, H., Zeng, S. J.	2020	4
170	Yang, Y., Jin, Z., Wang, J., Liu, S., Huang, H., Jin, X.	2018	1
171	Yao, L., Fu, H., Bai, L., Deng, W., Xie, F., Li, Y., Zhang, R., Xu, X., Wang, T., Lai, S., Wang, J.	2021	6
172	Yasui, E. M., Schwartzman, J. S., Brunoni, D., Arita, E. S., Frias, A. C., Uemura, S. T.	2008	2
173	Zablotsky, B., Waldman, H. B., Zablotsky, N., Perlman, S.	2012	7
174	Zappella, M.	1997	2
175	Zhou, N., Wong, H. M., McGrath, C.	2019	5

1. Without control group.
2. Without access to the full text.
3. Inappropriate study design.
4. Inadequate language.
5. Without oral condition data in ASD patients.
6. Without the outcome of interest.
7. Self-reported oral measures.
8. Inappropriate or not defined population.

Appendix 3

Subgroups analysis (Figures 1 to 23)

Figure 1. *Forest plot for caries prevalence comparison between ASD and non-ASD.* Subgroup analysis: Teeth.

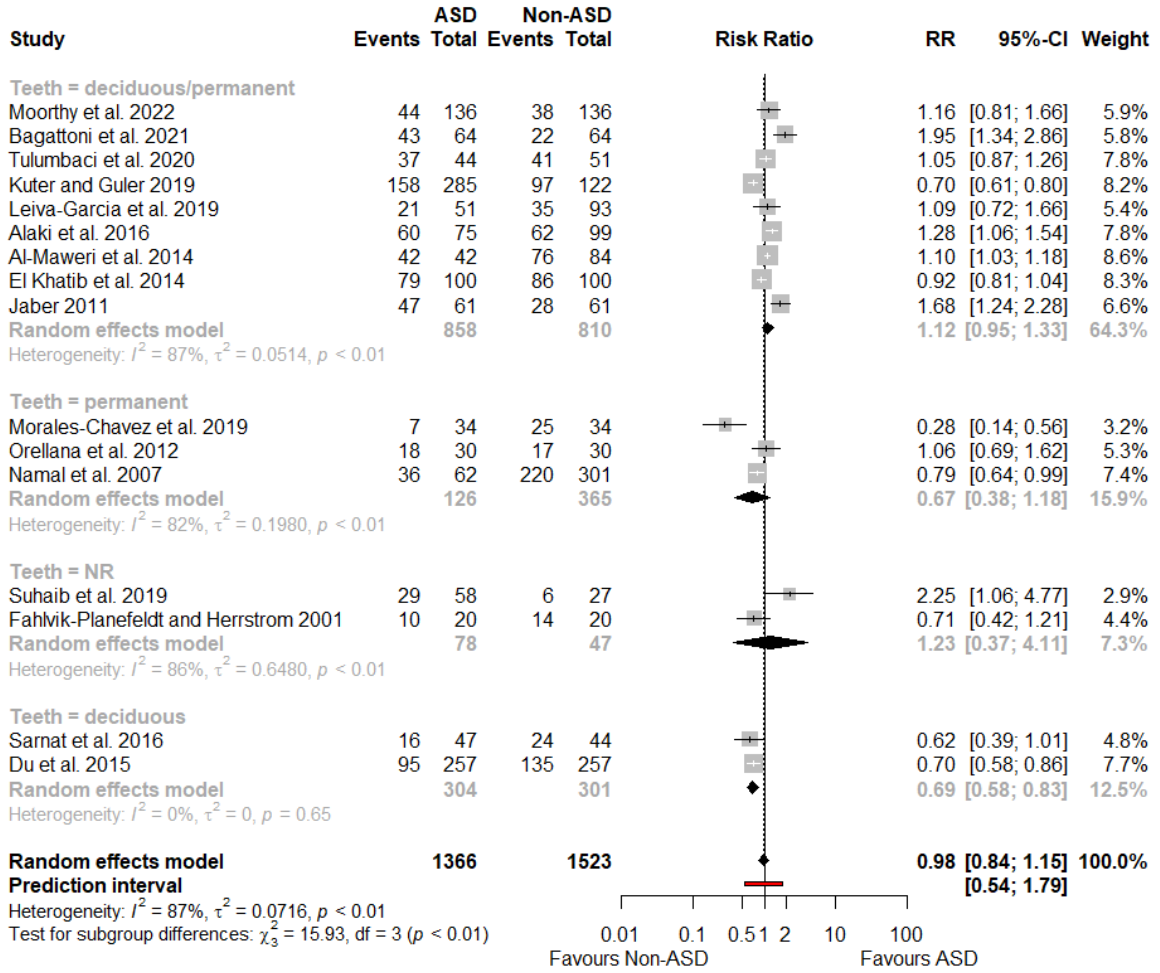


Figure 2. *Forest plot for caries prevalence comparison between ASD and non-ASD.* Subgroup analysis: Bias.

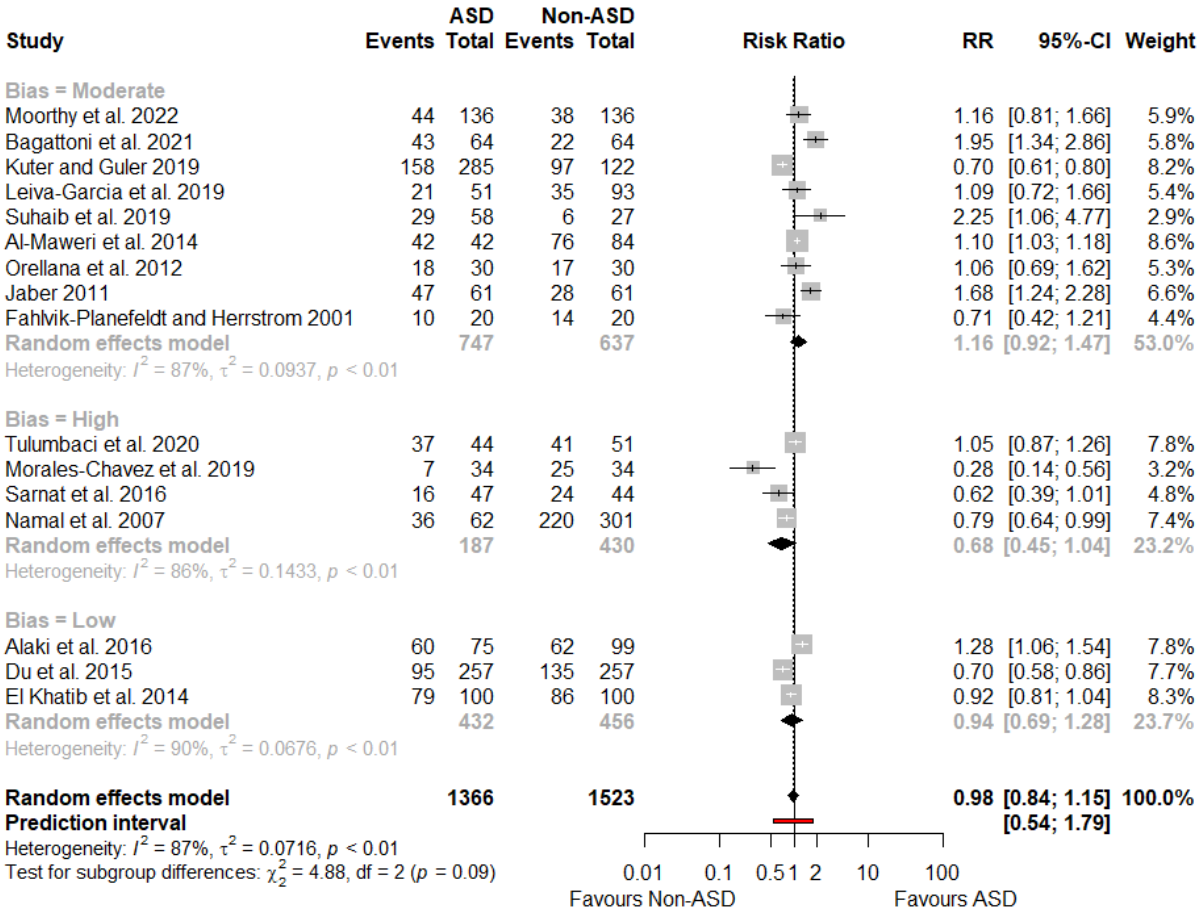


Figure 3. *Forest plot for caries prevalence comparison between ASD and non ASD.*

Subgroup analysis: Matching variables number.

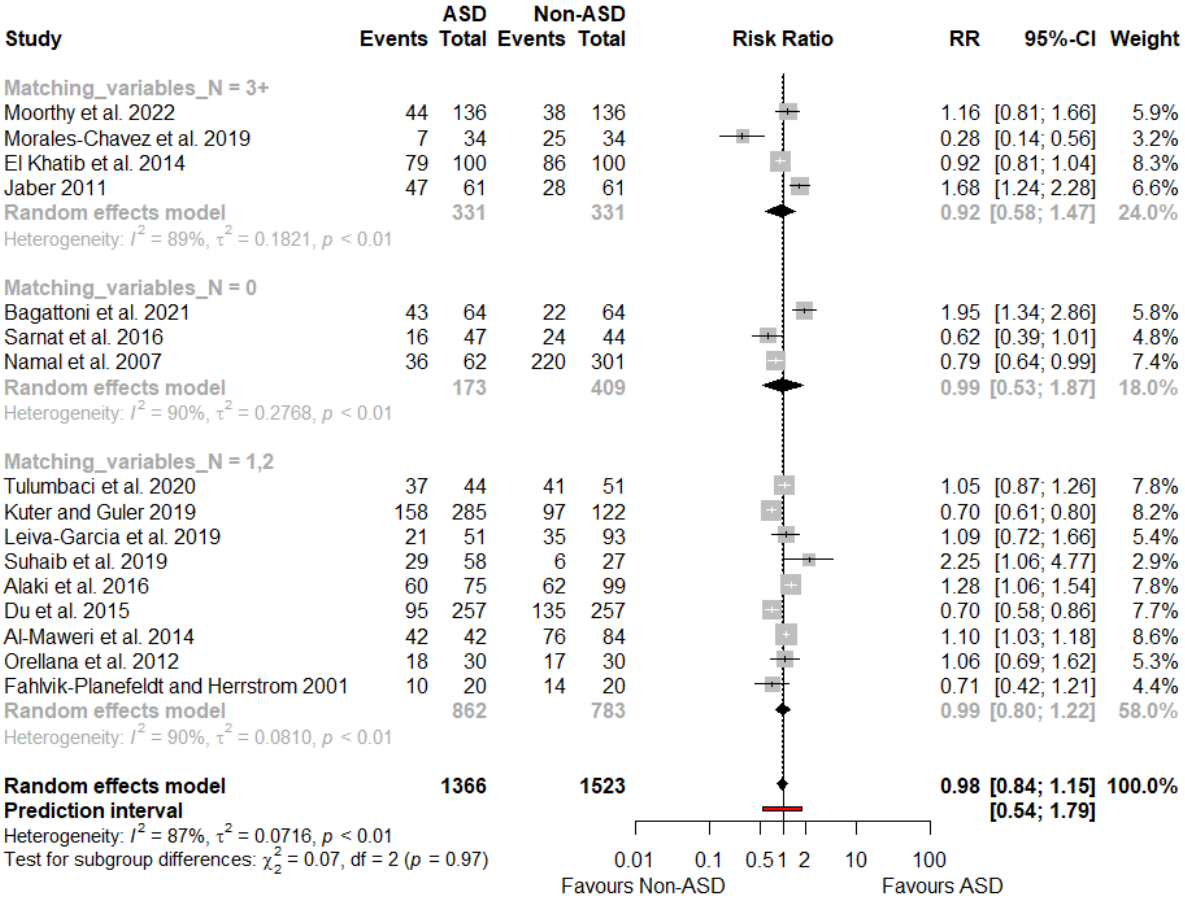


Figure 4. *Forest plot for caries prevalence comparison between ASD and non ASD.* Subgroup analysis: Socio demographic index (SDI).

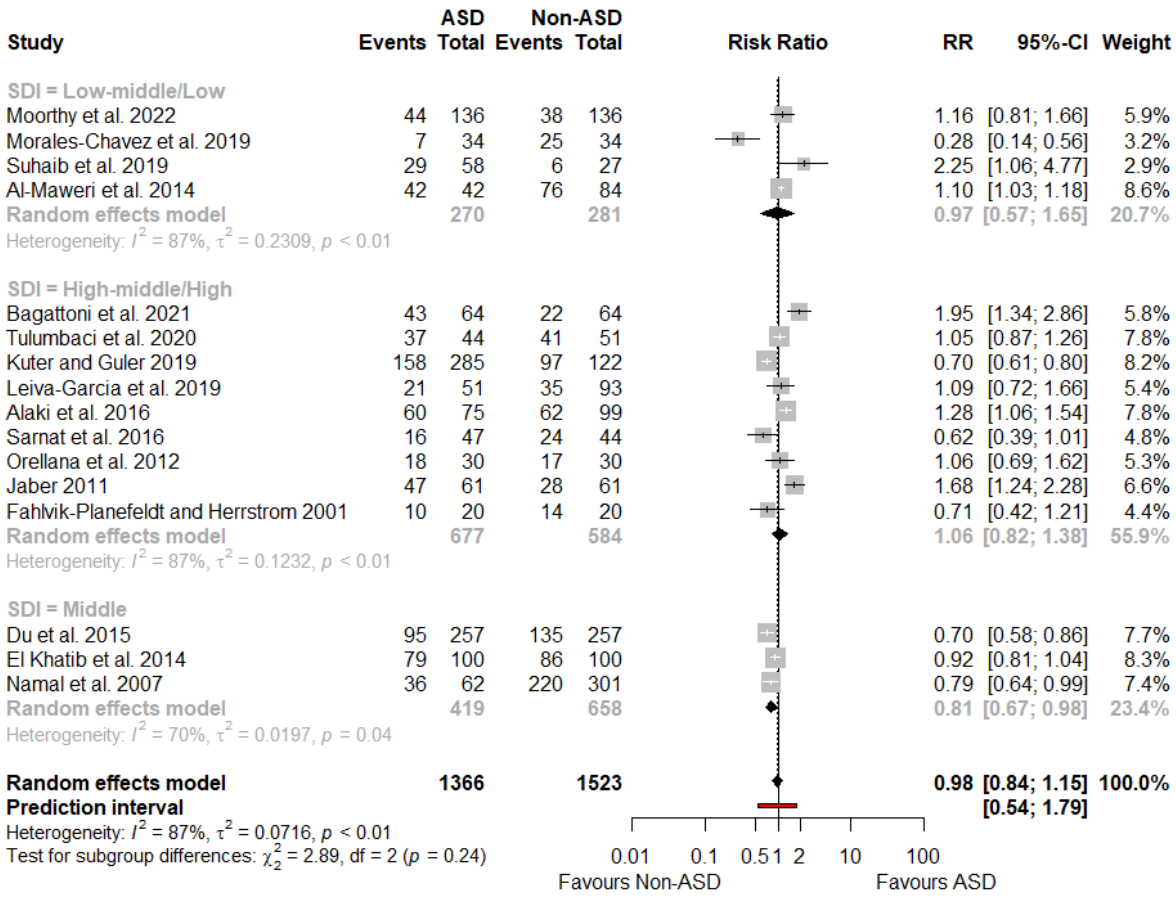


Figure 5. *Forest plot for caries severity comparison between ASD and non ASD.* Subgroup analysis: Socio demographic index (SDI).

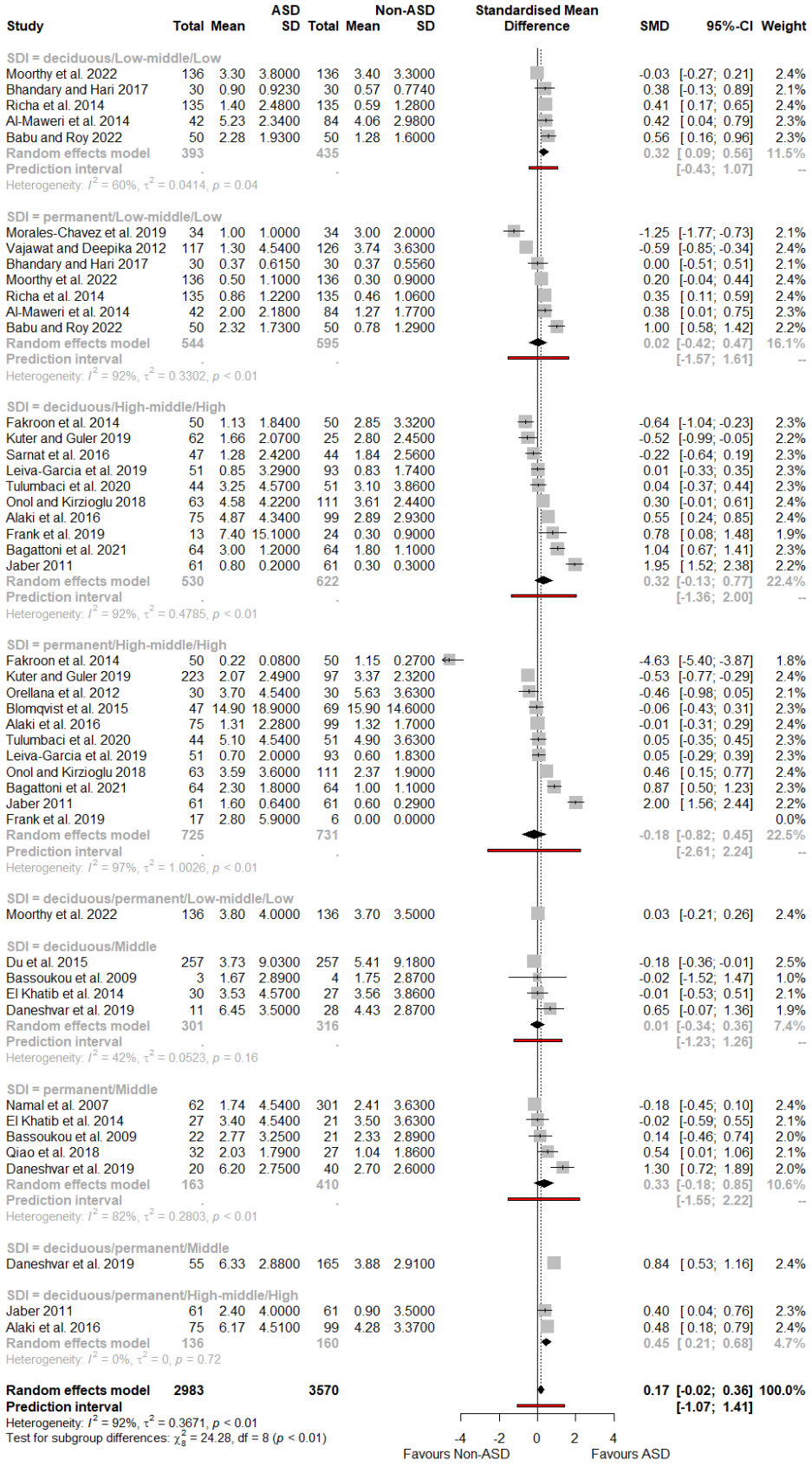


Figure 6. *Forest plot for caries severity comparison between ASD and non ASD.* Subgroup analysis: Matching variables number.

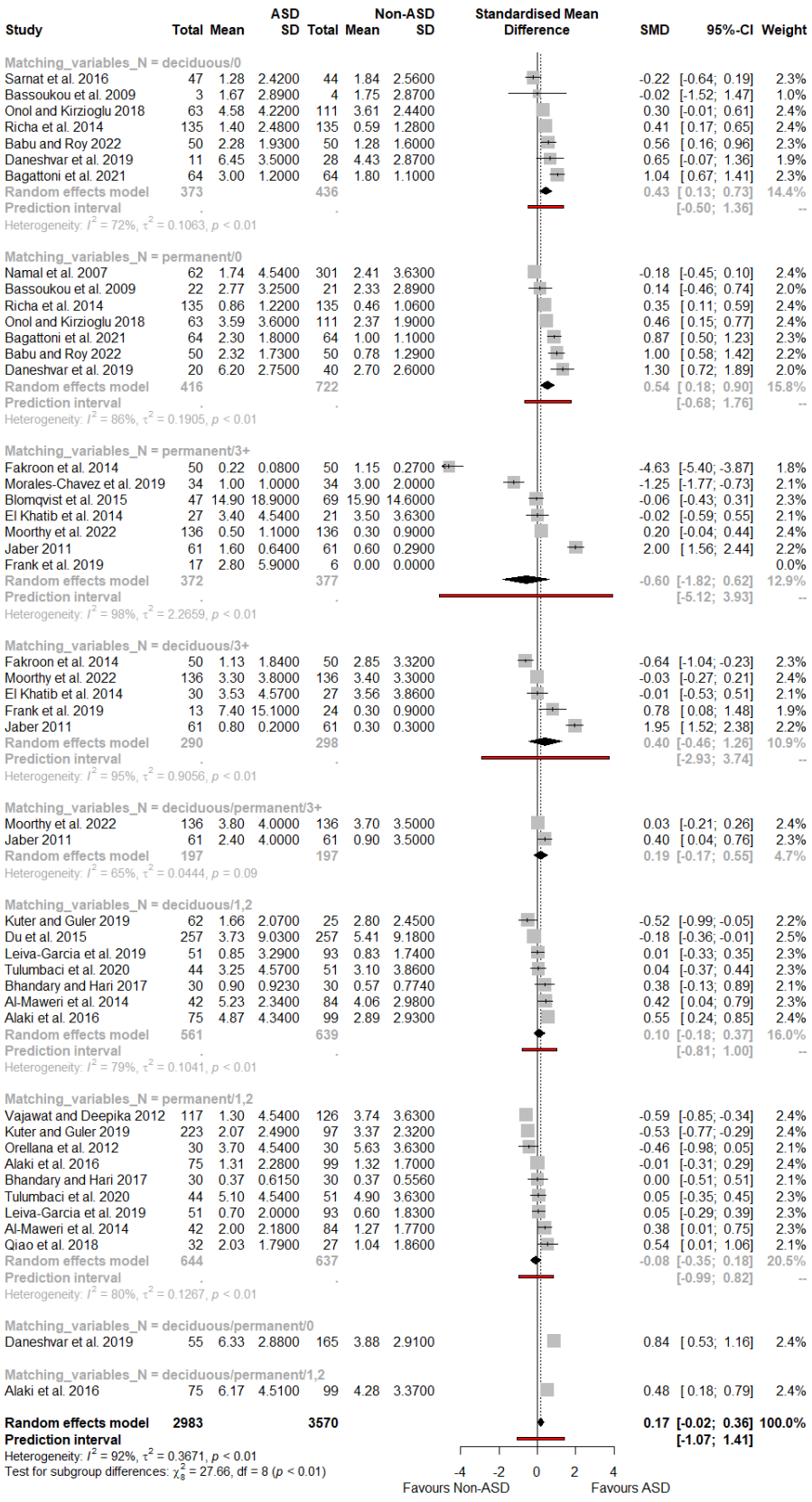


Figure 7. *Forest plot for caries severity comparison between ASD and non ASD.* Subgroup analysis: Bias.

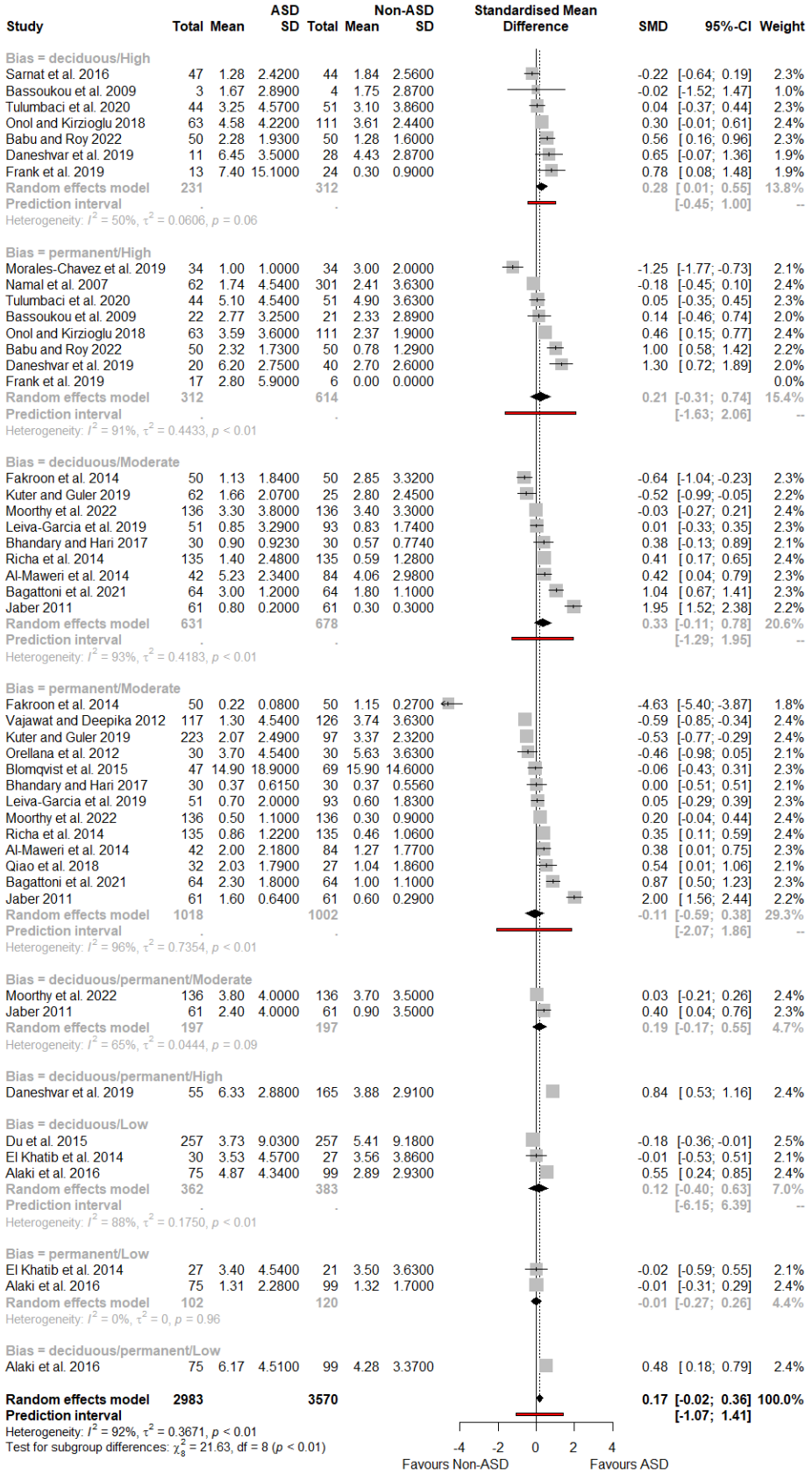


Figure 8. *Forest plot for non-treated caries severity comparison between ASD and non ASD.* Subgroup analysis: Bias.

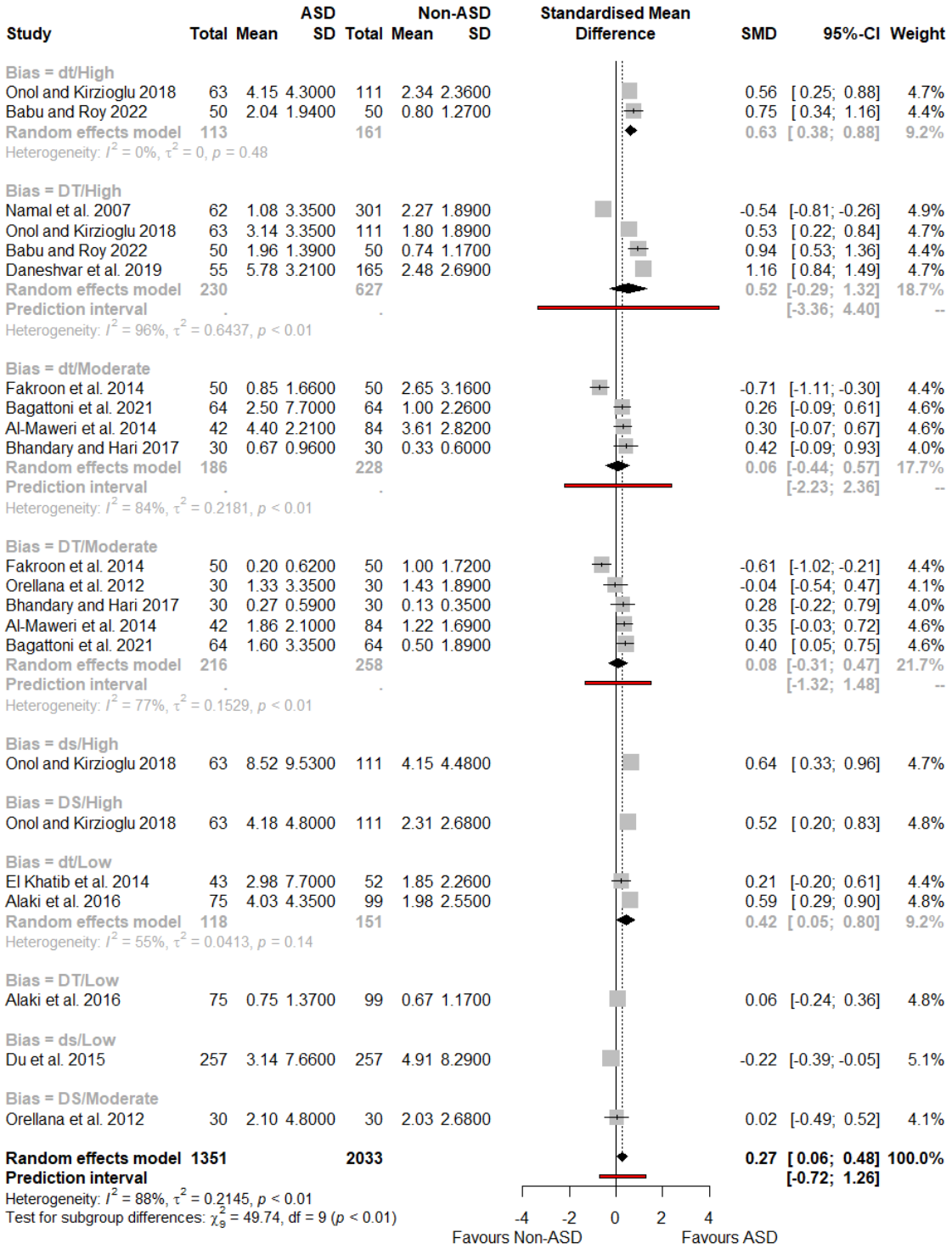


Figure 9. *Forest plot for non-treated caries severity comparison between ASD and non ASD.* Subgroup analysis: Matching variables number.

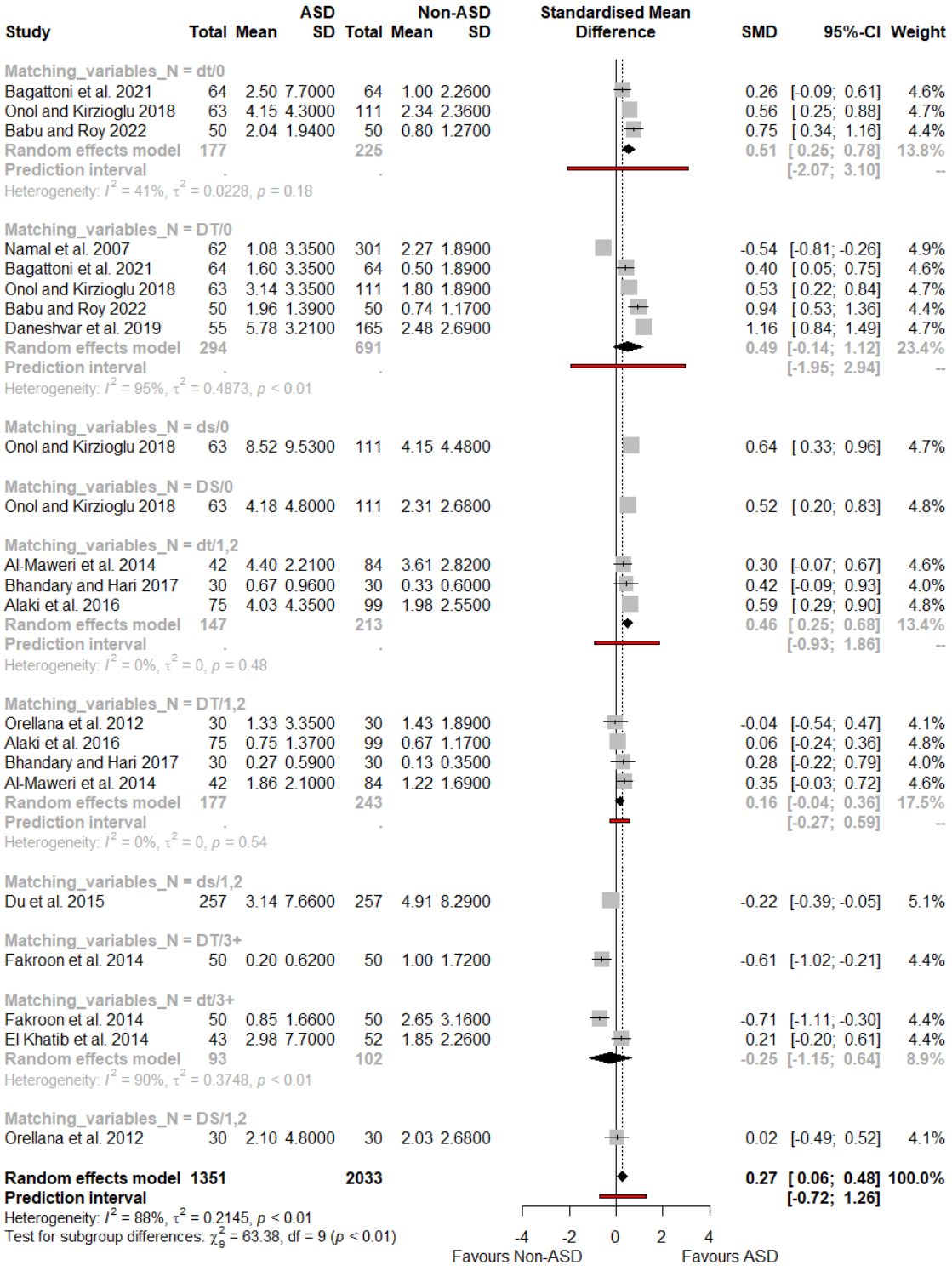


Figure 10. *Forest plot for non-treated caries severity comparison between ASD and non ASD.* Subgroup analysis: Socio demographic index (SDI).

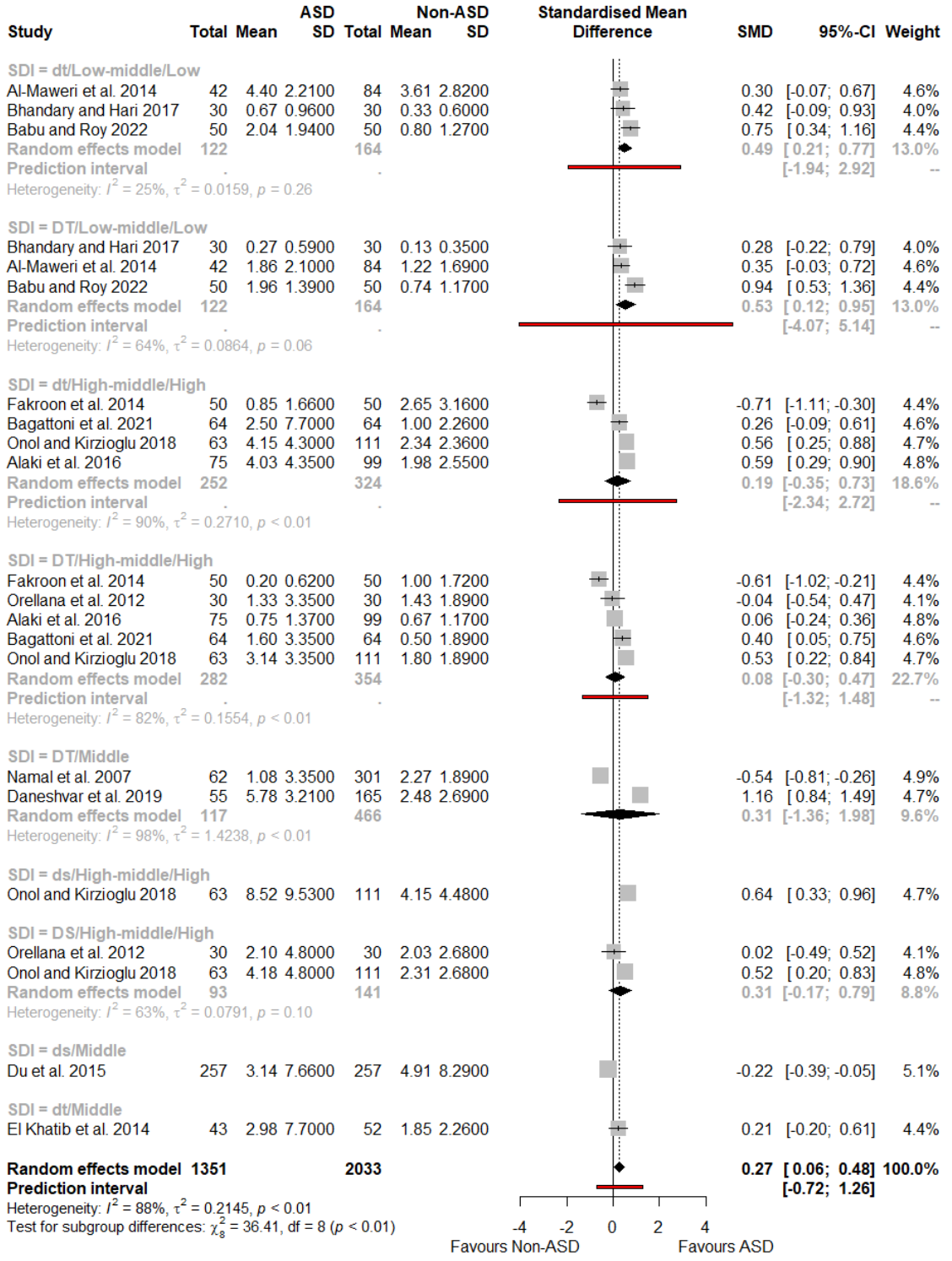


Figure 11. *Forest plot for filled severity comparison between ASD and non ASD.* Subgroup analysis: Bias

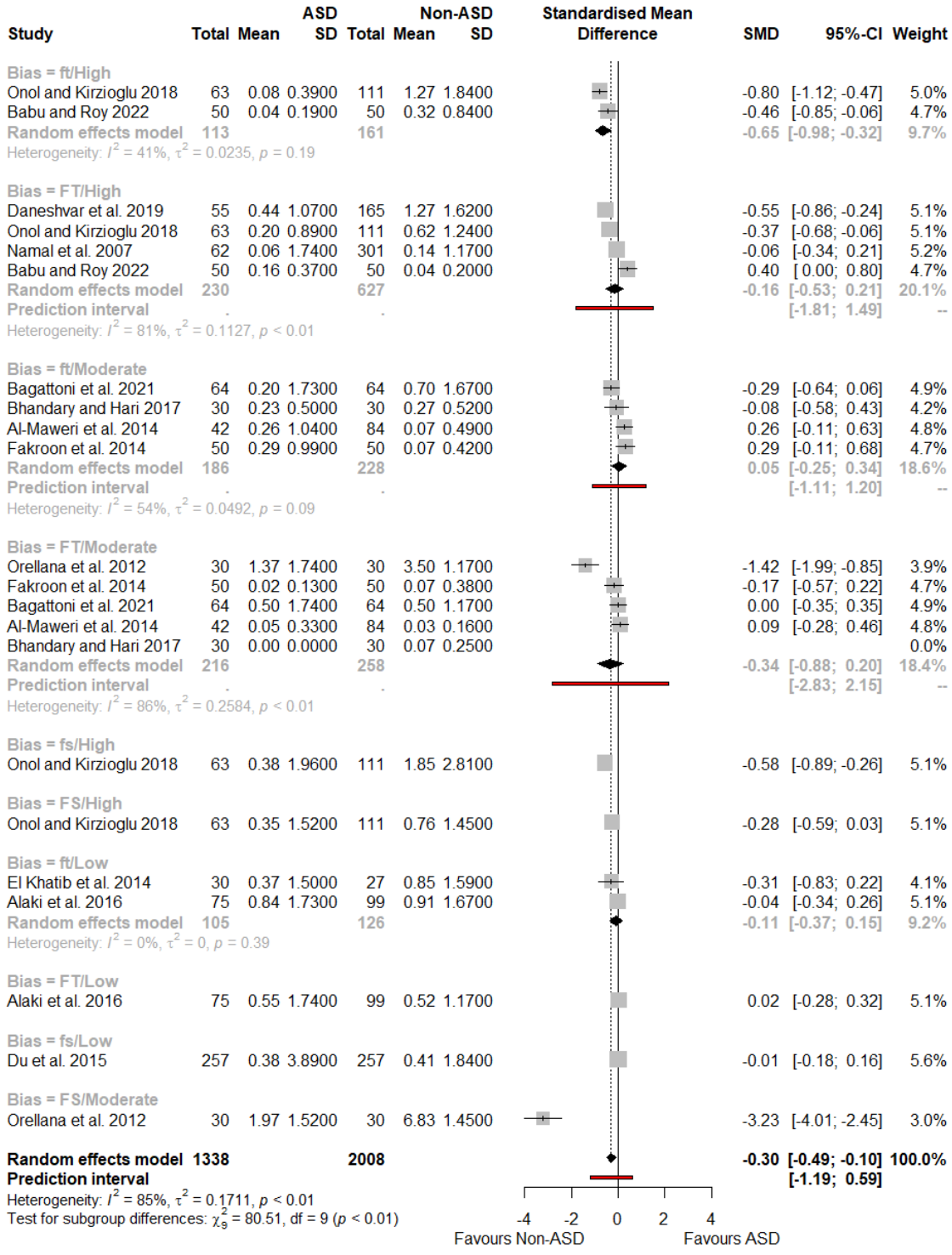


Figure 12. *Forest plot for filled severity comparison between ASD and non ASD.* Subgroup analysis: Matching variables number.

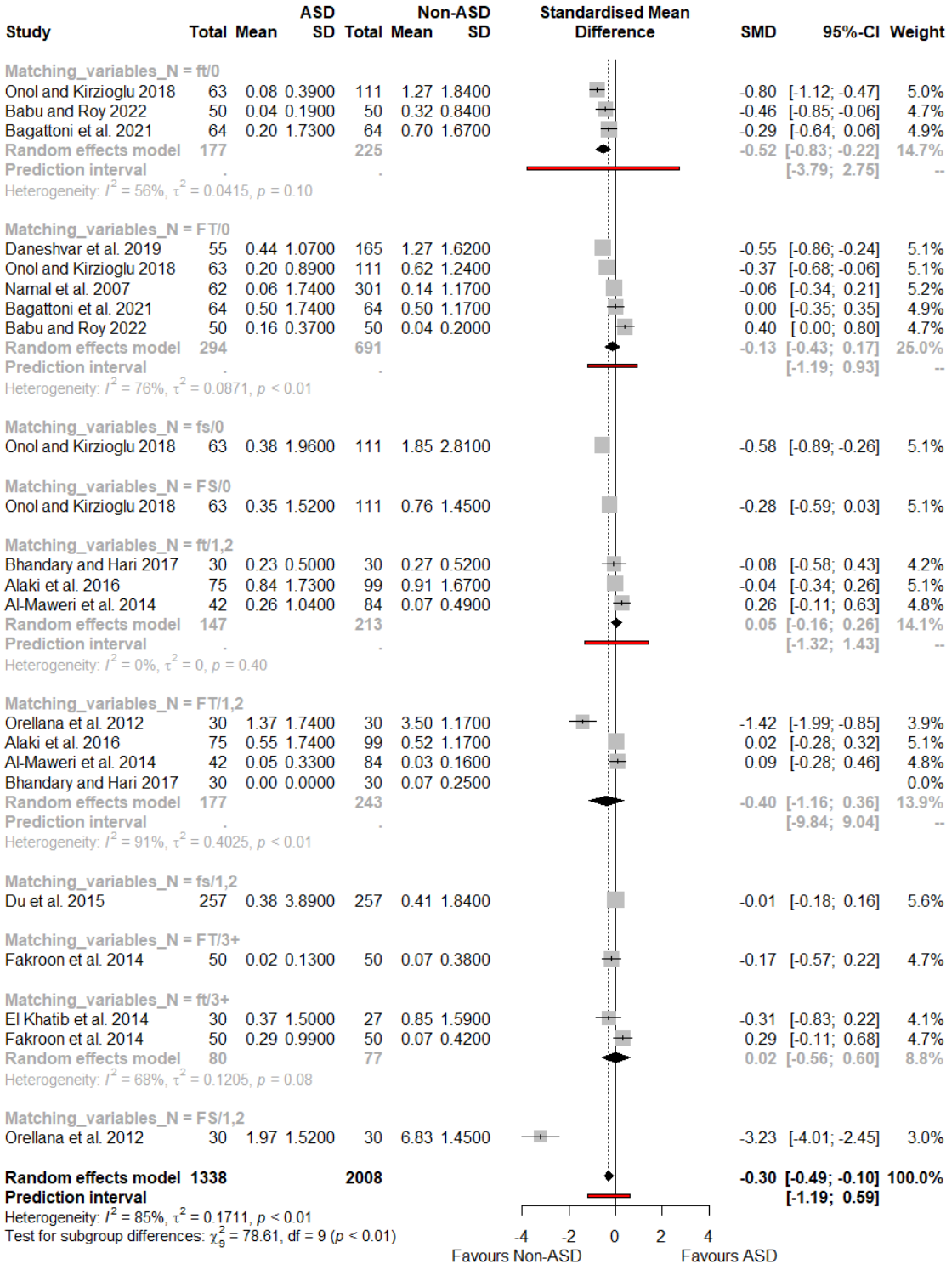


Figure 13. *Forest plot for filled severity comparison between ASD and non ASD.* Subgroup analysis: Socio demographic index (SDI).

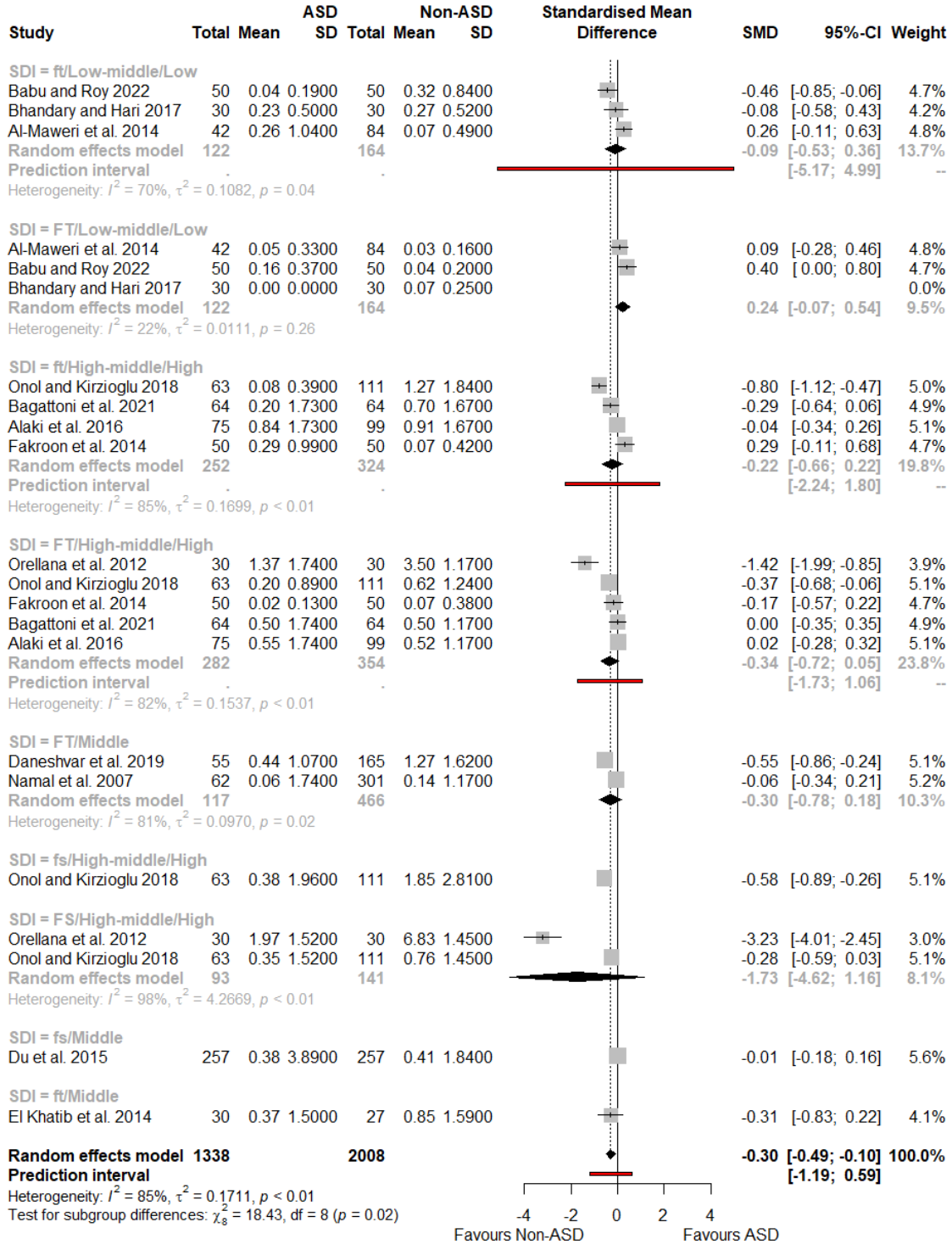


Figure 14. *Forest plot for tooth loss severity comparison between ASD and non ASD.*

Subgroup analysis: Bias.

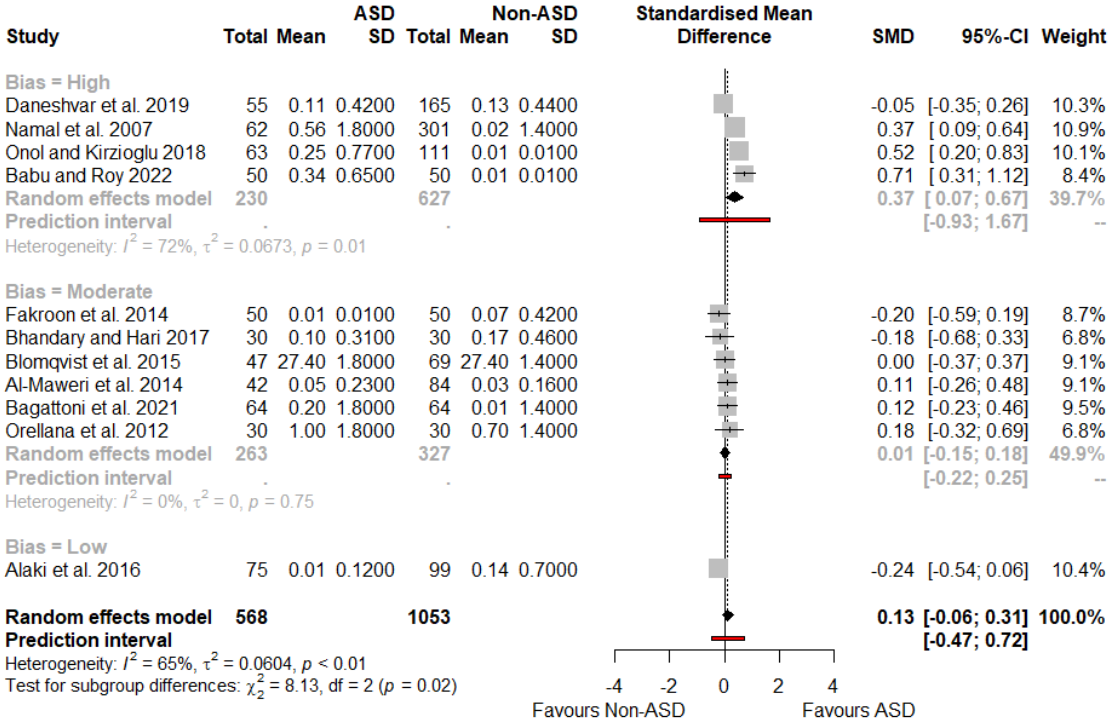


Figure 15. *Forest plot for tooth loss severity comparison between ASD and non ASD.* Subgroup analysis: Matching variables number.

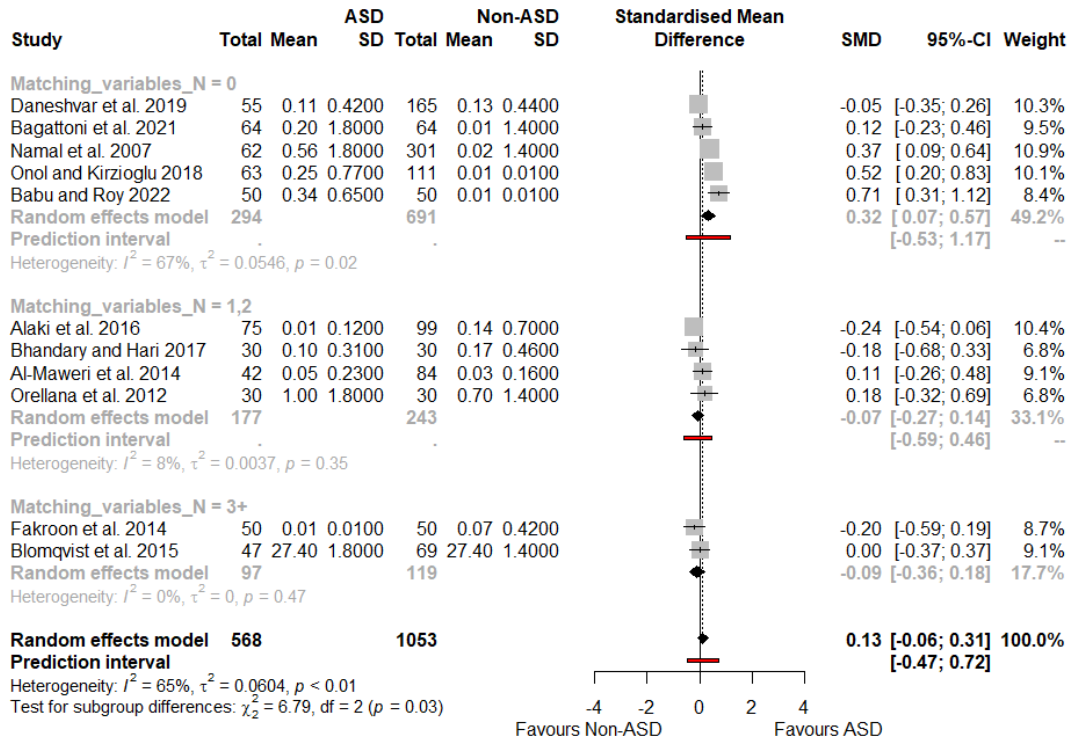


Figure 16. *Forest plot for malocclusion prevalence according Angle Class comparison between ASD and non ASD.* Subgroup analysis: Bias.

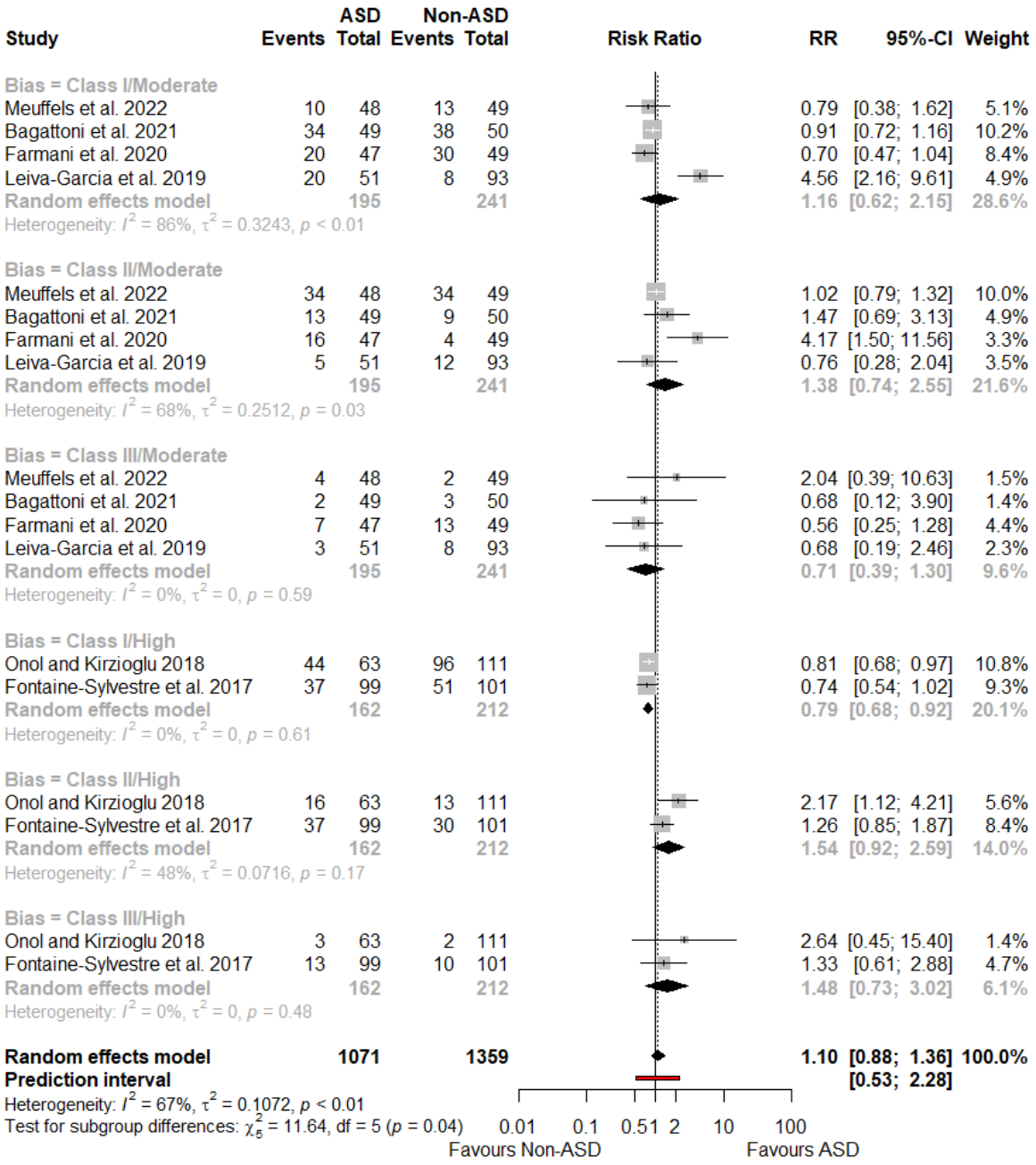


Figure 17. *Forest plot for malocclusion prevalence according Angle Class comparison between ASD and non ASD.* Subgroup analysis: Matching variables number.

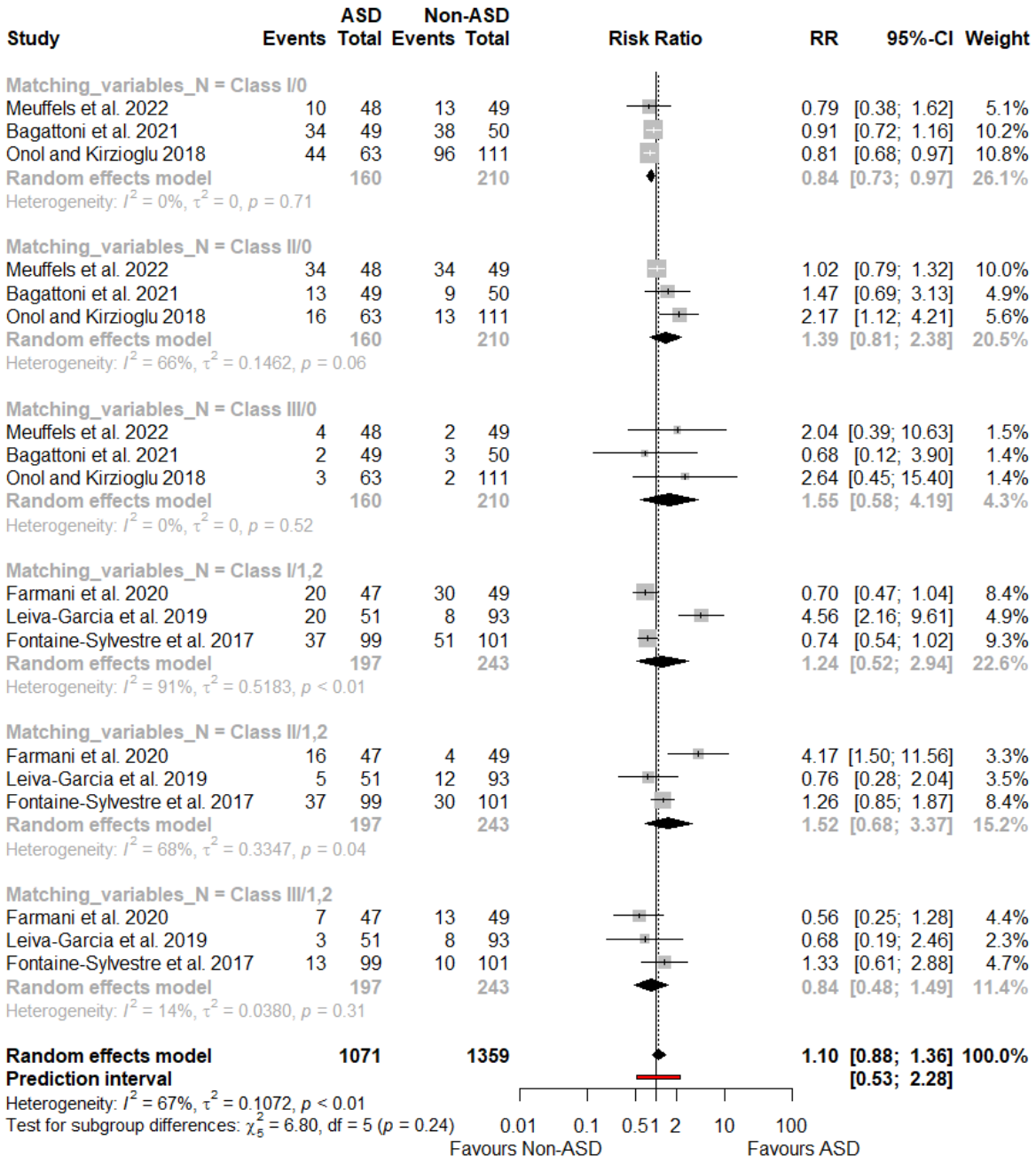


Figure 18. *Forest plot for malocclusion prevalence according Angle Class comparison between ASD and non ASD.* Subgroup analysis: Socio Demographic index (SDI).

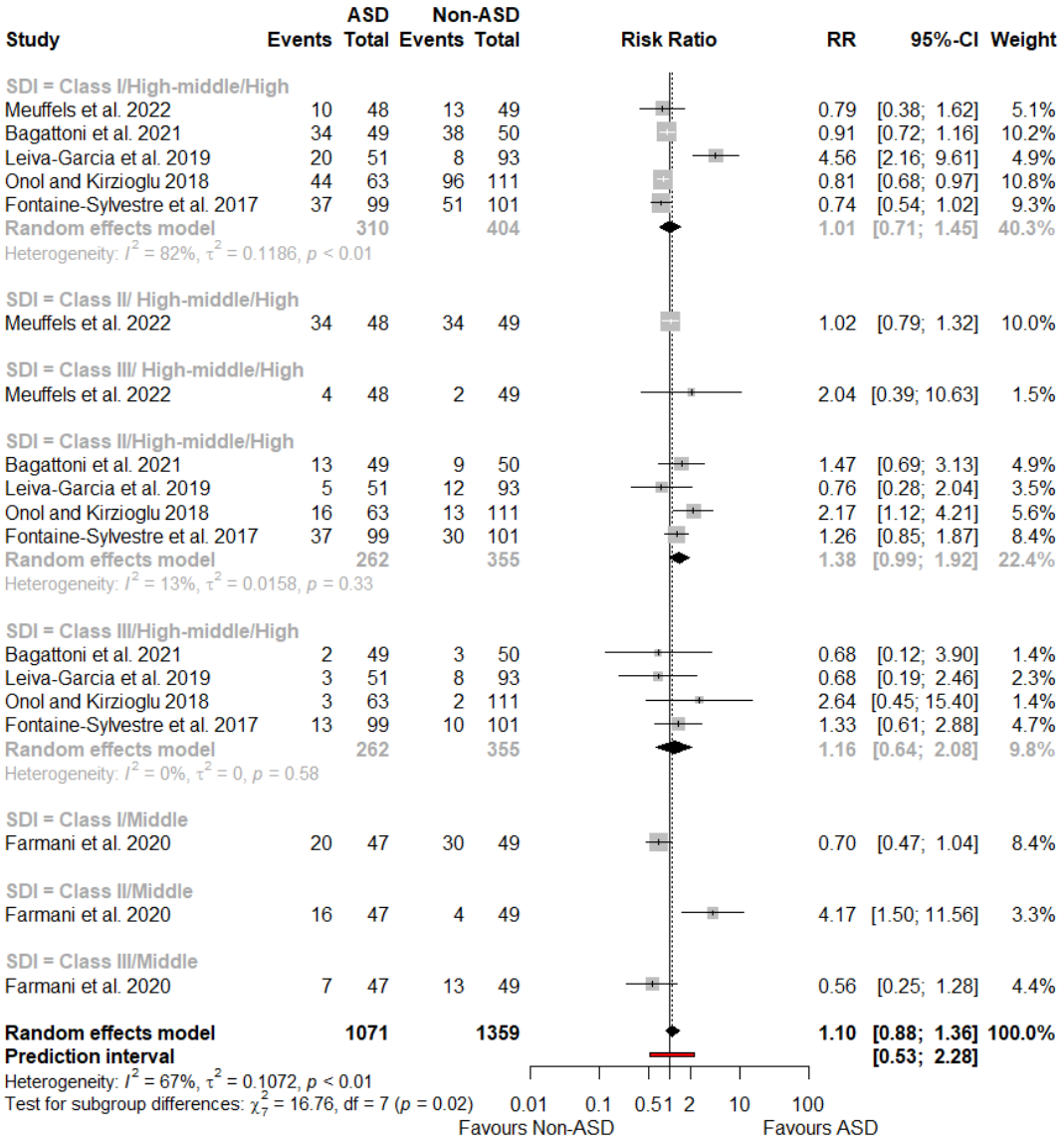


Figure 19. *Forest plot for crossbite prevalence comparison between ASD and non ASD.* Subgroup analysis: Bias.

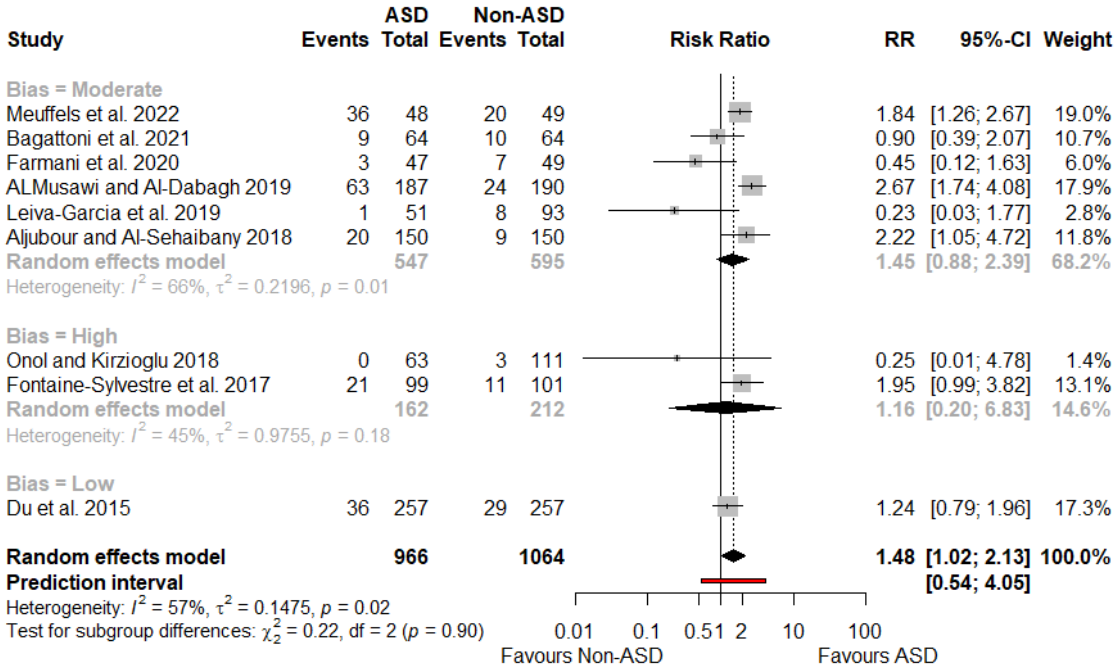
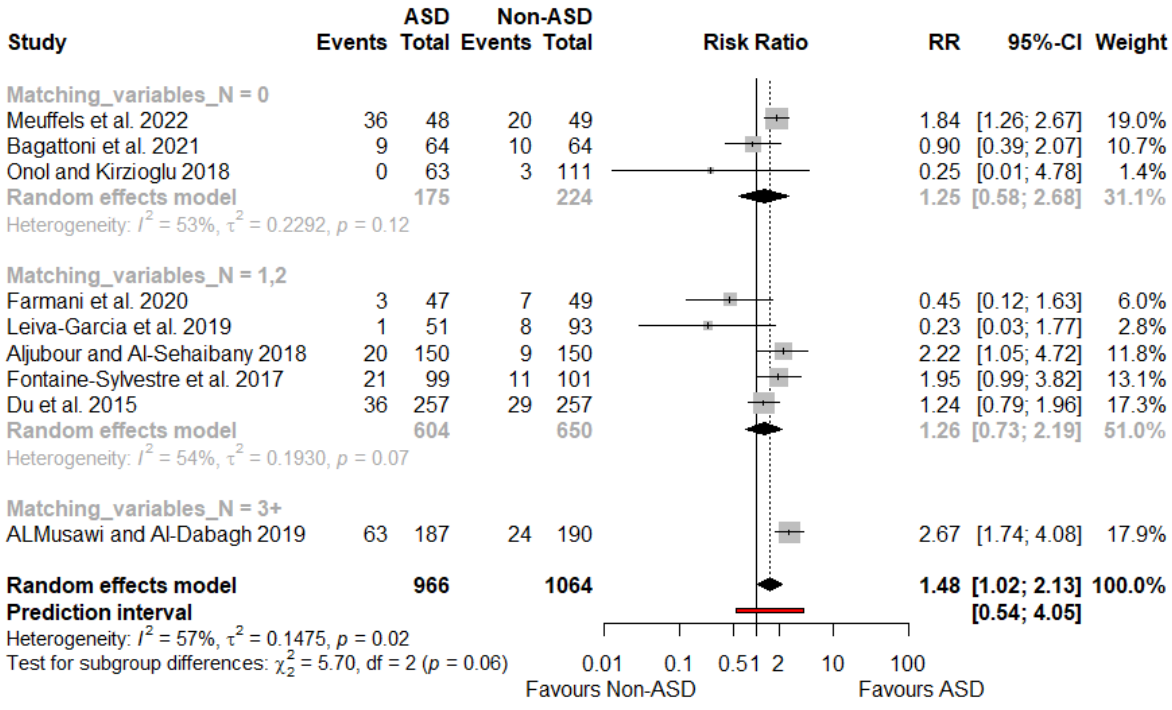


Figure 20. *Forest plot for crossbite prevalence comparison between ASD and non ASD.* Subgroup analysis: Matching variables number.



Appendix 4

Sensitivity analysis (Figures 1 to 7).

Figure 1. Sensitivity analysis of *caries prevalence*.

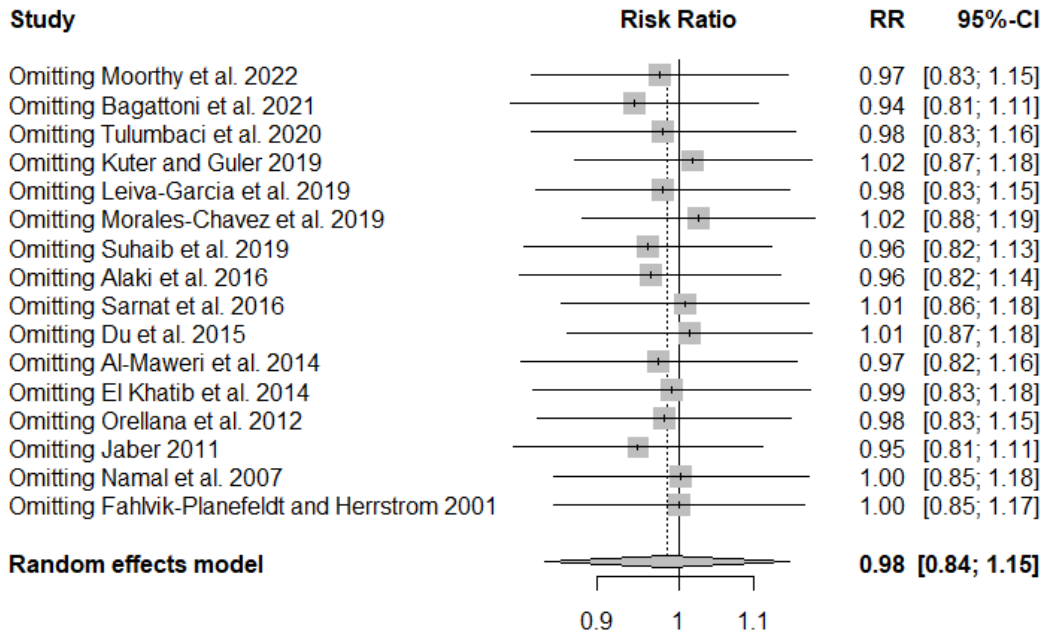


Figure 2. Sensitivity analysis of *tooth loss*.

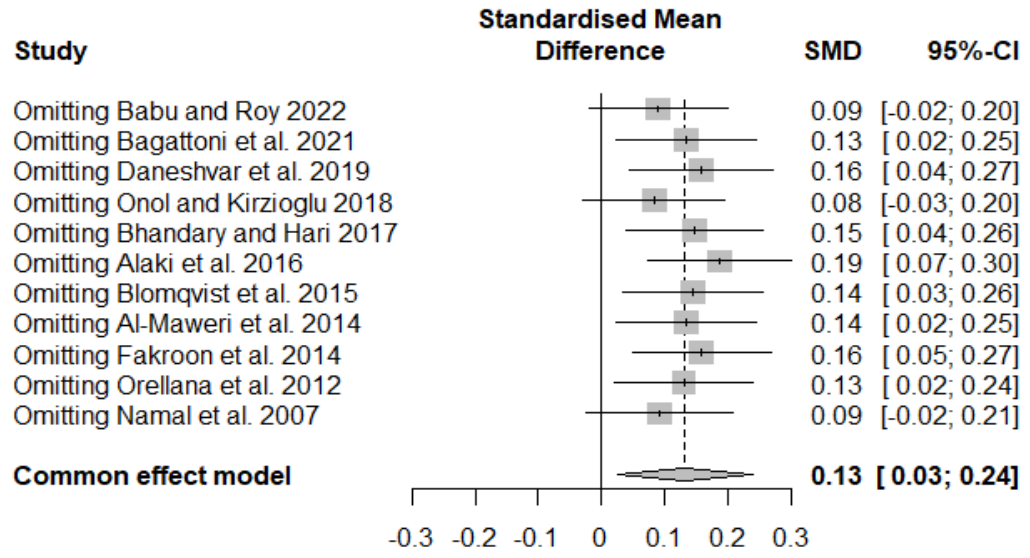


Figure 3. Sensitivity analysis of *overjet prevalence*.

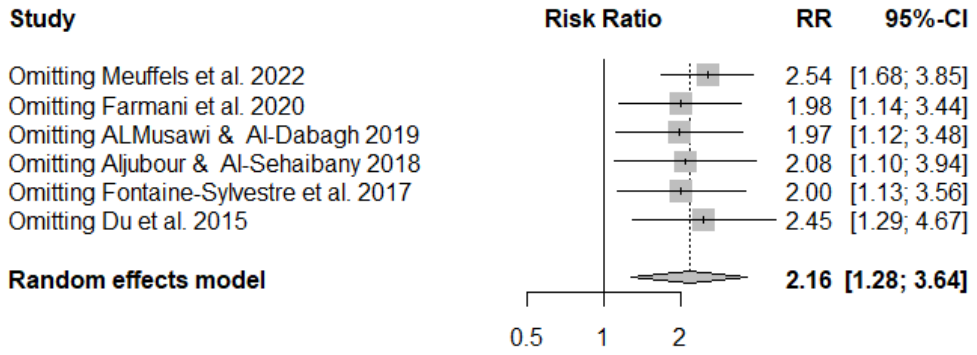


Figure 4. Sensitivity analysis of *overbite prevalence*.

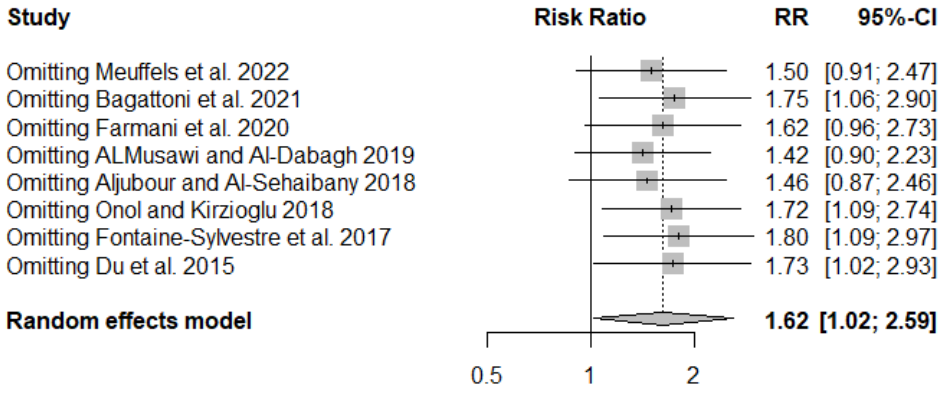


Figure 5. Sensitivity analysis of *crossbite prevalence*.

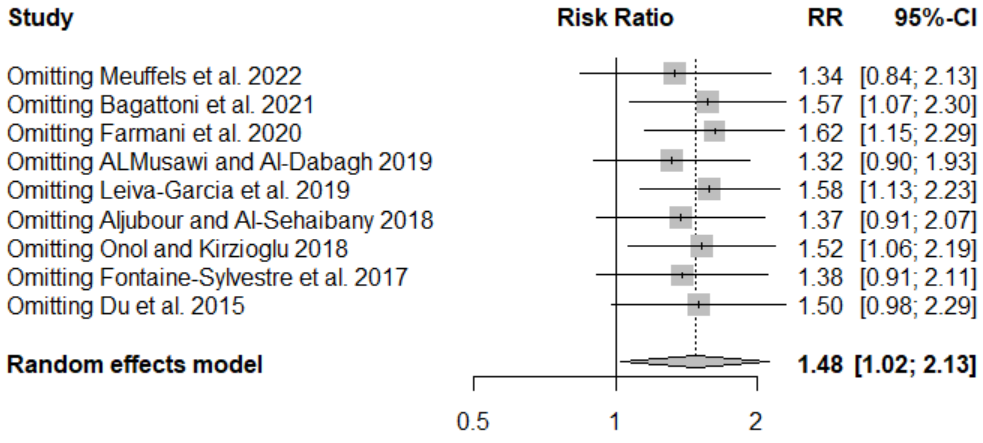


Figure 6. Sensitivity analysis of *openbite prevalence*.

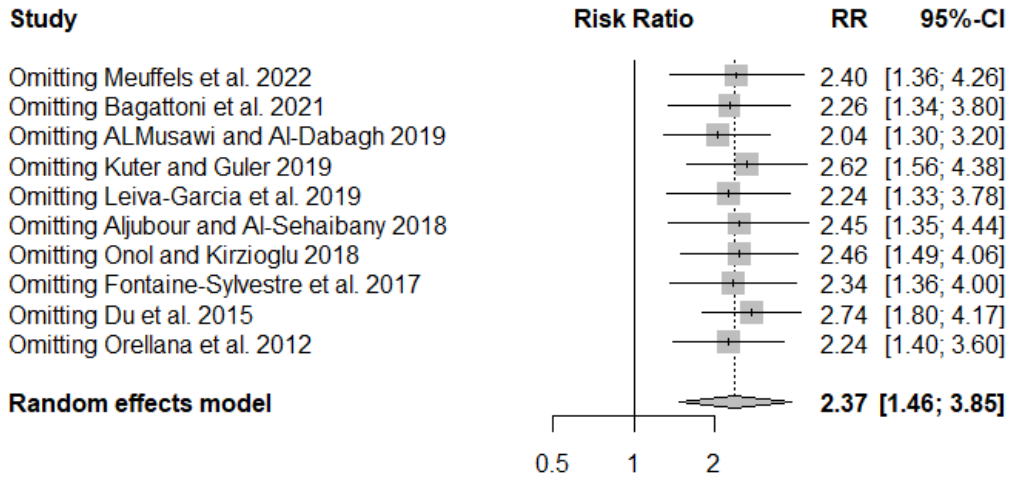
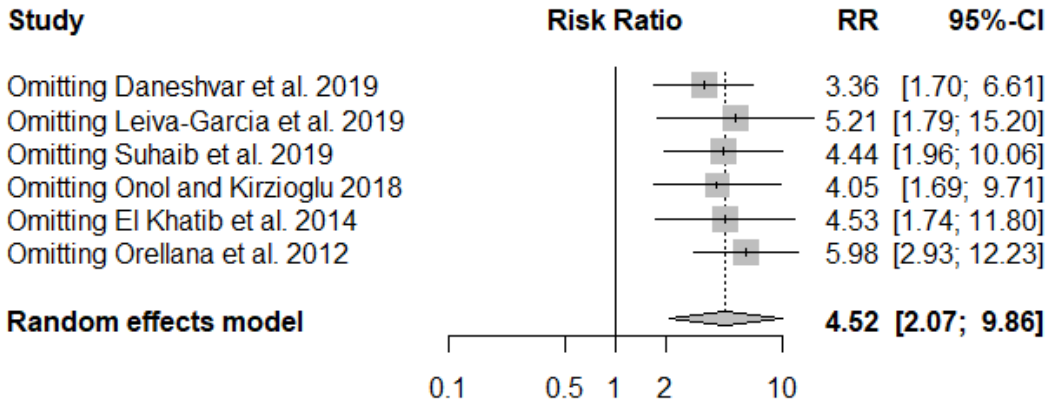


Figure 7. Sensitivity analysis of *bruxism prevalence*.



Appendix 5

Funnel plot (Figures 1 to 3).

Figure 1. *Funnel plot for caries prevalence.*

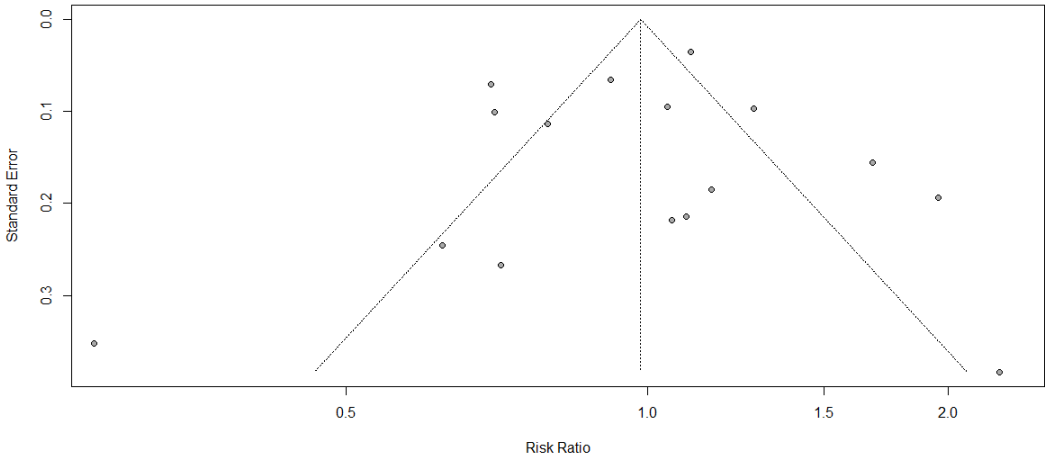


Figure 2. *Funnel plot for tooth loss severity.*

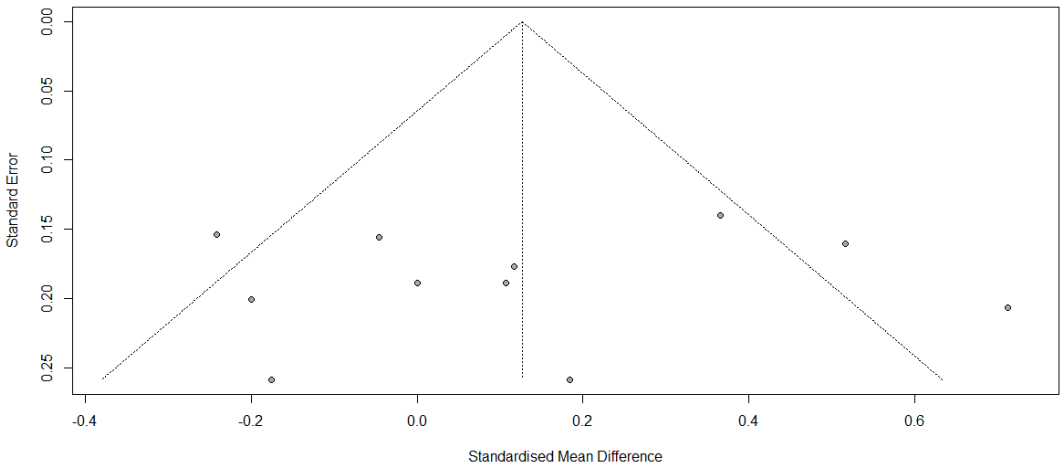
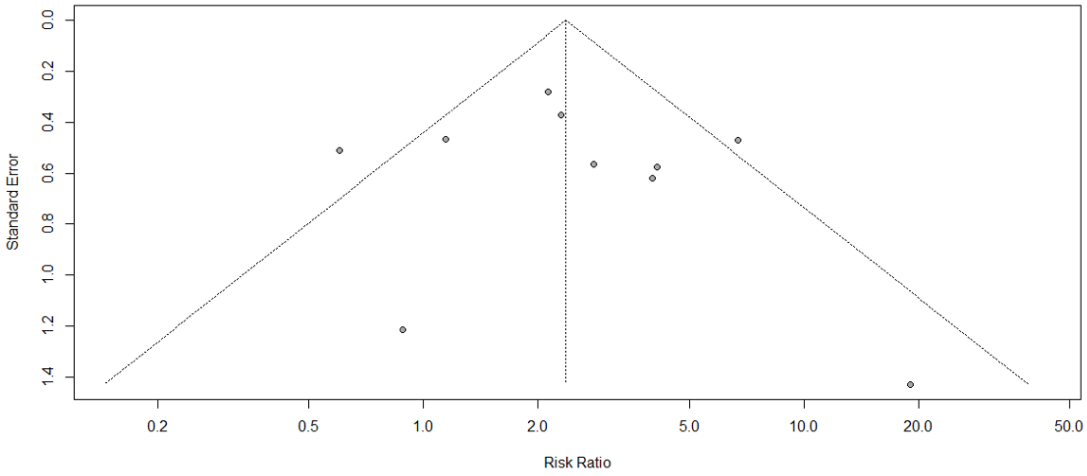


Figure 3. *Funnel plot for openbite prevalence.*



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ANEXO A – NORMAS PARA SUBMISSÃO NO PERIÓDICO *AUTISM.*

4. Preparing your manuscript for submission

4.1 Formatting

Autism asks that authors use the APA style for formatting. The APA Guide for New Authors can be found on the APA website, as can more general advice for authors.

4.2 Artwork, figures and other graphics

For guidance on the preparation of illustrations, pictures and graphs in electronic format, please visit SAGE's Manuscript Submission Guidelines.

Figures supplied in colour will appear in colour online regardless of whether or not these illustrations are reproduced in colour in the printed version. For specifically requested colour reproduction in print, you will receive information regarding the costs from SAGE after receipt of your accepted article.

4.3 Supplementary material

This journal is able to host additional materials online (e.g. datasets, podcasts, videos, images etc) alongside the full-text of the article. For more information please refer to our guidelines on submitting supplementary files.

4.4 Terminology

4.4.1 Terminology about autism and autistic people

Autism has researched and produced its own guidance on terminology and language used in autism research. Please consult the guide here: autism terminology guidelines.

4.4.2 Language used to discuss race and ethnicity

Likewise, *Autism* has also produced the following guidance to be considered when writing about race and ethnicity. Please consult the guide here: race and ethnicity language guidelines.

4.5 Reference style

Autism adheres to the APA reference style. View the APA guidelines to ensure your manuscript conforms to this reference style.

4.6 English language editing services

Authors seeking assistance with English language editing, translation, or figure and manuscript formatting to fit the journal's specifications should consider using SAGE Language Services. Visit SAGE Language Services on our Journal Author Gateway for further information.

5. Submitting your manuscript

Autism is hosted on SAGE Track, a web based online submission and peer review system powered by ScholarOne™ Manuscripts. Visit http://mc.manuscriptcentral.com/autism to login and submit your article online.

IMPORTANT: Please check whether you already have an account in the system before trying to create a new one. If you have reviewed or authored for the journal in the past year it is likely that you will have had an account created. For further guidance on submitting your manuscript online please visit ScholarOne Online Help.

5.1 ORCID

As part of our commitment to ensuring an ethical, transparent and fair peer review process SAGE is a supporting member of ORCID, the Open Researcher and Contributor ID. ORCID provides a unique and persistent digital identifier that distinguishes researchers from every other researcher, even those who share the same name, and, through integration in key research workflows such as manuscript and grant submission, supports automated linkages between researchers and their professional activities, ensuring that their work is recognized.

The collection of ORCID iDs from corresponding authors is now part of the submission process of this journal. If you already have an ORCID iD you will be asked to associate that to your submission during the online submission process. We also strongly encourage all co-authors to link their ORCID ID to their accounts in our online peer review platforms. It takes seconds to do: click the link when prompted, sign into your ORCID account and our systems are automatically updated. Your ORCID iD will become part of your accepted publication's metadata, making your work attributable to you and only you. Your ORCID iD is published with your article so that fellow researchers reading your work can link to your ORCID profile and from there link to your other publications.

If you do not already have an ORCID iD please follow this link to create one or visit our ORCID homepage to learn more.

5.2 Information required for completing your submission

You will be asked to provide contact details and academic affiliations for all co-authors via the submission system and identify who is to be the corresponding author. These details must match what appears on your manuscript. At this stage please ensure you have included all the required statements and declarations and uploaded any additional supplementary files (including reporting guidelines where relevant).

5.3 Lay Abstracts

As part of your submission you will be asked to provide a lay abstract of your article. Lay abstracts are a brief (max 250 words) description of the paper that is easily understandable. These abstracts will be made widely available (to the general public, and particularly to autistic people and their families). As such, lay abstracts should avoid both technical terminology and the reporting of statistics. Examples of lay abstracts are provided in recent issues of the journal. Authors may consider the following questions when composing their lay abstract.

a. What is already known about the topic?

b. What this paper adds?

c. Implications for practice, research or policy

Authors may also find the following resources helpful on this topic:

- How to write a summary paragraph
- Self Advocacy Resource and Technical Assistance Center (SARTAC): Plain Language
- Center for Plain Langauage: **Erro! A referência de hiperlink não é válida.**

5.4 Permissions

Please also ensure that you have obtained any necessary permission from copyright holders for reproducing any illustrations, tables, figures or lengthy quotations previously published elsewhere. For further information including guidance on fair dealing for criticism and review, please see the Copyright and Permissions page on the SAGE Author Gateway

ORDER OF PAGES

All papers, including student papers, generally include a title page, text, and references. They may include additional elements such as tables and figures depending on the assignment. Student papers generally do not include an abstract unless requested.

Arrange the pages of an APA Style paper in this order:

- title page
- abstract
- text
- references
- footnotes
- tables
- figures
- appendices

In general, start each section on a new page. However, the order of pages is flexible in these cases:

- **tables and figures:** Embed tables and figures within the text after they are first mentioned (or "called out"), or place each table and then each figure on separate pages after the references. If an embedded table or figure appears on the same page as text, place it at either the top or the bottom of the page, and insert a blank double-spaced line to separate the table or figure from the adjacent text.
- **footnotes:** Use the footnotes function of your word-processing program to insert a footnote at the bottom of the page of text on which the footnote appears, or list footnotes together on a separate page after the references.

TITLE PAGE SETUP:

Paper title

Place the title three to four lines down from the top of the title page. Center it and type it in bold font. Capitalize major words of the title. Place the main title and any subtitle on separate double-spaced lines if desired. There is no maximum length for titles; however, keep titles focused and include key terms.

Author names

Place one double-spaced blank line between the paper title and the author names. Center author names on their own line. If there are two authors, use the word "and" between authors; if there are three or more authors, place a comma between author names and use the word "and" before the final author name. When different authors have different affiliations, use superscript numerals after author names to connect the names to the appropriate affiliation(s). If all authors have the same affiliation, superscript numerals are not used (see Section 2.3 of the Publication Manual for more on how to set up bylines and affiliations).

Author affiliation

For a professional paper, the affiliation is the institution at which the research was conducted. Include both the name of any department and the name of the college, university, or other institution, separated by a comma. Center the affiliation on the next double-spaced line after the author names; when there are multiple affiliations, center each affiliation on its own line. When different authors have different affiliations, use superscript numerals before affiliations to connect the affiliations to the appropriate author(s). Do not use superscript numerals if all authors share the same affiliations (see Section 2.3 of the Publication Manual for more).

Author note

Place the author note in the bottom half of the title page. Center and bold the label "Author Note." Align the paragraphs of the author note to the left. For further information on the contents of the author note, see Section 2.7 of the Publication Manual.

Running head

The running head appears in all-capital letters in the page header of all pages, including the title page. Align the running head to the left margin. Do not use the label "Running head:" before the running head.

Page number

Use the page number 1 on the title page. Use the automatic page-numbering function of your word processing program to insert page numbers in the top right corner of the page header.

FONT

A variety of fonts are permitted in APA Style papers. Font options include the following:

- **sans serif fonts** such as 11-point Calibri, 11-point Arial, or 10-point Lucida Sans Unicode
- **serif fonts** such as 12-point Times New Roman, 11-point Georgia, or normal (10-point) Computer Modern (the default font for LaTeX)

We recommend these fonts because they are legible and widely available and because they include special characters such as math symbols and Greek letters. Historically, sans serif fonts have been preferred for online works and serif fonts for print works; however, modern screen resolutions can typically accommodate either type of font, and people who use assistive technologies can adjust font settings to their preferences. For more on how font relates to accessibility, visit the page on the accessibility of APA Style.

Use the same font throughout your paper, with the following exceptions:

- **figures:** Within figure images, use a sans serif font with a type size between 8 and 14 points.
- **computer code:** To present computer code, use a monospace font such as 10-point Lucida Console or 10-point Courier New.
- **footnotes:** When inserting footnotes with the footnotes function of your word-processing program, use the default font settings. The footnote font might be smaller than the text font (and have different line spacing), and it is not necessary to change it.

Instructors and publishers vary in how they specify length requirements. Different fonts take up different amounts of space on the page; thus, we recommend using word count rather than page count to gauge paper length if possible.

PAGE HEADER

The page header appears within the top margin of every page of the paper.

- For student papers, the page header consists of the page number only.
- For professional papers, the page header consists of the page number and running head.

Page numbers

Follow these guidelines to include page numbers in both student and professional APA Style papers:

- Use the page-numbering function of your word-processing program to insert page numbers.
- Insert page numbers in the top right corner. The page number should show on all pages.
- The title page carries page number 1.

Running head

The running head is an abbreviated version of the title of your paper (or the full title if the title is already short). The running head is not required for student papers unless the instructor or institution requests it. Thus, typically only professional papers include a running head.

Follow these guidelines to include a running head in an APA Style paper:

Type the running head in all-capital letters.

Ensure the running head is no more than 50 characters, including spaces and punctuation.

Avoid using abbreviations in the running head; however, the ampersand symbol (&) may be used rather than “and” if desired.

The running head appears in the same format on every page, including the first page.

Do not use the label “Running head:” before the running head.

Align the running head to the left margin of the page header, across from the right-aligned page number.

LINE SPACING

In general, double-space all parts of an APA Style paper, including the abstract; text; block quotations; table and figure numbers, titles, and notes; and reference list (including between and within entries). Do not add extra space before or after paragraphs.

Exceptions to double line spacing are as follows:

- **title page:** Insert a double-spaced blank line between the title and the byline on the title page. For professional papers, also include at least one double-spaced blank line above the author note (student papers do not include author notes). Double-space the rest of the title page.
- **tables:** The table body (cells) may be single-spaced, one-and-a-half-spaced, or double-spaced, depending on which is the most effective layout for the information. Double-space the table number, title, and notes.
- **figures:** Words within the image part of a figure may be single-spaced, one-and-a-half-spaced, or double-spaced, depending on which is the most effective layout for the information. Double-space the figure number, title, and notes.
- **footnotes:** When inserting footnotes with the footnotes function of your word-processing program, use the default font settings (usually single-spaced and a slightly smaller font than the text).
- **displayed equations:** It is permissible to apply triple- or quadruple-spacing in special circumstances, such as before and after a displayed equation.

MARGINS

Use 1-inch margins on every side of the page for an APA Style paper.

However, if you are writing a dissertation or thesis, your advisor or institution may specify different margins (e.g., a 1.5-inch left margin to accommodate binding).

PARAGRAPH ALIGNMENT AND INDENTATION

APA Style includes guidelines for paragraph alignment and indentation to ensure that papers are formatted in a consistent and readable manner. All writers should follow these guidelines.

Paragraph alignment

Align the text of an APA Style paper to the left margin. Leave the right margin uneven, or "ragged." Do not use full justification for student papers or manuscripts being submitted for publication.

Do not insert hyphens (manual breaks) in words at the end of line. However, it is acceptable if your word-processing program automatically inserts breaks in long hyperlinks (such as in a DOI or URL in a reference list entry).

Paragraph indentation

Indent the first line of each paragraph of text 0.5 in. from the left margin. Use the tab key or the automatic paragraph-formatting function of your word-processing program to achieve the indentation (the default setting is likely already 0.5 in.). Do not use the space bar to create indentation.

Exceptions to these paragraph-formatting requirements are as follows:

- **title page:** For professional papers, the title (in bold), byline, and affiliations should be centered on the title page. For student papers, the title (in bold), byline, affiliations, course number and name, instructor, and assignment due date should be centered on the title page.
- **section labels:** Section labels (e.g., "Abstract," "References") should be centered (and bold).
- **abstract:** The first line of the abstract should be flush left (not indented).
- **block quotations:** Indent a whole block quotation 0.5 in. from the left margin. If the block quotation spans more than one paragraph, the first line of the second and any subsequent paragraphs of the block quotation should be indented another 0.5 in., such that those first lines are indented a total of 1 in.
- **headings:** Level 1 headings should be centered (and in bold), and Level 2 and 3 headings should be left-aligned (and in bold or bold italic, respectively). Level 4 and 5 headings are indented like regular paragraphs.
- **tables and figures:** Table and figure numbers (in bold), titles (in italics), and notes should be flush left.
- **reference list:** Reference list entries should have a hanging indent of 0.5 in.
- **appendices:** Appendix labels and titles should be centered (and bold).

HEADINGS

Headings identify the content within sections of a paper.

Make your headings descriptive and concise. Headings that are well formatted and clearly worded aid both visual and nonvisual readers of all abilities.

Levels of heading

There are five levels of heading in APA Style. Level 1 is the highest or main level of heading, Level 2 is a subheading of Level 1, Level 3 is a subheading of Level 2, and so on through Levels 4 and 5. The number of headings to use in a paper depends on the length and complexity of the work.

- If only one level of heading is needed, use Level 1.
- If two levels of heading are needed, use Levels 1 and 2.
- If three levels of heading are needed, use Levels 1, 2, and 3 (and so on).

Use only the number of headings necessary to differentiate distinct sections in your paper; short student papers may not require any headings. Furthermore, avoid these common errors related to headings:

- Avoid having only one subsection heading within a section, just like in an outline.
- Do not label headings with numbers or letters.
- Double-space headings; do not switch to single spacing within headings.
- Do not add blank lines above or below headings, even if a heading falls at the end of a page.

Headings in the introduction

Because the first paragraphs of a paper are understood to be introductory, the heading "Introduction" is not needed. Do not begin a paper with an "Introduction" heading; the paper title at the top of the first page of text acts as a de facto Level 1 heading.

It is possible (but not required) to use headings within the introduction. For subsections within the introduction, use Level 2 headings for the first level of subsection, Level 3 for subsections of any Level 2 headings, and so on. After the introduction (regardless of whether it includes headings), use a Level 1 heading for the next main section of the paper (e.g., Method).

Creating accessible headings

Writers who use APA Style may use the automatic headings function of their word-processing program to create headings. This not only simplifies the task of formatting headings but also ensures that headings are coded appropriately in any electronic version of the paper, which aids readers who use navigation tools and assistive technologies such as screen readers.

Here are some tips on how to create headings in some common word-processing programs:

If you use Academic Writer to write your APA Style papers, the headings menu in the Writing Center will format headings for you in 7th edition APA Style.

If you use Microsoft Word to write your APA Style papers, use the Styles menu to format headings. Follow these headings directions from Microsoft to customize the heading formats for your future use.

To apply Level 4 and 5 headings (which are inline headings, meaning the heading appears on the same line as paragraph text), first type the heading and a few words of the text that follows.

Then highlight the text that you want to be your heading and select the appropriate heading level from the Styles menu.

Only the highlighted text will be formatted as the Level 4 or 5 heading.

TABLES

Table components

APA Style tables have the following basic components:

- number: The table number (e.g., Table 1) appears above the table title and body in bold font. Number tables in the order in which they are mentioned in your paper.
- title: The table title appears one double-spaced line below the table number. Give each table a brief but descriptive title, and capitalize the table title in italic title case.
- headings: Tables may include a variety of headings depending on the nature and arrangement of the data. All tables should include column headings, including a *stub heading* (heading for the leftmost, or stub, column). The heading "Variable" is often used for the stub column if no other heading is suitable. Some tables also include column spanners, decked heads, and table spanners; these are described in the *Publication Manual*. Center column headings and capitalize them in sentence case.
- body: The table body includes all the rows and columns of a table (including the headings row). A *cell* is the point of intersection between a row and a column.
    - The table body may be single-spaced, one-and-a-half-spaced, or double-spaced.
    - Left-align the information in the leftmost column or stub column of the table body (but center the heading).
    - In general, center information in all other cells of the table. However, left-align the information if doing so would improve readability, particularly when cells contain lots of text.
- note: Three types of notes (general, specific, and probability) appear below the table as needed to describe contents of the table that cannot be understood from the table title or body alone (e.g., definitions of abbreviations, copyright attribution, explanations of asterisks used to indicate *p* values). Include table notes only as needed.

Principles of table construction

The most important principle to follow when creating a table is to present information in a way that is easy for readers to understand. Provide sufficient information in the table itself so that readers do not need to read the text to understand it.

When creating a table, place entries that are to be compared next to each other. In general, place different indices (e.g., means and standard deviations) in different columns rather than in the same column. Use the same font in tables as in the rest of your paper.

Use the tables feature of your word-processing program to create tables in APA Style papers. Do not use the tab key or space bar to manually create the look of a table.

Table borders

Limit the use of borders or lines in a table to those needed for clarity. In general, use a border at the top and bottom of the table, beneath column headings (including decked heads), and above column spanners. You may also use a border to separate a row containing totals or other summary information from other rows in the table.

Do not use vertical borders to separate data, and do not use borders around every cell in a table. Use spacing between columns and rows and strict alignment to clarify relations among the elements in a table.

Long or wide tables

If a table is longer than one page, use the tables feature of your word-processing program to make the headings row repeat on the second and any subsequent pages. No other adjustments are necessary. If a table is too wide to fit on one page, use landscape orientation on the page with the wide table. It does not matter if the page header also moves when switching to landscape orientation.

Placement of tables in a paper

There are two options for the placement of tables (and figures) in a paper. The first is to embed tables in the text after each is first mentioned (or "called out"); the second is to place each table on a separate page after the reference list.

An embedded table may take up an entire page; if the table is short, however, text may appear on the same page as the table. In that case, place the table at either the top or bottom of the page rather than in the middle. Also add one blank double-spaced line between the table and any text to improve the visual presentation.

FIGURES

All types of visual displays other than tables are considered figures in APA Style. Common types of figures include line graphs, bar graphs, charts (e.g., flowcharts, pie charts), drawings, maps, plots (e.g., scatterplots), photographs, infographics, and other illustrations.

This page addresses the basics of figure setup, including figure components, principles of figure construction, and placement of figures in a paper. Note that tables and figures have the same overall setup.

Figure components

APA Style figures have these basic components:

- number: The figure number (e.g., Figure 1) appears above the figure title and image in bold font. Number figures in the order in which they are mentioned in your paper.

- title: The figure title appears one double-spaced line below the figure number. Give each figure a brief but descriptive title, and capitalize the figure title in italic title case.
- image: The image portion of the figure is the graph, chart, photograph, drawing, or other illustration itself. If text appears in the image of the figure (e.g., axis labels), use a sans serif font between 8 and 14 points.
- legend: A figure legend, or key, if present, should be positioned within the borders of the figure and explains any symbols used in the figure image. Capitalize words in the figure legend in title case.
- note: Three types of notes (general, specific, and probability) can appear below the figure to describe contents of the figure that cannot be understood from the figure title, image, and/or legend alone (e.g., definitions of abbreviations, copyright attribution, explanations of asterisks use to indicate *p* values). Include figure notes only as needed.

Principles of figure creation

The most important principle to follow when creating a figure is to present information in a way that is easy for readers to understand. Provide sufficient information in the figure itself so that readers do not need to read the text to understand it.

When creating a figure, ensure you meet the following standards:

- images are clear
- lines are smooth and sharp
- font is legible and simple
- units of measurement are provided
- axes are clearly labeled
- elements within the figure are clearly labeled or explained

Use graphics software to create figures in APA Style papers. For example, use the built-in graphics features of your word-processing program (e.g., Microsoft Word or Excel) or dedicated programs such as Photoshop or Inkscape.

Placement of figures in a paper

There are two options for the placement of figures (and tables) in a paper. The first is to embed figures in the text after each is first mentioned (or "called out"); the second is to place each figure on a separate page after the reference list.

An embedded figure may take up an entire page; if the figure is short, however, text may appear on the same page as the figure. In that case, place the figure at either the top or bottom of the page rather than in the middle. Also add one blank double-spaced line between the figure and any text to improve the visual presentation.

REFERENCES

Text citations

All references in the text and notes must be specified by the authors' last names and date of publication together with page numbers for direct quotations from print sources.

Do not use ibid., op. cit., infra., supra.

Note the following for the style of text citations:

1. If the author's name is in the text, follow with the year in parentheses.
2. If the author's name is not in the text, insert last name, comma and year.
3. For direct quotations, the page number follows the year, preceded by 'p.' (not a colon).
4. Where there are two authors, always cite both names, joined by 'and' if within running text and outside of parentheses; joined by an ampersand (&) if within parenthetical material, in tables and in captions, and in the reference list.
5. When a work has three or more authors, include only the surname of the first author followed by 'et al.' (not italicized and with a period after 'al') and the year.
6. If two references with three or more authors shorten to the same form, cite the surnames of the first authors and of as many of the subsequent authors as necessary to distinguish the two references, followed by a comma and 'et al.'.

However, because 'et al.' is plural (meaning 'and others'), it cannot stand for only one name. When only the final author is different, spell out all names in every citation.

7. If two or more references by the same author are cited together, separate the dates with a comma (in chronological order).
8. If there is more than one reference to the same author (or by the same two or more authors in the same order) and year, insert the suffixes 'a', 'b', 'c', etc., after the year of publication and repeat the year. The suffixes are assigned in the reference list, where these kinds of references are ordered alphabetically by title (of the article, chapter, or complete work).
9. List two or more works by different authors who are cited within the same parentheses in alphabetical order by the first author's surname, separated by semicolons.

Exception: You may separate a major citation from other citations within parentheses by inserting a phrase, such as 'see also' before the first of the remaining citations, which should be in alphabetical order.

10. When names of groups (e.g. government agencies, universities, etc.) serve as authors, these are usually spelled out each time they appear in a text citation. However, some group authors can be spelled out in the first citation and abbreviated thereafter.
11. When a work has no author as such, cite in the text the first few words of the reference list entry (usually the title) and the year. Use double quotation marks around the title of an article or chapter or web page and italicize the title of a journal, book, etc.

12. Citations from personal communications are not included in the reference list; cite in text only, giving the initials as well as the surname of the communicator and provide as exact a date as possible.

Reference list

General

1. Check that the list is in alphabetical order by surname of the first author (treat Mc and Mac alphabetically and literally, not as if they were all spelled 'Mac').

2. Names should be in initial cap then lower case.

3. Where several references have the same author(s), do not use ditto marks or em dashes; the name must be repeated each time.

4. Provide surnames and initials for up to and including 20 authors. When there are 21 or more authors, include the first 19 authors' names, insert an ellipsis (but no ampersand), and then add the final author's name.

5. Nothing precedes something when alphabetizing last names (e.g., Loft V. H. precedes Loftus, E. F.).

6. Names containing Jr or II should be listed as follows:

Author Last Name, Initials, Jr. (year).

Author Last Name, Initials, II (year).

7. When ordering several works by the same first author:

- Single-author references arranged in date order, the earliest first;
- Single-author entries precede multiple-author entries beginning with the same surname
- Two or more author references in alphabetical order according to the second author's last name, or if the second author is the same, the last name of the third author, and so on References with the same authors in the same order are arranged by year of publication, the earliest first:

8. Check that all periodical data are included – volume, issue and page numbers (complete span, not shortened), publisher, etc.

9. The date of retrieval of online material is not required, only the URL; see example below. Exceptions include dictionary entries, databases, and websites that are updated regularly but are not archived.

10. Include a DOI hyperlink for all works that have a DOI, regardless of whether the print version or the online version was used.

11. Check journal for examples.

Reference styles

Journal article

Author, A. A., Author, B. B., & Author, C. C. (year). Article title. Journal Name, vol. no.(issue no.), page range. DOI or URL

Book

Author, A. A., Author, B. B., & Author, C. C. (year). Book title. Publisher.

Chapter in book

Author, A. A., Author, B. B., & Author, C. C. (year). Chapter title. In A. Editor, B. Editor, & C. Editor (Eds.), Book title (pp. xxx–xxx). Publisher.

Website

Author, A. A., Author, B. B., & Author, C. C. (date). Title of work. Site Name. URL

Conference paper

Contributor, A. A., Contributor, B. B., & Contributor, C. C. (date). Title of contribution [Type of contribution]. Conference name, location. DOI or URL.